Dysphagia in severe traumatic brain injury

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ABSTRACT

يعد عسر البلع (Dysphagia) من المضاعفات الشائعة التي تحدث بعد الإصابات الشديدة في الدماغ، ويُصاحب هذه المشكلة زيادة خطر الإصابة بسوء التغذية والالتهاب الرئوي. يصعب التعامل مع المرضى المصابين بعسر البلع من جراء إصابات الدماغ الشديدة وذلك بسبب ظهور بعض الإعاقات في السلوك، والتواصل، والإدراك. يهدف هذه المقال إلى إطلاع الأطباء عن كيفية التعامل مع المرضى المُصابين بإصابات شديدة في الدماغ وما يحدث بعد ذلك من مضاعفات كعسر البلع وشرح هذه المشكلة، كما أنه يسترجع تاريخ دراسة عملية البلع، ومراحل البلع، والإعاقات التي قد تُصيب عملية البِلع، والتحكم العصبي بعملية البلع، وكيفية تقييم المرضى المُصابينِ بصعوبات في البلع والتعامل معهم. ويغطي هذا المقال أيضاً كيفية التعامل مع المرضى الذين أُجريت لهم عملية ثقب للقصبة الهوائية (tracheostomy) بسبب عسر البلع وكيفية تغذيتهم.

Dysphagia, or swallowing difficulty, is a common problem following severe traumatic brain injury and is associated with an increased risk of malnutrition and pneumonia. Management of patients with dysphagia following head injury is complicated by the presence of cognitive, communication, and behavioral impairments. The purpose of this review article is to help physicians taking care of traumatic brain injury patients understand and manage dysphagia. The article reviews the history of the study of swallowing, stages of swallowing, impairments of swallowing, neural control of swallowing, and the evaluation of patients with swallowing difficulties and their management. In addition to the general principles of dysphagia management, this article covers the management of dysphagic patients with tracheostomy and their nutritional management.

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Dysphagia, or swallowing difficulty, is a common problem following severe traumatic brain injury. The purpose of this review article is to help physicians taking care of traumatic brain injury patients understand and manage dysphagia. Swallowing was described by William Harvey (1578-1657), who compared the speed and complexity of the swallowing motions with those of the heart.[1] Hundreds of years later, this example remains accurate. Swallowing motions are rapid like the pumping of the heart, and the bolus has to travel through chambers and valves to reach the stomach (**Figure 1**, **Table 1**).[2] Swallowing impairment after severe traumatic brain injuries has been reported to be as high as 60% in adults and 68% in pediatric patients.[3,4] Risk factors for developing dysphagia following head injury include the severity of the injury on CT, lower Glasgow Coma Scale (GCS) and Rancho Los Amigos (RLA) scores on admission, abnormal tongue control, presence of a tracheostomy, feeding tubes and mechanical ventilation for more than 2 weeks.[3-6] In a prospective cohort study, Terre et al[7] found that 62% of the severe traumatic brain patients with dysphagia aspirate on instrumental examination, and 41% of the aspirators were silent aspirators. Severe head injury patients with swallowing impairments are at risk of developing malnutrition and pneumonia. Malnourishment is reported to affect 68% of severe traumatic brain injury patients.[8] Patients with severe malnutrition suffer from serious complications like pressure sores, infections, and contractures.[9] The incidence of pneumonia after traumatic brain injury is 44-60% in intensive care units (ICUs), and 12% in rehabilitation wards.[10] In addition, severe traumatic brain injury patients with dysphagia have more cognitive deficits, lower functional independence measure (FIM) scores, and longer hospital stay.[11] Similarly, stroke literature has shown that dysphagia is associated with increased risk of mortality and morbidity, and that patients with dysphagia have more cognitive impairments and lower FIM scores on admission and discharge from rehabilitation wards.[12]

