Neurogenic Dysphagia

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The understanding of normal physiology and pathophysiology of eating and swallowing disorders is essential for evaluating and managing disorders of eating and swallowing and developing dysphagia rehabilitation programs. Eating and swallowing are complex activities including both volitional and reflexive activities involving more than 30 nerves and muscles [1].

Disturbances in the mouth, tongue, pharynx, or esophagus can impair swallowing [*dysphagia*][1]. It can involve mechanical, musculoskeletal, or neurogenic mechanisms. This chapter will focus on neuromuscular and neurogenic causes of dysphagia because the diseases in these categories are seen by the neurologist.

Commonly used models to describe the physiology of normal eating and swallowing are four-stage model for drinking and swallowing liquid and the process model for eating and swallowing solid food. The normal swallow in humans is a three-stage sequential model. The swallowing process is divided into oral, pharyngeal, and esophageal stages according to the location of the bolus [2, 3]. The oral stage is subdivided into oral preparatory and oral propulsive stages, which resulted in four-stage model. Studies on the four-stage model adequately describe biomechanism and bolus movement during voluntary swallows of liquids. However, this model lacked understanding of the bolus movement and the process of eating of solid food. Therefore, the process model of feeding was established to describe the mechanism of eating and swallowing of solid food [4, 5].

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Oral Preparatory Stage

After liquid is taken into the mouth from a cup or by a straw, the liquid bolus is held in the anterior part of the floor of the mouth or on the tongue surface against the hard palate surrounded by the upper dental arch (upper teeth). The soft palate seals oral cavity and tongue contact to prevent the liquid bolus leaking into the oropharynx before the swallow. There can be leakage of liquid into the pharynx if the seal is imperfect. The impairment of this mechanism occurs with aging.

Oral Propulsive Stage

During oral propulsive stage, the tongue tip rises, touching the alveolar ridge of the hard palate just behind the upper teeth, while the posterior tongue drops to open the back of the oral cavity. The tongue surface moves upward, gradually expanding the area of tongue-palate contact from anterior to posterior, squeezing the liquid bolus back along the palate and into the pharynx. When drinking liquids, the pharyngeal stage normally begins during oral propulsion.

Oral Stage in Eating Solid Food (Process Model of Feeding)

The four-stage sequential model was unable to explain normal eating in humans, especially food transport and bolus formation in the oropharynx [4–6].

When healthy subjects eat solid food, it is chewed and moistened. It passes through the fauces for bolus formation in the oropharynx (including the valleculae) several seconds prior to the pharyngeal stage of a swallow. Additional portions of food can pass into the oropharynx and accumulate there while food remains in the oral cavity and chewing continues. Eating and swallowing solid food is a continuous process unlike swallowing liquids and, hence, cannot be explained by four-stage model. There is an overlap in oral preparatory, propulsive, and pharyngeal stage of swallowing solids. Hence, process model based on studies in mammalian is adapted.

Stage I Transport

The ingested food is carried and placed in occlusal surface of lower teeth for processing.

Food Processing

In this stage, food particles are chewed to small size and softened by salivation until the food consistency is optimal for swallowing. Chewing continues until it is of

optimal consistency for swallowing. Chewing is continuous process till it is ready for swallowing. The movements of jaw, tongue, cheek, soft palate, and hyoid bone are preferably coordinated.

Movements of the Jaw, Hyoid, and Tongue or Soft Palate Over Time

During this, there is an open passage between oral cavity and pharynx unlike closure of posterior oral cavity during drinking liquids [5, 7]. Movements of the jaw and tongue pump air into the nasal cavity through the pharynx, delivering the food's aroma to chemoreceptors in the nose [8–10].

The tongue movements are coordinated with jaw opening and closing in such a manner as to avoid tongue coordinated by suprahyoid and infrahyoid muscles. These muscles help to control the movements of the jaw and tongue as well [11, 12].

Stage II Transport

The masticated food is placed on the tongue surface and pushed back to the oropharynx. The basic mechanism of stage II transport is as described for the oral propulsive stage with a liquid bolus. (The anterior tongue surface first contacts the hard palate just behind the upper incisors. The area of tongue-palate contact gradually expands backward, squeezing the masticated food back along the palate to the oropharynx.) Stage II transport involves the tongue and does not require gravity [13, 14] and can be along with food processing cycles. The transported food accumulates on the pharyngeal surface of the tongue and in the valleculae. If food remains in the oral cavity, chewing continues and the bolus in the oropharynx is enlarged by subsequent stage II transport cycles. The duration of bolus aggregation in the oropharynx ranges from a fraction of a second to about 10 s in normal individuals eating solid food [5].

