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APPLIED SURGICAL ANATOMY AND PHYSIOLOGY

APPLIED SURGICAL ANATOMY AND PHYSIOLOGY

The oesophagus is a muscular tube connecting the pharynx to the stomach. It starts at the level of the cricoid cartilage (C6 vertebra) and ends at the oesophagogastric junction (OGJ) (opposite T11 thoracic vertebra). In the neck, it descends behind the trachea and anteriorly to the vertebral column following its curvature into the thorax. At the lower part of the thoracic cavity , it transverses the diaphragmatic hiatus, clasped by the crura of the diaphragm and into the stomach. There are slight curvatures of the oesophagus; in the neck, it leans slightly more towards the left side, while in the mid-chest it curves slightly towards the right and in the lower part of the thorax edges to the left again. Thus, in the neck exposure of the cervical oesophagus is generally easier from the left side. The recurrent laryngeal nerves lie in the tracheo-oesophageal grooves. On the right side, the recurrent laryngeal nerve at the lower neck is often at a small distance from the tracheo oesophageal groove, while on the left side the nerve is apposed closely to the oesophagus and trachea. This is an important anatomical detail when exposing the oesophagus and when cervical lymphadenectomy is performed. In perforation from Boerhaave’s syndrome , the perforation tends to a ff ect the lower oesophagus on the left side, as this part of the oesophagus is less well supported anatomically . There are multiple relative constrictions along the oesoph agus. First is the upper oesophageal sphincter (UOS); second is where the arch of the aorta/left main bronchus crosses the oesophagus; and third is the lower oesophageal sphincter (LOS). Foreign bodies such as food boluses tend to lodge at these sites . The UOS is a structural sphincter formed by a band of stri ated muscles of the inferior pharyngeal constrictor and crico pharyngeus. The LOS is much less well defined. It is a complex Hermann Boerhaave , 1668–1738, Professor of Medicine and Botany , the University of Leiden, The Netherlands. Friedrich Albert Zenker , 1825–1898, Professor of Pathology , Dresden, Germany . Norman Rupert Barrett , 1903–1979, surgeon, St Thomas’ Hospital, London, UK. interplay of the intrinsic muscle tone of the oesophageal wall, as well as the diaphragmatic crura. It is also variably exposed to the negative intrathoracic and positive intra-abdominal pressure. Thus, on oesophageal manometry , it is identified as a high-pressure zone; the diaphragmatic contribution is more obvious in the presence of a hiatus hernia, where two compo - nents of the high-pressure zone can be identified. Disorders of the coordination in UOS and pharyngeal function can result in dysphagia or aspiration symptoms. A common example is in elderly patients following a cerebrovascular accident or in those with neur ological diseases. Dysfunction of the UOS is also related to the pathogenesis of the pharyngeal pouch/upper oesophageal diverticulum (Zenker’s). Weakness of the LOS or inappropriate relaxation can result in oesophagogastric reflux disease (GORD), or tightness (incomplete relaxation) can lead to dysphagia, such as in achalasia. For clinical purposes, the oesophagus is divided into the cervical, the thoracic and the abdominal oesophagus (Figure - 66.1). These divisions are relevant for cancer staging purposes. The ‘cardia’ is the portion of the stomach that lies immediately belo w the

OGJ, but there is no clear definition of its extent. Tumours around this region are often referred to as cancer of the cardia; however, it may be better to refer to ‘cancer at the oesophagogastric junction’. Anatomically the OGJ is defined as the point where the tubular oesophagus becomes the saccular stomach; histologi - - cally it is the junction between the squamous mucosa and the columnar mucosa, but these levels will be di ff erent in Barrett’s oesophagus. The physiological LOS does not cor respond to the anatomical OGJ; the LOS is a high-pr essure zone defined physiologically but not anatomically . In practice, the most relevant definitions relate to findings on endoscopy . Endoscop - - ically the OGJ is defined as the top of the gastric folds (the - oesophagus is tubular and does not have folds while the stom - ach has rugae). In Japan, it is also commonly recognised as

