Swallowing is a complex process and dysphagia can be life-threatening. Shaheen Hamdy describes the swallowing process and the causes of dysphagia. She also discusses the problems associated with neurogenic dysphagia.

Swallowing is a complex sensorimotor process that involves the transport of material from the mouth to the stomach for digestion. It is also involved in protecting the airway and in rejecting noxious ingested substances. Humans swallow on average once every minute and this is supplemented by the production of saliva. When we eat, swallowing increases dramatically (six to eight times a minute).

The process is commonly divided into three phases: oral, pharyngolaryngeal and oesophageal, which represent the anatomical structures involved in swallowing. The process requires a sophisticated means of voluntary and reflex central control for coordinated execution (Miller, 1982) (Box 1). Disruption of swallowing can have serious effects, with complications such as malnutrition, pulmonary aspiration and the associated psychosocial stigma of being unable to eat.

Causes of dysphagia

This can be the cause by disease in either the anatomical structures involved in swallowing or, more commonly, the central nervous system (neurogenic dysphagia). Anatomical causes These can include almost any gastrointestinal disease process, from the oral cavity through to the duodenum. It is, therefore, important to exclude any intrinsic disease of the gut before making a diagnosis of neurogenic dysphagia in someone presenting with symptoms of swallowing difficulty.

Neurological causes Many neurological disorders can disrupt swallowing, including diseases that affect:

- The muscle or the neuromuscular junction (meeting point of a nerve fibre and a muscle fibre) — conditions such as polymyositis and myasthenia gravis;
- The peripheral nerves — conditions such as Guillain-Barré syndrome, polio and diphtheria;
- Central swallowing pathways — conditions such as stroke, head injury, motor neuron disease, Parkinson’s disease, multiple sclerosis and neurodegenerative diseases such as Alzheimer’s disease.

It is important to recognise that dysphagia can occur with use of many pharmacological agents, such as benzodiazepines, that might alter neuromuscular function. The most common cause of adult neurogenic dysphagia, however, is stroke (Kirshner, 1989).

Dysphagia and neurological damage

Traditionally, it has been taught that because the brain-stem swallowing centre has bilateral innervation from the cortex, dysphagia could occur only as a consequence of direct brain-stem or bilateral cortical damage. Over the past two decades the concept that unilateral cerebral lesions can cause dysphagia has been accepted. While the mechanism for dysphagia that occurs after unilateral cortical damage remains uncertain, studies show that one side of the cortex may be dominant for swallowing control (Hamdy et al, 1996). (This is not related to a person’s dominant hand.) Damage to the dominant cerebral hemisphere, therefore, will predispose that individual to dysphagia, whereas damage to the non-dominant hemisphere would leave the person with intact swallowing function.

Assessment of swallowing in neurogenic dysphagia

Neurological damage may hinder a patient’s recovery to normal function, at worst, resulting in death (Fig 2). However, it can be managed by enteric feeding. Most of the data available suggests that dysphagia...
occurs in 51–64 per cent of patients who have acute stroke (Mann et al, 2000). This figure falls markedly with time after the cortical event, so that three months after the stroke, less than 2 per cent of patients will have significant swallowing difficulties (Barer, 1989). The underlying mechanism for the recovery is unclear, but may be due to compensation from regions in the unaffected cerebral hemisphere that control swallowing (Hamdy, 1996).

**Diagnosis of neurogenic dysphagia** Diagnosis can be difficult, so it is important to consider the possibility that neurogenic dysphagia is the cause of swallowing problems in patients with an acute or chronic neurological disorder.

For example, the pattern of disordered swallowing in stroke is usually a combination of oral and pharyngeal abnormalities. This is associated with delayed swallowing reflex, with pooling or stasis of residue; reduced pharyngeal peristalsis (serial contraction of gut muscles that push food through the gastrointestinal tract); and weak or uncoordinated tongue control (Logemann, 1983). Occasionally, oesophageal abnormalities may be apparent.

Clinical suspicion of swallowing difficulty should be followed up with a thorough swallowing assessment and, where appropriate, videofluoroscopy.

**Bedside swallowing assessment** This should include the following clinical assessments of the patient:
- Nutritional status;
- Posture;
- Breathing and cooperation levels.

The patient’s oral musculature, oral reflexes, pharyngeal swallow should then be assessed. A trial feed with a 5–10ml water bolus should be given to the patient to swallow, and their response then assessed.

While the clinical swallowing assessment is cheap, easy to perform and involves no radiation exposure, it is unlikely to give detailed information about the pharyngeal stage of swallowing. It is possible to miss significant (often silent) aspiration problems.

**Videofluoroscopy** Videofluoroscopy gives a detailed anatomical assessment of the pharyngeal swallow, but it is expensive, involves radiation and uses a non-physiological medium (barium), which may not give a true picture of the patient’s swallowing performance.

