Curr Phys Med Rehabil Rep (2014) 2:219–230
DOI 10.1007/s40141-014-0064-z

SWALLOWING DISORDERS (RE MARTIN, SECTION EDITOR)

Management of Dysphagia Following Traumatic Brain Injury

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Published online: 22 October 2014

Abstract Dysphagia is a common morbidity and cause of mortality following traumatic brain injury (TBI). Despite this, there is a paucity of evidence demonstrating the efficacy of dysphagia management strategies and treatments in this population. Typically, subjects with dysphagia following TBI are placed into non-specific 'neurogenic' dysphagia subject groups, which include subjects with degenerative neurological diseases, neurological cancers, and cerebrovascular accident. However, dysphagia following TBI has a multifactorial presentation, with causative and contributory factors including cognitive-communication, behavioral, neurological, and mechanical issues. As such, the management for dysphagia post-TBI must be multifactorial, team-based and involve the patients' families and carers. Much of the research regarding the management and treatment of dysphagia in general is in its infancy: larger and more rigorous studies are required to demonstrate treatment efficacy. More studies specifically examining dysphagia and its management in the TBI population are required to ensure the future efficacy and accuracy of treatment.

Keywords Dysphagia · Traumatic brain injury · Cognitive-communication · Behavioral · Rehabilitation

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Introduction

Traumatic brain injury (TBI) and its complications contribute significantly to mortality and morbidity worldwide. In developed countries, the annual incidence rates are approximately 200 per 100,000 [1]. The greater the severity of TBI, the greater the economic burden on healthcare providers [2, 3], particularly acute care and rehabilitation services.

Dysphagia is a common complication following TBI, with an incidence as high as 93 % in patients admitted to brain injury rehabilitation [4]. Dysphagia following TBI can be multifactorial, but mainly occurs because of neurological impairment to any or all of the three phases of swallowing (the oral preparatory, the oral and pharyngeal phases) and cognitive-communication and behavioral dysfunction [5–9]. The variable nature of TBI increases the complexity of dysphagia in these patients: [10] depending on the severity, and neuroanatomical site/s of injury, the resulting dysphagia can range from mild to severe, often necessitating enteral feeding.

The complications from dysphagia are varied, costly and potentially fatal. Patients with TBI and dysphagia have longer average hospital admissions compared to those without dysphagia [11] and are at risk of weight loss, malnutrition and dehydration [12]. Dysphagia is causally linked with an increased risk of aspiration pneumonia; [13–16] the incidence of which can be as high as 12 % following severe TBI [14]. The social and psychological impacts of dysphagia can reduce patients' quality of life [17]. Post-discharge from rehabilitation, patients with TBI can be 79 times more likely to die from aspiration pneumonia compared to the general population [18].

Few studies have specifically assessed dysphagia following TBI. Studies of 'neurogenic dysphagia' typically

include heterogeneous subject groups, including subjects with cerebrovascular accident (CVA), progressive neurological diseases, brain tumors as well as TBI [19, 20]. Some studies involving TBI subjects even include other subjects with head and neck cancers [21] and dysphagia due to other structural/anatomical issues [22]., The majority of research into neurogenic dysphagia has, however, focused on subjects with CVA [23••, 24–27].

However, CVA and TBI populations are quite different. First, the pathophysiology of injury is dissimilar: CVA-related damage is usually focal whereas lesions in TBI represent a complex mixture of focal injury combined with diffuse axonal injury (DAI), with or without hypoxic injury. Thus, quite different neuromuscular and sensory deficits may present in each population [23••]. Second, population demographics are different, with CVA more common in older patients with degenerative co-morbidities. In contrast, TBI is frequently sustained by healthy, young males [28]. Third, post-TBI cognitive-communication and behavioral deficits contribute to or cause dysphagia [5, 6, 29]. This is particularly the case with patients with severe DAI who experience abnormal arousal, attention and cognitive issues [10]. Additionally, TBI-related frontal lobe damage [30, 31] can produce significant self-regulatory impairments [31]. Fourth, it has been suggested that post-stroke oromotor features of dysphagia differ from those following TBI [23••, 32]. Finally, patients with TBI may have concomitant injuries to the head and neck areas and/or necessitate prolonged endotracheal ventilation. All of these potential differences suggest that TBI-specific assessment and management of dysphagia should be recognized, along with the influence of these factors on the clinical management of such patients.