The clinical features, investigations and treatment of •

the distal end of the oesophagus where palisading vessels are found (Figure 66.2). Which definition used is not so import ant; rather, it requires experience from the endoscopist to define this point accurately . In a real-life situation, the junction is somewhat dynamic with the patient’s breathing excursions; an excessive amount of air insu ffl ation may ‘flatten’ the gastric folds, making it di ffi cult to define their ‘top’. The presence of a is important in defining hiatus hernia, assessing the presence of Barrett’s oesophagus, defining cancers around the OGJ and tumour staging. Histologically the oesophageal wall has layers: mucosa, submucosa, muscularis propria and adventitia. The mucosa consists of a non-keratinised stratified squamous epithelium, the lamina propria and the muscularis mucosae. The oesoph - ageal wall lacks a serosa; it is the m uscularis mucosae and sub - mucosa that give it strength for suture holding. The muscularis propria has an inner circular muscle and external longitudinal muscle layer (Figure 66.3). The muscle of the upper third of the oesophagus is made up of striated muscle, the middle third with a mixture of striated and smooth muscle and the lower third with smooth muscle. Connective tissue disease, such as scleroderma, mainly a ff ects smooth muscle, hence the lower oesophagus. The blood supply of the upper oesophagus is derived from the superior and inferior thyroid arteries. The middle oesoph - agus receives its supply from direct branches of the aorta and bronchial and intercostal vessels. The distal oesophagus has the arterial supply from the left gastric, left inferior phrenic and splenic vessels. The blood supply is usually excellent, and a long length of the oesophagus can be mobilised without compromising perfusion. An anastomotic leak from oesopha - geal anastomosis after oesophagectomy is rarely attributed to poor blood supply of the oesophagus; rather, it is the conduit - that lacks perfusion. V enous return to the systemic circulation forms a network of vessels within the oesophageal wall. The y drain to the inferior thyroid, azygos, hemiazygos and gastric veins. The communications of oesophageal veins and left gas - tric veins form part of the portal–systemic anastomosis. Cir - rhosis leads to their dilatation (varices).

0 cm Cricopharyngeal 15 cm constriction Aortic and bronchial

25 cm constriction Diaphragmatic and ‘sphincter’ constriction 40 cm Figure 66.1 Anatomy of the oesophagus, divisions of the oesopha gus and measurements endoscopically from the incisors. The three relatively ‘narrow’ parts of the oesophagus are at the level of the cricopharyngeus muscle (upper oesophageal sphincter), where the left main bronchus and aorta cross the oesophagus and the oesoph agogastric junction (lower oesophageal sphincter). (a) Figure 66.2 (a) Endoscopic image showing the ‘top of the gastric

folds’ (black arrows), indicating that this is the oesophagogastric junction. (b) The distal end of the ‘palisading vessels’ also indicates the oesophagogastric junction (blue arrows pointing at the end of the palisading vessels).

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(b)

The lymphatic drainage of the oesophagus is important, especially for cancer spread. There is a rich plexus of lym phatics in the submucosa, and direct drainage to the thoracic duct is also demonstrated. Submucosal spread of cancer along the oesophagus proximally and distally is common, and a lon ger resection margin reduces the chance of local recurrence. In the neck, the cervical oesophagus drains to the deep cervi cal and paratracheal nodes. In the thoracic oesophagus, lym phatic spread from a tumour can travel widely; potentially , it can spread to the neck, mediastinum and the nodes around the coeliac axis. Although upper thir d tumours tend to spread upwards and lower oesophageal cancer distally , this can be unpredictable and skip lesions can occur. Of particular impor tance is lymph node spread along the bilateral recurrent laryn geal nerves. This should be treated as a continuum of lymph node chains along both nerves, and thus they traverse from the chest into the neck. This widespread lymph node spread is the rationale behind the concept of three-field lymphadenectomy , whereby lymph nodes in the cervical region, mediastinum and around the coeliac axis are treated as regional nodes and dis section is recommended in curative surgery . The data mostly come from studies on squamous cell cancers in Japan. The lack of su ffi cient data for adenocarcinomas (mostly in the lower oesophagus) makes the benefit of such extended lymphadenec tomy less certain. The thoracic duct is the largest lympha tic vessel in the body . It is formed from the abdominal confluence of the left and right lumbar lymph trunks, as well as the left and right intestinal lymph trunks between T12 and L2. The conflu ence of lymph trunks is saccular and is refer red to as cisterna chyli. Through the diaphragmatic hiatus, the thoracic duct is formed as it ascends along the aorta, next to the azygos vein and oesophagus. It then cr osses to the left side at T4–T6, going Rudolf Virchow , 1821–1902, pathologist, Charité Hospital, Berlin, Germany . Charles Émile Troisier , 1844–1919, pathologist,

