The Impact of Swallowing Disorders in the Elderly

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Abstract

Introduction*: Swallowing disorders are common in the elderly but its prevalence is often underestimated. They can result in increased morbidity and mortality. *Methods*: This article summarises the findings of selected published papers in major international journals indexed on Medline on swallowing using the key words—swallowing, dysphagia, aged, geriatrics and deglutition. *Results*: There are age-related changes in the oral, pharyngeal and oesophageal functions. In the elderly, central nervous system diseases such as stroke, parkinsonism, dementia, medications, local oral and oesophageal factors are common causes of swallowing dysfunction. Swallowing disorders in the elderly are associated with increased mortality and morbidity. Aspiration, dehydration, pneumonia, malnutrition, functional decline and institutionalisation are often encountered in the elderly with dysphagia. There is a choice of different interventions available to reduce morbidity and mortality arising from swallowing impairments, and improving their quality of life. *Conclusion*: The effective management of swallowing impairment in the elderly requires a multidisciplinary team approach.

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Introduction

In 1898, Bastian[1] first reported on the case of a man who had been admitted to hospital with hemiplegia and aphasia, but who had transient difficulty in deglutition. Necropsy revealed that apart from two limited lesions in the left hemisphere, the patient's brain was normal. In 1951, Ardan and Kemp[2] using a series of X-ray cinefilms first demonstrated the mechanism of swallowing. Since then, research into the neurophysiology of swallowing has made great strides enabling clinicians to improve on the management of dysphagia particularly the neurogenic type. Recently, Hamdy and Rothwall[3] demonstrated that swallowing has a bilateral but asymmetrical interhemispheric representation within the motor and premotor cortex. Zald's and Pardo's work[4] also increased our understanding of the functional neuroanatomy of voluntary swallowing. Their work demonstrated that voluntary swallowing produced strong cerebral blood flow within the inferior precentral gyrus bilaterally, the right anterior insula/claustrum and the left cerebellum.

The Control of Swallowing

The neurophysiological control of swallowing is governed by the interplay and integration of activity arising from five distinct anatomical sites namely the cortex, cerebellum, medullary swallowing centre, motor efferents (V3, VII, IX, X, XII cranial nerves) and sensory afferents (V3, VII, IX, X cranial nerves).

Eating and swallowing are learned skills. In the newborn, swallowing is an important reflex to ensure survival but with maturation and growth, it increases in complexity such that in adults it is a form of learned behaviour. It consists of an oral preparation phase, which is solely voluntary followed by swallowing. Swallowing itself consist of an oral phase which is both voluntary and involuntary, involuntary pharyngeal and oesophageal phases. Patient, family and social factors also influence the process of feeding.

Ageing and its Effect on Swallowing

There are several changes to the oropharynx with ageing.[5-7] The elderly generally have poor dentition and sensory discrimination thresholds in the oral cavity also increased. There is also decreased perception of viscosity. This leads to altered size of bolus and preference for softer foods.[8] Reduced smell and increased taste thresholds cause changes in food preferences. Decreased salivary flow and muscle tone make swallowing sometimes uncomfortable and even

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painful.[9] Impaired pharyngeal peristalsis increases motor response time needed for chewing.[10] Delayed cricopharyngeal opening, slowed oesophageal transport, ligamentous laxity causing delayed laryngeal adduction and hiatal hernia with gastrointestinal reflux are other common changes associated with ageing.

These age-related functional and sensory changes result in decreased functional reserve. In the presence of diseases, the reduced functional reserve predisposes the elderly to increased incidence of swallowing dysfunction.

Prevalence of Swallowing Disorders

The prevalence of swallowing disorders in the elderly is often underestimated.[11] One reason is that patients can have non-specific symptoms such as loss of appetite and weight loss instead of dysphagia. These are often ignored by both patients and their carers. Patients tend not to complain and they compensate for their disability by modifying their diets such as eating pureed food. Atypical symptoms and subtle signs of a swallowing disorder are sometimes missed by clinicians. For instance, an elderly patient undergoing bypass surgery may have suffered a mild stroke with no neurological deficit but present only with coughing on drinking fluids.