Pharyngeal Stage

Pharyngeal swallow occurs in seconds. During this phase, bolus is propelled to pharynx and to esophagus simultaneously closing larynx and trachea preventing food from entering the airway. As the soft palate elevates, the nasopharynx closes at the same time and prevents bolus regurgitation in the nasal cavity. The base of the tongue retracts, pushing the bolus against the pharyngeal walls. The pharyngeal constrictor muscles contract sequentially from the top to the bottom, squeezing the bolus downward. This reduces the volume of pharyngeal cavity.

Prevention of aspiration during swallowing is very essential in human beings. (There are several airway protective mechanisms preventing aspiration of the foreign materials to the trachea before or during swallowing.) During swallowing, vocal folds adduct to close the glottis (space between the vocal folds) and the arytenoids tilt forward to contact the epiglottic base prior to opening of the UES [15, 16]. Suprahyoid muscles and thyrohyoid muscle contract to pull hyoid and larynx

upward and forward. (This displacement tucks the larynx under the base of the tongue. The epiglottis tilts backward to seal the laryngeal vestibule.)

Opening of the upper esophageal sphincter (UES) is essential for the bolus entry into the esophagus. The UES consists of the inferior pharyngeal constrictor muscles, cricopharyngeus muscle, and most proximal part of the esophagus. The UES is closed at rest by tonic muscle contraction [17, 18]. Three important factors contribute to the UES opening: (1) relaxation of the cricopharyngeus muscle; this relaxation normally precedes opening of the UES or arrival of the bolus; (2) contraction of the suprahyoid muscles and thyrohyoid muscles. These muscles pull the hyolaryngeal complex forward, opening the sphincter; and (3) the pressure of the descending bolus [19]. This pressure distends the UES, assisting its opening. The most important of these mechanisms is factor 2, the active opening process. This makes opening of the UES quite different from other sphincters (such as the external urethral sphincter which opens passively when it is pushed open by the descending fluid).

Esophageal Stage

The esophagus is a tubular structure from the lower part of the UES to the lower esophageal sphincter (LES). The lower esophageal sphincter is also tensioned at rest to prevent regurgitation from the stomach. It relaxes during a swallow and allows the bolus passage to the stomach. The cervical esophagus (upper one third) is mainly composed of striated muscle, but the thoracic esophagus (lower two thirds) is smooth muscle. Bolus transport in the thoracic esophagus is quite different from that of the pharynx, because it is true peristalsis regulated by the autonomic nervous system. Once the food bolus enters the esophagus passing the UES, a peristalsis wave carries the bolus down to the stomach through the LES. The peristaltic wave consists of two main parts, an initial wave of relaxation that accommodates the bolus, followed by a wave of contraction that propels it. Gravity assists peristalsis in upright position.

Bolus Location at Swallow Initiation in Normal Swallows

The position of the head of the bolus relative to the time of pharyngeal swallow onset is a measure of swallow elicitation. The point where the x-ray shadow of the ramus of the mandible crosses the pharyngeal surface of the tongue is commonly used as a marker for this measurement. At one time, it was believed that the pharyngeal swallow was normally triggered when the bolus head passes the fauces as seen on videofluoroscopy [3]. If the bolus head passed the lower border of the mandible more than 1 s before the swallow initiation, it was classified as delayed swallow initiation. Delayed swallow initiation is considered an important finding because the airway is open when the bolus approaches toward the larynx.

However, recent studies have revealed that pre-swallow bolus entry into the pharynx also occurs in healthy individuals drinking liquids [20–22]. Furthermore, as described above, during eating of solid food, chewed bolus is aggregated in the oropharynx or valleculae prior to swallowing. Bolus position at swallow initiation is now known to be quite variable in normal eating and swallowing. This is especially true when consuming a food that has both liquid and solid phases. Saitoh et al. [14] demonstrated that in healthy young adult eating a food that included soft solid and thin liquid components, the leading edge (liquid component) of the food often entered the hypopharynx before swallowing. As seen in, liquid enters the hypopharynx during chewing and approaches the laryngeal aditus at a time when the larynx remains open. The location of the bolus at swallow initiation is altered by sequential swallowing of liquid [20, 23–26]. The bolus head often reaches the valleculae before pharyngeal swallow initiation, especially when the larynx remains closed between swallows.