Important definitions in dysphagia. i) *Dysphagia:* any difficulty in swallowing. ii) *Aspiration*: entry of food material into the airway below the level of the true vocal cords.[13] iii) *Silent aspiration:* aspiration without a cough

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or other signs of distress or difficulty. Investigative tools, such as videofluoroscopy (VFS) or fiberoptic endoscopic evaluation of swallowing (FEES), are needed to diagnose silent aspiration.[14] iv) *Penetration:* a sign observed during instrumental investigation of swallowing (VFS, or FEES). Penetration means that food material went into the larynx but remained above the level of the vocal cords.[15]

Stages (mechanics) of swallowing. To François Magendie (1783-1855), we owe the classical division of swallowing to 3 stages: oral, pharyngeal, and esophageal.[16] In the oral phase, with the help of the saliva, the teeth and tongue transform the food into a homogenous bolus that can be swallowed easily. Next, the tongue pushes the bolus backward toward the pharynx, and the pharyngeal stage starts. In the pharyngeal stage, the soft palate seals the nasal cavity to prevent regurgitation of the food through the nose, the tongue base, and the pharyngeal muscles contract toward each other to push the bolus down, the larynx is pulled anterior and superior to avoid the coming bolus, and the upper esophageal sphincter relaxes. After that, the esophageal stage starts, and the bolus moves down via the concentric contractions of the esophageal smooth muscles and gravity until it reaches the lower esophageal sphincter and, finally, the stomach.[2,17]

Neural control of swallowing. Brain stem control.[18,19] i) *Afferent:* solitary nucleus (7, 9 & 10th cranial nerves). ii) *Efferent:* nucleus ambiguus (9, 10 & 11th cranial nerves). iii) *Central pattern generator (CPG):* interneuronal (premotor) reticular formation to coordinate different cranial nerves and synchronize both sides. **Table 2** demonstrates the function of the 6 cranial nerves involved in swallowing.[20]

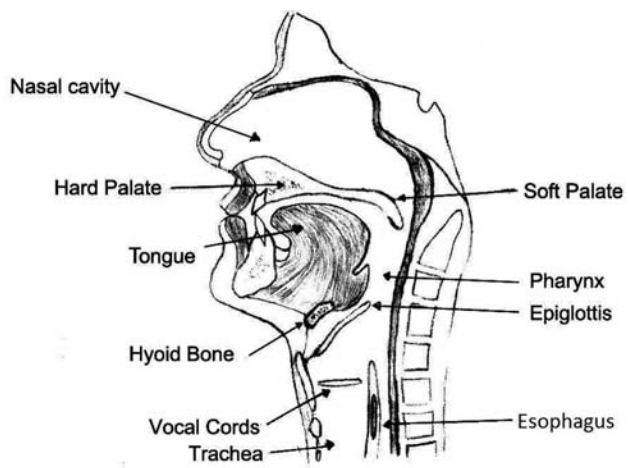

Figure 1 - The nasopharynx.

Cortical control of swallowing. Cortical control can start, inhibit, and modulate swallowing. Cortical involvement in swallowing has been studied using PET, and fMRI scans. Multiple and bilateral cortical/subcortical structures appear to be involved in the control of swallowing (sensorimotor cortex, posterior parietal, anterior insula, temporal cortex, anterior cingulate cortex, basal ganglia, thalamus and cerebellum). This diffuse representation explains why any cortical or sub-cortical insult can potentially cause dysphagia. In addition, it proves that swallowing is not a simple reflex; instead, it requires the interaction of many cortical and sub-cortical structures like any higher mental function.[18,19,21]

Dysphagia in relation to impairment. Oral and pharyngeal stage abnormalities are common in dysphagia secondary to traumatic brain injury.