**Fibreoptic nasendoscopy** This technique, often known as the FEES (fibre-optic endoscopic evaluation of swallowing), is used to assess the pattern of disordered swallowing in dysphagia. It involves passing a nasendoscope into the oropharynx where anatomical structures can be directly visualised. The swallowing mechanism can be studied when the patient ingests a small volume of a coloured physiological meal (Wilson et al, 1992). Although the oral phase of swallowing cannot be assessed directly, the results seem as good as with videofluoroscopy and the method has the advantage of being mobile and free of radiation.

**Patient assessment** Patients admitted with neurological disorders must have a swallowing assessment. This is often carried out by ward nurses, who may not have had appropriate training, so the results can be misleading. This may lead to inappropriate care pathways being instituted, and suboptimal therapy given.

It is, therefore, essential to ensure that all staff who may be expected to perform a bedside swallowing examination have appropriate training (Smithard et al, 1996). Guidance on when to make relevant referrals, for example, to speech and language therapists, dietitians and gastroenterologists, should also be available.

**Complications** Perhaps the most clinically apparent complication of neurogenic dysphagia is pulmonary aspiration. This may manifest itself acutely as choking or coughing, respiratory distress, wheezing, gasping or gurgling, loss of voice quality and tachycardia. The patient may have chronic symptoms (particularly in silent aspiration), including weight loss, hunger, production of excessive oral secretions and they may refuse to eat.

![Diagram of the central nervous system control of swallowing](https://example.com/diagram)

**REFERENCES**


The greatest risk factor for aspiration is abnormal pharyngeal function and, specifically, a delayed swallow reflex and increased pharyngeal transit times (Johnson et al, 1993). Patients with pharyngeal transit times of more than five seconds (normal time is less than 1.5 seconds) have a significant risk of aspiration.

Other clinical predictors for risk of aspiration in neurogenic dysphagia include a weak cough reflex; dysphonia; reduced gag reflex; and ‘wet hoarseness’. However, evidence suggests that even in healthy older people the gag reflex is frequently absent (Davies et al, 1995), casting doubt on its clinical significance.

Non-pharyngeal factors such as poor oral hygiene, dependence on carers for feeding, tube feeding and polypharmacy may also affect the risk of aspiration and outcome in older patients (Langmore, 1998).

**Dehydration and malnutrition** The complications of dehydration and malnutrition are less well defined. One would expect dehydration and malnutrition purely as a logical consequence of dysphagia, but the findings of studies are not clearcut. Gordon et al (1987) found that 58% of the dysphagia group after acute stroke had a blood urea of 10 mmol/L (normal 3.5–8.8mmol) or higher at one week compared with 38 per cent of those without dysphagia. This correlated with haematocrit measurements. (This rises in dehydrated patients.) Barer (1969) found that after two weeks, haematocrit levels were not significantly raised in patients with dysphagia, suggesting that dehydration was a minor problem when suitable supplemental fluid support was given.

Data from a recent diet trial (FOOD Trial Collaboration, 2003) suggests that nutritional status immediately after stroke is independently linked to long-term outcome, regardless of the presence or absence of dysphagia. Deaths rates six months after admission to hospital with stroke were 17 per cent higher in undernourished patients compared with those who had normal nutritional status.

**Management** Liaison with expert health professionals such as speech and language therapists is of major importance. In cases of severe dysphagia, the risk of aspiration is high and the patient must not have an oral diet or fluids — they should be prescribed parenteral fluids as soon as possible.

Therapeutic interventions may be tried in cases of less severe dysphagia, based on videofluoroscopic and bedside swallowing assessment outcomes. These are usually delivered by speech and language therapists and may include dietary changes and posture adjustments, as well as methods for sensitising or desensitising the oropharynx to alter the swallow reflex.

There is little data to support the benefits of any of these approaches with dysphagic patients (DePippo et al, 1994). If these methods are unhelpful, the patient should be considered for enteral feeding, which usually involves nasogastric tube feeding or percutaneous endoscopic gastrostomy (PEG) tube feeding.

Each method has its benefits, but studies suggest that more emphasis should be placed on increased and early (in the first two weeks) use of PEG (Norton et al, 1996). However, while this may improve a patient’s nutritional status, there is concern that the risk of pulmonary aspiration remains high (Finucane and Bynum, 1996). One study found mortality rate following PEG insertion for dysphagia to be as high as 28 per cent at 30 days and 52 per cent at six months (Sanders et al, 2000). Patients with dementia fared worst, which raises ethical considerations about the appropriateness and efficacy of enteral feeding in this patient group.

**Conclusion** Neurogenic dysphagia is a common, often underdiagnosed, problem. Early detection and appropriate treatment is crucial, as the condition can be distressing and life-threatening.

It is vital that health care professionals, especially nurses, are well-trained, to ensure optimal management of dysphagic patients. Therapies such as PEG feeding, aimed at improving a patient’s nutritional status offer a means of delivery but do not remove the risk of aspiration and mortality.