Coordination among Eating, Swallowing, and Breathing

Eating, swallowing, and breathing are tightly coordinated. Swallowing is dominant to respiration in normal individuals [27–29]. Breathing ceases briefly during swallowing, not only because of the physical closure of the airway by elevation of the soft palate and tilting of the epiglottis, but also of neural suppression of respiration in the brain stem [28]. When drinking a liquid bolus, swallowing usually starts during the expiratory phase of breathing. The respiratory pause continues for 0.5–1.5 s during swallowing, and respiration usually resumes with expiration [30–32]. This resumption is regarded as one of the mechanisms that prevent inhalation of food remaining in the pharynx after swallowing [33]. When performing sequential swallows while drinking from a cup, respiration can resume with inspiration [34].

Eating solid food also alters the respiratory rhythm. The rhythm is perturbed with onset of mastication. Respiratory cycle duration decreases during mastication, but with swallowing [29, 35, 36]. The “exhale-swallow-exhale” temporal relationship persists during eating. However, respiratory pauses are longer, often beginning substantially before swallow onset [10, 36, 37].

Causes of Neurogenic Dysphagia

Normal swallowing depends on the anatomical and functional integrity of numerous neural structures and extensive pathways in the central and peripheral nervous system. Lesions of the cerebral cortex, basal ganglia, brain stem, cerebellum, and lower cranial nerves may result in dysphagia. Degenerations of the myenteric ganglion cells in the esophagus, muscle diseases, and disorders of neuromuscular transmission, for example, myasthenia gravis and Eaton-Lambert syndrome, are other less common causes.

Cerebral Cortex

The stroke is the commonest cortical condition associated with dysphagia; one fourth to half cases of all strokes are associated with swallowing difficulty [38]. Dysphagia in these patients is usually associated with hemiplegia due to lesions of the brain stem or the involvement of one or both hemispheres. Rarely isolated dysphagia is presenting symptom of stroke. Dysphagia is seen in patients with lacunar infarcts in the periventricular white matter [39] and after discrete vascular brain stem lesions [40]. These patients may not have associated neurological deficit. The swallowing difficulty in acute stroke is usually transient lasting for 2 weeks in most.

The symptoms persist in about 8 % of patients for 6 months or more [41]. The occurrence of dysphagia in acute stroke does not appear to depend on the size or the site of the lesion. Right parietal strokes are associated with persistent dysphagia.

Basal Ganglia

Dysphagia is a common symptom in patients with Parkinson's disease particularly in the later stages of the disease. Occasionally, dysphagia may even be a presenting symptom of Parkinson's disease.

More than 80 % of patients with Parkinson's disease have mild dysphagia, and usually, patient's nutritional status is well maintained. However, in about 10 % of dysphagic Parkinsonian patients, the symptoms are severe, and this generally correlates with the severity and duration of the disease. Tremor and speech disturbances have been found to be the main predictors of dysphagia in these patients [42].

The swallowing difficulties seen in Parkinson's disease involve the oral phase (difficulties with lip closure and tongue movements) and the pharyngeal stage (complaints of food sticking in the throat). Dysphagia is due to abnormal bolus formation, multiple tongue elevations, delayed swallow reflex, and prolongation of the pharyngeal transit time with repetitive swallows to clear the throat as shown on videofluoroscopy. Drooling, which is commonly seen in patients with Parkinsonism, is not due to excessive salivation but is due to impaired swallowing due to bradykinesia of the oropharyngeal musculature. Other Parkinsonian syndromes, for example, progressive supranuclear palsy and multisystem atrophy, cause more severe symptoms.

Dysphagia is also common in spasmodic torticollis. Videofluoroscopy revealed impairment of swallowing in more than half the patients [43]. Interestingly, only two thirds of the study patients were symptomatic and were independent of patient's age or disease duration. The dysphagia may be due to dystonia of laryngeal and pharyngeal muscle involvement. The nomenclature now is cervical dystonia than spasmodic torticollis.

Cerebellum and Brain Stem

The oral phase is affected in cerebellum and brain stem lesions due to bulbar or pseudobulbar palsy; this leads to poor coordination of the oral and pharyngeal musculature resulting in poor lip seal, impaired initiation of the swallow reflex, poorly formed food bolus, and its propulsion to the pharynx.

Peripheral Nerves and Muscles

The rare causes of dysphagia are isolated peripheral nerve lesions and degeneration of autonomic ganglion cells in the lower two thirds of the esophagus (which results in achalasia). This causes stagnation of food and dilatation of esophagus due to abnormally reduced motility of the lower esophagus with tightening of the sphincter. The diagnosis is confirmed with endoscopy and studies of esophageal motility. Common symptom in addition includes halitosis.

