Journal of
neurology
neurosurgery
& psychiatry

Editorial

Neurogenic dysphagia: the role of the neurologist

The dual role of the oropharynx as a conduit for air and nutrition means that neurogenic dysphagia causes morbidity or death from (1) upper airway obstruction; (2) aspiration causing chemical pneumonitis or small airway obstruction with distal collapse and secondary infection; (3) dehydration and malnutrition

A slowed ability to eat a meal, loss of salivary control with drooling, episodic coughing, and choking and nasal regurgitation also cause considerable distress.

As well as their role in the diagnosis of the underlying disease, neurologists are well placed to help elucidate the mechanism of the dysphagia and to give advice in a multidisciplinary context about dysphagia management bearing in mind the patient's diagnosis, prognosis, and disability. Understanding of normal bulbar function and the methods of assessment and treatment have each improved in recent years.

Normal function[1–3]

Cranial nerve involvement in bolus preparation (V—motor and sensory— and VII), bolus propulsion (X and XII), and palatal elevation (X) is well appreciated. The airway is protected by apposition of the true and false vocal cords and of the arytenoids against the base of the epiglottis (the sphincteric action of the larynx); the epiglottis inhibits direct contact of the bolus with the laryngeal vestibule (all X). Upward and forward movement of the hyoid and larynx (V, VII, C1–3) enhances airway protection and pulls open the relaxed upper oesophageal sphincter. Breathing is centrally inhibited during the swallow (deglutition apnoea) and, after it, structures return to their original position passively, or aided by the infrahyoid muscles.

Swallowing was originally perceived as a brainstem reflex triggered by more or less "phagetic agents"[4]: subsequently the sequence of activation of the muscles involved in deglutition was delineated[5] and the concept developed of a central pattern generator anatomically related to the nucleus ambiguus and the nucleus of the tractus solitarius and under various peripheral and supranuclear controls.[6–8] Clinical observations and cortical mapping using magnetic stimulation[9 10] clearly point to roles for the cerebral cortex (primary motor, inferior frontal gyrus, insula[11]) in the modulation of the swallow whereas the frequency of dysphagia in basal ganglia and cerebellar disorders attests to involvement of these systems.

Diagnosis

Patients with obvious dysphagia or pulmonary or nutritional problems possibly due to dysphagia, should be seen first by an ear, nose, and throat surgeon or gastroenterologist to exclude structural disorders of the neck, mouth, pharynx, larynx, and oesophagus. Some of these can easily be missed initially, notable examples being tumours in the tongue base and hypopharynx, large anterior cervical osteophytes, and other retropharyngeal pathology,[12 13] upper or lower oesophageal sphincter dysfunction, lower oesophageal pathology including oesophageal reflux and its complications,[14] and external compression of the oesophagus (enlarged left atrium, aberrant subclavian artery, retrosternal goitre). Repeat examination, oesophageal manometry, or pH studies may be required.

If a structural lesion is excluded and there are no obvious features of neurological disease further enquiry is needed. The onset of symptoms and their rate of change is important to establish but the limited repertoire of dysphagia related symptoms may not be diagnostically very helpful. Symptoms such as pain on swallowing (glossopharyngeal neuralgia, Eagle's syndrome,[15] pterygoid hamulus bursitis),[16] improvement in swallowing with head turning to one side (unilateral X or XII palsy), or loss of sensation of bolus passage on one side (IX+/-X palsy), should be particularly noted. A history of thyroid disease, neck surgery including anterior discectomy,[17] or local radiotherapy,[18] a family history (myotonic or oculopharyngeal dystrophy), and a drug history (especially neuroleptic agents)[19 20] are potentially relevant.

