Disorders of swallowing

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Abstract

Disorders of swallowing are very common and, when looked for, occur regularly in most branches of surgery. Dysphagia is often not the patient's presenting complaint and can be easily missed. The consequences of missed or delayed diagnosis of dysphagia can be insidious but profound and, in some cases, fatal. The investigation and treatment of these patients is normally highly multidisciplinary, potentially involving gastroenterology, general surgery, otolaryngology, acute medicine, stroke medicine, paediatrics, speech and language therapy (SLT) and dietitians. While this article is aimed at surgeons and will thus concentrate mostly on those conditions seen by surgeons, it must be remembered that the most common cause of dysphagia is a neurological disturbance and is managed by physicians and SLT. That said, the incidence of these conditions rises with age, as does the incidence of many surgically treatable conditions. It is therefore common to assess a patient with a known neurological condition for the presence of a second pathology affecting their swallow. A basic knowledge of non-surgical conditions is therefore useful.

Keywords Dysphagia; food bolus impaction; pharyngeal pouch; reflux; squamous cell carcinoma; swallowing

Overview of treatment

The complex physiological coordination required for a successful swallow is mirrored by the need for careful organizational coordination required for the successful treatment of dysphagia. This is a condition best managed within a multidisciplinary team (MDT) so that the diagnostic and therapeutic expertise of the different team members mentioned above can be promptly accessed.

There are a large number of possible therapeutic options, from simple head positioning during swallowing and dietary modification, through gastrostomy tubes to parenteral nutrition and extensive head and neck surgery. No one professional will be an expert in all of these, so the MDT is vitally important.

Complications

Most complications of dysphagia are not acute emergencies, but they can be fatal and are even more dangerous because of their insidious onset. The most common complications are:

- dehydration
- malnutrition
- aspiration pneumonia.

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Malnutrition is a covert problem but has a significant effect on wound healing and may delay discharge from hospital.[1]

Problems with swallowing will also impact on administration of medication, leading to non-compliance with tablets/capsule medication, or the requirement of more expensive liquid formulations. In the acute situation, occlusion of the upper aerodigestive tract due to, for example, a food bolus/foreign object or deep space neck infection can lead to airway obstruction.

The social impact of dysphagia is easily missed or underestimated, with some sufferers unable to or choosing not to participate in social interactions involving food due to embarrassment or anxiety.

History

The chronicity or duration of symptoms is important — for example, a steady progression of symptoms in an elderly patient, initially involving solids then liquids, signifies malignancy until proven otherwise. In contrast, motility problems of swallowing tend to involve both solids and liquids from the start.

It is useful to determine the frequency of the problem: for example, patients with intermittent dysphagia are more likely to have a functional disorder such as diffuse oesophageal spasm or non-specific oesophageal motility disorder (NOMD).

If regurgitation is present, the volume and time delay from initiation of swallow to the onset of regurgitation may indicate the level of obstruction. For example, obstruction of the oesophagus is associated with a larger volume of regurgitation and a longer delay compared to obstruction at the level of the oropharynx or hypopharynx.

Coughing and choking associated with food or liquid intake may signify aspiration of food or drink into the trachea. A history of frequent chest infections would reinforce this. This can be a fatal complication, particularly in frail patients.

Determining the level of the dysfunction whereby the patient localizes the symptoms is not reliable.[2] In one study, up to 59% of patients with pharynx-localized dysphagia were found to have oesophageal disease distal to the level of perceived symptoms.[3] Patients presenting with dysphagia with a differential diagnosis of malignancy should therefore have direct visualization of the full oesophagus and the gastro-oesophageal junction (Box 1).

Examination

Physical examination should include direct visual inspection of the upper aerodigestive tract, with the use of adjuncts such as

Red flag symptoms suggestive of malignancy

Weight loss (>5% unintentional weight loss in 6 months)
Haemoptysis
Odynophagia with lateralizing symptoms
Hoarseness
Otalgia (referred pain)
Change of consistency of diet
Symptoms of lymphoma (fever, weight loss, night sweats)
Risk factors: smoking, alcohol intake, family history, previous malignancy

Box 1

endoscopic visualization of the hypopharynx, larynx and oesophagus.