Myasthenia gravis is a neuromuscular junction abnormality. Dysphagia is commonly associated with dysphonia and dysarthria. The weakness is often fluctuating and may not be evident at the time of examination. The swallowing is commonly affected in elderly myasthenics. The diagnosis of the underlying disorder can usually be confirmed with single-fiber electromyography.

Drugs and Dysphagia

Many drugs may precipitate or aggravate swallowing difficulties. This effect is usually dose dependent and is often reversible with discontinuation of the drug. Sometimes, reduction of the drug dose is sufficient. The mechanisms implicated in drug-induced dysphagia include reduced level of consciousness (sedatives and hypnotics) causing interference with the oropharyngeal phase of swallowing or as a direct effect on brain stem neurons or blocking of acetylcholine release at the neuromuscular junction. Some drugs mediate their effect on swallowing by more than one mechanism.

Initiation of swallowing reflex is delayed by neuroleptics [dopamine-blocking agents] in absence of extrapyramidal features. Dopaminergic drugs can cause orofacial dyskinesia which interferes with the preparation of the food bolus and its delivery to the pharynx. Anticholinergic drugs cause dryness of mouth with impaired bolus formation with dysphagia. The benzodiazepines can cause impaired level of consciousness and suppress brain stem neurons that regulate swallowing [44, 45].

Botulinum toxin type A causes dysphagia due to inhibition of neural transmission at the neuromuscular junction. It is the drug of choice for the treatment of cervical dystonia and may cause dysphagia in 10–28 % of these patients. This adverse effect is usually mild and transient, lasting 10–14 days. Clinical observations suggest that the incidence of dysphagia is increased when a large dose of the drug is injected. It should also be noted that cervical dystonia may also be associated with dysphagia.

Clinical Manifestations of Dysphagia and Pulmonary Aspiration

Patients with mild to moderate difficulty in swallowing may not be aware of the swallowing difficulty, and weight loss may be the only symptom. Patients tend to drool in sitting position and may cough in the night due to silent aspiration.

Swallowing assessment would include inspection of oral cavity and small quantity trial swallows. Pooling of saliva in the oral cavity would indicate difficulty in swallowing liquids. Different consistencies of food and liquids should be tried. Watch for coughing and choking while eating; they are obvious signs. Change in the voice and observation of breathing pattern may be early signs. Making patient swallow water in upright position and observing their speed of swallowing are a quick bedside method of assessing swallowing [46]. The speed of swallowing is reduced to 10 ml/s and may indicate neurogenic dysphagia. Regular and frequent monitoring of swallowing is possible by this simple method. The other methods to assess swallowing include videofluoroscopy, fiber-optic nasoendoscopy, and pulse oximetry [47]. Videofluoroscopy allows direct visualization of oral preparatory phase, reflex initiation of swallowing, and actual passage of bolus in the pharyngeal phase, and direct aspiration in the respiratory tract can be seen. The disadvantages of this test include its unsuitability for repeated assessments. Fiber-optic endoscopy involves placing endoscope just above the soft palate and observing pharyngeal pooling before and after swallowing.

Patients with neurogenic dysphagia find fluids more difficult to swallow than solids. A solid food bolus is more likely to trigger a swallow reflex than liquid. Dysphagia resulting from brain stem lesions or confluent periventricular infarction may affect predominantly the volitional initiation of swallowing. Reflex swallowing is normal in such patients. Swallowing is associated with severe emotional distress, and patient complains of a lump in the throat. These patients have a normal bolus transit time and do not complain of difficulties with eating or drinking.

Complications of Dysphagia

The most dreaded complication of difficulty in swallowing is pulmonary aspiration. In addition, patient's caloric intake may be affected resulting in loss of weight. Reduced liquid intake may result in dehydration.

Pulmonary aspiration is defined as passage of food or fluid into the airways below the true vocal cords. Silent aspiration may go undetected unless clinician has high index of suspicion and may only be detected on pulse oximetry. One third of patients with difficulty in swallowing tend to aspirate their food or liquids in their airway, and 40 % of these patients have silent aspiration. Silent aspiration does not trigger coughing or cause distress. The patients often do not complain of swallowing difficulties. Weak cough may be one of the symptoms of early silent aspiration.

Management

Multidisciplinary approach is essential for management of neurogenic dysphagia. The team includes speech and language therapist, a dietician, a nurse, and a physician.

