Swallowing and prevention of complications

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Dysphagia occurs in up to half of patients following a stroke. In most, it is transient with only about 1 in 10 of patients having any swallowing problems at 6 months. Persistent dysphagia may be due to lack of bilateral cerebral hemisphere representation of the oral and pharyngeal musculature involved in swallowing. Thus, the unaffected hemisphere is unable to take over the function of the damaged side. Bedside assessment is not a good predictor of aspiration on videofluoroscopy, but measurement of oxygen saturation may improve this. Nevertheless, clinical detection of dysphagia may be the more powerful predictor of an increased mortality and morbidity, including pneumonia, water depletion and poor nutrition. Dysphagia is also closely related to poor nutrition following stroke, but we do not know whether feeding support will improve outcome. Major trials are on-going.

Over the last decade, there has been a great interest in the effect of stroke on swallowing. The historical view was that dysphagia only occurred in patients with brainstem strokes, but it is now clear that many patients with hemispheric stroke also have swallowing difficulties. The dysphagia is often transient following an acute stroke\(^1\), but is a sign indicating a poor prognosis\(^2\), and an increased risk of complications including pneumonia\(^3\), water depletion\(^4\) and malnutrition\(^2\).

Current work is focused on whether any specific therapy might improve the recovery of dysphagia\(^5\) and whether supportive interventions such as percutaneous endoscopic gastrostomy (PEG) feeding might reduce the risk of complications, particularly poor nutrition and possibly pneumonia\(^6\).

This review will begin by looking at the neurological control of swallowing and how this might be affected by stroke, together with the reported incidence of dysphagia in stroke. Following on from this background, the questions that are addressed are whether swallowing problems are of any consequence or are they simply a marker for stroke severity. Finally, the review will look at how complications of dysphagia might be prevented or treated to improve the overall outlook from stroke.

Control of swallowing

Swallowing is the act of moving food from the oral cavity to the stomach. It is usually considered to be ‘voluntary’, but most swallows occur...
involuntarily in response to saliva production. This link with salivation, rather than solely with the presence of food, is important when considering the risk of aspiration pneumonia.

Swallowing is a complex act that utilises 31 paired striated muscles. Rapid and fine neuromuscular control leads to mastication of food to a consistency appropriate for swallowing and moving the bolus from the oral cavity to the pharynx, oesophagus and finally the stomach. During swallowing, respiration ceases momentarily, which, again, is important when considering aspiration pneumonia.

There are three anatomically and temporally distinct phases: (i) oral, including preparatory and propulsion; (ii) pharyngeal; and (iii) oesophageal.

**Oral**

**Preparatory**
Teeth, tongue, lips and cheeks respond to the sensation of taste by forming the food into a bolus. This is then held by the tip of the tongue against the alveolar ridge or superior incisors, which traps the food bolus against the hard palate.

**Propulsive**
The tongue lifts and presses the bolus against the roof of the mouth and sends it into the oropharynx using a sequential squeezing action. When the bolus reaches the faucial arches, sensory receptors trigger the pharyngeal stage, starting with descent of the tongue base and elevation of the uvula to create a pathway for the bolus that will be helped by gravity.

**Pharyngeal**
The start of the pharyngeal phase is defined as when the bolus passes the tonsillar pillars and finishes when it enters the oesophagus. A number of mechanisms protect the nasopharynx and airway. The soft palate approximates to the pharyngeal walls to prevent nasal regurgitation. The larynx and hyoid move upwards and anteriorly, and the larynx is closed from the vocal cords to the false cords. The laryngeal entrance is also protected by the epiglottis and aryepiglottic folds.

Pressure from the tongue base moves the bolus from the valleculae to the oesophageal inlet. Waves of pharyngeal peristalsis generate progressive contractile waves. Relaxation of the cricopharyngeal muscle leads to opening of the upper oesophageal sphincter. As the food moves into the oesophagus, the sphincter closes and the larynx and palate open so that respiration can restart. In total, like the oral phase, the pharyngeal phase lasts approximately 1 s.
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**Oesophageal**

A disordered oesophageal phase is not usually a major cause of dysphagia following a stroke. Sequential peristaltic waves move the bolus down the oesophagus and the phase usually lasts 8–20 s.

**Innervation and control**

In an elegant series of experiments using transcranial magnetic stimulation, Hamdy et al showed that the muscles involved in swallowing are discretely represented on the motor and premotor cerebral cortex with the topography of the oral, pharynx and oesophagus being arranged in set areas. Furthermore, there was marked asymmetry of representation in the two hemispheres, which was independent of handedness. This has major implications in predicting the occurrence of dysphagia following a stroke and also its recovery. Others have suggested that there may not be discrete cortex ‘swallowing’ centres, but a distributed neural network involving both hemispheres.