Dysphagia Resulting from TBI

Post-TBI dysphagia is caused and influenced by a number of factors. These include oropharyngeal neuromuscular and sensory deficits, cognitive-communication, and behavioral impairments, [5, 8, 9, 29, 33] physical injury to the head and neck regions [12], medications [5, 34], other concomitant injuries and prolonged endotracheal ventilation [12, 34–39]. Tracheostomies, while not causing dysphagia on their own [37–39], are common in this population and will also be discussed.

Cognitive-Communication and Behavioral Issues

Early TBI studies revealed that the most prevalent issue interfering with swallowing function was reduced cognition, followed by motor-control impairments [6]. In patients with normal or near normal swallowing physiology, these cognitive-communication and behavioral issues actually cause or worsen the dysphagia [40]. The strong relationship between cognition and safe oral feeding is reported frequently. [6, 8, 9, 33, 41, 42] For example, lower admission scores on the Ranchos Los Amigos (RLA) scale are a risk factor for dysphagia [41, 42] and that as cognitive function improves, so do functional oral feeding skills, [6, 29, 41, 42] such that RLA scores represent the most significant independent predictor of the time to return to full oral feeding [29].

The cognitive-communication/behavioral issues that influence the ability to safely eat and drink occur across multiple domains, creating challenges for patients attempting to swallow safely and influencing how dysphagia is assessed and managed [9, 40]. For example, post-TBI attentional impairments mean that some patients may be so impaired that they are unaware of food in front of them [8, 9]. Low alertness levels can slow the triggering of the pharyngeal swallow [40]. Highly distractible patients may slow their rate of intake so they eat and drink less [40], placing them at risk of malnutrition or dehydration. Patients can also be at risk of aspiration if they are so distracted they forget to swallow [40]. Disordered sensory perception in some patients may result in difficulty registering that food/fluid remains in their mouth [40]. The absence of an automatically triggered swallow risks choking or aspirating if they start to speak [40].

Memory issues may result in patients forgetting about safe food consistencies [8, 9, 40], or when/how much they last ate [40], increasing aspiration risk and over/under-eating, respectively. Short-term memory and receptive language deficits may inhibit understanding, learning, recall and ability to generalize dysphagia management strategies [8, 9, 33, 40]. Higher level cognitive impairments involving organizational and sequencing skills may cause difficulty for patients undertaking appropriate strategies [8, 9, 40]. Similarly, impaired executive functions such as self-regulation and mental flexibility may limit patients' ability to apply strategies to everyday eating situations [33]. Additionally, such deficits can make it hard for patients to be independent in skills such as appropriate diet selection and meal planning [33].

Agitated patients, and those with verbal and/or physical outbursts, are at risk of choking or aspirating if outbursts occur during mealtimes [8, 9, 40]. This is particularly important when the patient also has impaired swallowing physiology [8, 9, 40]. Combativeness and refusal to be fed by a caregiver can also cause safe feeding problems when complicated by impulsivity [40]. Impulsivity may affect patients' judgment regarding the amount and rate of food/fluid intake, presenting additional choking/aspiration risks, [8, 9, 11, 32, 33, 40] particularly if the patient's impaired swallowing physiology cannot cope with such a volume and rate of feeding [8, 40].

Motor Features

The most frequent post-TBI oropharyngeal motor deficits are thought to be reduced range and/or control of tongue movements [11, 41, 42], in isolation or combined [32] with deficits such as delayed or absent pharyngeal swallow [6, 11, 32, 41–43]. Aspiration is very common, present in 38–63 % of patients [11, 32, 41, 42]. Other less frequently observed deficits include decreased laryngeal elevation [6, 11, 32, 42], reduced base of tongue retraction [11, 42], decreased pharyngeal peristalsis [11, 32], prolonged pharyngeal transit time [41], prolonged oral transit time [11, 41], unilateral pharyngeal paralysis [42], absent or weak reflexive or voluntary cough [6], cricopharyngeal dysfunction [11, 32] and primitive oral reflexes (biting, pursing and rooting) [6]. Delayed or disorganized oral preparatory or oral phases of the swallow and premature spillage of the bolus into the pharynx can indicate poor tongue control [44]. Similar oromotor presentations have been found in pediatric TBI patients [10, 44, 45]. Abnormal facial muscle tone can produce hypertonicity, hypotonicity, reduced contraction of oppositional muscles and/or facial asymmetry [46].