The causes of dysphagia are oral problems which result in poor food bolus formation. Poorly fitted dentures should be corrected, and mouth ulcers and candida infection should be treated. Avoid feeding patients when distracted particularly while watching television or talking. This increases the risk of aspiration. The suction should be carried out to remove saliva. Parkinson's patients experience on/off phenomenon. The swallowing may be normal in "on" state and impaired remarkably in "off" state.

Aspiration of saliva makes it necessary to do frequent suctioning of oral cavity. In dysphagic patients who have a tracheostomy, occlusion of the stoma with a speech valve during swallowing reduces the risk of pulmonary aspiration presumably by normalizing the pressure in the upper airways. Posture during swallowing is very important. For example, "chin tuck" decreases the pharyngeal transit time of the food bolus, whereas "chin up" has the opposite effect. Head tilt to one side to maximize the effect of gravity on the unaffected side of pharynx is also a useful strategy on some occasions.

It has been shown that patients with weak tongue movements and those with poor pharyngeal clearance of the food bolus benefit from the use of gravity and posture to facilitate safe swallowing. Lying down on one side (at 45° from flat) may be associated with less risk of aspiration than feeding in the upright position [48, 49].

Sedative and other drugs that reduce the patient's level of consciousness should be discontinued. In patients with Parkinson's disease, drug-induced dyskinesia may aggravate dysphagia, and the successful management of this complication usually improves swallowing. Sometimes, it is sufficient to avoid feeding during periods of peak-dose dyskinesia. Drooling in Parkinsonian patients is primarily due to swallowing difficulties rather than the excessive production of saliva. Anticholinergic drugs can aggravate dysphagia by increasing the viscosity of oral secretions. Viscid secretions interfere with bolus preparation and predispose to the formation of a mucous plug. Hence, these drugs are avoided in Parkinson patients with dysphagia. Benzodiazepines should be avoided in dysphagic patients, and anticonvulsants should be taken as a single dose at bedtime if possible.

Dietary Modification

Avoidance of dry and sticky food and eating food with uniform consistency and the use of starch-based fluid thickeners are also an important management strategy. Tube feeding is usually required in only a minority of patients.

Patients with neurogenic dysphagia experience more difficulties with fluids than with solid food. This is probably due to the difficulty in controlling a thin bolus and a delay or absence of triggering the swallow reflex. The rationale for the use of fluid thickeners is that by increasing the viscosity of ingested fluids, the resistance to flow of the bolus is increased. In addition, the duration of cricopharyngeal opening and the oropharyngeal transit time are increased. However, the optimal viscosity of fluids that ensures safe swallowing in patients with neurogenic dysphagia has not been established. In practice, the required fluid thickness is judged subjectively and recorded using descriptive terms such as syrup or yogurt consistency. This has the disadvantage that fluids with low viscosity may be served and result in pulmonary aspiration. Thick fluids are usually unpalatable and are often disliked by patients. A viscometer may be utilized to prepare correct thickness and has been shown to improve the dietary management in these cases [50].

Tube Feeding

Patients who are at risk of pulmonary aspiration if fed orally should be tube fed. Increased transit time of the food bolus on videofluoroscopy [51] may be indication for tube feeding. In some cases, easy fatigability makes swallowing unsafe; tube feeding can be used to supplement the daily oral intake. The patients are able to take their favorite foods orally, and the rest of the calorie requirements will be given through the tube.

The use of a gastrostomy tube is preferred to nasoesophageal intubation, especially in prolonged dysphagia. Nasogastric tube feeding is poorly tolerated. Patient may get irritable or agitated. Patients commonly extubate themselves; the volume of feeds delivered is inadequate. Patients fed using a nasogastric tube received less feeds as compared to those fed with a gastrostomy tube [52]; nasogastric tube uses fine-bore tubes, which are more likely to dislodge, kink, or block. They also deliver feeds at a relatively slow rate.

Some patients with neurological disease develop gastrointestinal ileus, and in these patients, enteral nutrition could be established with the intrajejunal administration of low-residue solutions.

Prolonged nasogastric tube feeding often results in nasopharyngitis, esophagitis, esophageal strictures, epistaxis, pneumothorax, and nasopharyngeal edema with associated otitis media. Furthermore, nasogastric tube feeding does not fully protect against aspiration, and the association between nasogastric tube feeding and this complication is well documented. Forty-three percent of dysphagic patients aspirated in the first 2 weeks after nasogastric tube feeding was started Ciocon et al. [53]. Elevation of the head of the bed during and for 1–2 h after feeding reduces the risk of aspiration in these patients.