Regarding the clinical examination some bulbar signs merit comment. We failed to find a healthy adult in whom the palatal and pharyngeal reflex could not be elicited at least once after examination on five separate occasions and so interpret their consistent absence in an alert subject as of potential importance.[21] Whereas a truly absent palatal and pharyngeal reflex may impair the ability to eject a bolus from the pharynx back into the mouth (a function probably more dependent on cough and postural change), inferences about swallowing function from such signs of innervation are unjustified. Reduced voluntary palatal or pharyngeal movement with brisk reflex responses is seen in pseudobulbar palsy. Unilateral pharyngeal wall paresis causes the paralysed side to move towards the healthy side, Vernet's mouvement de rideau. It is not clear whether the bedside sensory examination can reliably distinguish IXth and Xth nerve lesions but lack of posterior wall pharyngeal sensation is reported to have prognostic implications in stroke.[22] A unilateral XIIth nerve lesion causes the tongue to deviate to the healthy side on retraction (unapposed action of styloglossus), as well as to the affected side on protrusion (genioglossus). In a hemispheric stroke, because of the action of genioglossus, the tongue is the only structure which tends towards rather than away from the side of the limb weakness.[23] As well as observation of swallowing (see below) from both a diagnostic and impairment viewpoint it is important to formally assess articulation, phonation, and resonance (nasality). Volitional cough may

Table 1 Factors influencing swallowing capacity in neurological patients

1	Conscious level
2	Cognition and behaviour (eg, inappropriate "stuffing", talking while eating)
3	Head/neck posture and mobility (eg, tracheostomy, neck lines, neck surgery)
4	Local oropharyngeal and laryngeal factors (eg, poor oral hygiene, dry mouth, secretions, drooling, dentition, dentures, mouth ulcers, sore throat, intubation)
5	Neurological factors (eg, spasticity, rigidity, weakness (central or peripheral), sensory loss, movement disorder, coordination, loss of voluntary control, exaggeration of oropharyngeal and laryngeal reflexes)
6	Accompanying symptoms (vertigo, nausea, neuralgic pain (V, IX), syncope (IX))
7	Gastro-oesophageal function (eg, acid reflux disease)
8	Breathing and effectiveness of cough (eg, control of respiratory cycle, inspiratory and expiratory capacity, pulmonary function, laryngeal function)
9	Appetite (affected by illness and medication)
10	Ability to self feed (eg, arm and hand function)
11	Medication (eg, anticholinergic drugs, neuroleptic medication, benzodiazepines)
12	Psychological factors and social factors (eg, confidence, embarrassment, fear)

be feeble due to a depressed conscious level or respiratory or laryngeal weakness, whereas a "bovine" cough suggests vocal cord paresis. Patients with corticobulbar lesions may exhibit impaired voluntary control of facial movements, cough, and respiration while retaining emotional or reflex responses. Breathing may be stridulous (for example, multisystem atrophy, laryngeal dystonia), obstructed (laryngeal or pharyngeal occlusion), or abnormal in pattern (respiratory dyskinesia). Dyskinesia or dystonia of the throat, face, jaw, and head and neck may be important clues to the underlying neurological disorder—for example, Whipples disease, coeliac disease with encephalopathy, and multisystem atrophy.

Having recognised a neurogenic swallowing problem the diagnosis of causation proceeds along well tried lines of investigation.

Mechanism of dysphagia in the individual patient

The dominant mechanism causing neurogenic dysphagia (table 1) may differ in two patients with the same disease and can clearly change over time. The input of the speech and language therapist is valuable in determining the mechanisms of dysphagia and input from the family, nurses, dietician, and physiotherapist is important. Appropriate management is most likely if the dominant mechanism can be identified; thus cricopharyngeal myotomy in motor neuron disease may be inappropriate if the apparent spasm of the upper oesophageal sphincter is in fact due to reduced movement of the hyoid and larynx, particularly as such patients usually have oral stage problems as well.[24 25]

Several techniques are available to acquire more information about the mechanism of dysphagia. Videofluoroscopy is unmatched as a means of visualising the bolus path and related structures from mouth to stomach. Combined with various food textures, postural adjustment, and specific swallowing manoeuvres the technique can yield therapeutic information in the hands of a skilled speech and language therapist.[26] Combined videofluoroscopy and manometry has allowed the timing and magnitude of changes in intraluminal pressure to be precisely correlated with movement of the bolus and different structures.[27] Endoscopy[28] may be the investigation of choice to exclude structural lesions but palatal elevation during swallowing obscures the view of the larynx and pharynx. Scintigraphy has been used to obtain indices of pharyngeal clearance,[29] ultrasound to study the movements of the tongue and pharyngeal wall,[30] cervical auscultation to detect aspiration,[31] and electrical impedance tomography[32] to measure the impedance changes in the neck during swallowing, but these remain essentially research tools.