University of Paris, Paris, France. the left neck, it drains into the junction between the left jugular vein and left subclavian vein. Around 75% of the lymph from the entire body (aside from the right upper limb, right breast, right lung and right side of the head and neck) passes through the thoracic duct. The cells of the immune system circulate through the lymphatic system. Also, large molecular products of digestion, such as fats, first need to be absorbed into the lym - phatic system, and then reach the systemic circulation through the venous system. During oesophagectomy , the thoracic duct and its tributar - ies may be damaged, postoperatively presenting as chylotho - rax. The thoracic duct may also be injured during cervical lymphadenectomy , leading to a chylous leak from the neck wound after surgery . Prolonged chylous drainage cannot be tolerated because there is loss not only of fluid and electrolytes but also important proteins as well as lymphocytes, which can - not be replaced. The path of drainage of lymph explains the finding of Virchow’s node (Troisier’s sign), when a metastatic node is found in the left supraclavicular fossa, both from intra- abdominal malignancies as well as from oesophageal cancer .

Strati /f_i ed squamous epithelium Lamina propria Muscularis mucosae Submucosa Muscularis propria: inner circular muscle layer Muscularis propria: outer longitudinal muscle layer Adventitia Figure 66.3 Histological layers of the oesophagus (courtesy of Dr Anthony Lo, Department of Pathology, Queen Mary Hospital, Hong Kong SAR, China).

Achalasia

Achalasia

Pathology and aetiology The term achalasia originated from the Greek word ‘khalasis’, meaning ‘failure to relax’. It is uncommon, with a prevalence of 1.8–12.6 per 100 000 persons per year. The aetiology Leopold Auerbach , 1828–1897, neuropathologist, Breslau, Germany (now Wroc ł aw , Poland). Carlos Chagas , 1879–1934, microbiologist, Rio de Janeiro, Brazil. Jeremy Allgrove , contemporary , paediatric endocrinologist, London, UK. Volker F Eckardt , b. 1942, gastroenterologist, Wiesbaden, Germany . inhibitory ganglion cells in the myenteric (Auerbach’s) plexus, possibly related to a virus-induced autoimmune e ff ect. Histol - ogy of m uscle specimens generally shows a reduction in the number of ganglion cells with a variable degree of chronic inflammation. During a normal swallow , the food bolus will trigger primar y peristalsis in the oesophagus by sequential activation of excitatory lower motor neurones. At the same time, relaxation of the LOS allows oesophageal emptying. The mismatch in excitatory and inhibitory activity results in the failure of LOS relaxation and absent peristalsis. With time, the oesophagus dilates and contractions disappear, so that the oesophagus empties mainly by the hydrostatic pressure of its contents. This is nearly always incomplete, leaving residual food and fluid. The air–fluid level in the stomach evidenced on radiography taken in the erect position in normal individuals is frequently absent, as no bolus with its accompanying air passes through the LOS. The oesophagus becomes progres - sively more tortuous and dilated (megaoesophagus); persistent retention oesophagitis due to fermentation of food residues - may predispose to the increased incidence of carcinoma of the oesophagus. In South America, chronic infection with the parasite Trypanosoma cruzi causes Chagas’ disease and the destruction of the myenteric plexus has marked clinical similarities to achalasia. A rare genetic syndrome (Allgrove syndrome) is associated with familial adrenal insu ffi ciency , alacrimia and achalasia. Clinical features The disease is most commonly diagnosed between 30 and 60 years of age. It typically presents with dysphagia (to both solid and liquid), regurgitation and heartburn (often mistaken for GORD), although chest pain/odynophagia is also common in the early stages. Patients often present late and, having had relatively mild symptoms, remain untreated for many years. Patients may or may not have experienced weight loss. Frequently , patients will adjust their diet according to symptoms and can maintain their body weight after an initial drop. An ‘Eckardt score’ was developed to assess the severity of symptoms and monitor treatment outcome (Table 66.1). - Aspiration-related respiratory symptoms and pneumonia can also occur when there is significant stasis of food residue in the dilated oesophagus. The retained food substance can cause fermentation and therefore halitosis. Patients may report regurgitating food that they have ingested before. Diagnosis A high index of suspicion is needed in the diagnosis of achalasia as symptoms can be mild and chronic and can be easily mis - diagnosed as GORD. Endoscopy typically shows frothy saliva pooling in the oesophagus and the presence of food residue. The oesophagus may be dilated and can be tortuous. T he OGJ appears tight and spastic but can usually allow an endoscope to pass with gentle pressure. A normal endoscopy however does not exclude achalasia, as 30–40% of endoscopies are reported as normal before a final diagnosis of achalasia is made. It is an important