The prevalence of swallowing disorders in the elderly varies according to the setting of the study. In community surveys (>50 years), it is about 10%.[12] In acutely hospitalised elderly, it is also about 10% but in more disabled elderly living in nursing homes, the prevalence rates range between 30% and 60%.[13,14] In an acute geriatric unit in Singapore, about a third of the elderly patients have dysphagia but only 7.1% would volunteer this information at admission.[15]

Aetiology of Swallowing Disorders

Based on location, the swallowing process can be divided into three phases, namely, oral, oropharyngeal, and oesophageal phases. Dysphagia can be classified into two categories based on the location of the dysfunction. If the dysfunction arises prior to the bolus reaching the upper oesophagus, the term "oropharyngeal dysphagia" is used and "oesophageal dysphagia" is present if the dysfunction arises afterwards. In the elderly, oropharyngeal dysphagia is usually caused by neurological lesions such as stroke, parkinsonism and dementia. However, oesophageal dysphagia is commonly caused by reflux oesophagitis, motility disorders and tumours. Table I shows a list of the causes of dysphagia in the elderly.

TABLE I: CAUSES OF DYSPHAGIA IN THE ELDERLY

Oropharyngeal	Oesophageal
Mechanical	Intrinsic lesion
Extrinsic	Tumours—benign/malignant
Goitre	Webs and rings
Cervical osteophytes	Strictures
Neck cancer	Foreign bodies
Instrinsic	Extrinsic lesion
Post-cricoid cartilage web	Mediastinal tumour
Zenker diverticulum	Aberrant subclavius
	Right atrial dilatation
Neurogenic	Neuromuscular (motility)
Strokes	Achalasia
Cerebral neoplasm	Diffuse oesophageal spasm
Head injury	Scleroderma
Alzheimer's disease	Chagas disease
Parkinson's disease	Diabetes mellitus
Multiple sclerosis	Radiation oesophagitis
Pseudobulbar/Bulbar palsy	
Medicine with central nervous system side effects	
Neuromuscular junction	
Myasthenia gravis	
Eaton-Lambert syndrome	
Muscular	
Myotonic dystrophy	
Polymyositis/dermatomyositis	
Hypo or hyperthyroidism	
Cushing's syndrome	
Abnormal relaxation of the upper oesophageal sphincter	
Reflux oesophagitis	
Medications with anti-cholinergic side effects	

Common Diseases Causing Dysphagia in the Elderly

Stroke

Stroke is the most common cause of neurogenic dysphagia.[16] Bilateral hemispherical strokes are associated with higher incidence and greater severity of aspiration than unilateral strokes.[17] Unilateral hemispheral strokes can also cause dysphagia.[17]

Generally, left-sided strokes impair the oral phase of swallowing and patients also have apraxia of swallowing. They swallow better when eating automatically without any verbal commands to swallow. Patients return to oral intake early. Right-sided strokes affect pharyngeal function and laryngeal elevation is delayed resulting in aspiration. They have a longer delay in returning to oral intake.[17-19]

Subcortical strokes affect motor and sensory pathways to and from the cortex resulting in mild delay in oral transit time, mild delay in triggering of pharyngeal swallow and impaired pharyngeal swallow. A few patients develop aspiration because of this delay in pharyngeal swallow. Full recovery occurs by 3 to 6 weeks post-stroke.[20]

Brain stem strokes cause dysphagia as the corticobulbar tracts, cranial nerve nuclei and swallowing centres are affected.[21-23] They have absent pharyngeal swallow in the

first week of stroke, with recovery occurring in the second week post-stroke.[20]

Patients with recurrent multiple strokes have more significant swallowing abnormalities (oral, pharynx and larynx) and recovery to oral intake is more protracted.[20]

Parkinson's Disease

Aspiration pneumonia is a common cause of death in advanced Parkinson's disease.[24] In this degenerative disease, the orolingual muscles are also affected, causing difficulty with bolus preparation and its transfer through the pharynx.[25,26] Silent aspiration is common because of reduced oropharyngeal sensation with impaired volitional cough.[27] The severity of dysphagia increases with worsening of the disease and cognitive changes and depression are also important compounding factors.[28]

Alzheimer's Disease

Self-feeding and swallowing changes may occur early in the course of Alzheimer's disease. In a study which compared self-feeding and swallowing of healthy elderly with a group of individuals with mild Alzheimer's disease, significantly more partner-initiated cues or direct assistance, significantly prolonged oral transit duration, pharyngeal response duration, and total swallow duration were noted in the latter.[29] Aspiration occurred in 28.6% of patients with Alzheimer's disease.[30] Swallowing abnormalities were associated significantly with duration of dementia and eating dependency.[30] Masquerading as dementia, depression and cognitive impairment due to side effects of medication compound the difficulties of swallowing. A major dilemma is whether feeding gastrostomy is indicated in advanced dementia.[31]

Medications

Treating medication-related oropharyngeal dysphagia is the easiest and most important aspect of management of dysfunctional swallowing but is most frequently overlooked.[32] Medications can affect swallowing usually as a result of their adverse side effects through various pathways.