Oral stage abnormalities.[2] i) Impairment of the sensations of the oral mucosa causes a prolonged oral phase and delays the initiation of swallowing. ii) Facial

Table 1 - Chambers, and valves in relation to swallowing.[2]

Chambers	Valves
Mouth	Lips & soft palate
Nose	Soft palate
Pharynx	Base of the tongue
Trachea	Larynx
Esophagus	Upper esophageal sphincter

Table 2 - Cranial nerves involvement in swallowing.[20]

Cranial nerve	Swallowing involvement
Trigeminal	Motor to the muscles of mastication, and sensory to the face and oral mucosa.
Facial	Motor to the muscles of facial expression, taste from the anterior 2/3 of the tongue, and parasympathetic supply to all salivary glands excluding the parotid.
Glossopharyngeal	Taste from the posterior 1/3 of the tongue, sensory to the pharynx, motor to the stylopharyngeus muscle and parasympathetic supply to the parotid gland.
Vagus	Taste from the epiglottis & pharynx, sensory to the larynx & pharynx, and motor to the pharyngeal & laryngeal muscles.
Accessory	Joins the vagus nerve (functionally considered as a part of the vagus nerve).
Hypoglossal	Motor to the intrinsic muscles of the tongue.

Note that 10 cranial nerves are located in the brain stem, and 6 of them are involved in swallowing (olfactory & optic nerves are in the brain).

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muscles weakness leads to leakage of food and drooling, as well as pocketing of food inside the mouth. iii) Tongue weakness leads to difficulty in the formation and transportation of the bolus. iv) Loss of teeth causes difficulty in eating a regular diet.

Pharyngeal phase abnormalities.[17,22] i) Inability of the soft palate to seal the nasal cavity due to weakness results in regurgitation of food through the nose. ii) Food residue tends to accumulate in the vallecula in cases of tongue base weakness. iii) Lack of normal sensation in the pharynx and larynx leads to delayed swallowing, pooling of food residue, and loss of the protective cough reflex. iv) Pharyngeal muscles weakness cause pooling of food residue. v) When the upper esophageal sphincter fails to relax, food residue accumulates in the pyriformis sinus. vi) Incomplete epiglottic closure and vocal cord paralysis increase the risk of aspiration. vii) Weakness of the submandibular/suprahyoid group of muscles leads to decreased laryngeal protective movement (superior/anterior). Note that accumulation of residue anywhere in the mouth or pharynx increases the risk of aspiration.

Patient evaluation. History. The patient's age, medical problems, and medications are essential components of the history. Severe traumatic brain injury patients with dysphagia are usually not able to communicate due to the decreased level of consciousness and/or the presence of tracheostomy. Information about the mechanism of trauma, extent of the injury(ies), feeding difficulties and change in weight can be obtained from the medical chart and care givers. Symptoms suggestive of dysphagia include a history of chocking/coughing during or after eating, tube feeding, weight loss, recurrent pneumonia, drooling and need for suction.[23,24]

Examination. At the bedside, patients are observed for the presence of skull deformities, O_2 supplement, drooling, tracheostomy, feeding tube and physical restraints. Patient's vital parameters and GCS score need to be documented. In the presence of a tracheostomy, the frequency of suction and color of secretions are also documented. Patients are examined for cognitive and cranial nerves abnormalities. In addition, the oral cavity is examined for lack of hygiene, loss of sensation, loss of teeth and presence of fixation (jaw wires). A blue dye test,[23] can be performed for tracheostomized patients. It is a sensitive, but not a specific test. Patients are given a fluid colored with an inert blue dye through the mouth. If the colored material comes out of the trachea during suction, then this test is diagnostic for aspiration.[25] If the test is negative, the patient may still be aspirating. In that case, the physician may need to order VFS or FEES to rule out aspiration. A feeding trial is performed by a trained clinician (speech pathologist, nurse or a physician), if the patient is medically stable and cognitively able to cooperate. It is the last and most functional part of the exam. During the trial the patient is given different fluid and food consistencies, and is observed for cognitive/behavioral deficits, oropharyngeal coordination, change of voice (wet voice), and spontaneous cough during or after feeding.[26,27] At the end of the trial, recommendations can be given regarding safe fluid and food consistencies, use of specific rehab strategies, or the need for further instrumental examination of swallowing using VFS or FEES.