The oral cavity should be inspected to assess the state of the dentition, mucosal dryness, mucosal lesions, adequacy of lip closure, tongue fasciculations and palatal elevation. Next, the oropharynx should be visualized for lesions of the tonsil or tongue base. Cranial nerve examination is important in the evaluation of a patient with a suspected neuromuscular cause for dysphagia. The neck should also be examined for cervical lymphadenopathy, goitre or cervical borborygmi (Boyce's sign in pharyngeal pouch).

Additionally, a bedside swallow test with sip of water or swallow of solid food may help in evaluation of the actual process of swallowing. The patient should be able to hold the bolus in their mouth without dribbling and then move it smoothly to their pharynx in one go and swallow it without coughing or regurgitation. If they need multiple swallowing efforts for a small bolus, must throat-clear/cough or they bring the bolus back then further investigation is needed.

Investigations

There are many investigations that examine the upper aerodigestive tract (UADT) in different ways.

Which investigation to order is critically dependent upon the question to be answered.

Clinic investigations

Flexible nasendoscopy (FNE): The next step up is an FNE, which is available in all ENT departments. It allows direct visualization of the nasopharynx, oropharynx and most of the hypopharynx, but crucially not the post cricoid region.

Functional endoscopic evaluation of swallowing (FEES): This is an extension of FNE normally performed with SLT. Various textures (commonly liquid, yogurt and cake) are given to the patient and directly observed as they pass through the oropharynx and hypopharynx. Penetration and aspiration can be clearly seen, as can the patient's response to this. The absence of a cough reflex in response to penetration or aspiration is clearly a significant risk factor for aspiration pneumonia.

Radiological

Contrast swallow: This is a commonly ordered test with limited usefulness. Its main value is in assessing for a pharyngeal pouch.

CT/MRI: Cross-sectional imaging is the gold standard for assessment of significant anatomical derangement of the UADT, but always remember that smaller mucosal lesions that may be obvious on direct inspection may be completely invisible on CT/MRI.

Video fluoroscopy (VF) is a fluoroscopic assessment by SLT and radiology of swallowing various textures that have been soaked in contrast medium. It gives a very thorough assessment of the passage of food/drink through the whole UADT but is relatively resource intensive.

pH manometry

This procedure involves the placement of a probe similar to a nasogastric tube into a patient's oesophagus. The pressure and pH at various levels can then be recorded, allowing assessment of reflux as well as objective assessment of oesophageal dysmotility.

Oesophagoscopy

Transnasal oesophagoscopy (TNO): This is a relatively new technique that allows passage of a flexible nasendoscope down the entire oesophagus under local anaesthetic only, as a clinic procedure. It allows a limited assessment of the hypopharynx, but evidence shows that this is enough to exclude post cricoid carcinoma.[4] It also includes an FNE as a standard part of the procedure.

Oesophago-gastro duodenoscopy (OGD): This procedure examines the oesophagus very well, but the transoral passage of a large scope reduces the view in the pharynx. Large lesions can be noticed but not assessed, and small lesions may be missed altogether. It normally requires sedation and thus may not be suitable for frail patients.

Rigid pharyngo-oesophagoscopy: This allows excellent examination and treatment of many UADT lesions but requires a general anaesthetic. There are significant risks of dental damage and oesophageal perforation, particularly if used to visualize the lower oesophagus. It can be used to extract some sharp foreign bodies as it protects the oesophagus during their extraction (Table 1).

Food bolus impaction

Food bolus or foreign body impaction in the digestive tract can occur at any age and because of almost any cause of dysphagia. It is best viewed as a symptom of an underlying problem rather than a diagnosis in its own right. It usually presents with complete aphagia and resultant drooling of saliva. If not treated appropriately, dehydration happens within 24–48 hours. The nature of the foreign body is extremely important. Sharp or caustic objects (e.g. batteries) require immediate removal. Most food boluses in the UK are meat and can initially be managed conservatively with IV rehydration and muscle relaxants such as hyoscine butylbromide 20 mg IV or glucagon 1 mg IV. Neither of these is clearly better than placebo and the important point is waiting to see if the bolus spontaneously clears over a few hours. This occurs in around 50% of patients. If it does not, the safest treatment is retrieval of the foreign body or pushing it more distally into the stomach with a flexible endoscope (OGD or TNO). Occasionally, rigid oesophagoscopy may be required for sharp or solidly impacted boluses. Once the bolus has been cleared, it is important to assess the need for further investigation of the patient.