Sedatives affect swallowing by decreasing arousal and by suppressing brain stem function leading to oropharyngeal dysfunction, and this resolves soon after the medications are stopped.[32,33] Neuroleptics are often given to control agitation in patients with dementia and their side effects such as tardive dyskinesia can also cause dysphagia.[34] Tricyclic antidepressants, antihistamines and anticholinergic drugs decrease saliva production making swallowing difficult and even painful. Antibiotics such as neomycin, streptomycin and certain tetracyclines can interfere with neuromuscular transmission, resulting in pharyngeal muscular weakness.[35] Drugs such as colchicine, steroids and lipid-lowering agents cause toxic myopathy that involves the pharyngeal muscles.[36]

Gastro-oesophageal Reflux Disease

Heartburn and regurgitation are classical symptoms of gastro-oesophageal reflux disease. However, many elderly have silent reflux and present with cough, voice hoarseness and discomfort resulting in frequent throat clearing instead of the classical pain of heartburn. Transient lower oesophageal relaxation is the dominant cause for the reflux and is more common in the presence of hiatal hernia.

Diagnosis of Swallowing Disorders

An accurate and detailed history, including the site of discomfort, its onset, associated symptoms, relationship to the type of food and progress, is crucial in the diagnosis. Oropharyngeal dysphagia typically causes difficulty in swallowing liquids with complaints of food "sticking" in the throat above the sternal notch. This usually happens immediately after swallowing and in severely disabled patients, may be accompanied by coughing, choking or nasal regurgitation. An acute onset of these symptoms usually suggests a stroke and a slowly progressive course points to a more sinister cause such as head or neck tumour.

Typically, oesophageal dysphagia presents with discomfort in the substernal region but sometimes the pain may be referred to the suprasternal area and, as a result, it may be confused with oropharyngeal dysphagia. Very often, the discomfort usually presents shortly after swallowing and, depending on the aetiology, may be present with solids only, if it is obstructive, or both solids and liquids, if it is motility related. The presence of angina-like chest pain usually suggests oesophageal spasm or gastro-oesophageal reflux.

The physical examination should include the various organs involved in swallowing, and the detection of signs of systemic disease and possible consequences arising from dysphagia such as aspiration pneumonia and malnutrition.

Any evidence of previous surgery, tracheostomy or radiation to the head and neck is noted. An examination of the neck for tumours, goitre or lympadenopathy is conducted. It is also important to conduct an oromotor function assessment. The status of oral anatomy including dentition and locations of any pooled secretions are noted. The range, rate, strength and co-ordination of movements of the lips, tongue, jaw, palate and larynx are tested. In evaluating laryngeal function, vocal quality, phonation time, loudness, strength of volitional cough and throat clearing are also tested. Finally, oral sensory evaluation of palatal and gag reflexes, and light touch in the mouth, is conducted. Whenever possible, the vocal cords are also

assessed. Acid reflux disease can give rise to erythema and inflammation of the vocal cords. The gag reflex is absent in up to 40% of healthy adults, especially in the older patient. It has little value in the prediction of pharyngeal dysfunction or aspiration risk.[37] Finally, a thorough neurological examination, including a detailed cranial nerve examination, is essential.

Investigation of Swallowing Disorders

The tensilon test for myasthenia gravis and thyroid function test for thyroid disease enable clinicians to confirm the diagnosis of these medical conditions which cause dysphagia. Other more invasive tests such as videofluoroscopic swallowing study (VFSS), barium-contrast oesophagogram, upper endoscopy or manometry are currently used in the diagnostic work-up and the choice of the investigation is guided by the history and physical findings.