Investigations. i) Videofluoroscopy, also known as, MBS (modified barium swallow) is considered a gold standard test for studying swallowing disorders. In 1898, Cannon[28] was the first to use fluoroscopy to study swallowing. Over the last century, several other pioneers continued to work on this test,[29-31] however, the way we administer and interpret the test today is based on the work of Professor Logemann.[32,33] The VFS is performed in the radiology department using a fluoroscopy machine and radiopaque material (barium). During the exam, the patient is given food of different consistencies with the addition of barium. The patient should be cooperative to be able to undergo the exam. The details of how to administer and interpret the exam can be found in other articles.[32,33] The VFS is a diagnostic and therapeutic tool that can diagnose silent aspiration and document the efficacy of compensatory strategies used to manage dysphagia.[5,34] ii) Fiberoptic endoscopic evaluation of swallowing was originally described by Professor Langmore in 1988.[35] Since then, a great number of studies and reviews have been published documenting the benefits of FEES.[36,37] The test is conducted using a flexible fiberoptic laryngoscope and a food coloring material. The FEES is a diagnostic and therapeutic tool that can diagnose silent aspiration and document the efficacy of compensatory strategies used to manage dysphagia.[37] iii) Both the VFS and FEES are complementary to each other. **Table 3** illustrates important clinical differences between the 2 tests.[34,37] iv) If the patient is coughing, tachypneic, tachycardic, febrile, on oxygen or in need of frequent suction, a chest x-ray is performed to rule out aspiration pneumonia.

Table 3 - Important clinical differences between videofluoroscopy (VFS) and fibro-optic endoscopic evaluation of swallowing (FEES).[34,37]

FEES	VFS
1. Direct sensory and anatomical exam.	1. Detect aspiration before, during and after swallowing.
2. No radiation risk (repeatable).	2. Oral phase abnormalities are demonstrated.
3. Portable to bed side.	

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Rehabilitation management. General principles. Functional improvement after an injury can occur due to a structural change that regains function, or a behavioral change that compensates for the function. This improvement is achieved in rehabilitation of swallowing disorders by using compensatory strategies and rehabilitation exercises. The compensatory strategies aim to change eating behaviors by modifying bolus volume, texture, and head/body posture. On the other hand, rehab exercises aim to induce structural change by intensifying motor and sensory input to the damaged area.[3,38] Pharyngeal muscular stimulation is an emerging modality in the field of swallowing exercises. Pharyngeal muscular stimulation, whether it is superficial (VitalStim), deep on pharyngeal walls or surgically implanted, requires further research before it can be generally applied in the management of dysphagic patients.[23,39] Swallowing is much more complex than a simple motor task. In addition to the motor part, swallowing has cognitive, sensory, and co-ordination components. This complexity of swallowing may interfere with the efficacy of the pharyngeal muscular stimulation. Finally, dysphagia following severe traumatic brain injury is complicated by the presence of cognitive, communication, and behavioral impairments.[40] The best way to minimize the effects of these complications is to use a multidisciplinary (Dysphagia/Tracheostomy) team that consists of (ENT specialist, Respiratory therapist, Speech & Language Pathologist and a Dietitian) when managing severe traumatic brain injury patients with dysphagia.