investigation to exclude ‘pseudo-achalasia’, often referring to cancer of the gastric cardia mimicking achalasia (Figure 66.24). Barium contrast study typically shows a hold-up of contrast in the distal oesophagus, abnormal contractions in the oesophageal body and a tapering stricture in the distal oesophagus, described as a ‘bird’s beak’ or ‘rat’s tail’ (Figure 66.25). Progressive dila tation leads to a ‘sigmoidal’ shaped oesophagus. A timed barium oesophagogram is used to quantify the height of the retained contrast at a specific time after ingestion to determine the severity of the disease. All these investigations are suggestive of achalasia but definitive diagnosis relies on HRM. Treatment The treatment goal of achalasia is for symptom palliation since there is no therapy to reverse the neuronal degeneration. Therapies target the LOS, aiming to reduce its contractility by pharmacological means or by destroying the muscle fibres using endoscopic or surgical methods. Medical therapy Pharmacological therapy has a limited role. Calcium channel blockers, nitrates or 5 /uni2032 -phosphodiesterase inhibitors are used to - reduce the LOS pressure. Most have limited e ffi cacy in symp - tom improvement. There are also significant side e ff ects, such as headache, oedema and hypotension, after repeated doses. Medical therapy should be reserved for selected patients who are poor candidates for endoscopic or surgical tr eatments.

Disorders of OGJ out /f_l ow Yes 100% failed peristalsis Achalasia I without POP 100% absent peristalsis Yes Yes 100% failed peristalsis All swallows are either Achalasia II with POP in ≥ 20% swallows failed or premature ≥ 20% swallows with Yes premature contractions. Achalasia III Failed peristalsis ± POP may be present Step 2: (if not done) Wet swallows in secondary position + MRS/RDC Elevated LOS IRP persists in No varying positions + elevated IBP/POP Yes No Yes Abnormal TBO or FLIP OGJOO Figure 66.23 Hierarchy diagnostic algorithm of oesophageal motility disorders according to the Chicago classi /f_i cation 4.0. The broad categories of ‘disorders of oesophagogastric junction (OGJ) out /f_l ow’ and ‘disorders of oesophageal peristalsis’ are differentiated by the integrated relax ation pressure (IRP). FLIP , functional lumen imaging planimetry; IBP , intrabolus pressurisation; LOS, lower oesophageal sphincter; MRS, multiple rapid swallow; OGJOO, oesophagogastric out /f_l ow obstruction; POP , pan-oesophageal pressurisation; RDC, rapid drink challenge; TBO, timed barium oesophagram. Table 66.1 Clinical scoring system for achalasia (Eckardt score). Score Symptom Weight loss (kg) Dysphagia 0 None None 1 <5 Occasionally 2 5–10 Daily 3

> 10 Each meal Disorders of peristalsis Abnormal median IRP Yes No Step 2: Wet swallows in secondary position + MRS/RDC Yes Elevated LOS IRP in varying positions ± elevated IBP/POP No No Yes Absent 100% failed peristalsis contractility No Distal ≥ 20% swallows with Yes oesophageal premature contractions spasm No ≥ 20% swallows with Hypercontractile Yes hypercontractility oesophagus No No evidence of OGJ Ineffective 70% ineffective or ≥ 50% Yes out /f_l ow obstruction oesophageal failed swallows motility No No evidence of disorder of peristalsis Consider meal challenges based on symptom