Table 2 Home based compensatory processes to overcome neurogenic dysphagia

Type of compensation	*Example*
Behavioural	Slowing down eating and drinking Spontaneous dietary adjustments Help with feeding Altered social setting (eg, sitting alone)
Postural	Sitting up or lying on side Removal of neck restrictions Control of abnormal movements
Oropharyngeal	Head turning, supraglottic swallow, sensory stimulation through textures and diet
Breathing	Prompt treatment of pulmonary problems, effective coughing techniques: first aid measures (eg, Heimlich manoeuvre)

Breathing should be assessed hand in hand with swallowing. Basic observation of breathing rate and pattern, cough, forced vital capacity (supine and sitting), diaphragm function, blood gases, and chest radiography may be required. Loss of integration of breathing and swallowing may result in an abnormal respiratory pattern around the swallow increasing the risk of inhalation; sensitive techniques to investigate this problem have been developed.[33]

Assessment of severity and advice regarding interventions

Compensatory mechanisms (table 2) are recruited by the dysphagic patient spontaneously, or following simple advice, which may reduce the likelihood of pneumonia or malnutrition, the major decompensatory events. These may require time, instruction, and encouragement to be brought into play and therefore may be absent acutely. Qualitative water swallow tests have proved useful for dysphagia screening[34–36] and have been extended to include a quantitative element.[37–39] In one version[39] a known volume of water is swallowed in a measured time and the number of swallows counted. The swallowing capacity ($ml.s^{-1}$), and the average volume/swallow (ml) can be expressed in relation to a normal range. Reduced capacity and volume per swallow are probably best interpreted as compensatory mechanisms designed to reduce the risk of laryngeal penetration or aspiration whereas cough, wet hoarse voice, or breathlessness are evident decompensations. Water has the advantage that it is a stringent test of laryngeal closure, has face validity (people drink it), and it is accessible during a clinic or ward round.

The roles of clinical assessment and videofluoroscopy in the process of decision making about the "risks" of oral feeding are much discussed. It is argued that bedside assessment is an insensitive indicator of videofluoroscopically defined aspiration[40] and that, because aspiration is sometimes associated with chest infections, videofluoroscopy should usually be undertaken. However, we think that care should be taken not to place excessive weight on the presence or absence of aspiration alone. It is the clinical consequence of aspiration, rather than radiological aspiration itself, which is important: this may depend on multiple factors not assessed at videofluoroscopy. When the main issue is the assessment of risk of oral feeding a review of the severity and frequency of relevant symptoms, of the occurrence of chest infections, and of recent oral intake and weight change, followed by formal observation of eating and drinking over a representative period probably provide more relevant information than videofluoroscopy.[41–44] Videofluoroscopy seems helpful when unusual swallowing techniques are adopted to help patients regain oral

Table 3 Active external interventions in neurogenic dysphagia

Intervention category	*Example*
Conservative	Dietary intervention, supplements, thickeners
Enhanced swallowing	Palatal prostheses, electrical stimulation, cricopharyngeal myotomy
Enteral feeding	Nasogastric tube, gastrostomy, jejunostomy
Parenteral feeding	
Pulmonary function	Domiciliary (nocturnal) ventilation, improved bronchial access through tracheostomy
Airway protection from aspiration	Vocal cord manipulation (Teflon), partial cord closure, laryngectomy
Salivary secretions	Reduce, thicken, divert

feeding,[45] when the effect of a treatment or manoeuvre has to be demonstrated to patient and carer to reinforce advice based on clinical judgement, or in those patients who, for whatever reason, are unable to cooperate with clinical examination or other methods of investigation.

From a practical point of view if a patient is alert, able to sit up, and to phonate normally, has a reasonable cough and no major pulmonary problem, medical or nursing staff can undertake test boluses of water and proceed to oral feeding if these are taken without difficulty. If a water test is clearly abnormal (very slow or produces coughing, choking, or a wet hoarse voice) the patient should be "nil by mouth", fed by an alternative route and reassessed later or referred to a speech and language therapist. In many acute neurological disorders such as stroke the swallowing problem will often improve spontaneously over a few weeks. The best interim management to be adopted requires research through randomised clinical studies.[46]