Videofluoroscopic swallowing study, also known as "modified barium swallow", is currently the "gold standard" for diagnosis of oropharyngeal dysphagia. It was first described by Ekberg et al in 1982[38] for the assessment of the oropharyngeal phase of swallowing. During the test, the patient is asked to swallow various food items of different consistencies coated with barium. A videofluoroscopic recording is made in both antero-posterior and lateral views allowing the clinician to observe the progress of the radio-opaque bolus during swallowing. The study also allows one to evaluate the response to certain correctional techniques such as chin tucking. However, it requires the co-operation of an alert patient and VFSS is dangerous in a drowsy and uncooperative patient because of a high risk of aspiration.

Videoendoscopic swallowing study (VESS), first described by Bastian in 1991,[39] allows direct visualisation of the oropharynx in action with and without swallowing using a fibreoptic scope inserted nasally. The addition of methylene blue water may improve the sensitivity of this test. This test can be carried out at the bedside safely.[40]

Barium-contrast oesophagogram (barium swallow) is the initial recommended test if oesophageal dysphagia is suspected. The patient is asked to drink liquid barium while pictures are taken in both upright and supine positions. Barium-contrast oesophagogram identifies most cases of mechanical obstruction, such as strictures, rings and webs.

If the radiographic studies are inconclusive, oesophagoscopy is indicated. It is superior to barium swallow for evaluating small lesions of the mucosa, and sequelae of acid reflux disease. Barium swallow is, however, complementary for diagnosing intramural lesions and extrinsic obstruction, and is almost of no value in diagnosing motility disorders. Endoscopy provides the opportunity for therapeutic intervention if necessary.

Oesophageal manometry is based on the principle of recording pressures throughout the oesophageal lumen using a solid-state or perfusion technique. It gives a recording of the peristaltic contractions in the oesophageal body including its duration, velocity and amplitude. Manometry is indicated when an oesophageal aetiology for dysphagia is suspected despite inconclusive barium swallow and endoscopic evaluation.

Consequences of Swallowing Disorders in the Elderly

Swallowing disorders in the elderly are associated with increased mortality and morbidity. Aspiration, dehydration, pneumonia, malnutrition, functional decline and institutionalisation are often encountered in the elderly with dysphagia.

Mortality and Swallowing Disorders

In a retrospective study of 149 hospital patients deemed non-oral feeders based on their videofluoroscopic swallowing study, Cowen et al[41] demonstrated that the overall 1-year mortality was 62%. Schmidt et al[42] showed that the odds ratio for death was 9.2 times greater in those who aspirated thickened liquids or food of more solid consistency compared with those who did not aspirate or who aspirated thin liquids only.

Aspiration

Elderly patients without underlying high-risk conditions have excess mortality of 9 per 100,000 from lower respiratory tract infections. The rate increases to 217 per 100,000 with one high-risk condition such as malnutrition but if both malnutrition and aspiration are present, the mortality rate increases to 979 per 100,000.[43]

The symptoms of aspiration are cough, choking and intermittent fever but silent aspiration which is not associated with a cough response is commonly seen in the dysphagic elderly. There are many factors correlated with aspiration namely depressed consciousness, immobility, tube feeding, trachea tube or ventilation, stroke disease, reduced pharyngeal sensation and abnormal voluntary cough response; and a patient with several of these factors has a high risk of developing aspiration.

Pneumonia

Aspiration and pneumonia are closely linked because the former leads to the development of the latter. The odds ratio for developing pneumonia post-stroke was over 6.95 times in aspirators compared with non-aspirators.[44] In a study of community-acquired pneumonia in the elderly, large-volume aspiration and low serum albumin (<30 mg/dl) were independent risk factors associated with the development of pneumonia.[45] About 6% of all patients

suffering from stroke die from aspiration pneumonia within the first year.[46] Besides increased mortality, aspiration pneumonia also results in prolonged hospitalisation.

In the healthy elderly, the lower respiratory tract has an effective host defence system that maintains a sterile tracheobronchial tree and the oropharynx is colonised by non-pathogenic Gram-positive and anaerobic bacteria.[47,48] However, in the hospitalised and institutionalised elderly, there is impaired lower respiratory defence and the oropharynx is colonised by enteric Gram-negative bacteria.[49,50] Together with changes in pulmonary function such as decreased effectiveness of cough and ciliary clearance, decreased diffusion capacity and oxygen saturation, the presence of swallowing disorders causing aspirations increases the likelihood of pneumonia, especially with Gram-negative bacteria.