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Retrosternal pain Regurgitation None None Occasionally Occasionally Daily Daily Each meal Each meal

Botulinum toxin Botulinum toxin is a potent presynaptic inhibitor of acetylcho line release from nerve endings. When injected endoscopically into the LOS, it interferes with the LOS cholinergic excitatory neural activity and paralyses the sphincter muscle. The reported symptom relief decr eased from 70% in 3 months to around 40% in a year. The injection usually has to be repeated after a few months. Because the e ff ect is temporary , it is sometimes used when the diagnosis of achalasia is in doubt. Repeated Ernst Heller , 1877–1964, surgeon, St George's Krankenhaus, Leipzig, Germany . injection may result in scarring, making subsequent treatments more di ffi cult. It should not be o ff ered as first-line treatment in patients who are suitable for myotomy or pneumatic dilatation and its indication is usually r estricted to elderly patients with comorbidities. Pneumatic dilatation This involves stretching the LOS with a non-compliant balloon to disrupt the sphincter muscle and render it less competent. Plastic (polyethene) balloons with a precisely controlled exter - nal diameter are used. If the pressure in the balloon is too high, the balloon is designed to split along its length rather than expanding further. Balloons of 30–40 /uni00A0 mm in diameter are available and are inserted over a guidewire. There is no standardised dila tation protocol. Generally , it is preferred to have serial dilatations in a graded manner, from 30 /uni00A0 mm to 35 /uni00A0 mm and 40 /uni00A0 mm. Serial pneumatic dilatation has similar e ffi cacy to surgical myotomy in selected patients. Features that predict optimal response ar e: patients older than 45 years, female, those with an undilated oesophagus, those who have responded to first dil - atation and those with type II achalasia. Perforation is uncom - mon; the reported incidence averaged about 1.9% (0–16%). With a 30-mm balloon, the chance of perforation should be less than 0.5%. The risk of perforation incr eases with big - ger balloons, which should be used cautiously for progressive - dilatation over weeks. It is important to have an experienced endoscopist performing the procedure and surgical back-up in case of perforation. Heller's myotomy This involves cutting the muscle of the lower oesophagus and gastric cardia (Figure 66.26).Typically , anterior myotomy is

Figure 66.24 Pseudo-achalasia in a patient with cancer of the oesophagogastric junction. The patient was referred as having possible achalasia based on a barium contrast study the obstruction, prompting a computed tomography scan surgical specimen (c) . Figure 66.25 Barium contrast study showing the typical 'rat's tail' appearance of achalasia. (a) . Endoscopy could not get past (b) , making a diagnosis of cancer. The resected

and 2–3 /uni00A0 cm distally into the gastric cardia. Transabdominal or transthoracic approaches have been advocated. Currently , the standard procedure is a laparoscopic approach. The major complication is GORD, which can occur in up to 40% of patients. The addition of a partial fundoplication (anterior Dor or posterior Toupet) has been shown to be e ff ective in reducing the incidence of GORD. A complete 360° fundoplication (Nissen) is considered contraindicated because the increase in outflow resistance against an aperistaltic oesophageal body will probably result in postoperative dysphagia. Laparoscopic myotomy is superior to single pneumatic dilatation in e ffi cacy and durability . The surgical outcome is better in types I and II achalasia than in type III. For the latter, a longer extended proximal myotomy is often needed for adequate treatment. Peroral endoscopic myotomy Peroral endoscopic myotomy (POEM) involves opening the mucosa at a short distance proximal to the intended myotomy site. Entrance is gained into the submucosal plane, which is extended distally to about 2–3 /uni00A0 cm into the gastric cardia. The circular +/– longitudinal muscles are then cut using ESD instruments. Typically , the myotomy extends a