Most clinicians would consider gastrostomy tube feeding in stroke patients if there are no signs of recovery of swallowing after the first week. In patients with motor neuron disease, the option of percutaneous endoscopic gastrostomy (PEG) tube feeding should be offered early after the onset of dysphagia to supplement the

oral intake and help maintain the muscle mass. Insertion of the feeding tube through a PEG, rather than a surgical gastrostomy, is a relatively simple, safe, and cost-effective technique. PEG tube feeding is effective and is usually acceptable to patients and their caretakers. Transient, self-limiting abdominal pain and diarrhea [54] may occur in the early postoperative period. Long-term complications include tube obstruction and wound infection.

In some patients who are fed via a PEG tube, pulmonary aspiration may occur, and routine intrajejunal feeding has been suggested for these cases. An additional advantage is that bolus gastric tube feeding is more physiological, particularly with respect to insulin secretion. Furthermore, because the feeds can be given intermittently, it allows greater patient freedom (intrajejunal feeding should be given continuously rather than intermittently). Direct intrajejunal delivery of nutrients should probably be reserved for patients with gastroesophageal reflux, hiatus hernia, or recurrent aspiration on gastrostomy feeding.

Swallowing Therapy

Swallowing exercises are used to strengthen the orofacial musculature, maneuvers to improve poor laryngeal elevation and laryngeal closure during swallowing, and techniques to stimulate the swallow reflex. These methods are usually used before starting direct swallowing.

Exercises to enhance the function of the orofacial muscles are used to improve lip seal, mastication, and tongue movements. A simple technique known as “the supraglottic swallow” may improve the elevation and closure of the larynx during swallowing. During this maneuver, the subject holds his/her breath and swallows, and he/she releases the air by coughing. Patients with delayed or absent swallow reflex often benefit from thermal stimulation of the oropharyngeal receptors. The procedure has been claimed to improve triggering of the swallowing action and to reduce the bolus transit time. It involves the repeated application of a small laryngeal mirror dipped in ice to the anterior faucial arch. Sensitization may be repeated between swallows. Direct swallowing therapy can be started with small amounts of food (of the right consistency) under the supervision of a speech and language therapist when the risk of pulmonary aspiration is deemed to be low.

Surgical Treatment of Neurogenic Dysphagia

Cricopharyngeal myotomy is an effective method of treatment of dysphagia in patients with stroke, muscular dystrophy, and in patients with motor neuron disease. Careful selection of patients is essential prior to this procedure. Two important things should be looked for, that is, failure of relaxation of the pharyngeal sphincter must be demonstrated on videofluoroscopy. Secondly, the oral phase of swallowing, lip seal, voluntary initiation of swallowing, and the propulsive action of the tongue must also be preserved. Poor tongue movement (demonstrated on videofluoroscopy

by the inability to propel or retrieve the food bolus) is a contraindication to cricopharyngeal myotomy. Patients with absent pharyngeal peristalsis or delayed triggering of the swallow reflex by 10 s or more are also unlikely to benefit from this treatment. Surgery for cricopharyngeal dysfunction after stroke and traumatic brain injury should be considered after the first 3 months of the disease onset.

Relaxation of the cricopharyngeus can also be achieved with "chemical cricopharyngeal myotomy" using botulinum toxin type A injections [55]. The location of the cricopharyngeal muscle is determined with direct esophagoscopy and electromyography (using a hooked wire electrode), and the toxin is injected transcutaneously into the dorsomedial part and into the ventrolateral part of the muscle on both sides. A total dose of botulinum toxin type A of 80–120 units is usually sufficient, and the mean beneficial effect of treatment is 5 months. This may be used in patients with lateral medullary syndrome.