Causes of dysphagia

There are distinct causes or associations with dysphagia depending on patient age. The causes of dysphagia in infancy, childhood and adolescence include congenital causes, acute infectious causes, injury and neurodevelopmental delay. Gastroenterological and immunological causes start to manifest in middle age, whereas neurological and oncological causes become more frequent in the elderly.[5] The prevalence of dysphagia in the

Relative strengths of investigations for dysphagia

		Examination in clinic		Radiology			pH Manometry	Oesophagoscopy		
		FNE	FEES with SLT	Contrast swallow	CT/MRI	VF		TNO	OGD	Rigid under GA
Function		+	+++	++		++++	++++	+	+	
Anatomy	Pharynx	+++	+++	+	++++	+		+++	+	+++
	Hypopharynx	+	+	++	++++	++		+++	+	++++
	Oesophagus			++	++++	++		+++	+++	++
Biopsy								++	+++	++++
Reflux		+		+		+	++++	+	+	+
Pharyngeal pouch				+++	+	+++	+	+	+	++++

Table 1

general population has been found to range from 1.7% to 11.3% in various studies. It most commonly affects the elderly population, in which oropharyngeal dysphagia is associated with muscle atrophy, cognitive decline and increased aspiration risk in as many as 35% of patients older than 75 years.[6] (Table 2).

Neurogenic

As well as focal neurological conditions affecting the UADT, any global or central neurological condition may present with dysphagia. The most common of these is stroke. The age-adjusted incidence of stroke is about 0.12% and there are around 1.2 million stroke survivors in the UK. The prevalence of dysphagia following stroke varies with time; on diagnosis it is around 50%, dropping to 25% at 7 days and 6% at 6 months.[7] Other neurological conditions with a high incidence of dysphagia include head injury (29%), Parkinson's disease (50%), multiple sclerosis (33%) and dementia (35%).

Autoimmune

Sjögren's syndrome has a strong association with dysphagia. It should be suspected if the patient reports dryness of the mucus membranes with no other obvious cause. There is no single definitive diagnostic test but the combination of history, antinuclear antibodies and a lip/salivary gland biopsy is normally undertaken. Treatment is generally symptomatic.

Severe rheumatoid arthritis may also disturb swallowing, as can the thickened collagen deposited in tissues in scleroderma.

Infectious

These conditions usually present with pain and odynophagia. Conditions involving the upper aerodigestive tract include quinsy, supraglottitis and deep space neck infection. These are usually diagnosed clinically and managed by otolaryngology in the secondary care setting. A full explanation of investigation and management of these is beyond the scope of this article.

Infective oesophagitis is usually associated with candidiasis; immune compromise is the most important risk factor. Definitive diagnosis requires biopsy for microbiologic and histologic evaluation; treatment is based on severity of infection and host immune factors.

Iatrogenic causes

The dynamic nature of swallowing requires mobile anatomy. Laryngotracheal elevation and pharyngeal contracture occur during a normal swallow; limitation or impairment of mobility leads to dysphagia. The presence of a tracheostomy, scarring, fibrosis or mucosal trauma from ingestion of caustic materials can impede normal movement and result in dysphagia.

Structural

Mucosal-reflux: Any cause of oesophagitis can lead to dysphagia. The most common of these is gastro-oesophageal reflux disease (GORD). This can often be diagnosed on history and examination alone, but oesophagoscopy is often performed to exclude any malignant or premalignant conditions. The gold standard is pH monitoring. Treatment is with a combination of:

- Lifestyle changes – including weight loss, smoking cessation and raising the head of the bed. Dietary modification is only helpful if the patient knows specific foods to be trigger factors.
- Medication – proton pump inhibitors are the most effective treatment for GORD if indicated, particularly if used twice daily, but they should be maintained on the lowest clinically effective dose. H_2 antagonists may also help, as can antacids combined with alginate. The literature on laryngopharyngeal reflux (LPR) is less clear and alginates may be more effective than PPIs for this condition.
- Surgery – is recommended for patients who do not improve with medical management. The standard treatment is a laparoscopic Nissen fundoplication, in which the gastric fundus is wrapped around the lower oesophageal sphincter. This reinforces the action of the lower oesophageal sphincter, reducing reflux. The oesophageal hiatus in the diaphragm is also narrowed to prevent a sliding hiatus hernia. Partial fundoplication is generally safer than the full 360° wrap described by Nissen, and posterior wrapping seems more effective than anterior.