minimum of 6 /uni00A0 cm in the oesophagus proximally and 2 /uni00A0 cm into the gastric cardia distally (Figure 66.27). The mucosal opening is then closed with endoclips. In type III achalasia, there is a spastic component at the distal oesophagus that responds less well to pneumatic dilatation and Heller’s myotomy . POEM has the advantage in that it can extend the length of the myotomy proximally , tailored to preoperative HRM and barium swallow parameters. POEM can also be utilised to treat other types of ‘spastic’ oesophageal motility disorders such as distal oesoph ageal spasm and hypercontractile oesophagus. Randomised controlled trials have demonstrated similar e ffi cacy of POEM to pneumatic dilatation and Heller’s myotomy in relieving dysphagia. Without any antir eflux procedure, the incidence of GORD is expectedly higher in POEM compared with Heller’s myotomy with partial fundoplication. The incidence of oesophagitis at 3 months after POEM can be as high as 57%, which may subject patients to lifelong acid suppression therapy or subsequent antireflux operation. Oesophagectomy Oesophagectomy is reserved only for the treatment of patients with ‘end-stage’ achalasia with a sigmoidal or megaoesopha gus that is not responding to other methods (Figure 66.28 Depending on the chronicity of the disease, the symptoms of achalasia may be tolerated. However, a grossly dilated oesoph agus pr edisposes to regurgitation and aspiration pneumonia. Balancing the risk of an oesophagectomy with the patient’s quality of life and risk of aspiration complication, surgery can be a reasonable option for surgically fit patients. Follow-up Treatment success is usually defined by symptom relief. The Eckardt score is quantified and compared with the preoperative score. Patients should be counselled on a post-treatment diet as the oesophageal body motility remains defective. Ideally , - -). - HRM, barium contrast study , endoscopy and 24-hour pH monitoring should be performed postoperatively to objectively assess LOS function, bolus retention, response to treatment, presence of oesophagitis and acid reflux. This depends on the availability of resources and patients’ preference.

(b) (c) Figure 66.26 Laparoscopic myotomy and Dor hemi-fundoplication. (a) The lower oesophageal myotomy extending onto the stomach for at least 2 cm. (b) Completion of the myotomy; the light of the endo

scope can be seen shining through the thin mucosa. (c) Completion of the Dor anterior fundoplication.

Summary box 66.5 Achalasia /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 66.27 The procedure of peroral endoscopic myotomy (POEM). mucosa and the muscle layer down to the stomach. (c) Myotomy starts a short distance below the mucosal opening. into the proximal stomach. (e) The mucosal opening is closed by endoclips. Achalasia is the most common oesophageal motility disorder A normal endoscopy does not exclude the diagnosis of achalasia Beware of pseudo-achalasia HRM is the gold standard for the diagnosis of oesophageal motility disorder Laparoscopic myotomy, pneumatic balloon dilatation and POEM are effective treatments of achalasia Type III achalasia may be better treated with long myotomy by POEM

Aetiology

Aetiology

Iatrogenic perforation secondary to endoscopic procedures such as dilatation of strictures or achalasia is the most common cause. Other endoscopic procedures such as EMR/ESD/ - POEM may result in leakage if there is transmural disruption and mucosal defects are not closed properly . Spontaneous emetogenic perforation (Boerhaave’s syndrome) results from a sudden increase in oesophageal pressure against a closed glottis from vomiting. Perforation from direct penetrating trauma is - rare as the oesophagus is a deep-seated organ. Blunt external trauma rarely causes oesophageal perforation. Foreign body ingestion, especially with sharp objects, may perforate the oesophagus. Corrosive ingestion can also lead to transmural necrosis and disruption of the oesophageal wall. Patients with EOO may present with spontaneous perforation. Oesophageal cancer can perforate, and the prognosis is usually poor since it reflects the underlying advanced disease. Aetiology

The aetiological factors for the development of oesophageal cancer vary between the two main cell types (Table 66.3). Genetic predisposition may be important in the pathogenesis of oesophageal squamous cell cancer. While smoking and alcohol intake are independent contributing factors, genetic polymorphism is important in individuals with chronic alcohol consumption. Approximately 36% of East Asians show a physiological response to drinking that includes facial flush - ing, nausea and tachycardia. This facial flushing response is - predominantly related to an inherited deficiency in the enzyme aldehyde dehydrogenase 2 (ALDH2). Alcohol is metabolised to acetaldehyde by alcohol dehydrogenase and the acetaldehyde y ALDH2 to acetate. Individuals with is in turn metabolised b variants of the ALDH2 gene may have a suboptimal level of the enzyme, leading to the accumulation of the carcinogen acetaldehyde.