The oropharyngeal deficits in CVA are thought to differ from those occurring in TBI populations. One study, examining TBI dysphagia deficits, commented that reduced tongue control was frequent and severe, whereas reduced pharyngeal peristalsis was less problematic [32]. Conversely, in dysphagia following CVA [47] the tongue control deficits were less severe and reduced pharyngeal peristalsis was more frequent [32]. Another TBI study [23••] identified aphonia as a single independent predictor of severe dysphagia. Other variables, predictive of dysphagia following CVA (for example, dysarthria and coughing post-swallow), were not found to be significant predictors following TBI. Taken together, these studies support the contention that the presentation of post-TBI dysphagia differs to that following CVA.

Mechanical Causes of Dysphagia

Physical Damage to Head and Neck

Trauma patients may experience injury-related physical damage to their head and neck. For example, injuries to the jaw may interfere with chewing; [12] injury to the neck may impair laryngeal closure and cricopharyngeal opening [43]. In this way, physical injury may add complexity to the management of neurological dysphagia.

Prolonged Endotracheal Intubation and Ventilation

Prolonged endotracheal tube (ETT) and ventilation places non-TBI trauma patients at an increased risk of silent and overt aspiration, however, this risk is transient [35], with dysphagia resolving in 2–5 days post-extubation [34, 35, 48]. Such dysphagia in trauma patients with prolonged ETT intubation is often multifactorial [35], resulting from prolonged contact of the ETT with chemo- and/or mechanoreceptors in the pharyngeal and laryngeal mucosae, critical for triggering the swallowing reflex [34].Physical injury including vocal fold ulceration and laryngeal edema [36] and impaired laryngeal elevation and/or closure may impede swallowing function [12]. Medications such as sedatives required for intubation may also temporarily depress the swallowing reflex [5, 34]. The potential effect of prolonged intubation in TBI patients with dysphagia has not been investigated.

Tracheostomy

Long-term tracheostomies can cause physical injuries such as tracheostenosis, tracheomalacia, and/or granuloma [49–52]. However, whether tracheostomies actually cause dysphagia and aspiration remains controversial. For many years, a causative link between tracheostomies and dysphagia and aspiration was believed to exist [53–55]. Studies suggested that even in the absence of neurological conditions, patients with tracheostomies risked dysphagia [56]. However, recent studies [37–39, 57–59] have failed to demonstrate causality between aspiration and tracheostomies per se as tracheostomies do not impair hyoid bone movement or laryngeal excursion during swallowing [60]. Instead, the severe illness necessitating the tracheostomy, whether neurological or not, and/or high dose medications [5] such as sedatives and neuromuscular blocking agents cause dysphagia, not the tracheostomy itself [37, 38, 58]. Another study found that tracheostomy removal did not change subjects' aspiration or dysphagic status [58]. Tracheotomised patients with TBI are likely to be dysphagic because of their neurological impairment, medications [5], intercurrent medical co-morbidities [37], or a combination of these factors. Thus, even when a patient with TBI is decannulated, they are highly likely to remain dysphagic and at risk of aspiration.

Predictors of Resolution of Dysphagia in TBI

The likelihood and severity of dysphagia appears linked to injury severity, in that various injury severity markers have been identified as independent predictors. For example, increased intracranial pressure is associated with moderate to severe dysphagia and a coma duration of >24 h associated with more severe dysphagia [32]. Hansen et al. [4] found that global injury severity indicators [Glasgow Coma Score (GCS), RLA, Functional Independence Measure and

Functional Oral Intake Scale scores] were all predictors of time to achieving functional oral intake.

Mackay et al. [42] reported four risk factors for dysphagia following severe TBI: lower admitting GCS (3–5), lower admitting RLA (levels I or II), tracheostomy and ventilation >2 weeks. However, the authors commented that tracheostomy and longer ventilation were potentially not causative factors for dysphagia, but were indicative of a greater severity of brain injury [42]. Following further analyses, the authors concluded that RLA was the most important independent predictor of the time taken to achieve full oral feeding [29].

Similarly, studies have reported swallowing function improves alongside improved cognition. For example, Winstein [6] reported that 94 % of dysphagic patients progressed to full oral feeding within 5 months post-injury. Ward et al. [61] found that duration to the first swallowing assessment (DFSA) was a predictor for achieving normal oral feeding. DFSA was the point where patients could cognitively and medically tolerate a swallowing assessment. Additionally, patients with more severe injury (on GCS) took longer to initiate oral feeding. Terre and Mearin [41] found that dysphagia improved along with better cognitive status on the RLA scale. They reported that feeding mode at discharge correlated with RLA level on admission and discharge and Disability Rating Scale (DRS) score on discharge [41].

