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Editorial

Neurogenic dysphagia

The function of swallowing is the safe transfer of food and liquids from the mouth into the oesophagus. Since the airway and the "foodway" effectively share a common path in the mouth and pharynx, an elaborate mechanism exists to separate the two during swallowing thus preventing airway penetration by swallowed material: at the same time breathing and speech are necessarily arrested. The details of the oral preparatory phase, the oral phase of swallowing, and the swallowing reflex have been studied in considerable detail using clinical observation and radiological techniques (videofluoroscopy, ultrasound and isotope scanning) supplemented by manometry and electromyography.^{1,2}

The tongue plays an essential role in bolus preparation for swallowing, without which the bolus of material may not be properly presented to the oropharynx and thus may fail to trigger the essential swallowing reflex. Initial patterns of tongue movements vary between individuals, with age and in the presence of oral/pharyngeal tubes: the timing of laryngeal and cricopharyngeal events appears to be influenced by bolus volume.³ As the bolus passes through the pillars of the fauces a reflex mechanism comes into play. The essential elements of which are exclusion of the nasopharynx by velopharyngeal closure, propulsion of the bolus into the pharynx by the pumping action of the tongue as it strips backwards along the palate, closure of the larynx, and pharyngeal peristalsis which sweeps the bolus through the cricopharyngeal sphincter into the oesophagus. The pumping/peristaltic action of the tongue is perceived as producing the "tongue driving pressure" (measured manometrically) in the closed off oropharynx⁴; therapeutically if the larynx is held elevated voluntarily the base of the tongue can be used to push a bolus down the pharynx.⁵

Prevention of aspiration is achieved by the upward and anterior movement of the laryngeal aditus (mirrored clinically by the observed superior and anterior movement of the hyoid bone and thyroid cartilage) so that it is protected under the root of the tongue, diversion of the bolus by the epiglottis away from the laryngeal opening into the valleculae and lateral channels⁶ (which are formed by the aryepiglottic folds medially and the pharyngeal walls laterally) and occlusion of the airway. The upper airway is closed by approximation and stiffening of the aryepiglottic folds, below them the false cords (which form a flap-like valve tending to prevent the egress of air from the trachea) and finally closure of the true vocal cords which because of their direction act as a valve preventing ingress of material into the airway.⁷ Penetration of the airway by foreign material should normally elicit coughing. The ability to cough depends on the ability to build up expiratory volume and pressure behind a closed glottis and hence depends on vital capacity and the strength of powerful expiratory muscles including the abdominal muscles, latissimus dorsi, and pectorals. The upward and anterior movement of the

larynx during the swallowing reflex promotes opening of the cricopharyngeal sphincter⁸ and is associated with the development of a transient negative pressure in the hypopharynx and upper oesophagus which assists the passage of the bolus.

Coordination of swallowing depends on the integrity of sensory pathways from the tongue, mouth, pharynx and larynx (cranial nerves V, VII, IX, X) and coordinated voluntary and reflex contractions involving cranial nerves V, VII, and X-XII. A medullary "swallowing centre" has been proposed in the region of the nucleus of the tractus solitarius closely adjacent to the respiratory centre⁹; swallowing is coordinated with phase of respiration so that deglutition apnoea is normally followed by expiration thus helping to prevent aspiration.^{10,11} Reflex swallowing remains even in a persistent vegetative state. Voluntary initiation of swallowing is poorly understood but there is significant lateralisation of supranuclear control of the lower cranial nerves.¹² Unilateral lesions in the lowest part of the precentral gyrus and the posterior part of the inferior frontal gyrus may be associated with severe dysphagia unassociated with any buccolingual apraxia, speech impairment or local paresis.¹³ Dysphagia was present in the first few days in 37/91 consecutive patients with unilateral (right or left) hemisphere stroke and was associated with greater limb paresis and higher frequency of dysarthria, impairment of gag reflex, palatal movement, tongue protrusion and cough than those without dysphagia¹⁴; more patients with dysphagia died but in those who did not, dysphagia was generally transient lasting on average 8.5 days. Dysphagia was commonly found (29% of cases) in another large stroke series when patients were assessed on the day of admission¹⁵ and had a negative effect on outcome independent of other factors associated with stroke severity. Silent aspiration has been commonly observed in some selected stroke series^{16,17} and presumably the common sequence of stroke followed by "chest infection" is likely to relate to aspiration especially in bilateral stroke.¹⁸ Whether previous contralateral subclinical lesions impair the ability to compensate for a new lesion causing dysphagia is unclear: age related local factors (for example, dentures, poor oral hygiene, pharyngeal/laryngeal disease, cervical osteophytosis), may also reduce the ability to compensate for dysphagia in the elderly.^{19,20}

