Dysphagia

Dysphagia is a distressing symptom, which can have far-reaching effects on a patient's life.

ALAN McCULLOCH, ***MB BCh, FCS (SA) ORL***
ENT Surgeon in private practice, Sandton Medi-Clinic, Johannesburg
Alan McCulloch is a member of the multidisciplinary Dysphagia Treatment Unit at RehabMatters Therapy Centre in Rivonia, Johannesburg.
E-mail: alanent@gmail.com

Dysphagia is the general term for disordered swallowing. The inability to swallow safely will have an impact on the ability to maintain both hydration and nutrition at an adequate level. Additionally, not being able to meet and socialise over a meal or a drink, or enjoy a family meal together has vast ramifications on one's enjoyment of life and social interaction.

This article provides a simple overview of the swallowing process and common clinical problems and a review of some of the current evaluations, treatments and rehabilitation options available to patients with dysphagia.

It is estimated that between 6 and 15 million adults and an unknown number of children in the USA alone are affected by dysphagia.[1]

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Stages of swallow

Swallowing can be divided into 3 functional phases:

- oral phase
- pharyngeal phase
- oesophageal phase.

While this is a very practical classification, it should be emphasised that all phases are functionally connected and that dysfunction in one phase will have a significant impact on function in all the others.

The process of swallowing needs to be considered as part of a pressure-generating system. The bolus will always move from one high-pressure chamber to the next low-pressure chamber, with muscles compressing and expanding the various compartments and their associated valves. Any anatomical or neuromuscular dysfunction in one area will result in a loss of the pressure gradient and as a result, transit of the bolus may be slowed or inco-ordinated.

The path a bolus takes (Fig. 1)

The bolus is introduced to the oral cavity; thereafter there is interaction between orbicularis oris, buccinator and the muscles of mastication to adequately prepare the bolus for the pharyngeal stage.

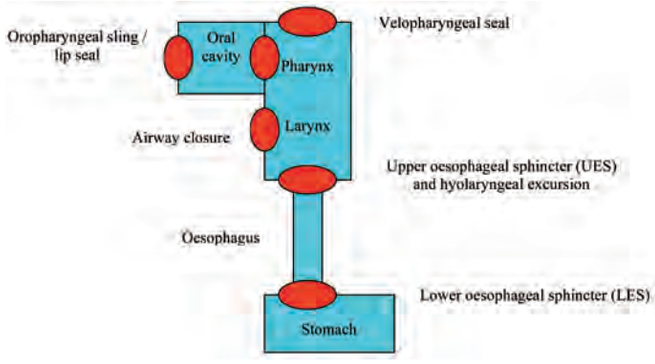

Fig. 1. The path of a bolus. (Printed with kind permission from Y Wijting, Vitalstim Training Manual.)

This stage of swallowing, the oral phase, is entirely under voluntary control via the cortical and subcortical areas of the brain.

Once sensory information reports the bolus to be adequately prepared, the bolus will be moved posteriorly. As the bolus passes the posterior faucal (tonsil) arches the swallow becomes reflexive under the control of the brainstem. It should be noted that in the over-60s, the swallow may only become reflexive as the bolus reaches the vallecula (between the back of the tongue and epiglottis). Simultaneously, velopharyngeal closure occurs, i.e. the soft palate lifts and closes the posterior nasal space to prevent movement of the bolus into the nasal cavity. The lips should remain tightly closed, the teeth closely approximated and the tongue in contact with the palate for pressure in the oral chamber to be maintained. Failure of these processes will result in pocketing of food and residuals being left in the oral cavity. Oral-pharyngeal seal is completed by the full retraction of the tongue base onto the posterior pharyngeal wall.

A number of processes assist in the transit of the bolus into the pharynx. Firstly, elevation and forward movement of the hyoid creates a negative sucking pressure in the hypopharynx. Secondly, closure of the airway and an apnoeic interval during the pharyngeal phase prevents the bolus being sucked towards the negative pressure within the airway and finally, the sequential stripping action of the superior, middle and inferior pharyngeal constrictors moves the bolus into the upper oesophagus. Dysfunction in the pharyngeal phase can lead to multiple problems in the safe transit of the bolus, the most crucial being the increased likelihood of penetration or aspiration.