Management of patients with tracheostomy. Up to 50% of patients undergo tracheotomy following severe traumatic brain injury (**Figure 2**).[5] Tracheostomy decreases the patient's ability to communicate with others, which may lead to depression and social isolation. It may also increase the risk of aspiration due to the impairment of laryngeal mobility, sensitivity, and the loss of the subglottic pressure.[41] In addition, prolonged tracheostomy can cause an anterior fistula to the innominate artery and a posterior fistula to the esophagus. An anterior fistula can result in a fatal bleed, and a posterior fistula can result in recurrent pneumonia.[42] Due to these risks and complications, patients with tracheostomy need to be weaned off tracheostomy as soon as they are ready for the weaning process. **Table 4** presents criteria to attempt weaning from tracheostomy.[42,43] If a patient cannot produce a voice when his tracheostomy opening is closed for a short period, he may have an obstruction at or below the level of the vocal cords. In such cases, a scope must be performed by ENT to rule out vocal cord paralysis due to recurrent laryngeal nerve injury at the time of tracheotomy. If the vocal cords are mobile, the scope has to pass below the level of the vocal cords to rule out an obstruction caused by clot, granuloma, or mucus plug. An obstruction at or below the level of the vocal cords may cause failure of the tracheostomy weaning process. The process of weaning from tracheostomy differs between centers. Safe decannulation is the ultimate goal of all weaning protocols. There is no evidence to suggest that one method is superior to the other.[42] The use of multidisciplinary Tracheostomy/Dysphagia teams leads to fast and safe decannulation of patients, with a significant reduction in the number of tracheostomy related complications.[44-46] Tracheostomy teams in each hospital have their own protocols that should be followed to ensure the delivery of needed care for tracheostomy patients. **Table 5** illustrates steps that can be taken during the weaning process.[42,43]

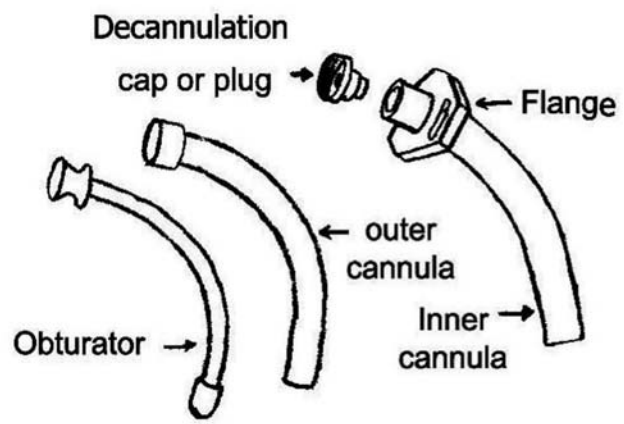

Figure 2 - Tracheostomy tube.

Table 4 - Criteria to attempt weaning from tracheostomy.[42,43]

M C Ø F A M O S	
M	Good **M**ental state *
C	Good **C**ough
Ø	Absence of the following (null set)
F	Respiratory **F**ailure
A	**A**spiration (his own secretions)
M	**M**edical problems (fever, pneumonia, low BP)
O	**O**bstruction (vocal cord paralysis, tracheomalacia, granuloma, clot, mucous plug)
S	**S**uction (more than twice/shift)

*Comatose patients may tolerate weaning, if all remaining criteria are fulfilled.

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Table 5 - Steps that can be taken during the weaning process.[42,43]

Steps to follow during weaning from tracheostomy
1. Consider the criteria to attempt weaning.
2. May downsize the tracheostomy for more use of the upper airways.
3. May use a fenestrated tube to decrease air resistance and the work of breathing.
4. May use a speaking valve to help voicing.
5. Cork (plug) the tracheostomy with appropriate monitoring, depending on your hospital protocol.
6. Decannulate if well tolerated, again with monitoring according to your hospital protocol.