(c) Tumour resection (d) Finish resection (e) Mucosa closure and haemostasis

For squamous cell cancer, dietary and environmental fac tors are important. Nitrosamines and their precursors (nitrate, nitrite and secondary amines), commonly found in preserved food, such as pickled vegetables, have been identified as predis posing factors. Nutritional depletion of certain micronutrients, particularly vitamins A, C and E, niacin, riboflavin, molybde num, manganese, zinc, magnesium and selenium, as well as dietary deficiencies of fresh fruit and vegetables, together with an inadequate protein intake, predisposes the oesophageal e thelium to neoplastic transformation. Other dietary risk factors include consumption of hot beverages, chewing betel nuts and drinking yerba mate in South American countries. Patients with other aerodigestive malignancies are at particularly high risk, presumab ly because of exposure to similar environmental carcinogens. Using oesophageal cancer as the index tumour, multiple primary cancers are found in about 10% of patients, of which 70% are in the aerodigestive tract. The overall incidence of synchronous or metachronous oesophageal cancer in patients with primary head and neck cancer is estimated to be 3%. The rise in incidence of adenocarcinoma coincides

with the increase in obesity , GORD and Barrett’s oesophagus in Western populations. GORD a ff ects up to 44% of the general population in the USA and approximately 5–8% will develop Barrett’s oesophagus, with an estimated annual rate of neo plastic transformation of 0.2–0.5% per year. Ye r b a m a t e is a herbal tea made from the leaves and twigs of the Ilex paraguariensis

Factor Squamous Adenocarcinoma cell cancer + Smoking +++ +++ – Alcohol + – Hot beverages + – N-nitroso-containing food (e.g. pickled vegetables) + – Chewing betel nut + – Drinking yerba mate + – Dietary de /f_i ciency of fresh green vegetables, fruits and vitamins + – Low socioeconomic class + – Fungal toxin or virus + + History of radiation to mediastinum + – Lye corrosive stricture +++ – History of upper aerodigestive malignancy + – Plummer–Vinson syndrome + – Achalasia – ++ Obesity – +++ Gastro-oesophageal re /f_l ux Barrett’s oesophagus – ++++

Ambulatory reflux and combined pH– impedance monito

Ambulatory reflux and combined pH– impedance monitoring

Ambulatory reflux monitoring is considered one of the most important confirmatory tests for GORD. There are two types of monitoring devices: catheter based and wireless capsule (Figure 66.11). Both measure the pH value at 5–6 /uni00A0 cm above the upper border of the LOS, as defined by manometry . For the catheter-based device, data are captured conventionally for 24 hours. The wireless capsule device is anchored onto the mucosa of the oesophagus by a pin and can transmit pH data for up to 96 hours. Various parameters are measured, such as the number of reflux episodes (when pH drops below 4) and oesophageal acid exposure time (the percentage of time exposed to pH /uni00A0 < /uni00A0 4) (Figure 66.12). An oesophageal exposure time of more than 4% can be considered abnormal, and one more than 6% is considered diagnostic. A composite score (Johnson–DeMeester) consists of six parameters that can be calculated. The patient needs to record their symptoms Lawrence F Johnson , contemporary , gastroenterologist, Birmingham, AL, USA. To m R D e M e e s t e r , contemporary , surgeon, Los Angeles, CA, USA. throughout the study period, so that symptom correlation with pH data can be calculated. The catheter-based pH monitoring device also incorpo - rates measurement of impedance. Impedance is inversely pro - portional to the electrical conductivity of the luminal contents and the cross-sectional area. Liquid refluxate has high con - ductivity and therefore low impedance. On the other hand, air has low conductivity and high impedance. With the change in the temporal–spatial patterns in di ff er ent impedance sensors spreading across di ff erent levels of the monitoring catheter, an y bolus transit can be assessed in its direction (antegrade or retrograde) as well as by its nature (air or liquid). Thus, acid reflux, aerophagia, belching and liquid passage can be distin - guished (Figure 66.13).