Intramural – malignancy

Oral cavity – In addition to dysphagia, symptoms and signs associated with oropharyngeal malignancy include lymphadenopathy, odynophagia, otalgia and change in voice.[8] In contrast to oropharyngeal and hypopharyngeal tumours, patients with oral cancers do not usually get problems with the pharyngealty phase of swallowing.

Oropharyngeal – These do not typically present with dysphagia initially. Patients may complain more of discomfort or

Causes of dysphagia

Neurogenic/myopathic	Stroke, Parkinson's disease, Alzheimer's disease, multiple sclerosis, traumatic brain injury, cerebral palsy, Inflammatory myopathies, myasthenia gravis
Autoimmune	Sjögren's syndrome, Rheumatoid arthritis, scleroderma/CREST syndrome, Crohn's, Behcet's, pemphigoid
Infectious	Pharyngitis, tonsillitis, peritonsillar abscess, supraglottitis, deep space neck infection, oesophagitis
Iatrogenic	Post-surgical (head and neck surgery, tracheostomy, cervical spine surgery, surgery for reflux e.g. Nissen fundoplication) Side-effect of chemotherapy/radiotherapy Side-effect of medication (anticholinergics, antispasmodics, antipsychotics, antiparkinsonian medication, diuretics, botulinum toxin, leading to mucosal dryness, problems with bolus manipulation, tardive dyskinesia, muscle paralysis)
Trauma	Blunt and penetrating trauma, cranial nerve injury
Structural	Mucosal • Heterotopic gastric mucosa • Gastro-oesophageal reflux Intramural • Malignancy (oropharyngeal, hypopharyngeal, laryngeal, oesophageal, thyroid) • Eosinophilic oesophagitis • Benign stricture (related to reflux, radiotherapy, long-term sequela of caustic ingestion, CREST syndrome) • Pharyngeal pouch, cricopharyngeal bar or cricopharyngeal dysfunction • Pharyngeal/oesophageal web Compression from extrinsic structures • Mediastinal lymphadenopathy • Large goitre • Aortic aneurysm • Osteophytes
Congenital	Prematurity, oesophageal stenosis/atresia, mitochondrial disease, cleft lip and palate, laryngeal cleft, vascular anomalies
Functional	Diffuse oesophageal spasm, achalasia, non-specific oesophageal motility disorder
Other	Xerostomia (salivary gland disease), psychogenic factors

Table 2

difficulty when chewing, perhaps trismus, or discovery of a lesion/ulcer in the mucosa of the oral cavity. Nonetheless, these problems certainly impact on the oral phase of swallowing; even the pharyngeal phase can be affected if the base of the tongue is involved. Most cases of oropharyngeal malignancy are squamous cell carcinoma.

Hypopharynx — This is almost invariably squamous cell carcinoma (SCC) and often presents late, with an advanced tumour and thus has a poor prognosis. Early stage disease has a 5-year survival around 40–50% and advanced disease is approximately 20%. The cardinal symptom is of progressive dysphagia initially for solids then liquids, and significant weight loss. It can present with a myriad of different symptoms and requires at least a flexible laryngoscopy and sometimes a pharyngoscopy to diagnose. Pooling of saliva in the pyriform fossa can indicate a hidden postcricoid lesion, as can a vocal cord palsy with no obvious laryngeal cause. Investigation is with rigid pharyngo-oesophagoscopy, biopsy and cross-sectional imaging. Treatment is managed within a large multidisciplinary team. Curative options are surgery and radiotherapy, with early lesions being treated with a single modality and advanced lesions receiving both, often with adjuvant chemotherapy as well. Surgery is often a total pharyngolaryngectomy and free flap reconstruction, but partial pharyngectomy may be possible in early stage disease.

Oesophagus — Unlike the remainder of the UADT this can be either SCC (normally in the upper two-thirds of the oesophagus) or adenocarcinoma from the submucosal glandular tissue in the lower one-third. Adenocarcinoma often develops from lower oesophageal cells that have undergone gastric metaplasia as a result of reflux and become an intestinal cell type (Barrett's oesophagus). Diagnosis is by endoscopy and patients known to have Barrett's should undergo regular screening endoscopy. Somewhat paradoxically *Helicobacter pylori* infection is somewhat protective for adenocarcinoma, despite being a risk factor for gastritis. Management is within an MDT. Localized small SCCs can be endoscopically excised with curative intent, but beyond this partial or total oesophagectomy may be required depending on the size and extent of the lesion. Radical radiotherapy may be used if the patient is unsuitable for surgery. For palliative cases, a self-expanding oesophageal stent may be placed to control stenosis or to close off fistulae. The overall prognosis is poor with a 5-year survival of 15%.