Elevation and protraction of the hyoid has also been shown to play a crucial role in the opening of the upper oesophageal sphincter (UES) through the anatomical stretch that occurs with the up-and-forward movement of the larynx. Failure of UES to open

adequately is a major cause of aspiration and penetration and contributes significantly to a dysfunctional swallow.

Once the bolus has entered the oesophagus, the UES must be closed and remain closed to counteract the increased pressures that come about through the stripping action of the oesophageal musculature. The lower oesophageal sphincter needs to be open for the bolus to leave the oesophagus and enter the stomach.

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Neural control of swallow

In the past, the swallow was thought to be a pure reflex but has now been shown to be a pattern-elicited response.[3] The oral phase is controlled primarily by cortical and subcortical areas. The reflexive pharyngeal and oesophageal stages are under brainstem control and require bilateral innervation due to the need for the two sides to work in unison in order to provide the pressure gradient required.

Fig. 2 shows a brief overview of the cranial nerves and their involvement in the swallow process.

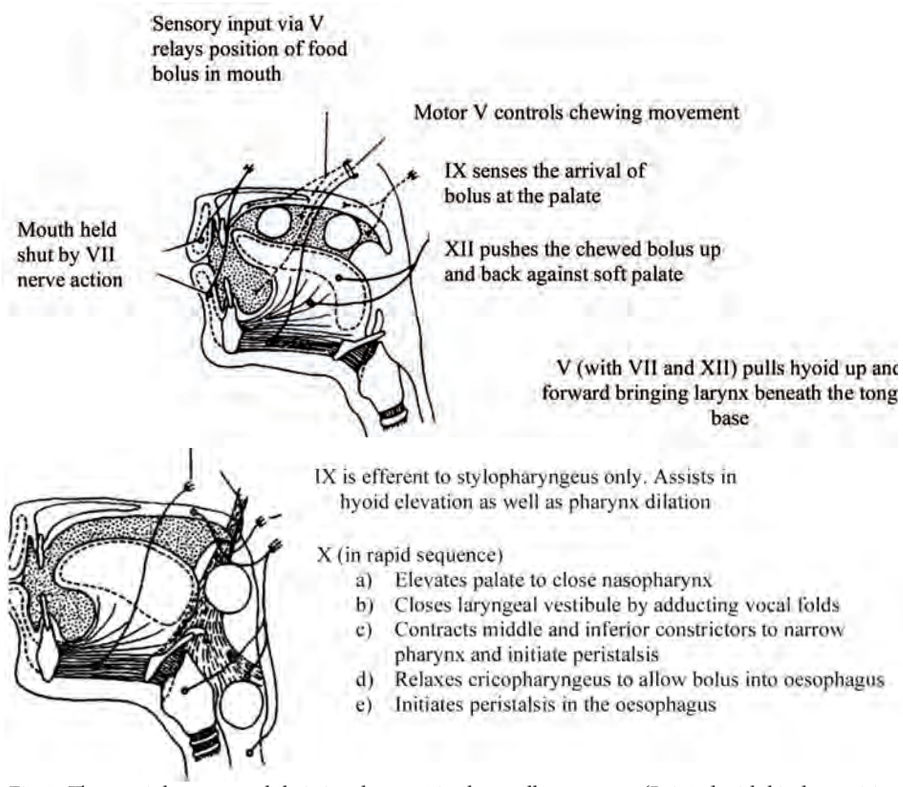

Fig. 2. The cranial nerves and their involvement in the swallow process. (Printed with kind permision from Y. Wijting.)

Who is at risk for dysphagia?

Many patients with dysphagia are overlooked, so be sure to consider this diagnosis in someone with recurrent episodes of pneumonia, dehydration and weight loss. There are numerous causes of dysphagia; some of the more common ones have been summarised in Table I.