Nutritional management. Due to the decreased level of consciousness, severe traumatic brain injury patients may undergo tube feeding for long periods. Early percutaneous endoscopic gastrostomy (PEG) insertion is better than prolonged nasogastric (NG) feeding for those patients.[8,47-49] Some centers perform the tracheostomy procedure and PEG insertion at the same time for the comatose traumatic brain injury patients in the ICU if their coma is expected to be prolonged. The NG tubes are not comfortable for patients and may increase patients' agitation and necessitate the use of restraints. Furthermore, the NG tube may cause delay in the swallowing stages and if misplaced, it may lead to worsening of dysphagia.[50,51] In addition, the risk of malnutrition may increase due to the repeated removal of NG tubes by confused patients. Severe traumatic brain injury patients are at a high risk for developing malnutrition if they lose more than 10% of their body weight.[52] The prevalence of malnourishment in severe traumatic brain injury patients is 68%.[8] The high prevalence of malnourishment is probably due to increased metabolic expenditure (mean of 140%) in comatose head injury patients and to improper nutritional management.[53] Krakau et al[52] assessed the nutritional management of patients with severe traumatic brain injuries and found that nutritional assessment routines (body mass index & energy requirement) for patients were deficient. Incomplete nutritional assessment may lead to improper nutritional management and contribute to the risk for malnutrition. Terre et al[7] found that on discharge from rehabilitation, 72% of the severe traumatic brain injury patients with dysphagia were on oral diet, 14% were on combined oral and tube feeding, and only 14% were fed exclusively via a gastrostomy tube. Also, in a recent study,[6] he documented that their body mass index increases as their dysphagia improves.

In conclusion, dysphagia is a common problem following severe traumatic brain injury and is associated with increased morbidity. A multidisciplinary team approach is essential for the management of dysphagic patients. More teaching and research is needed in this area to increase our clinical knowledge and to improve patients' outcomes.

References

1. Saunders J, Davis C, Miller E. The mechanism of deglutition (second stage) as revealed by cine-radiography. ***Ann Otol Rhinol Laryngol*** 1951; 60: 897-916.
2. Logemann JA. Swallowing disorders. ***Best Pract Res Clin Gastroenterol*** 2007; 21: 563-573.
3. Mackay LE, Morgan AS, Bernstein BA. Swallowing disorders in severe brain injury: risk factors affecting return to oral intake. ***Arch Phys Med Rehabil*** 1999; 80: 365-371.
4. Morgan A, Ward E, Murdoch B, Kennedy B, Murison R. Incidence, characteristics, and predictive factors for dysphagia after pediatric traumatic brain injury. ***J Head Trauma Rehabil*** 2003; 18: 239-251.
5. Ward EC, Grean K, Morton AL. Patterns and predictors of swallowing resolution following adult traumatic brain injury. ***J Head Trauma Rehabil*** 2007; 22: 184-191.
6. Terré R, Mearin F. Evolution of tracheal aspiration in severe traumatic brain injury-related oropharyngeal dysphagia: one-year longitudinal follow-up study. ***Neurogastroenterol Motil*** 2009; 21: 361-369.
7. Terré R, Mearin F. Prospective evaluation of oro-pharyngeal dysphagia after severe traumatic brain injury. ***Brain Inj*** 2007; 21: 1411-1417.
8. Krakau K, Hansson A, Karlsson T, Boussard CN, Tengvar C, Borg J. Nutritional treatment of patients with severe traumatic brain injury during the first six months after injury. ***Nutrition*** 2007; 23: 308-317.
9. Denes Z. The influence of severe malnutrition on rehabilitation in patients with severe head injury. ***Disabil Rehabil*** 2004; 26: 1163-1165.
10. Hansen TS, Larsen K, Engberg AW. The association of functional oral intake and pnuemonia in patients with severe traumatic brain injury. ***Arch Phys Med Rehabil*** 2008; 89: 2114-2120.
11. Cherney LR, Halper AS. Swallowing problems in adults with traumatic brain injury. ***Semin Neurol*** 1996; 16: 349-353.
12. Falsetti P, Acciai C, Palilla R, Bosi M, Carpinteri F, Zingarelli A, et al. Oropharyngeal dysphagia after stroke: incidence, diagnosis, and clinical predictors in patients admitted to a neurorehabilitation unit. ***J Stroke Cerebrovasc Dis*** 2009; 18: 329-335.
13. Finestone HM, Greene-Finestone LS. Rehabilitation medicine: 2. Diagnosis of dysphagia and its nutritional management for stroke patients. ***CMAJ*** 2003; 169: 1041-1044. Review.
14. Ramsey D, Smithard DG, Kalra L. Silent Aspiration: what do we know? Dysphagia. ***Stroke*** 2005; 20: 218-225.
15. Rugiu MG. Role of videofluoroscopy in evaluation of neurologic dysphagia. ***Acta Otorhinolaryngologica Ital*** 2007; 27: 306-316.
16. Magendie F. Precis elementaire de physiologie. Paris (FR): Mequignon-Marvis; 1816-1817; 2: 58-67.
17. Matsuo K, Palmer JB. Anatomy and physiology of feeding and swallowing: normal and abnormal. ***Phys Med Rehabil Clin N Am*** 2008; 19: 691-707.
18. Ertekin C, Aydogdu I. Neurophysiology of swallowing. ***Clin Neurophysiol*** 2003; 114: 2226-2244.
19. Mistry S, Hamdy S. Neural control of feeding and swallowing. ***Phys Med Rehabil Clin N Am*** 2008; 19: 709-728.