The importance of interventions to maintain nutrition and fluid intake (table 3) is well recognised and action must not be unduly deferred while diagnostic issues are considered, unless declined by the patient or clinically inappropriate. Extra urgency is implied if sepsis, wasting, injury, or systemic illness cause increased metabolic requirement. Intravenous fluids and a nasogastric tube are effective short term interventions if the patient is either fully cooperative or comatose. If a patient obviously faces prolonged recovery or is confused and keeps pulling a nasogastric tube out a gastrostomy is more acceptable and allows a far higher proportion of the prescribed diet to be delivered.[14 47–50] In chronic neurodegenerative conditions it often helps to anticipate interventional feeding issues at an early stage by discussion with the patient and carers. In deciding on the timing of gastrostomy the amount of food and liquid taken, the time taken to consume it, recent weight loss, hunger, indices of nutrition and metabolic demand, and whether or not patients wish to continue oral feeding are the most relevant factors to consider. Gastrostomy insertion can be endoscopic, radiological or surgical.[51] All techniques carry a small risk: elderly and neurological patients, especially those with concomitant breathing disorders, can be at risk of sudden deterioration with sedation.[52–54] Feeding via gastrostomy in patients unused to large gastric loads may lead to oesophageal reflux[55] with aspiration particularly in those fed overnight in the supine position. We favour bolus feeding in the upright posture by day at around normal meal times although gastric volumes may need to be built up gradually. From a rehabilitation standpoint hours spent feeding by the oral route should not be replaced with hours spent "tied" to a pump infusing a percutaneous endoscopic gastrostomy. Whether there are really different risks from these feeding patterns needs to be established through randomised trials. If regurgitation or aspiration are persistent stomach emptying may be enhanced pharmacologically (metoclopramide, cisapride); in some cases jejunostomy helps.

Apart from an ongoing aspiration risk patients who become "nil by mouth" can still face difficulty controlling oropharyngeal secretions; this may result in drooling, or recurrent coughing and choking attacks[56] particularly if laryngeal reflexes are hyperactive as may be the case in motor neuron disease. Secretions can be reduced using anticholinergic medication. Suitable preparations for dysphagic patients include atropine solution emptied into the mouth or hyoscine tablets. After recommendation from a colleague in palliative care (Professor I Finlay) we have found patients who benefit from subcutaneous injections of glycopyrronium bromide (Robinul, unlicensed use) starting with 50–100 μg daily: self administration can be learnt. Thicker secretions may be helped by β-blockers.[57] If combined with suction apparatus for use by the patient or carer these measures are often enough to control symptoms and prevent aspiration of secretions. Excessive saliva reduction can result in thick sticky strands which are difficult to clear and may trigger choking attacks. Redirecting the salivary ducts may reduce drooling but in those who cannot effectively swallow may further threaten maintenance of the airway. Irradiation of the salivary glands is occasionally used to reduce salivary flow but a dry mouth may be equally troublesome. Palatal training devices may of value in some circumstances.[58]

Some patients with dysphagia have had a tracheostomy placed to allow better access to the airway or for ventilation. The optimal sequence for testing and resuming oral feeding is often a source of argument mainly for lack of clear evidence: the input, guidance and continuity provided by an experienced speech and language therapist is usually invaluable. We do not favour oral feeding while an inflated cuffed tube is still in situ; it does not prevent aspiration and indeed may promote it by allowing secretions to pool above the cuff, and by impeding laryngeal movement during swallowing.[59 60] We prefer to move to an uncuffed tube and establish whether oral secretions are controlled with the lungs remaining clear; if so oral feeding with suitable textures can be commenced. If pulmonary complications supervene oral feeding should cease. Dye tests occasionally seem of value; in this context a videofluoroscopy may be of more use if serious "silent" aspiration is suspected.

More invasive measures can sometimes be helpful for intractable aspiration or recurrent disabling choking and coughing. Such situations usually arise when there is bilateral involvement of the intrinsic laryngeal muscles as after brainstem trauma or haemorrhage, or with overactivity of airway protective reflexes. Teflon injection into a paretic vocal cord may reduce aspiration risk. Surgical manipulation of the arytenoids and epiglottis, and partial or complete closure of the laryngeal entrance in those with tracheostomies may be an option if speech can be compromised. Oesophageal speech is usually not an option for neurological patients. If the patient is irreversibly aphonic the option of laryngectomy with an end tracheostome is worth considering purely for relief of symptoms. Alternatively the larynx can be sectioned and closed below the true vocal cords; this leaves the option of reconnecting the larynx and trachea if subsequent recovery of the intrinsic laryngeal muscles occurs.[61] If the overall prognosis is poor it is particularly important to try and spare the patient from a debilitating series of hospital admissions for assessment and progressively elaborate interventions.[62–64]