References

1. Jones B, editor. Normal and abnormal swallowing: imaging in diagnosis and therapy. 2nd ed. New York: Springer; 2003.
2. Dodds WJ, Stewart ET, Logemann JA. Physiology and radiology of the normal oral and pharyngeal phases of swallowing. AJR Am J Roentgenol. 1990;154(5):953–63.
3. Logemann JA. Evaluation and treatment of swallowing disorders. 2nd ed. Austin: Pro-Ed; 1998.
4. Palmer JB, Rudin NJ, Lara G, Crompton AW. Coordination of mastication and swallowing. Dysphagia. 1992;7(4):187–200.
5. Hiiemae KM, Palmer JB. Food transport and bolus formation during complete feeding sequences on foods of different initial consistency. Dysphagia. 1999;14(1):31–42.
6. Dua KS, Ren J, Bardan E, Xie P, Shaker R. Coordination of deglutitive glottal function and pharyngeal bolus transit during normal eating. Gastroenterology. 1997;112(1):73–83.
7. Matsuo K, Hiiemae KM, Palmer JB. Cyclic motion of the soft palate in feeding. J Dent Res. 2005;84(1):39–42.
8. Buettner A, Beer A, Hannig C, Settles M. Observation of the swallowing process by application of videofluoroscopy and real-time magnetic resonance imaging-consequences for retronasal aroma stimulation. Chem Senses. 2001;26(9):1211–9.
9. Hodgson M, Linforth RS, Taylor AJ. Simultaneous real-time measurements of mastication, swallowing, nasal airflow, and aroma release. J Agric Food Chem. 2003;51(17):5052–7.
10. Palmer JB, Hiiemae KM. Eating and breathing: interactions between respiration and feeding on solid food. Dysphagia. 2003;18(3):169–78.
11. Palmer JB, Hiiemae KM, Liu J. Tongue-jaw linkages in human feeding: a preliminary videofluorographic study. Arch Oral Biol. 1997;42(6):429–41.
12. Mioche L, Hiiemae KM, Palmer JB. A postero-anterior videofluorographic study of the intraoral management of food in man. Arch Oral Biol. 2002;47(4):267–80.
13. Palmer JB. Bolus aggregation in the oropharynx does not depend on gravity. Arch Phys Med Rehabil. 1998;79(6):691–6.
14. Saitoh E, Shibata S, Matsuo K, Baba M, Fujii W, Palmer JB. Chewing and food consistency: effects on bolus transport and swallow initiation. Dysphagia. 2007;22(2):100–7.
15. Shaker R, Dodds WJ, Dantas RO, Hogan WJ, Arndorfer RC. Coordination of deglutitive glottic closure with oropharyngeal swallowing. Gastroenterology. 1990;98(6):1478–84.
16. Ohmae Y, Logemann JA, Kaiser P, Hanson DG, Kahrilas PJ. Timing of glottic closure during normal swallow. Head Neck. 1995;17(5):394–402.

17. Cook IJ, Dodds WJ, Dantas RO, et al. Opening mechanisms of the human upper esophageal sphincter. Am J Physiol. 1989;257(5 Pt 1):G748–59.
18. Ertekin C, Aydogdu I. Electromyography of human cricopharyngeal muscle of the upper esophageal sphincter. Muscle Nerve. 2002;26(6):729–39.
19. Shaw DW, Cook IJ, Gabb M, et al. Influence of normal aging on oral-pharyngeal and upper esophageal sphincter function during swallowing. Am J Physiol. 1995;268(3 Pt 1):G389–96.
20. Daniels SK, Foundas AL. Swallowing physiology of sequential straw drinking. Dysphagia. 2001;16(3):176–82.
21. Martin-Harris B, Brodsky MB, Michel Y, Lee FS, Walters B. Delayed initiation of the pharyngeal swallow: normal variability in adult swallows. J Speech Lang Hear Res. 2007;50(3):585–94.
22. Stephen JR, Taves DH, Smith RC, Martin RE. Bolus location at the initiation of the pharyngeal stage of swallowing in healthy older adults. Dysphagia. 2005;20(4):266–72.
23. Chi-Fishman G, Stone M, McCall GN. Lingual action in normal sequential swallowing. J Speech Lang Hear Res. 1998;41(4):771–85.
24. Chi-Fishman G, Sonies BC. Motor strategy in rapid sequential swallowing: new insights. J Speech Lang Hear Res. 2000;43(6):1481–92.
25. Chi-Fishman G, Sonies BC. Kinematic strategies for hyoid movement in rapid sequential swallowing. J Speech Lang Hear Res. 2002;45(3):457–68.
26. Daniels SK, Corey DM, Hadskey LD, et al. Mechanism of sequential swallowing during straw drinking in healthy young and older adults. J Speech Lang Hear Res. 2004;47(1):33–45.
27. Nishino T, Yonezawa T, Honda Y. Effects of swallowing on the pattern of continuous respiration in human adults. Am Rev Respir Dis. 1985;132(6):1219–22.
28. Nishino T, Hiraga K. Coordination of swallowing and respiration in unconscious subjects. J Appl Physiol. 1991;70(3):988–93.
29. McFarland DH, Lund JP. Modification of mastication and respiration during swallowing in the adult human. J Neurophysiol. 1995;74(4):1509–17.
30. Selley WG, Flack FC, Ellis RE, Brooks WA. Respiratory patterns associated with swallowing: Part 1. The normal adult pattern and changes with age. Age Ageing. 1989;18(3):168–72.
31. Klahn MS, Perlman AL. Temporal and durational patterns associating respiration and swallowing. Dysphagia. 1999;14(3):131–8.
32. Martin-Harris B, Brodsky MB, Michel Y, Ford CL, Walters B, Heffner J. Breathing and swallowing dynamics across the adult lifespan. Arch Otolaryngol Head Neck Surg. 2005;131(9):762–70.
33. Shaker R, Li Q, Ren J, et al. Coordination of deglutition and phases of respiration: effect of aging, tachypnea, bolus volume, and chronic obstructive pulmonary disease. Am J Physiol. 1992;263(5 Pt 1):G750–5.
34. Dozier TS, Brodsky MB, Michel Y, Walters Jr BC, Martin-Harris B. Coordination of swallowing and respiration in normal sequential cup swallows. Laryngoscope. 2006;116(8):1489–93.
35. Smith J, Wolkove N, Colacone A, Kreisman H. Coordination of eating, drinking and breathing in adults. Chest. 1989;96(3):578–82.
36. Matsuo K, Hiiemae KM, Gonzalez-Fernandez M, Palmer JB. Respiration during feeding on solid food: alterations in breathing during mastication, pharyngeal bolus aggregation and swallowing. J Appl Physiol. 1985;104(3):674–81. Epub 2007 Dec 27
37. Charbonneau I, Lund JP, McFarland DH. Persistence of respiratory-swallowing coordination after laryngectomy. J Speech Lang Hear Res. 2005;48(1):34–44.
38. Kidd D, Lawson J, Nesbitt R, et al. Aspiration in acute stroke: a clinical study with videofluoroscopy. Q J Med. 1993;86:825–9.
39. Celifarco A, Gerard G, Faegenburg D, et al. Dysphagia as the sole manifestation of bilateral strokes. Am J Gastroenterol. 1990;85:610–3.
40. Buchholz DW. Clinically probable brain stem stroke presenting as dysphagia and nonvisualised by MRI. Dysphagia. 1993;8:235–8.
41. Smithard DG, O'Neill PA, Martin DF, et al. The natural history of dysphagia following stroke. Dysphagia. 1997;12:188–93.