Terre and Mearin [7] undertook a longitudinal cohort study of swallowing recovery. Initially, greater than one third were silent aspirators. At 1 year follow-up, none were silent aspirators and some had recovered their cough reflex. The greatest swallowing improvements were noted during the first 6 months post-injury, with more gradual improvements after this time. The authors found that baseline DRS score, RLA level, tongue control impairment, the absence of gag reflex and an increase in duration of pharyngeal delay time were predictive of whether a patient would continue aspirating at 1 year follow up.

Assessment

The multifactorial nature of post-TBI dysphagia necessitates a comprehensive assessment of all the potential causal factors [23••]. Logemann [62] notes that in order to correctly and effectively treat and manage dysphagia, 'the exact aetiology of the problem in swallowing as well as the particular muscles, muscle groups or structures involved' should be identified. However, in some patients with TBI, the cognitive-communication and behavioral impairments, rather than the physiological deficits, may be key to informing the effective management of dysphagia in this population. [8, 9, 33, 40]

Clinical Bedside Assessment

Speech pathologist bedside assessment involves history taking, cognitive-communication screening, observing for behavioral issues, oromotor assessment and, where indicated, trialing the patient with food and/or fluid. These will be considered in turn.

Medical and Background History

A thorough premorbid and current medical history including the nature and severity of the TBI are obtained from the medical file. Patient observation should note issues such as their level of responsiveness, positioning, nasogastric tube or gastrostomy tube, duration of endotracheal intubation and respiratory status (e.g., ventilated or oxygen via nasal prongs). These factors help identify patients at risk of dysphagia [12].

Informal Cognitive-Communication and Behavioral Assessment

Patients' behavioral and cognitive-communication skills are informally assessed at the bedside [40]. For example, taking the history from the patient may reveal basic expressive and/or receptive language issues, as well as memory and orientation deficits. Asking the patient to perform movements in the oromotor assessment provides information about their ability to attend, follow and sequence instructions [40]. Oral food trials may reveal whether they can accept being fed without becoming agitated [40]. If cognitive-communication or behavioral impairments are pronounced, patients will require more detailed assessment [40].

Oromotor Assessment

This assesses the oral structures and their functions (e.g., symmetry, sensation), the cranial nerves involved in swallowing, oral hygiene, dentition and, if appropriate, an oral food and/or fluid trial of varying consistencies [62, 63]. If the dysphagia manifests in pharyngeal and/or laryngeal deficits, further instrumental assessments may be undertaken [62, 63]. These can include videofluoroscopic swallow study (VFSS) (modified barium swallow), fiber-optic endoscopic evaluation of swallowing (FEES), fiber-optic endoscopic evaluation of swallowing with sensory testing (FEESST), cervical auscultation, pharyngeal manometry, pulse oximetry and electromyography [63]. VFSS and FEES are the most relevant instrumental assessments for patients following TBI [64]. If a patient is tracheotomised, blue dye tests may also be used to screen for aspiration.

Instrumental Assessment

Videofluoroscopic Swallow Study

Videofluoroscopic swallow studies (VFSS) assesses the speed and coordination of movements during chewing and swallowing in the oral cavity, tongue base, pharynx, hyoid, larynx, and cricopharyngeal region [43]. It provides information on transit times and the amount, etiology and type (silent or overt) of aspiration [43]. As well as diagnosing features of dysphagia, VFSS can assess the efficacy of management strategies, for example, varying the speed of bolus presentation [65] and/or the use of various postures (including chin down, head rotation, head tilt or lying down). [62] Trialing different consistencies, viscosities and volumes of food and fluid allows optimization of the patient's swallowing regime. However, the efficacy of these approaches is dependent on the patient having sufficient behavioral control and cognitive-communication skills to attend to, comprehend and recall and sequence commands consistently [8, 9, 40]. Thus, they are not always appropriate or possible for patients with TBI.