Malnutrition

Eating and swallowing disorders can lead to malnutrition and this carries a poor prognosis. But malnutrition itself can also affect swallowing. Veldee and Peth[51] postulated that the muscles involved in deglutition have a moderate to high percentage of type II (fast twitch) muscle fibres.[51] Type II muscle fibres are affected by malnutrition to a much greater extent than type I (slow twitch) fibres.[52] Therefore, in malnutrition, deglutitive muscles may be among the first to atrophy, compromising the integrity of the swallow, further decreasing intake of food and increasing the risk of aspiration. The diaphragm has more fast twitch fibres (47%) compared to slow twitch fibres (23%).[53] Thus, in malnourished patients, there is an additional factor of increased weakness of the respiratory muscles leading to a decreased ability to clear aspirated material.

Other Consequences of Swallowing Disorders

Dehydration, poorer mobility status, longer length of hospital stay and greater need for institution care are often seen in patients with swallowing disorders.

Swallowing Disorders in the Elderly in Singapore

In a study on swallowing impairment and feeding dependency in the hospitalised elderly by Lee et al,[15] a mortality rate of 8.1% was noted in patients with swallowing impairment. It was also significantly associated with the presence of dehydration (RR = 2.82), chest infection on admission (RR = 2.85), development of nosocomial chest infection (RR = 6.75), discharge to institutional care (RR = 2.8) and increased mortality (RR = 3.77).

Treatment of Swallowing Disorders

The approach to the management of swallowing disorders is a multi-disciplinary team approach. The speech therapist plays an important central role. Other members of the team include dietician, neurologist, gastroenterologist, otolaryngologist, geriatrician and trained nurse. If a reversible or treatable cause of oropharyngeal dysphagia such as thyroid disease or myasthenia gravis is identified, it should be corrected. The management options for different conditions causing oesophageal dysphagia are summarised in Table II.

TABLE II: MANAGEMENT OPTIONS FOR OESOPHAGEAL DYSPHAGIA

Condition	Medical treatment
Achalasia	Soft food, anti-cholinergics, calcium channel blockers, botulinium injections, myotomy
Diffuse oesophageal spasm	Calcium channel blockers, nitrates, surgical dilations, myotomy
Gastro-oesophageal reflux disease	H2 blockers, proton pump blockers, domperidone, fundoplication
Infectious oesophagitis	Antibiotics (nystatin for candidiasis)
Pharyngoesophageal (Zenker's) diverticulum	Cricopharyngeal myotomy and repair
Schatzki's ring	Soft food and surgical dilation
Scleroderma	Anti-reflux treatment and medical management of scleroderma

There are numerous swallowing therapy techniques currently used by speech therapists in swallowing rehabilitation such as biofeedback modalities, compensatory intervention techniques and dietary modifications. Rehabilitation is guided by the presenting aetiology of the dysphagic physiology, the cognition, motivation and support system of each patient. These techniques are very often employed and are effective in treating neurogenic dysphagia due to strokes, parkinsonism or dementia. Some examples of such manoeuvres include modified Valsalva manoeuvre, Masako manoeuvre, vocal adduction exercises, Mendelson manoeuvre, neurosensory stimulation and head-lifting. A few may even incorporate biofeedback modalities such as surface electromyography, cervical auscultation, endoscopy, ultrasonography and pulse oximetry to improve the success of swallowing rehabilitation. Bolus control techniques are also effective and they include modification of bolus size, adaptation in rate of intake, slurp and swallow. They are often used with volitional airway protection strategies such as supraglottic swallow, super-supraglottic swallow, pharyngeal expectoration and vocal quality check.

However, patients with neurologic deficits sometimes have cognitive deficits that may hinder rehabilitative efforts.

This is because some of these techniques require the patient to be able to execute sequencing of the motor cortex and conceptualisation of the swallowing process. In a patient who cannot co-operate and learn the above techniques due to severe cognitive impairment, diet modifications can be still be used to help the patient swallow orally, and this is done by modifying the texture of food and the viscosity of feeds with thickeners. Rarely, prosthetic devices such as palatal lifts and tracheostomy valves may be useful.