42. Coates C, Bakheit AMO. Dysphagia in Parkinson's disease. Eur Neurol. 1997;38:49–52.
43. Riski JE, Horner J, Nashold BS. Swallowing function in patients with spasmodic torticollis. Neurology. 1990;40:1443–5.
44. Wyllie E, Wyllie R, Cruse RP, et al. The mechanism of nitrazepam-induced drooling and aspiration. N Engl J Med. 1986;14:35–8.
45. Hockman CH, Bieger D. Inhibitory effect of diazepam on reflexly-induced deglutition. Proc Can Fed Biol Sci. 1979;2:85.
46. Nathadwarawala KM, Nicklin J, Wiles CM. A timed test of swallowing capacity for neurological patients. J Neurol Neurosurg Psychiatry. 1992;55:822–5.
47. Collins MJ, Bakheit AMO. Does pulse oximetry reliably detect aspiration in dysphagic stroke patients? Stroke. 1997;28:1773–5.
48. Ekberg O. Posture of the head and pharyngeal swallowing. Acta Radiol. 1986;27:691–6.
49. Drake W, O'Donoghue S, Bartram C, et al. Eating in side lying facilitates rehabilitation in neurogenic dysphagia. Brain Inj. 1997;11:137–42.
50. Goulding R, Bakheit AMO. Evaluation of the benefits of monitoring fluid thickness in the dietary management of dysphagic stroke patients. Clin Rehabil. 2000;14:119–24.
51. Park RHR, Allison MC, Lang J, et al. Randomised comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding in patients with persisting neurological dysphagia. BMJ. 1993;304:1406–9.
52. Wilson PS, Johnson AP, Bruce-Lockhart FJ. Videofluoroscopy in motor neurone disease prior to cricopharyngeal myotomy. Ann R Coll Surg Engl. 1990;72:375–7.
53. Ciocon JO, Silverstone FA, Graver LM, et al. Tube feeding in elderly patients. Indications, benefits and complications. Arch Intern Med. 1988;148:429–33.
54. Keohane PP, Attrill H, Love M, et al. Relation between osmolality of diet and gastrointestinal side effects in enteral nutrition. BMJ. 1984;288:678–80.
55. Schneider I, Thumfart WF, Potoschnig C, et al. Treatment of dysfunction of the cricopharyngeal muscle with botulinum A toxin: introduction of a new non-invasive method. Ann Otol Rhinol Laryngol. 1994;103:31–5.