Larynx — Large laryngeal tumours, particularly of the supraglottis, can invade the pharynx and cause dysphagia. This is an

unusual presenting symptom and is normally only seen in combination with advanced hoarseness and/or stridor.

Effects of treatment for malignancy – In oral/oropharyngeal cancer, dysphagia is caused by extensive tissue loss, limited excursion of the remaining tissue, and sensory paralysis of the tongue, soft palate and pharynx after surgical resection or radiotherapeutic treatment of the primary site. Swallowing is affected by the degree of resection required, and the nature of the reconstruction. A degree of aspiration may occur in patients undergoing hemi- or total glossectomy or extensive supraglottic surgery. Nasal leakage of food/drink can happen after palatal surgery. Pharyngolaryngectomy, once healed, prevents aspiration as the airway and pharynx are separated completely, but there is a loss of propulsion through the pharynx and strictures can occur at the anastomosis to the oesophagus. Chemotherapy and radiotherapy for advanced disease can cause scarring and fixation of the pharynx and larynx, severe narrowing, or even complete obliteration of the hypopharyngeal lumen, severe xerostomia and dysgeusia. These are often the patient's main complaints after curative treatment.

Intramural – eosinophilic oesophagitis: This is an allergic response within the oesophageal mucosa. Patients present with intermittent dysphagia often with partial food bolus obstruction. The underlying cause is unclear, but it is thought to be related to food allergies. The diagnosis is made histologically on oesophageal biopsies, but the classical ridged appearance of multiple concentric rings (a trachealized oesophagus) may be seen at endoscopy or on contrast radiography. This condition often responds to a PPI but may also require a topical steroid, such as fluticasone or budesonide taken with a deliberately poor inhaler technique with no respiratory effort and then swallowed. Severe cases may require dilatation.

Intramural – cricopharyngeal dysfunction/spasm: Failure of relaxation of the cricopharyngeal muscle can lead to elevated bolus transit times and increased food residue in the pyriform fossae. Apart from swallowing, this may lead to penetration of food material into the larynx with subsequent aspiration.

A cricopharyngeal bar is a common incidental finding during radiological examination of the UADT. This finding may be associated with dysphagia, but is not always the cause of the swallowing difficulty and is not related to functional failure of relaxation.

Cricopharyngeal dysfunction can be successfully diagnosed and treated with a trial of botulinum toxin injection. However, as paralysis from the toxin is temporary, repeated injections may be needed. Alternative strategies such as endoscopic balloon dilatation of the cricopharyngeus may be performed, or a definitive procedure such as cricopharyngeal myotomy (open or endoscopic), depending on the suitability of the patient for the operation.

Intramural – pharyngeal pouch (Zenker's diverticulum): This is an outpouching of the posterior oesophageal mucosa just above the cricopharyngeus (upper oesophageal sphincter). It is thought to occur as a result of failure of the cricopharyngeus to relax when a food bolus is propelled into the hypopharynx. There is a natural weakness at this point between the lower fibres of the inferior pharyngeal constrictor and the cricopharyngeus (Killian's dehiscence, Figure 1). The pressure from the food bolus therefore causes the mucosa to progressively bulge out at this point and eventually form a pouch which can trap food (Figures 2 and 3). This leads to the classic symptoms of a pharyngeal pouch: dysphagia, regurgitation of undigested food some time after eating, halitosis and sometimes a cough as the pouch contents spill over into the larynx. It is important to establish the degree of impairment of the patient and to balance this against their fitness for anaesthesia. The best investigation is a contrast swallow to assess the size of the pouch.