VFSS can assess other management approaches where appropriate. If sensory issues are suspected, modifying bolus temperature, taste and carbonation can be trialed to determine if these strategies assist with bolus detection and improve swallow safety [62]. Swallowing maneuvers, which encourage voluntary control over certain parts of the pharyngeal swallow, can be visualized during VFSS. These maneuvers may not be appropriate for all patients with TBI [8, 9, 40, 43] and details will be discussed in the management section of this paper.

Fiber-Optic Endoscopic Evaluation of Swallowing (FEES) and Fiber-Optic Endoscopic Evaluation of Swallowing with Sensory Testing (FEESST)

This examination involves passing a flexible scope through the nose to the level of the soft palate [43] to view the hypopharynx, larynx and proximal trachea [66] during swallowing. Both techniques are effective tools for assessing dysphagia, detecting aspiration and trialing management strategies in patients with TBI [35]. FEESST can also tests laryngopharyngeal sensory function [66]. The benefits of using FEES are multiple, including its simplicity of use at the bedside [67, 68] and in ventilated patients [64]. Barium contrast is not required, improving patient compliance with food and fluid trials [67, 68], particularly if they are orally or tactilely defensive [64], combative or agitated. As patients are not irradiated, assessments and implementation of management strategies need not be curtailed; allowing clinicians to assess for pre-swallow pooled pharyngeal secretions [67, 68].

Disadvantages of FEES include the inability to observe the oral cavity, tongue base movement, pharyngeal wall contraction and degree of laryngeal elevation or cricopharyngeal opening during swallowing [62, 69]. Some patients, particularly those with tactile defensiveness, agitation or confusion may find the transnasal placement of the endoscopy too uncomfortable [64]. The choice of VFSS or FEES depends on availability of equipment and clinician training, as well as the patients' medical status and sensory, cognitive-communication, and behavioral issues [64].

Blue Dye Tests

This test involves placing drops of blue dye on a patient's tongue fourth hourly and periodic tracheal suctioning, noting blue-stained secretions suggestive of aspiration [70, 71]. The Modified Evans Blue Dye Test uses blue dyed food and fluid instead [70, 72]. As a bedside assessment, it is a simple and economical way of screening patients with tracheostomies for aspiration [73]. However, reliability and validity of testing is variable, necessitating the use of stronger assessment methods such as VFSS or FEES wherever possible [70, 73].

Dysphagia Management

As Terre and Mearin [41] noted, 'there is no specific treatment for TBI-related dysphagia,' nor have randomized controlled trials examined treatment efficacy in TBI-exclusive populations [74•]. As discussed previously, post-TBI dysphagia may not be equivalent to other neurogenic dysphagias, and the multifactorial nature of post-TBI dysphagia requires patient- specific treatment and management. This includes taking into account their neuromuscular, cognitive-communicative and behavioral presentation. [41] In some patients with TBI, the cognitive-communication and behavioral impairments may dictate dysphagia management, rather than the physiological deficits [33]. Despite this, common treatment and management strategies for patients with neurogenic dysphagia have relevance to TBI. These will be discussed, along with potential future directions and emerging treatment options.

Meal-Time Strategies That Target Cognitive-Communication and Behavioral Issues

In patients where oral feeding is contraindicated due to low responsiveness, sensory stimulation (tactile, olfactory, gustatory, auditory and visual) to has been suggested to improve patient responsivity [8, 9]. More frequent, smaller meals may be recommended if alertness fluctuates during the day [40]. This can also be a useful strategy for patients with limited attention spans [40].

Reducing environmental distractions can assist patients with deficits in divided or alternating attention [8, 9]. Meals in a quiet room, with closed curtains, away from televisions, other people, noisy traffic and other visual/auditory distractions can help [40]. As attentional skills improve, distractors can be gradually re-introduced to the meal-time environment. This fosters real-world skills such as eating while performing other activities, such as having a conversation [9].

Controlling stimuli and the meal-time environment is also important for patients with agitation and combativeness. Identification and minimization of triggers for verbal or physical outbursts can reduce the likelihood of an outburst occurring during meal-times [9, 40]. Supervised meals with a caregiver who cues to the patient to other issues such as speaking with the mouth full, may be required [8, 9]. Giving small amounts of food or one utensil at a time [8, 9] at a time, cueing to reduce the amount and rate of food intake and to put utensils and cups down between mouthfuls are strategies that may reduce impulsive grabbing and 'shoveling' of food. [8, 9] This provides training to encourage independent eating [40].