Dysphagia20100318 235 10/17/10 2:52:35 PM

20. Blumenfeld H. Brain stem surface anatomy and cranial nerves. In: Blumenfeld H, editor. Neuroanatomy through Clinical Cases Book. Sunderland (MA): Sinauer Associates; 2002.
21. Smithard DG. Swallowing and stroke. Neurological effects and recovery. ***Cerebrovasc Dis*** 2002; 14: 1-8.
22. Logemann JA. Oropharyngeal dysphagia and nutritional management. ***Curr Opin Clin Nutr Metab Care*** 2007; 10: 611-614.
23. Cichero J. Assessment of swallowing disorders. In: Cichero J, Murcoch B, editors. Dysphagia: Foundation, Theory and Practice. Chichester (UK): John Wiley & Sons; 2006. p. 149-165.
24. Ward E, Morgan A. Dysphagia assessment and intervention. In: Ward E, Morgan A, editors. Dysphagia Post Trauma. San Diego (CA): Plural Publishing Inc; 2009. p. 1-30.
25. Schindler A, Vincon E, Grosso E, Miletto AM, Di Rosa R, Schindler O. Rehabilitative management of oropharyngeal dysphagia in acute care settings: data from a large Italian teaching hospital. ***Dysphagia*** 2008; 23: 230-236.
26. McCullough GH, Wertz RT, Rosenbek JC. Sensitivity and specificity of clinical/bedside examination signs for detecting aspiration in adults subsequent to stroke. ***J Commun Disord*** 2001; 34: 55-72.
27. McCullough GH, Rosenbek JC, Wertz RT, McCoy S, Mann G, McCullough K. Utility of clinical swallowing examination measures for detecting aspiration post-stroke. ***J Speech Lang Hear Res*** 2005; 48: 1280-1293.
28. Cannon WB, Moses AT. The movements of food in the esophagus. ***Am J Physiol*** 1898; 1: 435-444.
29. Mosher HP, MacMillan AS. X-ray study of movements of the tongue, epiglottis and hyoid bone in swallowing. ***Laryngoscope*** 1927; 37: 235-262.
30. Ardran GM, Kemp FH. The mechanism of swallowing. ***Proc R Soc Med*** 1951; 44: 1038-1040.
31. Donner MW, Siegel CI. The evaluation of pharyngeal neuromuscular disorders by cinefluorography. ***Am J Roentgenol*** 1965; 94: 299-307.
32. Logemann JA, editor. Manual for the videofluorographic study of swallowing. 2nd ed. Austin (TX): ProEd; 1993.
33. Logemann JA, editor. Evaluation and treatment of swallowing disorders. Austin (TX): ProEd; 1998.
34. Martin-Harris B, Jones B. The videofluorographic swallowing study. ***Phys Med Rehabil Clin N Am*** 2008; 19: 769-785.
35. Langmore SE, Schatz K, Olsen N. Fiberoptic endoscopic examination of swallowing safety: a new procedure. ***Dysphagia*** 1988; 2: 216-219.
36. Hiss SG, Postma GN. Fiberoptic endoscopic evaluation of swallowing. ***Laryngoscope*** 2003; 113: 1386-1393. Review.
37. Leder SB, Murray JT. Fiberoptic endoscopic evaluation of swallowing. ***Phys Med Rehabil Clin N Am*** 2008; 19: 787-801. Review.