Future research

Much published work on neurogenic dysphagia has been anecdotal, uncontrolled, and based on highly selected,

usually small, groups of patients. Some series also reflect rather particular local patterns of referral relevant to the country of origin or the lack (or otherwise) of resources, which make it difficult to generalise data to a wide set of clinical problems. Whereas new technologies have clearly enhanced our understanding of dysphagia and its relation to nutrition and breathing at a descriptive level, there is a real need for more randomised controlled clinical studies focused on commonly used interventions if best practice is to be evidence based.

We thank Professor R Eccles for his support and Mrs Anna McGroary for helpful comments about the manuscript. CMW and TATH have both been funded for research into dysphagia by Procter and Gamble Health and Beauty Care Ltd.

T A T HUGHES
C M WILES

University of Wales College of Medicine, Heath Park, Cardiff, CF4 4XN, Wales

Correspondence to:
Professor C M Wiles, University of Wales College of Medicine, Heath Park Cardiff, Wales CF4 4XN. Telephone 0044 1222 743798; fax 01222 744166; email wiles@cardiff.ac.uk

1 Donner MW, Bosma JF, Robertson DL. Anatomy and physiology of the pharynx. *Gastrointestinal Radiology* 1985;**10**:196–212.
2 Logemann JA. Swallowing physiology and pathophysiology. *Otolaryngol Clin North Am* 1988;**21**:613–23.
3 Dodds WJ, Stewart ET, Logemann JA. Physiology and radiology of the normal oral and pharyngeal phases of swallowing. *AJR Am J Roentgenol* 1990; **154**:953–63.
4 Miller FR, Sherrington CS. Some observations on the bucco-pharyngeal stage of reflex deglutition in the cat. *Quarterly Journal of Experimental Physiology* 1916;**9**:147–86.
5 Doty RW, Bosma JF. An electromyographic analysis of reflex deglutition. *J Neurophysiol* 1956;**19**:44–60.
6 Miller AJ. Deglutition. *Physiol Rev* 1982;**62**:129–84.
7 Jean A. Brainstem control of swallowing: localisation and organisation of the central pattern generator for swallowing. In: Taylor A, ed. *Neurophysiology of the jaws and teeth*. New York: McMillan, 1990:294–321.
8 Miller AJ. The search for the central swallowing pathway: the quest for clarity. *Dysphagia* 1993;**8**:185–94.
9 Hamdy S, Aziz Q, Rothwell JC, *et al*. The cortical topography of human swallowing musculature in health and disease. *Nat Med* 1996;**2**:1217–24.
10 Urban PP, Hopf HC, Connemann B, *et al*. The course of corticohypoglossal projections in the human brainstem: functional testing using transcranial magnetic stimulation. *Brain* 1996;**119**:1031–8.
11 Daniels SK, Foundas AL. The role of the insular cortex in dysphagia. *Dysphagia* 1997;**12**:146–56.
12 Artenian DJ, Lipman JK, Scidmore GK, *et al*. Acute neck pain and dysphagia due to tendinitis of the longus colli: CT and MRI findings. *Neuroradiology* 1989;**31**:166–9.
13 Hughes TAT, Wiles CM, Lawrie BW, *et al*. Dysphagia and sleep apnoea associated with cervical osteophytes due to diffuse idiopathic skeletal hyperostosis. *J Neurol Neurosurg Psychiatry* 1994;**57**:384.
14 Singh S, Stein HJ, De Meester TR, *et al*. Non-obstructive dysphagia with gastroesophageal reflux disease: a study with combined ambulatory pH and motility monitoring. *Am J Gastroenterol* 1992;**87**:562–7.
15 Balbuena L, Hayes D, Ramirez SG, *et al*. Eagle's syndrome (elongated styloid process). *South Med J* 1997;**90**:331–4.
16 Shankland WE. Pterygoid hamulus bursitis: one cause of craniofacial pain. *Journal of Prosthetic Dentistry* 1996;**75**:205–10.