In patients with memory or higher level cognitive deficits, written and visual cues can provide reminders of meal time strategies (for example, "take small mouthfuls") [9]. Providing simplified, written instructions of a series of steps that a patient may have to learn, perform and recall is an effective way of assisting patients to learn new sequences of actions [40] such as adjusting posture for safer swallowing.

Strategies and Treatments Targeting Swallowing Physiology

Behavioral Management

Behavioral dysphagia management techniques targeting swallowing physiology are often divided into 'compensatory' and 'rehabilitation'/direct therapy strategies [65, 75]. However, the principles of neural plasticity to swallowing rehabilitation may blur the line between this traditional divide. [76] Irrespective of this, supportive evidence for these management strategies is limited and has not necessarily provided recommendations on how best to prescribe and apply them [77].

Postural Adjustments Depending on the patient, postures including chin down, head rotation to the damaged side, head tilt to the stronger side, or lying down may be trialed [62]. These postures aim to improve airway protection or redirect food toward the stronger side of the pharynx [62]. However, to be effective the patient must have sufficient behavioral control and cognitive-communication skills to attend to, comprehend and recall and sequence commands consistently [8, 9, 40]. Thus, postures may not be suitable for some patients with TBI. Additionally, these postures are not effective in every patient [78–80] and there is a paucity of rigorous studies to demonstrate their efficacy, outcomes and limitations [78].

Compensatory Swallowing Maneuvers As with the postural adjustments, these maneuvers provide an 'immediate but only transient approach to the underlying physiologic deficit.' [81] The supraglottic swallow functions to close the vocal folds before and during the swallow [43] and clear bolus residue from the airway post-swallow [82]. An effortful breath hold with the super-supraglottic swallow aims to close the airway before and during the swallow [43]. Other swallowing maneuvers (the Mendelsohn maneuver, effortful swallow and Masako maneuver), can be considered compensatory, but when 'repeated in the context of an exercise regime, may facilitate overall change in swallowing physiology.' [81] These will be discussed in following rehabilitation section.

The supraglottic and super-supraglottic swallows are recommended in patients with reduced airway closure and/or a delayed pharyngeal swallow [43]. However, two small studies suggest that the supraglottic swallow doesn't produce measurable pharyngeal or intrabolus pressure effects in healthy subjects [82, 83]. There is also evidence that these maneuvers may work differently, depending on individual technique [82]. Training has been suggested to obtain consistent and effective performance: [82] a factor meaning they may be unsuitable for some patients with TBI, particularly as the supraglottic swallow was shown to be difficult for neurological patients to learn and perform [84].

Increased Sensory Input Modification of the bolus temperature, taste or carbonation, applying downward pressure on the tongue when feeding with a spoon, allowing self-feeding (hand to mouth movement may provide additional sensory input), thermal/tactile stimulation (vertically rubbing the faucial arches with a cold laryngeal mirror to increase oral awareness prior to swallowing) and a presenting a bolus that requires chewing are all techniques that can be trialed to assist with bolus detection and improve swallow safety [62, 65]. Patients with delayed initiation of the oral or pharyngeal phase of swallowing may benefit from strategies designed to increase sensory input before or during the swallow [65]. Modifying bolus temperature alone will not alter swallowing physiology; [76, 85] however, application of cold and pressure to the faucial arches can increase the speed of the onset of tongue movement and the pharyngeal phase of the swallow in the short-term [86]. Sour boluses have been shown to prompt faster

initiation of bolus propulsion by the tongue and a faster pharyngeal phase of the swallow [87, 88]. Studies into the effects of carbonated boluses have been criticized [89], and further, better designed studies are required before conclusions can be drawn [76].

Modifying The Volume and Speed of Bolus Presentation Reducing food/fluid intake volume and speed can help prevent pharyngeal pooling and aspiration in patients with delayed or weak pharyngeal swallows [65]. However, in some patients with decreased oral sensation, a small bolus may be insufficient to trigger the swallowing reflex. Conversely, increasing bolus volume can increase the extent of lingual, submental and pharyngeal movement and cricopharyngeal opening [76]. Determining whether a patient would benefit from large or small bolus volumes illustrates the importance of accurately identifying the nature of their swallowing disorder.