Table I. Common causes of dysphagia

- Neurological diseases - stroke, Parkinson's amyotrophic lateral sclerosis, multiple sclerosis, traumatic brain injury
- Head and neck tumours and malignancies
- Sequelae of radiotherapy
- Autoimmune diseases – lupus, myasthenia gravis, Sjögren's
- Laryngopharyngeal reflux
- Infectious diseases – *Haemophilus, Streptococcus,* fungal
- Thyroid diseases
- Vascular abnormalities – aberrant vessels and compressive rings
- Congenital abnormalities – palatal/laryngeal clefts
- Psychogenic – developmental, conditioned dysphagia, primary conversion reaction, secondary conversion reaction
- Disorders of oral, pharyngeal, oesophageal motility
- Upper oesophageal sphincter dysfunction
- Pharyngeal, oesophageal diverticulae/pouches, rings and webs

Signs and symptoms

- Coughing during or after swallowing
- Choking on food or liquid
- Voice change ('gurgly' vocal quality) during meals
- Sensation of food getting stuck in the throat
- Sensation of having a lump in the throat
- Painful swallowing
- Recurrent chest infections of unknown origin
- Weight loss and fatigue.

How to evaluate and investigate a patient with dysphagia

Bedside clinical evaluation

The main purpose of a clinical swallowing evaluation is to identify patients who are at risk for dysphagia and who may require either further investigations or a referral for specialist management or rehabilitation. The swallowing evaluation should include a careful history, clinical examination and possibly additional imaging studies.

Daniels *et al.* identified six clinical features associated with an increased risk of aspiration in stroke patients. The presence of any two of these six features correctly identified the risk of aspiration with 92% accuracy.[4] The indicators listed in Table II should form the basis of a simple bedside examination.

Although aspiration and penetration remain the most life-threatening signs, they are not the only signs that should qualify the patient for further investigations and referral for treatment.

Diagnostic swallowing evaluations

The primary purpose of any imaging examination should be to directly evaluate

the kinesiology of the swallowing mechanism, determine swallowing safety and identify the effects of possible compensatory strategies. Contrary to common practice, the primary function of any imaging evaluation should not be to solely determine whether aspiration or penetration is taking place. The investigations should determine the cause and area of dysfunction, so that the most appropriate treatment can be employed.

The main two imaging tools used to evaluate dysphagia are:

- modified barium swallow (MBS) using videofluoroscopy
- video-endoscopy
  - flexible endoscopic evaluation of swallowing (FEES)
  - flexible endoscopic evaluation of swallowing with sensory testing (FEEST).

There are advantages and disadvantages to each modality and it is difficult to determine the superiority of either technique in the evaluation of dysphagia (Table III). A thorough working knowledge of the processes and timing of normal swallow mechanisms is necessary for the interpretation of either examination.

When performing a MBS study, it is best conducted with a suitably experienced speech therapist, radiographer and radiologist. The patient is tested against a recommended protocol involving the use of varying quantities and viscosities of liquid and solid boluses. If swallowing dysfunction is evident during the instrument evaluation, therapeutic intervention **must** be attempted during the diagnostic study in order to assess the efficacy of the intervention relative to the particular swallowing impairment.

It should be noted that in the over-60s, the swallow may only become **reflexive as the** bolus reaches the vallecula.

Table II. Basic indicators of a simple bedside examination for dysphagia

Indicator	Finding
Abnormal volitional (voluntary) cough	A weak response, verbalised response, or no response on command to cough
Abnormal gag reflex	Either absent or weakened velar (soft palate) or pharyngeal wall contraction, unilateral or bilateral, in response to tactile stimulation of the tongue base, faucal (tonsil) arches or posterior pharyngeal wall
Dysphonia	Voice disorder reflected by impaired vocal quality, pitch, or intensity
Dysarthria	Speech disorder reflected by impaired respiration, articulation, phonation, resonance or prosody
Cough after swallow	Cough immediately or within 1 min of ingestion of calibrated water volumes (5, 10, 20 ml presented in duplicate)
Voice change after swallow	Alteration in voice quality from baseline following ingestion of calibrated water volumes

Management of dysphagia

The dysphagia patient is best managed in a multidisciplinary team environment, including speech therapists and physiotherapists. The employment of a treatment strategy must be specific to the actual biomechanical disorder and not merely applied in a random fashion.

Acute management of dysphagia must consider the following first and foremost:

- hydration
- nutrition
- the impact of limiting the swallow through nasogastric feeds or nil per os orders.

Medical treatment

There are very few good randomised clinical trials that have been conducted into the effectiveness of various medical and surgical interventions. Medical treatments generally involve the management of symptoms as well as treating the underlying cause, e.g. Parkinson's or gastro-oesophageal reflux disease (GORD).