Bilateral upper motor neuron lesions commonly cause dysphagia, usually associated with a brisk jaw jerk, dysarthria and emotional lability (pseudobulbar palsy). Tongue movements are usually strikingly slowed with impairment of facial and lip control: movements of the palate and pharynx on phonation are very variable but often reduced whilst direct pharyngeal stimulation may elicit a brisk response, or no response at all. The swallowing reflex may be delayed or even absent even though a "gag

reflex" can be elicited emphasising the unreliability of the latter test. Cerebellar pathway and extrapyramidal disorders²¹ frequently disrupt swallowing although other impairments are usually more prominent and the incidence of aspiration related symptoms less clear. In medullary disease the tongue may deviate to the side of the lesion, while the palate and uvula deviate away from the side of the lesion on phonation. Palatal and pharyngeal sensation and reflexes may be reduced, the speech dysarthrophic and the larynx incompetent with impaired cough; occasionally there is concomitant disturbance of respiration with loss of metabolic control and apnoea.²² Respiratory patterns associated with swallowing change both with age and in the dysphagic patient.

The clinical impact of dysphagia depends on its severity, its speed of onset and the state of the respiratory system. A very gradual onset over a period of years permits numerous adaptive and compensatory mechanisms to be introduced and nutrition maintained. Whilst fully alert patients with intact respiratory function, a normal vital capacity and strong cough may be able to tolerate acute mild or moderate dysphagia many neurological disorders especially in the elderly^{23,24} make aspiration and its consequences more likely by impairing compensatory mechanisms. Specifically, there may be a depressed level of consciousness, weak respiratory muscles and hence impaired cough, previous pulmonary disease, splinting or irritation of the pharynx and larynx by naso- or oro-tracheal intubation, tracheostomy or feeding tubes. Local secondary factors such as oral candidiasis, ill fitting dentures, pharyngitis or trauma to the larynx from intubation, oesophageal disorders, reduced salivation (notably due to drugs) or abnormal head posture may all serve to inhibit residual swallowing further and promote aspiration. Incidental non neurological causes of dysphagia should be excluded in all cases. Patients with conditions such as myotonic dystrophy, myasthenia gravis, Guillain-Barré syndrome, poliomyelitis, motor neuron disease, multiple sclerosis, brainstem vascular disease and structural disease of the medulla and upper cervical cord such as tumours, Chiari malformations and syrinx where bulbar and respiratory dysfunction may coexist are thus peculiarly susceptible to the occurrence and consequences of dysphagia. It is probably no exaggeration that any "chest infection" in these patients may be due to aspiration until proved otherwise. Complete assessment of swallowing thus implies an assessment of the respiratory system.

The swallowing mechanism has considerable reserve capacity which is used before frank decompensation (aspiration and inability to obtain adequate nutrition and fluid) occurs. In this connection a simple timed test of swallowing capacity may have some merit for the routine monitoring and screening of potentially at risk patients before the stage of overt severe dysphagic symptoms.^{25,26} Food residues in the mouth or secretions pooled in the oropharynx are obvious features of dysphagia whether due to akinesia of swallowing (as in Parkinsonism) or facial/bulbar muscular weakness. Whether or not drooling occurs will depend on posture: thus in the erect position the oral cavity may empty by drooling whereas in the supine or unconscious patient secretions can only escape into the pharynx, oesophagus, the trachea or into a suction machine. Coughing or choking after swallowing suggests laryngeal penetration and nasal regurgitation implies palatal weakness. However, more subtle symptoms and signs often precede such obvious features. Weight loss due to reduced nutrition may precede the complaint of dysphagia. The patient may require increasing time for oral preparation of the bolus before swallowing and eat smaller mouthfuls, may avoid certain foods and drinks or need a drink to help "wash" food down. Other features may

include a reduced rate of spontaneous swallowing, nodding movements of the head or extension of the neck to project the mouth contents into the pharynx, and sideways turning of the head to close off the lateral food channel on the functionally impaired side, sometimes with head tilting to the less impaired side. The breath may be held while swallowing, followed immediately by releasing the breath into a cough to expectorate any material threatening the larynx. More than one swallow may be utilised to empty the oral cavity. Sleep is a vulnerable time for aspiration even in the healthy²⁷ and recurrent waking due to coughing should be noted. If there has been laryngeal penetration during a swallow or spill over from the valleculae or pyriform fossae the voice quality may be altered and have a "wet hoarse" character in addition to any dysphonia or dysarthria normally present. The patient at risk of aspirating may have difficulty modulating the volume of the voice, coordinating breathing and talking. The extent and rapidity of tongue movement should be noted together with any facial weakness and it may be helpful to palpate the hyoid bone during a swallow to assess movement.