The basic swallowing mechanism starts with trains of impulses from the cortices that produce a temporal summation on the brainstem swallowing centre to trigger a swallow via the cranial nerves directly controlling the muscles involved. If there is residual material in the pharynx, sensory receptors detect this and initiate a second swallow to clear the material. If food goes ‘the wrong way’, then sensory mechanisms will elicit a cough reflex to protect the airway from aspiration and to clear the pharynx. It is very unusual for a fit person to aspirate or to have detectable pharyngeal foodstuffs after swallowing.

**Dysphagia and stroke**

**Assessment**

In dysphagia research, it is important to separate out studies that have used bedside (clinical) assessment tools to determine the presence of swallowing problems (e.g. Gordon et al and Wade & Hewer) and those that have used videofluoroscopy (VF; e.g. Splaingard et al). Research carried out using VF has mainly used aspiration or penetration of food into the larynx as outcome measures, which may or may not have reflected the clinical presence of dysphagia. One blinded study found that a standardised assessment of swallowing by speech and language therapists (SLTs) identified only 42% of patients who were aspirating on VF. Similarly, Smithard et al found that the bedside
assessment by a SLT gave a sensitivity of only 47% and a positive predictive value of 50% for the presence of aspiration in acute stroke. It is possible that measurement of oxygen desaturation alone or combined with bedside assessment will improve the clinical detection of aspiration\textsuperscript{13}, though others have found it to be unhelpful\textsuperscript{16}.

The other factor is whether VF should be regarded as the gold standard. The imaging is carried out in a controlled environment with good positioning of the patient and filming time of less than 3 min. This probably does not reflect swallowing function whilst feeding on a ward or at home, so the false negative rate may be high.

Given these concerns about the usefulness of very detailed bedside assessments, the most useful factors\textsuperscript{14,17-19} in pointing towards the presence of a high risk of aspiration are: (i) any disturbance of conscious level; (ii) ‘wet voice’; (iii) weak voluntary cough; (iv) cough on drinking small volumes of water’ and (v) a timed water swallowing test. Despite the general moderate results from screening instruments, work continues on trying to delineate a good screening procedure using multiple items\textsuperscript{20}.

**Epidemiology**

Many studies have highlighted the high prevalence of swallowing problems following stroke, depending on the phase and type of assessment. In acute stroke, between one-quarter and a half of patients have dysphagia\textsuperscript{2,4,11}. One important factor is the timing of the assessment; dysphagia is transient in many patients, resolving in the first couple of days\textsuperscript{11}.

Following the acute phase of stroke, many studies have used VF to determine the presence of swallowing difficulties. Prevalence rates of a half to two-thirds of patients\textsuperscript{21-23} have been found, but the studies have been conducted months to years after a stroke on patients referred to specialist centres. In an unselected stroke population, Smithard et al\textsuperscript{11} found that the initial prevalence of 51% had decreased to 11% at 6 months. However, more recently, Mann et al\textsuperscript{24} showed that, in many dysphagic patients, the swallowing problems were still present at 6 months, particularly in elderly male patients with initial laryngeal penetration.

**Prognosis**

Dysphagia has been found in several studies to be associated with excess morbidity and increased mortality rates compared to stroke patients without swallowing difficulties\textsuperscript{2,4,17}. When combined with other...
accepted indicators of poor prognosis (weakness, neglect, hemianopia, incontinence, apraxia, age and sex) in a multivariate logistic regression analysis, the presence of an unsafe swallow, but not aspiration on VF, remained a significant predictor of mortality. Patients with reduced level of consciousness had already been excluded from the study population. Similarly, almost one-third of alert patients with dysphagia were dead by 6 months following their stroke compared to less than 10% of those with a normal swallow.

Swallowing difficulties are also an indicator of a worse functional outcome. Barer, Wade and Hewer, and Smithard et al all reported that patients with dysphagia were more disabled as assessed by the Barthel index score. In addition, stroke patients with dysphagia were more likely to remain in institutionalised care.

Complications

Dysphagia is associated with: (i) aspiration and associated bronchopulmonary infections; (ii) fluid depletion; and (iii) malnutrition. Aspiration pneumonia may account for about 1 in 20 deaths following stroke. Gordon et al found that 1 in 5 patients with dysphagia developed pneumonia compared with 1 in 12 without swallowing problems following stroke. In a case-matched study, Schmidt et al reported that aspiration predicted the development of pneumonia with an odds ratio of over 5 in those patients who aspirated on thickened fluids. Although aspiration on VF is a predictor for pneumonia, it is likely that dysphagia detected at the bedside is a more powerful pointer to an increased risk: both Smithard et al and Kidd et al found that the risk of pneumonia remained whether or not aspiration was present on VF.