Food Consistency and Fluid Viscosity Changes Speech pathologists commonly recommend thickening fluids and/or softening or pureeing food [90]. VFSS may indicate swallowing difficulty with particular consistencies such as thin fluids, so these may be eliminated from the patient's diet. Thickening liquids slows the flow of the liquid through the pharynx and may help avoid aspiration [90]. Softened or pureed foods are recommended if a patient has difficulty manipulating challenging food consistencies [90] such as hard, chewy or crumbly foods or foods with dual consistencies (e.g., soup containing solid vegetable pieces).

Oral Motor Exercise Programs The use of oral motor exercises, such as range of movement and strength exercises as a treatment for dysphagia is controversial [91]. A systematic review of oral motor exercises and sensory motor interventions found 'insufficient evidence to draw any conclusions on the value of these interventions in dysphagia treatment' [92] and that further studies are required to determine their efficacy. To this end, small studies are emerging suggesting that strengthening exercises targeting the muscle level of the swallowing function are beginning to show some effect in some populations [76]. However, strategies to target weakness from decreased excitatory input to motor neurons are not well understood [76] or researched. A study by Robbins et al. [93] found that isometric tongue exercises improved tongue strength and swallowing function in patients post-CVA. However, it was unclear whether these results were due to improvements at the 'muscle level alone or neuroplastic modifications as well' [93].

Other Exercise Programs The McNeill Dysphagia Therapy Program has demonstrated improved swallowing physiology and functional swallowing [94•]. The program uses swallowing of different consistencies and volumes as the exercise [94•]. Studies have found increased rate of oropharyngeal movements [95], lingual-palatal pressures and laryngeal and hyoid elevations [96] and functional improvements in swallowing were noted [21]. However, as with many dysphagia studies, groups were heterogeneous, small and the authors noted the research was 'exploratory in nature and lack the rigor of larger controlled studies.' [96] Thus, while promising, further research into this program is required.

The Shaker Head Lift is not a direct swallowing task, but rather requires the patient to repeatedly raise their head and hold from a supine position. The aim is to increase cricopharyngeal opening 'by strengthening suprahyoid musculature with resulting increased hyolaryngeal excursion' [94•], thereby eliminating dysphagic symptoms [97]. A systematic review of nine studies found promising results, but that further controlled studies were required to determine efficacy [97].

The Masako maneuver involves the patient holding their tongue between their front teeth in an anterior position while swallowing [77]. It increases posterior pharyngeal wall movement during swallowing which is helpful for patients with reduced base of tongue retraction [98]. It is intended as a saliva swallowing exercise (i.e., no bolus) to strengthen pharyngeal muscles [77, 98]. Further research into the effect of long-term training is indicated [98].

Oral Motor Exercises and TBI Patients with diffuse TBIs present heterogeneous pathoanatomical injury features and pathophysiological mechanisms behind their neurological symptoms [99]. Motor impairment following TBI depends on the site/s of injury and can include spasticity, weakness, ataxia, apraxia and extrapyramidal movement disorders [100]. These impairments commonly occur in combination because 'selective injury of particular neural tracts is rare.' [100] In keeping with this, post-TBI oropharyngeal deficits could result from a variety of neuromuscular dysfunctions. Identifying exactly why (at a neurological level) oropharyngeal movements are impaired can be difficult. For example, there is very little, if any, data on what constitutes normal tone in the swallowing musculature [91], and abnormal oropharyngeal muscle tone is difficult to identify. Additionally 'the effects of hypotonia…may be difficult to distinguish perceptually from those of weakness.' [91] Thus, if the exact nature of the neuromuscular presentation is unknown, recommending specific oral motor exercises can be very difficult, if not impossible. Strengthening exercises for patients who display hypertonia or rigidity are contraindicated as it may increase tone, discomfort and further reduce the range of movement [91, 101]. Thus,

without an accurate identification of the nature of the problem use of oral motor exercises as a form of treatment is not prudent.

Swallowing Maneuvers Swallowing maneuvers encourage voluntary control over the timing or coordination of certain parts of the pharyngeal swallow [65]. The Mendelsohn maneuver aims to increase submental muscle activation [102], hyoid movement and the duration of cricopharyngeal sphincter opening [62]. The effortful swallow increases oral pressure during swallowing, the amplitude of submental muscle activation [102], tongue base retraction, duration of pharyngeal pressure, among other changes [76]. Again, evidence demonstrating the efficacy, reliability and limitations of these maneuvers is limited [78]. As discussed previously, they may be unsuitable for patients with TBI with cognitive-communication and behavioral issues [8, 9, 40, 43].