Finally, recurrent chest infections or inhalation of food items may be the presenting feature of aspiration without specific symptoms or signs pointing to neurogenic dysphagia. Aspiration can thus be clinically silent^{28,29} and not associated with coughing and spluttering even in disorders which are presumed to affect only motor function; possibly recurrent aspiration, infection and instrumentation desensitise the upper airway and elevate the cough threshold. It is therefore evident that observation of swallowing judged in the context of respiratory function is far more important in determining the degree of dysphagia and the threat from aspiration than the specific examination of individual cranial nerve function which is pre-eminent for diagnosis. The presence of a palatal, gag or pharyngeal reflex (which is not in any case functionally equivalent to a swallowing reflex) is no guarantee of a safe swallow and its absence is not a reliable indicator of an impaired swallow. If silent aspiration is considered likely the possibility can be pursued with videofluoroscopy under supervision.

Assessment is more difficult if the patient is intubated with a cuffed oro/naso-tracheal tube or has a tracheostomy. The intubated supine semi-sedated patient in ITU cannot be expected to swallow well and some pooling of secretions is usual. A tracheostomy especially with an inflated cuff inhibits upward movement of the larynx during the swallow, may cause some backward pressure on the upper oesophagus and impairs coughing; in addition the lack of normal airflow through the larynx and mechanical desensitisation due to the tube and cuff may actually promote aspiration. The cuff is thus best deflated to assess swallowing. In addition, the long term need for a tracheostomy should be carefully reviewed if it is not required for ventilation but primarily for managing the secondary effects of aspiration.³⁰⁻³² Sedation then is minimised, the patient is sat up in a comfortable position for swallowing and a clear explanation given as to what is required. If the patient appears to control oral secretions and spontaneously swallows them the next stage can be to give small quantities of water or even thickened liquid feed and gradually increase volume. If, however, doubts remain a few drops of a dye such as Evans or methylene blue (some use black-current juice) can be placed on the tongue every few hours to determine whether this emerges in the tracheal aspirate. Finally, videofluoroscopy by an experienced radiologist with a speech therapist and physiotherapist in attendance and utilising different textures of bolus may be helpful although such a study only represents a very limited "snapshot" of the patient's overall swallowing ability and cannot always be relied on to be representative. As

previously noted, the decision to allow the patient to start eating solids or fluids, especially after a long period of prohibition, should depend not only on the swallowing but on respiratory function. If vital capacity is very low the swallowing will need to be judged that much safer than if the vital capacity is normal. Supervision in the early stages of refeeding by a speech therapist in collaboration with a dietician to modify the food consistency and texture seems to help progress and overcomes some of the patient's anxiety about choking.

Patients with significant dysphagia should only be fed by mouth if it is clear that they are not aspirating, yet it is not unusual to see a confused patient with a stroke, semi-recumbent in bed, being "encouraged" to take fluids by mouth while coughing and spluttering due to their "chest infection" for which they may be receiving vigorous physiotherapy! If there is doubt, oral feeding must cease and a fine bore nasogastric tube should be inserted. Modification of the diet texture and swallowing procedure on the advice of the speech therapist may allow oral feeding to start once the patient has been assessed; the alternative is to wait and reassess after a few days.

Cricopharyngeal myotomy³³ may permit the continuation of oral feeding if videofluoroscopy shows hypopharyngeal pooling with failure of relaxation of the cricopharyngeal sphincter.³⁴ The procedure is pointless if the cause of dysphagia is essentially at the oral or early pharyngeal phase. If long term tube feeding is necessary some patients will be unhappy with a nasogastric tube for reasons of appearance and convenience and an endoscopically performed feeding gastrostomy may be carried out without much risk to the patient: alternatives such as cervical oesophagostomy or jejunostomy are less often performed. The relief to some patients of giving up the daily struggle with eating and drinking is striking.

Most dysphagic patients with reasonable respiratory function, once fed by tube, can adequately protect their airway from their own secretions with postural advice and the use of suction devices. As previously noted tracheostomy per se (and a minitracheostomy more so) may worsen residual swallowing but may make it easier to cope with the consequences of aspiration. Alternative surgical measures to protect from aspiration such as laryngeal suspension, closure or diversion are available.^{35 36} The appropriateness of such procedures needs to be critically considered against the immediate cause of the dysphagia, the ventilatory state, the diagnosis and prognosis, and the consequences for the patient of further impairment or loss of speech from any surgery.²²

In summary, dysphagia and/or aspiration cause disability in a wide range of acute and chronic neurological diseases and have probably been under-recognised as causes of morbidity and mortality in the past. Recent research has highlighted much detailed information about the stages of swallowing and their assessment. The consequences of dysphagia must be evaluated bearing in mind respiratory function. Effective management is best carried out in the context of a multidisciplinary group which may include at various stages the neurologist, speech therapist,

dietician, radiologist and ENT surgeon.

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