17 Buchholz DW Oropharyngeal dysphagia due to iatrogenic neurological dysfunction. *Dysphagia* 1995;**10**:248–54.
18 Shapiro BE, Rordorf G, Schwamm L, *et al*. Delayed radiation-induced bulbar palsy. *Neurology* 1996;**46**:1604–6.
19 Sokoloff LG, Pavlakovic R. Neuroleptic-induced dysphagia. *Dysphagia* 1997;**12**:177–9.
20 Stoschus B, Allescher H-D. Drug induced dysphagia. *Dysphagia* 1993;**8**: 154–9.
21 Hughes TAT, Wiles CM. Palatal and pharyngeal reflexes in health and in motor neuron disease. *J Neurol Neurosurg Psychiatry* 1996;**61**:96–8.
22 Kidd D, Lawson J, Nesbitt R, *et al*. Aspiration in acute stroke: a clinical study with videofluoroscopy. *Q J Med* 1993;**86**:825–9.
23 Willoughby EW, Anderson NE. Lower cranial nerve motor function in unilateral vascular lesions of the cerebral hemisphere. *BMJ* 1984;**289**:791–4.
24 Born LJ, Harned RH, Rikkers LF, *et al*. Cricopharyngeal dysfunction in Parkinson's disease: role in dysphagia and response to myotomy. *Mov Disord* 1996;**11**:53–8.
25 Ali GN, Wallace KL, Laundl TM, *et al*. Predictors of outcome following cricopharyngeal disruption for pharyngeal dysphagia. *Dysphagia* 1997;**12**: 133–9.
26 Chen MY, Peele VN, Donati D, *et al*. Clinical and videofluoroscopic evaluation of swallowing in 41 patients with neurologic disease. *Gastrointestinal Radiology* 1992;**17**:95–8.
27 Castell JA, Olsson R, Castell DO, *et al*. Simultaneous video radiography and manometry in dysphagia with normal barium swallow. *Am J Gastroenterol* 1993;**88**:1483.
28 Langmore S, Schatz K, Olsen N. A comparison of endoscopic and videofluoroscopic evaluations of swallowing. *Annals of Otorhinolaryngology* 1991;**100**:678–81.
29 Hamlet S, Choi J, Zormeier M, *et al*. Normal adult swallowing of liquid and viscous material: scintigraphic data on bolus transit and oropharyngeal residues. *Dysphagia* 1996;**11**:41–7.
30 Miller JL, Watkin KL. Lateral wall pharyngeal motion during swallowing using real time ultrasound. *Dysphagia* 1997;**12**:125–32.
31 Zenner PM, Losinski DS, Mills RH. Using cervical auscultation in the clinical dysphagia examination in long-term care. *Dysphagia* 1995;**10**:27–31.
32 Hughes TAT, Liu P, Griffiths H, *et al*. Simultaneous electrical impedance tomography and videofluoroscopy in the assessment of swallowing. *Physiol Meas* 1996;**17**:109–19.
33 Tarrant SC, Ellis RE, Flack FC, et al. Comparative review of techniques for recording events at rest and during deglutition. *Dysphagia* 1996;**12**:24–38.
34 Gordon C, Langton Hewer R, *et al*. Dysphagia in acute stroke. *BMJ* 1987; **295**:411–14.
35 DePippo KL, Holas MA, Reding MJ. The Burke dysphagia screening test; validation for its use in patients with stroke. *Arch Phys Med Rehabil* 1994;**75**:1284–6.
36 Kidd D, Lawson J, Nesbitt R, *et al*. The natural history and clinical consequences of aspiration in acute stroke. *Q J Med* 1995;**8 8**:409–13.
37 Ertekin C, Aydogdu I, Yuceyar N. Piecemeal deglutition and dysphagia limit in normal subjects and in patients with swallowing disorders. *J Neurol Neurosurg Psychiatry* 1996;**61**:491–6.
38 Nilsson H, Ekberg O, Olsson R, *et al*. Quantitative assessment of swallowing in healthy adults. *Dysphagia* 1996;**11**:110–6.
39 Hughes TAT, Wiles CM. Clinical measurement of swallowing in health and in neurogenic dysphagia. *Q J Med* 1996;**89**:109–16.
40 Splaingard ML, Hutchins B, Sulton LD, *et al*. Aspiration in rehabilitation patients: videofluoroscopy vs bedside clinical assessment. *Arch Phys Med Rehabil* 1988;**69**:637–40.