Recently, there are reports of some pharmacological agents that can be used to improve swallowing in patients with strokes. Nitrates have been reported to improve oropharyngeal dysphagia in ischaemic brainstem stroke.[54] Patients with strokes given slow-release nifedipine orally have shown improvement in the initiation of pharyngeal contractions and reduction in bolus transit time to the pharynx.[55] Arai et al[56] have also reported that angiotensin-converting enzyme inhibitors may cure symptomless dysphagia in hypertensive patients with stroke.

In general, the management of oesophageal dysphagia is dependent on the endoscopy findings. In the case of achalasia, additional results of manometry are important. Reflux oesophagitis, a very common cause of oesophageal dysphagia is first treated with anti-reflux therapy (proton pump inhibitors and domperidone) and by fundoplication if the former fails. Oesophageal infections should be treated with appropriate chemotherapy. Tumours require surgery. Patients with achalasia with low operative risks should be recommended for surgery. Pneumatic dilatation is rarely used nowadays because of the risk of perforation. For patients with high operative risks, medical therapy with nitrates and/or calcium channel blockers can be started. If this is unsuccessful, bougienage with a 45 F to 60 F dilator can be tried. Injection with botulinium toxin is a safe option in a poor surgical risk patient.[57] Patients with structural lesions (tumour or Zenker's diverticulum) should be referred for surgery.

In patients with dysphagia, there is a need to identify whether the patient can obtain adequate nutrition through the oral route and how high the risk of aspiration pneumonia appears to be. If the ability to obtain adequate nutrition is limited or if the aspiration risk is high, non-oral feeding should be considered. For short duration transient dysphagia, nasogastric tube feeding is the option of choice. However, patients with permanent dysphagia benefit from feeding gastrostomy.

Controversy on Tube Feeding

There is much controversy on the benefits and dangers of tube feeding. In a review, Finucane and Bynum[58] concluded that "although the use of feeding tubes is widespread, no data show that they reduce the risk of aspiration pneumonia in neurogenic dysphagia. There are data to the contrary, and the burdens of feeding tube placement can be severe. For almost all conscious patients, we suggest a dedicated attempt at feeding by hand." Nevertheless, Rudberg et al[59] demonstrated that tube feeding could be life prolonging, even if the quality of life is not substantial. Finally, there are ethical and legal issues to consider whether to give or withhold fluid and nutrients through a tube.[60] This remains to be resolved.

Conclusion

Swallowing disorders are common in the elderly. They are often neglected till serious complications arise. They are associated with significant morbidity and mortality. Currently, with appropriate evaluation, there are safe and effective ways of managing these disorders through a multi-disciplinary approach, thus improving the patients' quality of life.

REFERENCES

1. Bastian H C. A treatise on aphasia and other speech defects. London: 87 Lewis, 1898.
2. Ardran G M, Kemp F. The mechanism of swallowing. Proc R Soc Med 1951; 44:1038-40.
3. Hamdy S, Rothwall J C. Gut feelings about recovery after stroke: The organization and reorganization of human swallowing motor cortex. Trends Neurosci 1998; 21:278-82.
4. Zald D H, Pardo J V. The functional neuroanatomy of voluntary swallowing. Ann Neurol 1999; 46:281-6.
5. Newman H F. Palatal sensitivity to touch: Correlation with age. J Am Geriatr Soc 1979; 27:319-23.
6. Feldman R S, Kapur K K, Alman J E. Aging and mastication: Changes in performance and the swallowing threshold with natural dentition. J Am Geriatr Soc 1980; 28:97-103.
7. Iggo A. Peripheral nerve injuries. Cutaneous sensory mechanisms. J Bone Joint Surg Br 1986; 68:19-21.
8. Logerman J A. Effects of aging on the swallowing mechanism. Otolaryngol Clin North Am 1990; 23:1045-56.
9. Robbins J, Hamilton J W, Lof G C. Oropharyngeal swallowing in normal adult different ages. Gastroenterology 1999; 103:823-9.
10. Shaker R, Ren J, Podorson B, Dodds W J, Hogan W J, Kern M. Effect of aging and bolus variables on pharyngeal and upper esophageal sphincter motor function. Am J Physiol 1993; 264:G427-9.
11. Jahnke V. Dysphagia in the elderly. HNO 1991; 39:442-4.
12. Paterson W G. Dysphagia in the elderly. Can Fam Physician 1996; 42:925-32.
13. Lugger K E. Dysphagia in the elderly stroke patient. J Neurosci Nurs 1994; 26:78-84.
14. Mendez L, Friedman L S, Castell D O. Swallowing disorders in the elderly. Clin Geriatr Med 1991; 7:215-30.
15. Lee A, Sitoh Y Y, Lieu P K, Phua S Y, Chin J J. Swallowing impairment and feeding dependency in the hospitalised elderly. Ann Acad Med Singapore 1999; 28:371-6.
16. Horner J, Massey E W, Brazer S R. Aspiration in bilateral stroke patients. Neurology 1990; 40:1686-8.