A number of studies have suggested that dehydration is associated with dysphagia following a stroke, but the differences compared with the non-dysphagic group did not reach statistical significance. Neither Schmidt et al nor Smithard et al found any links between swallowing problems and dehydration; the latter authors ascribed this to an active unit policy of fluid support as judged by the use of parenteral fluids.

Elsewhere in this issue, the relationship between stroke and malnutrition are reviewed. Axelsonn et al noted a gradual deterioration in nutritional status with time following stroke, but considered this to be a function of the severity of brain damage and age of the patient rather than the ability to swallow. Davalos et al also reported a worsening of nutritional indices after admission and also an increased risk of death. Smithard et al found that the risk of malnutrition in the first month following stroke was associated with dysphagia, and suggested
that this was due to poor food intake consequent upon the swallowing impairment.

**Prevention and management of complications**

Although the management of a dysphagic patient by a multidisciplinary team including a trained SLT is regarded as being a marker of good quality of care, there is limited evidence for this. Lucas and Rodgers did report a trend towards better outcome measures when an SLT was involved and Jacobsson et al. suggested that outcomes were improved with individualised interventions in patients with severe eating problems.

The management of dysphagia and the prevention of complications can be considered in terms of: (i) specific therapies to improve recovery from dysphagia; and (ii) supportive measures to reduce the consequences of dysphagia. Many different therapies have been described in the literature for swallowing difficulties following a stroke and there is not a clear evidence base for selecting one of these to use in a patient with particular characteristics. Some authors suggest that a therapy should be selected on the basis of bedside assessment, whilst others argue that such decisions should be determined by VF findings.

Logemann (38) put forward a framework consisting of 3 categories of therapies:

1. **Compensatory**: in which the aim is to eliminate the symptoms of dysphagia through altering the direction of food flow. An example is by paying careful attention to the position of the head and body during feeding.

2. **Indirect**: in which the aim is to improve neuromuscular control without giving the patient any food/fluid or causing him/her to swallow. An example is getting the patient to move their tongue or jaw against resistance.

3. **Direct**: in which the aim is to directly improve the pathophysiology of swallowing. The principal example is thermal (cold) stimulation of the oral cavity.

The work on thermal stimulation illustrates how further research is required to determine whether any of these therapies are of value (and under what circumstances). Lazzara et al. reported significant improvement in the triggering of swallowing after cold therapy. This study was criticised on the grounds that the subjects were heterogeneous in terms of their stroke and nature of their swallowing problems. Furthermore, the duration and type of stimulation used were not clearly described. In a later paper, Rosebek et al. did not find any clear effect of thermal stimulation. More recently, oral electrical stimulation and nifedipine have been added to the number of therapies.
There have been few studies looking at the impact of specific interventions on aspiration pneumonia. An important factor is the origin of the infection. Murray et al found that accumulated oropharyngeal secretions predicted aspiration\textsuperscript{45}, which implies that pooling of saliva \textit{per se} may be a major factor in developing pneumonia rather than feeding. Other studies have suggested that feeding tubes may not prevent aspiration\textsuperscript{46} and that a major predictor of aspiration pneumonia is a previous episode\textsuperscript{47}. In relation to acute stroke, it is possible that the aspiration of saliva occurs soon after the onset of symptoms, making it unlikely that stopping oral intake several hours later will have any impact on the development of pneumonia.

As with the prevention of aspiration pneumonia, it is not clear how an optimal level of hydration should be achieved in dysphagic patients following a stroke. Clearly, some fluid support is needed in those patients unable to take anything orally. However, due to the neuroendocrine response to acute stroke, some patients may have very high vasopressin levels\textsuperscript{48}. It follows that these patients may need to be fluid restricted so that overhydration and hyponatraemia does not occur.

The major area in which a major impact on outcome following stroke may be possible is nutritional support. This is reviewed by Dennis\textsuperscript{49} elsewhere in this issue, but, currently, there is only limited evidence for the use of PEG feeding or other nutritional support in patients with stroke.

**Key points for clinical practice**

- Screen all acute stroke patients for dysphagia using: disturbance of consciousness, wet voice, weak cough and cough on taking small amounts of water. Use pulse oximetry if available.
- If any doubt about presence of swallowing impairment, refer to a speech and language therapist.
- Use videofluoroscopy only where the speech and language therapist needs information to guide choice of therapy.
- Consider entering patients into the FOOD study\textsuperscript{6-49} to determine fluid and nutritional support.
- Monitor (and treat) for aspiration pneumonia.

**References**

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