Surgical treatment

Surgical interventions must be directed towards the specific deglutition dysfunctions. They are primarily aimed at addressing anatomical dysfunctions or at dealing with the nutritional requirements of the patient, e.g. percutaneous endoscopic gastrostomy (PEG) insertion. PEG should only be considered when long-term oral feeding is deemed unsafe. Studies looking at the PEG decision-making process concluded that the decision to insert a PEG tube was often made in a time of crisis, with incomplete information and no reasonable alternative to tube feeding being perceived.[5]

PEG-related complications are reported in up to 70% of patients and are a considerable cause of morbidity and mortality. Aspiration pneumonia is reported in about 20 - 30% of PEG-fed patients and is frequently a terminal event.[6,7]

Table III. Comparison of MBS and FEES/FEEST studies

Only MBS can	Only FEES/FEEST can
• Visualise bolus during oral and oesophageal stages	• Visualise secretions
• Assess completeness of tongue base retraction	• Directly assess sensation
• Assess completeness of upper oesophageal sphincter opening	• Identify mucosal anomalies
• Assess completeness of hyo-laryngeal elevation and protraction	• Identify the effect of altered anatomy on bolus flow, glottic closure and airway protection
• Assess extent of aspiration	• View location of bolus within hypopharynx

Other surgical procedures that can be considered include:

- tracheostomy
- vocal fold medialisation
- surgical management of cricopharyngeal dysfunction (CPD) or spasm
  - dilatation
  - Botox injection
  - cricopharyngeal myotomy
- laryngotracheal separation (for severe life-threatening aspiration).

Many patients with dysphagia are overlooked – be sure to consider this diagnosis in someone with recurrent episodes of pneumonia, dehydration and weight loss.

Rehabilitative treatment

The primary goal of the rehabilitation therapist should be to keep the patient swallowing safely for as long as possible or to restore the swallow as quickly as possible.

The majority of muscle fibres involved in the swallowing process are type II fibres, which can lose up to 10% of their muscle bulk in a 24-hour period. If nasogastric (NG) feeding is to be introduced, it should always be accompanied by a referral to a rehabilitation therapist who can start the strengthening or compensatory strategies earlier rather than later so as to limit disuse atrophy and muscle weakness.

At this stage it may also be appropriate to consider the introduction of the Frazier Free Water Protocol. While it is a commonly held belief that the aspiration of clean water is harmful, this is untrue. The Frazier Free Water Protocol allows people with dysphagia free access to water under *specific* conditions, thereby maintaining active swallowing for longer periods.

The therapeutic management of dysphagia (Table IV) can be subdivided into:

- treatment techniques to improve the strength and co-ordination of the swallow
- compensatory techniques.

Table IV. Therapeutic management of dysphagia

Treatment techniques	Compensatory techniqes
• Myofascial techniques	• Modify sequence of delivery
• Exercises	• Alteration of bolus volume, texture, taste or temperature
• Swallow manoeuvres	• Modify rate of delivery
• Sensory stimulation techniques	• Alter positioning – head turn, chin tuck
• Electrical stimulation (NMES) *	• Advice on procedure of swallow, e.g. multiple swallows
• Biofeedback	• Alter method of food/liquid delivery

* The use of neuromuscular electrical stimulation (NMES) in the management of dysphagia is a relatively new therapy and is now available in South Africa through a number of VitalStim-certified therapists. VitalStim(®) therapy is an FDA-approved method to promote swallowing therapy through the application of NMES to the swallowing muscles with the goal of strengthening and re-educating the muscular system and improving motor control of the swallowing mechanism. This is not a stand-alone therapy and should form part of a well-planned exercise and biofeedback-based programme. It is most suited to dysphagias associated with upper motor neuron lesions.

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In a nutshell

- A thorough working knowledge of the normal swallowing mechanism is the basis for understanding and treating dysphagia.
- All phases of swallowing are functionally connected and problems in one area will impact on other areas.
- Dysphagia is best managed within a multidisciplinary team environment.
- Any decision to substantially alter or stop a patient's oral feeding should be done in consultation with or referral to a speech therapist.
- Diagnostic imaging studies need to assess swallowing dysfunction and the effect of compensatory strategies and not merely the presence of aspiration or penetration.