17. Robbins J, Levine R L, Maser A, Rosenbek J C, Kempster G B. Swallowing after unilateral stroke of the cerebral cortex. Arch Phys Med Rehabil 1993; 74:1295-300.
18. Smithard D G, O'Neill P A, Martin D F, England R. Aspiration following stroke: is it related to the side of the stroke? Clin Rehabil 1997; 11:73-6.
19. Daniels S K, Foundas A L, Iglesia G C, Sullivan M A. Lesion site in unilateral stroke patients with dysphagia. J Stroke Cerebrovasc Dis 1996; 6:30-4.
20. Logemann J A. Management of dysphagia poststroke. In: Chapey R, editor. Language Intervention Strategies in Adult Aphasia. 3rd ed. Baltimore, MD: Williams & Wilkins, 1994:503-12.
21. Horner J, Buoyer F G, Alberts M J, Helms M J. Dysphagia following brain-stem stroke. Clinical correlates and outcome. Arch Neurol 1991; 48:1170-3.
22. Teasell R W, Bach D, McRae M. Prevalence and recovery of aspiration poststroke: a retrospective analysis. Dysphagia 1994; 9:35-9.
23. Chua K S, Kong K H. Functional outcome in brain stem stroke patients after rehabilitation. Arch Phys Med Rehabil 1996; 77:194-7.
24. Hoehn M M, Yahr M D. Parkinsonism: onset, progression and mortality. Neurology 1967; 17:427-42.
25. Donner M W, Silbiger M L. Cinefluorographic analysis of pharyngeal swallowing in neuromuscular disorders. Am J Med Sci 1966; 251:606-16.
26. Blonsky E R, Logemann J A, Boshes B, Fisher H B. Comparison of speech and swallowing function in patients with tremor disorders and in normal geriatric patients: a cinefluorographic study. J Gerontol 1975; 30:299-303.
27. Robbins J A, Logemann J A, Kirshner H S. Swallowing and speech production in Parkinson's disease. Ann Neurol 1986; 19:283-7.
28. Nilsson H, Ekberg O, Olsson R, Hindfelt B. Quantitative assessment of oral and pharyngeal function in Parkinson's disease. Dysphagia 1996; 11:144-50.
29. Priefer B A, Robbins J. Eating changes in mild-stage Alzheimer's disease: a pilot study. Dysphagia 1997; 12:212-21.
30. Horner J, Alberts M J, Dawson D V, Cook G M. Swallowing in Alzheimer's disease. Alzheimer Dis Assoc Disord 1994; 8:177-89.
31. Hasan M, Meara R J, Bhowmick B K, Woodhouse K. Percutaneous endoscopic gastrostomy in geriatric patients: attitudes of health care professionals. Gerontology 1995; 41:326-31.
32. Buchholz D W. Oropharyngeal dysphagia due to iatrogenic neurological dysfunction. Dysphagia 1995; 10:248-54.
33. Wyllie E, Wyllie R, Cruse R P, Rothner A D, Erenberg G. The mechanism of nitrazepam-induced drooling and aspiration. N Engl J Med 1986; 314:35-8.
34. Gregory R P, Smith P T, Rudge P. Tardive dyskinesia presenting as severe dysphagia. J Neurol Neurosurg Psychiatry 1992; 55:1203-4.
35. McQuillen M P, Cantor H E, O'Rourke J R Jr. Myasthenic syndrome associated with antibiotic. Arch Neurol 1968; 18:402-15.
36. Kuncl R W, Wiggins W W. Toxic myopathies. Neurol Clin 1988; 6: 593-619.
37. Bleach N R. The gag reflex and aspiration: a retrospective analysis of 120 patients assessed by videofluoroscopy. Clin Otolaryngol 1993; 18:303-7.
38. Ekberg O. Defective closure of the laryngeal vestibule during deglutition. Acta Otolaryngol 1982; 93:309-17.
39. Bastian R W. Videoendoscopic evaluation of patients with dysphagia: an adjunct to the modified barium swallow. Otolaryngol Head Neck Surg 1991; 104:339-50.