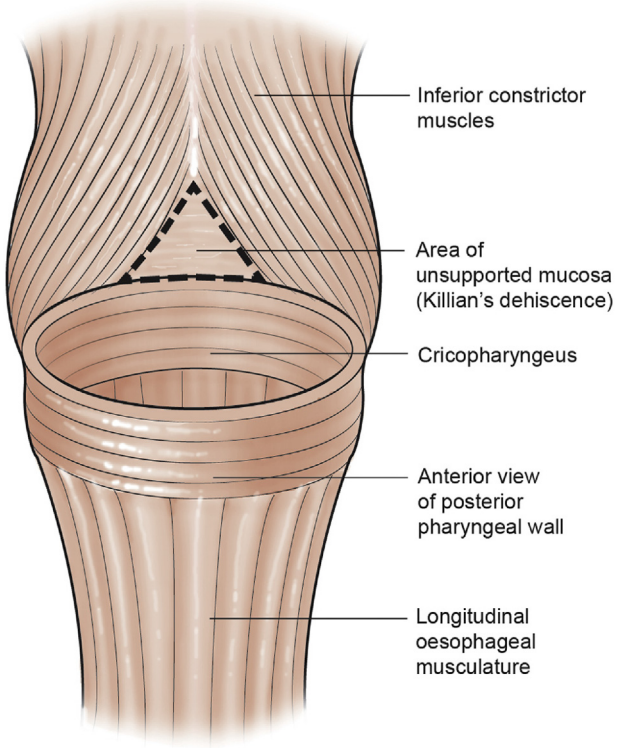

Figure 1 Anterior view of posterior hypopharyngeal wall.

The pathogenesis of this condition means that treatment must address not only food being trapped in the pouch but the overactive cricopharyngeus as well. The treatment options are:

- Watchful waiting – appropriate for very frail patients or those with minimal symptoms.
- Endoscopic pouch stapling – performed transorally under general anaesthetic, the blades of an endoscopic staple gun are placed in the oesophagus and the pouch. The anterior pouch wall is thus divided between two rows of staples. As shown in Figure 2e, the posterior part of the cricopharyngeus forms the superior lip of the pouch's anterior wall, thus the cricopharyngeus is also divided. Very small pouches (Figure 2c or d) are not treatable like this as the pouch is too small for placement of the staple gun. This procedure is only possible if the area can be seen with a rigid pharyngoscope. This is not always possible where there is cervical spondylosis or retrognathia.
- External cricopharyngeal myotomy and pouch excision – the pouch can be excised (and subjected to histological

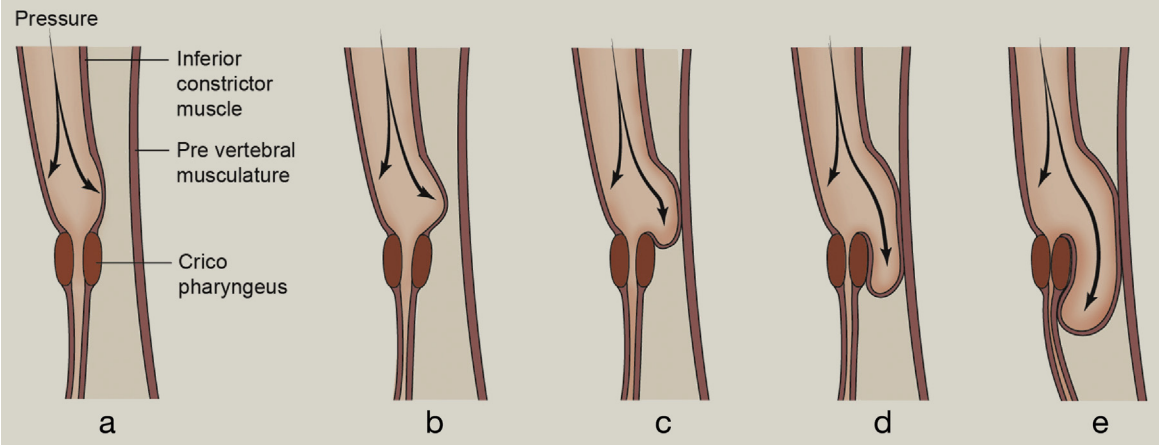

Figure 2 Lateral view of hypopharynx showing progressive enlargement of pharyngeal pouch with time.