38. Robbins J, Butler SG, Daniels SK, Diez Gross R, Langmore S, Lazarus CL, et al. Swallowing and dysphagia rehabilitation: translating principles of neural plasticity into clinically oriented evidence. ***J Speech Lang Hear Res*** 2008; 51: S276-S300.
39. Logemann J. Treatment of oral and pharyngeal dysphagia. ***Phys Med Rehabil Clin N Am*** 2008; 19: 803-816. Review.
40. Mayer V. The challenges of managing dysphagia in brain-injured patients. ***Br J Community Nurs*** 2004; 9: 67-73.
41. Donzelli J, Brady S, Wesling M, Theisen M. Effects of the removal of the tracheotomy tube on swallowing during the fiberoptic endoscopic exam of the swallow (FEES). ***Dysphagia*** 2005; 20: 283-289.
42. Bourjeily G, Fadlallah H, Gerald S. Review of Tracheostomy Usage: Complications and Decannulation Procedures, Part II. ***Clin Pulm Med*** 2002; 9: 273-278.
43. Christopher KL. Tracheostomy decannulation. ***Respirat Care*** 2005; 50: 538-541.
44. Norwood MG, Spiers P, Bailiss J, Sayers RD. Evaluation of the role of a specialist tracheostomy service. From critical care to outreach and beyond. ***Postgrad Med J*** 2004; 80: 478-480.
45. Frank U, Mäder M, Sticher H. Dysphagic patients with tracheotomies: a multidisciplinary approach to treatment and decannulation management. ***Dysphagia*** 2007; 22: 20-29.
46. Garrubba M, Turner T, Grieveson C. Multidisciplinary care for tracheostomy patients: a systematic review. ***Crit Care*** 2009; 13: R177.
47. Loser C, Aschl G, Hebuterne X, Mathus-Vleigen EM, Muscaritoli M, Niv Y, et al. ESPEN guidelines on artificial enteral nutrition--percutaneous endoscopic gastrostomy (PEG). ***Clin Nutr*** 2005; 24: 848-861.
48. Akkersdijk WL, Roukema JA, van der Werken C. Percutaneous endoscopic gastrostomy for patients with severe cerebral injury. ***Injury*** 1998; 29: 11-14.
49. Foley N, Teasell R, Salter K, Kruger E, Martino R. Dysphagia treatment post stroke: a systematic review of randomised controlled trials. ***Age Ageing*** 2008; 37: 258-264.
50. Huggins PS, Tuomi SK, Young C. Effects of nasogastric tubes on the young, normal swallowing mechanism. ***Dysphagia*** 1999; 14: 157-161.
51. Dziewas R, Warnecke T, Hamacher C, Oelenberg S, Teismann I, Kraemer C. Do nasogastric tubes worsen dysphagia in patients with acute stroke? ***BMC Neurol*** 2008; 8: 28.
52. Krakau K, Hansson A, Olin AO, Karlsson T, de Boussard CN, Borg J. Resources and routines for nutritional assessment of patients with severe traumatic brain injury. ***Scand J Caring Sci*** 2010; 24: 3-13.
53. Gencosmanoglu R. Percutaneous endoscopic gastrostomy: a safe and effective bridge for enteral nutrition in neurological or non-neurological conditions. ***Neurocrit Care*** 2004; 1: 309-317. Review.

Dysphagia20100318 236 10/17/10 2:52:36 PM