40. Aviv J E, Kaplan S T, Thomson J E, Spitzer J, Diamond B, Close L G. The safety of flexible endoscopic evaluation of swallowing with sensory testing (FEESST): an analysis of 500 consecutive evaluations. Dysphagia 2000; 15:39-44.
41. Cowen M E, Simpson S L, Vettese T E. Survival estimates for patients with abnormal swallowing studies. J Gen Intern Med 1997; 12:88-94.
42. Schmidt J, Holas M, Halvorson K, Reding M. Videofluoroscopic evidence of aspiration predicts pneumonia and death but not dehydration following stroke. Dysphagia 1994; 9:7-11.
43. Schneider E L. Infectious diseases in the elderly. Ann Intern Med 1983; 98:395-400.
44. Holas M A, DePippo K L, Reding M J. Aspiration and relative risk of medical complications following stroke. Arch Neurol 1994; 51:1051-3.
45. Riquelme R, Torres A, El-Ebiary M, de la Bellacasa J P, Estruch R, Mensa J, et al. Community-acquired pneumonia in the elderly. A multivariate analysis of risk and prognostic factors. Am J Respir Crit Care Med 1996; 154:1450-5.
46. Hannig C, Wuttge-Hannig A, Hoffman M, Herman I F. Cinematographic study of the pathologic mechanisms of aspiration pneumonia. ROFO Fortschr Geb Rontgenstr Nuklearmed 1989; 150:260-7.
47. Laurenzi G A, Potter R T, Kass E H. Bacteriologic flora of the lower respiratory tract. N Engl J Med 1961; 265:1273-8.
48. Lee A W, McNaught W. Bacteriology of lower respiratory tract secretions, sputum and upper respiratory tract secretions in 'normals' and chronic bronchitis. Lancet 1959; 2:1112-5.
49. Johanson W G, Pierce A K, Sanford J P. Changing pharyngeal bacterial flora of hospitalized patients. Emergence of gram-negative bacilli. N Engl J Med 1969; 281:1137-40.
50. Johanson W G, Pierce A K, Sanford J P. Nosocomial respiratory infection with gram negative bacilli: the significance of colonization of the respiratory tract. Ann Intern Med 1972; 77:701-6.
51. Veldee M S, Peth L D. Can protein-calorie malnutrition cause dysphagia? Dysphagia 1992; 7:86-101.
52. Russell D M, Walker P M, Leiter L A, Sima A A, Tanner W K, Mickle D A, et al. Metabolic and structural changes in skeletal muscle during hypocaloric dieting. Am J Clin Nutr 1984; 39:503-13.
53. Lewis M I, Belman M J. Nutrition and the respiratory muscles. Clin Chest Med 1988; 9:337-48.
54. Jamrozik Z, Czyzewski K, Zakrzewska-Pniewska B, Kwiecinski H. A case of neurogenic dysphagia responding to nitrates. Neurol Neurochir Pol 1999; 33:1435-41.
55. Perez I, Smithard D G, Davies H, Kalra L. Pharmacological treatment of dysphagia in stroke. Dysphagia 1998; 13:12-6.
56. Arai T, Yasuda Y, Takaya T, Toshima S, Kashiki Y, Yoshimi N, et al. ACE inhibitors and symptomless dysphagia. Lancet 1998; 352: 115-6.
57. Hoogerwerf W A, Pasricha P J. Achalasia: treatment options revisited. Can J Gastroenterol 2000; 14:406-9.
58. Finucane T E, Bynum J P. Use of tube feeding to prevent aspiration pneumonia. Lancet 1996; 348:1421-4.
59. Rudberg M A, Egleston B L, Grant M D, Brody J A. Effectiveness of feeding tubes in nursing home residents with swallowing disorders. J Parenter Enteral Nutr 2000; 24:97-102.
60. Lennard-Jones J E. Giving or withholding fluid and nutrients: ethical and legal aspects. J R Coll Physicians Lond 1999; 33:39-45.