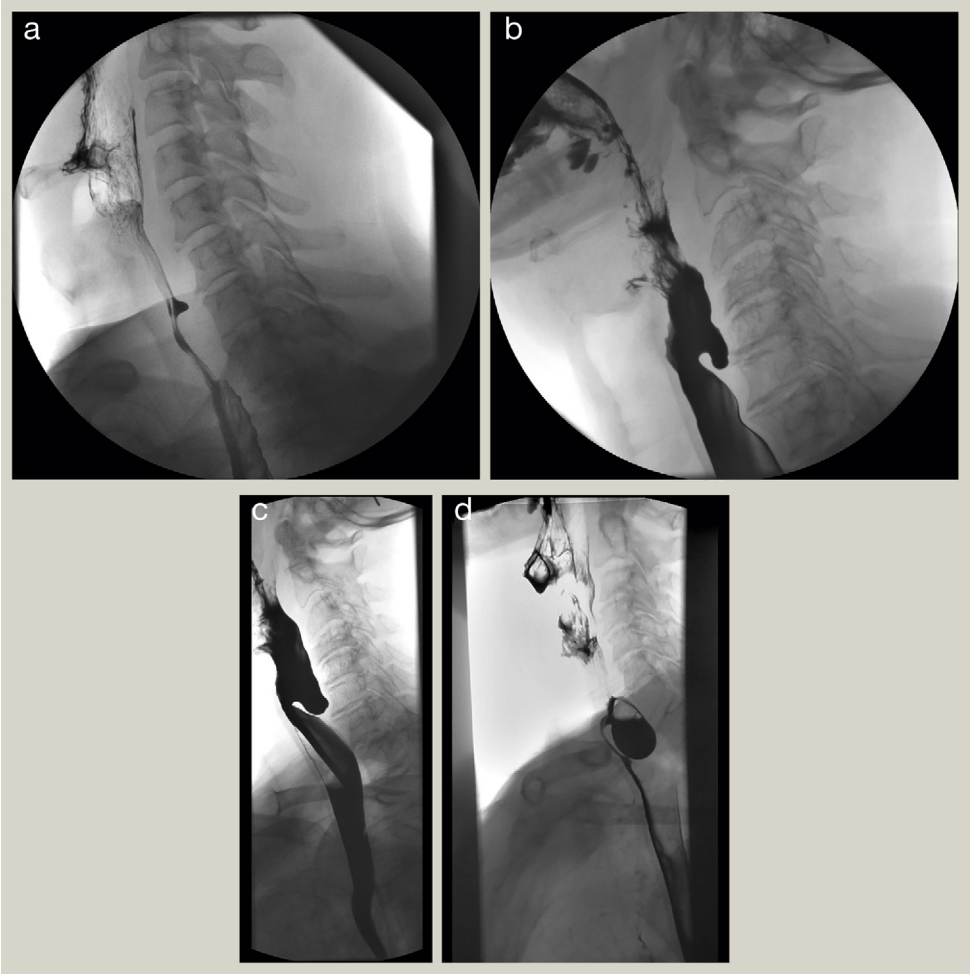

Figure 3 Contrast swallow showing progressive growth of pharyngeal pouch. Note constricted oesophageal lumen in d.

analysis) and the cricopharyngeus divided via an anterolateral approach through the neck. This carries a much higher risk of postoperative leak and requires a much longer stay in hospital.

- Botulinum toxin injection – Under general anaesthetic or electromyographic guidance, this is a temporary (4–6 months) but safe treatment option and can also be used for cricopharyngeal spasm.

Intramural – oesophageal atresia: This is a rare congenital condition with an incidence of 2.5/10000 live births.[9] The oesophagus fails to develop fully and there is a fistula into the

trachea. The most common variant by far is a fistula from the lower segment, but any variant is possible, including two fistulae, or a fistula with a patent oesophagus. Treatment is usually by open surgery, although non-invasive techniques are being developed for cases with a small gap between the segments.

Intramural – oesophageal webs and rings: Webs are most common in Patterson-Brown-Kelly (Plummer-Vinson) syndrome, a rare combination of dysphagia, iron deficiency anaemia, glossitis, angular cheilitis and oesophageal webs. The cause is unclear, but oral iron replacement and mechanical dilatation of the oesophagus normally control the problem well. Ten per cent of patients develop oesophageal cancer so long-term monitoring is essential.

They can also occur in coeliac disease and bullous diseases (e.g. epidermolysis bullosa). Congenital webs are rarer and usually more inferiorly placed. All of these respond well to dilation.