Surface Electromyography Biofeedback (sEMG) sEMG is an adjunctive therapy tool that can increase motor learning via biofeedback during dysphagia treatment tasks [103, 104]. Electrodes are placed in the submental region [104] to record muscle activity during rehabilitative swallowing tasks such as the Mendelsohn maneuver, Masako maneuver, effortful swallow or Shaker Head Lift [81]. While performing these tasks, the patient watches real-time sEMG feedback of their muscle activity [103]. Electrode placement can be difficult as the muscles are small and overlapping [105]. Techniques to maximize signal detection and avoid misinterpretation of signals are also important [105].

Limited evidence suggests that sEMG can be an effective adjunct to these exercises [81, 103]. However, the neuromuscular processes behind why improvement in is observed in these sEMG studies is not well understood [81] and further research is necessary [103, 105].

Neuromuscular Electrical Stimulation (NMES) NMES applies an electrical current to stimulate motor and/or sensory nerves or nerve endings [106]. The purported aims of transcutaneous NMES are often vague and generic: to 'enhance movement by increasing muscle contraction' [94•] to 'improve function by strengthening the swallowing musculature or by stimulating the sensory pathways relevant to swallowing, or both;' [107] and to 're-educate patients to use their pharyngeal muscles in the throat for patterned activity to initiate or re-establish swallowing.' [24] Humbert [108] comments that the intended use of transcutaneous NMES is not clear and the evidence limited and conflicting when used in dysphagia [94•, 108, 109]. Few studies have demonstrated the physiological benefits of transcutaneous NMES for swallowing [94•, 110] and no studies have demonstrated a functional improvement in swallowing (for example, increased oral intake). Heterogeneous subject groups, small subject numbers, lack of specificity of transcutaneous NMES at a tissue level [108] and differences in electrode placement and stimulation parameters are just a few of the reasons why better designed studies are required [94•] before the efficacy of this treatment option will be known.

Oral Hygiene

If oral hygiene or dentition issues are observed at the bedside, referral to dental services is required, as excessive colonization of microorganisms in the oral cavity can contribute to respiratory infections [111]. Implementation of an oral care program to maintain and improve oral health reduced the risk of pneumonia in an aged care population [112]. In populations who are physically or cognitively unable to independently perform oral care activities, regular oral care is critical in reducing the risk of aspiration pneumonia and other respiratory infections [113].

Team-Based Management

A multidisciplinary team approach to dysphagia management involving speech pathologists, physiotherapists, dietitians, physicians, and nursing staff resulted in improved weight and caloric intake [114]. Involvement of the patient, family and caregivers, is also critical to ensuring that management strategies generalize to functional settings, such as the home [115]. This is particularly the case post- TBI, as attentional and memory impairments may affect their ability to recall and implement strategies. Guidance for the family and caregivers regarding adequate communication is important, for example, use of short verbal instructions, simple, written instructions, repetition and avoidance abstract language can be discussed and modeled [9].

Family education programs must explain dysphagia and provide training in feeding techniques and management strategies where appropriate, as increasing the family's awareness of these potential issues may assist with compliance with therapy and strategies in the home environment [115]. Discussions must address psychosocial issues, such as the probable change in the patient's role in the family and dependency issues following the TBI [115].

Conclusion

Dysphagia following TBI is complex and multiple factors influence how it manifests and is assessed and managed.

Research into the treatment and management of dysphagia in TBI-specific populations tends to be limited and often lack scientific rigor. Traditional treatments for 'neurogenic dysphagia' such as postural adjustments, swallowing maneuvers and oral motor exercises are not necessarily appropriate or effective for TBI patients. The management of dysphagia following TBI requires a multifaceted, individualized approach that incorporates the contributory and causative cognitive-communication, behavioral, physiological, and pharmacological factors, any concomitant injuries and the pathoanatomic features of the TBI. Careful and detailed assessment of the impact of these factors on the dysphagia should guide treatment and management plans. Given the scarcity of evidence for many traditional forms of dysphagia management, regular monitoring and assessment of therapeutic strategies for individuals is recommended to maximize efficacy and avoid unwanted outcomes. The multifactorial nature of dysphagia following TBI, and its far-reaching effects on quality of life, necessitates multidisciplinary management involving the patient, family or caregivers wherever possible.

Compliance with Ethics Guidelines

Conflict of Interest I Baguley, A Howle, and L Brown all declare no conflicts 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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- •• Of major importance

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