41 Holas MA, DePippo KL, Reding MJ. Aspiration and relative risk of medical complication following stroke. *Arch Neurol* 1994;**51**:1051–3.
42 Hughes TAT, Lawrie BW, Wiles CM. Laryngeal aspiration of liquid during videofluoroscopy in patients with motor neuron disease. *European Journal of Neurology* 1996;**3**(suppl 2):115–6.
43 Smithard DG, O'Neill PA, Park C, *et al*. Complications and outcome after acute stroke: does dysphagia matter? *Stroke* 1996;**27**:1200–4.
44 Mari F, Matei M, Ceravolo MG, *et al*. Predictive value of clinical indices in detecting aspiration in patients with neurological disorders. *J Neurol Neurosurg Psychiatry* 1997;**63**:456–60.
45 Drake W, O'Donoghue S, Bartram C, *et al*. Eating in sidelying facilitates rehabilitation in neurogenic dysphagia. *Brain Inj* 1997;**11**:137–42.
46 DePippo KL, Holas MA, Reding MJ, *et al*. Dysphagia therapy following stroke; a controlled trial. *Neurology* 1994;**44**:1655–9.
47 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* 1992;**304**:1406–9.
48 Raha SK, Woodhouse K. The use of percutaneous endoscopic gastrostomy (PEG) in 161 consecutive elderly patients. *Age Aging* 1994;**23**:162–3.
49 Norton B, Homer-Ward M, Donnelly MT, *et al*. A randomised prospective comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding after acute dysphagic stroke. *BMJ* 1996;**312**:13–6.
50 Britton JER, Lipscomb G, Mohr PD, *et al*. The use of percutaneous endoscopic gastrostomy (PEG) feeding tubes in patients with neurological disease. *J Neurol* 1997;**244**:431–4.
51 Wollman B, D'Agostino HB, Walus-Wigle JR, *et al*. Radiologic, endoscopic and surgical gastrostomy: an institutional evaluation and meta-analysis of the literature. *Radiology* 1995;**197**:699–704.
52 Foutch M. Complications of percutaneous endoscopic gastostomy and jejunostomy. Recognition, prevention and treatment. *Gastrointest Endosc Clin N Am* 1992;**2**:231–48.
53 Stuart SP, Tiley EH, Boland JP. Feeding gastrostomy: a critical review of its indications and mortality rate. *South Med J* 1993;**86**:169–72.
54 Tham TCK, Taitelbaum G, Carr-Locke DL. Percutaneous endoscopic gastrostomies: are they being done for the right reasons? *Q J Med* 1997;**90**: 495–6.
55 Short TP, Patel NR, Thomas E. Prevalence of gastroesophageal reflux in patients who develop pneumonia following percutaneous endoscopic gastrostomy: a 24 hour pH monitoring study. *Dysphagia* 1996;**11**:87–9.
56 Ekberg O, Feinberg M. Clinical and demographic data in 75 patients with near-fatal choking episodes. *Dysphagia* 1992;**7**:205–8.
57 Newall AR, Orser R, Hunt M. The control of oral secretions in bulbar ALS/ MND. *J Neurol Sci* 1996;**139**(suppl):43–4.
58 Oliver RG. Theoretical aspects and clinical experience with the palatal training appliance for saliva control in persons with cerebral palsy. *Special Care in Dentistry* 1987;**1**:271–4.
59 Cameron JL, Reynolds J, Zuidema GD. Aspiration in patients with tracheostomies. *Surgical Gynaecology and Obstetrics* 1973;**136**:68–70.
60 Betts RH. Post tracheostomy aspiration. *N Engl J Med* 1965;**273**:155.
61 Hawthorne M, Gray R, Cottam C. Conservative laryngectomy (an effective treatment for severe aspiration in motor neurone disease). *J Laryngol Otol* 1987;**101**:283–5.
62 Baredes S. Surgical management of swallowing disorders. *Otolaryngol Clin North Am* 1988;**21**:711–20.
63 Blitzer A, Krespi KP, Oppenheimer RW, *et al*. Surgical management of aspiration. *Otolaryngol Clin North Am* 1988;**21**:743–50.
64 Short SO, Hillel AD. Palliative surgery in patients with bulbar amyotrophic lateral sclerosis. *Head Neck* 1989;**11**:364–9.