Intramural – oesophageal achalasia: Failure of the lower oesophageal smooth muscle (including the lower oesophageal sphincter) to relax eventually leads to massive dilatation of the oesophagus and regurgitation of undigested food. There is associated chest discomfort and weight loss. Oesophageal manometry is the gold standard test, but contrast radiography may show a typical 'rat's tail' appearance of the lower oesophagus and oesophagoscopy may demonstrate the dilatation. The underlying pathology is loss of the noradrenergic inhibitory cells in the myenteric plexus, but why these cells should be lost is currently unclear.

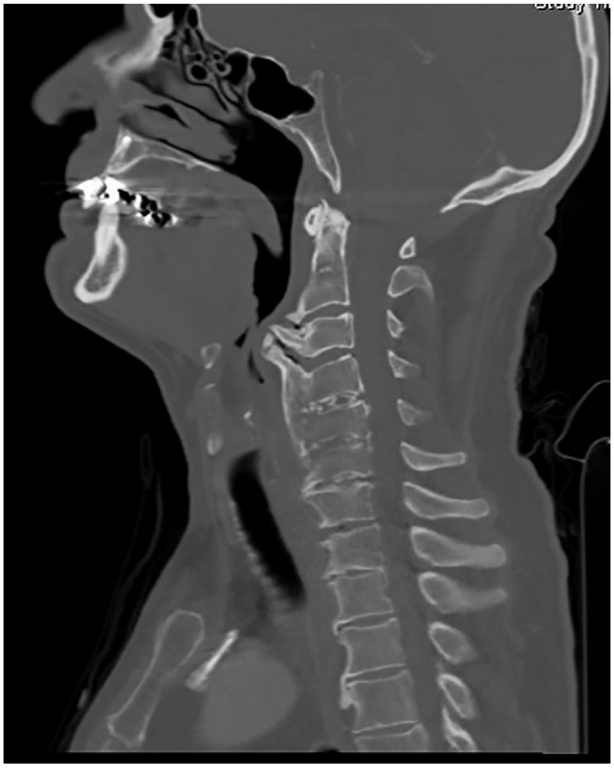

Figure 4 Anterior cervical osteophytes causing hypopharyngeal compression and dysphagia.

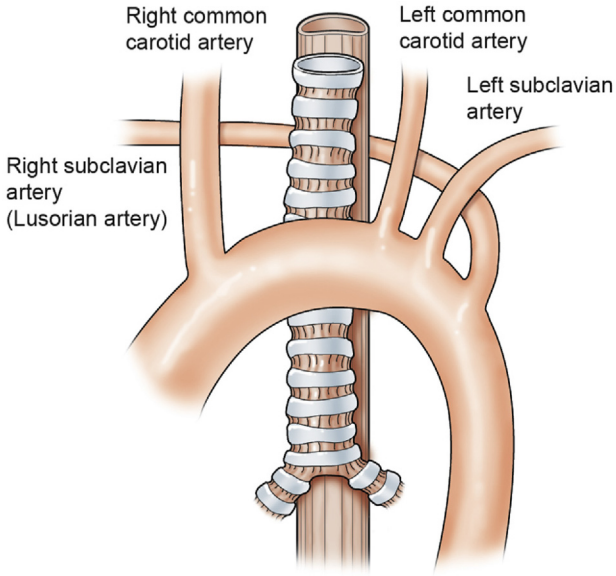

Figure 5 Aberrant right subclavian artery causing compression of the oesophagus.

Initial treatment is with calcium channel blockers such as nifedipine,[10] but if this fails either pneumatic dilatation or surgical myotomy are equally effective. Surgery can be laparoscopic (Heller's procedure) or endoscopic, but in both cases may need repeating after a few years.

Extramural – external compression: Aortic aneurysms, mediastinal lymphadenopathy, large bronchial primaries and intrathoracic goitres can all cause oesophageal dysphagia. Cervical osteophytes can grow large enough to impinge on the posterior hypopharynx or even divert food into the larynx, thus causing higher level symptoms (Figure 4).

Extramural – vascular anomalies: Several rare vascular anomalies have been described where one of the major vessels causes pressure on the oesophagus and thus dysphagia. The best described is dysphagia lusoria, where an aberrant right subclavian artery arises from the left of the aortic arch and then travels immediately posterior or anterior to the oesophagus as it crosses back over the midline (Figure 5). Diagnosis is best seen on contrast CT or MR angiography and treatment, when significantly symptomatic, is by ligating the origin of the aberrant vessel and reattaching it to a more anatomically normal position. ◆

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