18.0 Pharynx UOS 21.0 23.0 DCI 28.0 33.0 Oesophagus CDP 38.0 43.0 43.0 LOS PIP 44.4 44.3 OGJ 46.1 48.0 Stomach Gastric 52.3 53.0 0.0 150.0

BARRETT’S OESOPHAGUS Diagnosis and definitions

BARRETT’S OESOPHAGUS Diagnosis and definitions

Barrett’s oesophagus is a known complication of GORD. First described in 1950 as peptic ulceration in a tubular organ lined by columnar epithelium, it was interpreted as an intrathoracic tubular stomach with a congenitally short oesophagus. Later it was correctly identified as ‘oesophagus lined with a gastric mucous membrane’.Currently , the commonlyagreed definition of Barrett’s oesophagus is the proximal migration of columnar epithelium (salmon-coloured mucosa) in the lower oesophagus extending more than 1 /uni00A0 cm above the OGJ. The additional criterion of the biopsy-proven presence of mucus-secreting goblet cells or intestinal metaplasia is controversial. Endoscopically , the OGJ is defined as the proximal end of the longitudinal gastric folds under minimal air insu ffl ation. It should not be confused with the diaphragmatic hiatal pinch or the squamocolumnar junction. The Prague C&M Classifica tion for Barrett’s length is based on validated, explicit, consen sus-driven criteria, including assessment of the circumferential (C) and maximal (M) extent of the endoscopically visualised Barrett’s segment (Figure 66.21). The length of Barrett’s oesophagus is a risk factor for de veloping neoplasia.

Benign pathologies

Benign pathologies

Benign epithelial lesions include papillomas, fibrovascular polyps, glycogen acanthosis, parakeratosis, lipomas, lymph - angiomas and haemangiomas. They are benign, though parakeratosis is associated with malignancy of the oesophagus and head and neck region. Inlet patches (also referred to as heterotopic gastric mucosa) are common and are most often found within a short distance e 66.40). They consist of of the postcricoid region (Figur

(b) (a) Inlet patch on white light endoscopy at 9–10 o’clock.

embryonic gastric mucosa, though there are conflicting views as to whether they are truly embryonic or acquired in origin. The discovery of inlet patches is usually incidental on endos copy . Biopsies will show corpus- or fundus-type gastric mucosa, sometimes even with parietal cells. Most are incidental and can be observed. An association with globus sensation, c hronic cough and laryngopharyngeal reflux has been suggested. The patches can be ablated with RFA, argon plasma coagulation or multipolar electrocoagulation. Resolution of symptoms, how ever, is unpredictable. Oesophageal duplication cysts are congenital anomalies that arise during early embryonic development. They are located within the oesophageal wall, covered by two muscle lay ers, and contain squamous epithelium or a lining compatible with that found in the embryonic oesophagus. Sometimes heterotopic gastric or pancreatic mucosa can be found. Most duplication cysts do not communicate with the oesophageal lumen, run parallel with the oesophagus and are asymptomatic unless large. Symptomatic duplication cysts can be resected. Granular cell tumours are rare. On endoscopy they are typically sessile, yellowish-white and submucosal. They feel firm when prodded with a biopsy for ceps. On EUS, they are hyperechoic and arise from the submucosal layer. They most likely arise from Schwann cells, suggesting a neural origin. Rarely they undergo malignant transformation. Schwannomas are often found incidentally; if large, surgical removal is indicated (Figure 66.41). Theodor Schwann , 1801–1882, physiologist, Berlin, Germany , later Leuven and Liège, Belgium. Leiomyomas are the most common solid benign tumours of the oesophagus (Figure 66.42). They are mostly found - incidentally as a submucosal mass on endoscopy but may produce compressiv e symptoms when large. EUS shows a hypoechoic mass arising from the muscularis propria or the submucosal layer. They rarely become malignant; however, resection is indicated if enlarging on serial assessment. Small leiomyomas can be enucleated with a thoracoscopic approach, - keeping the mucosa intact. Preoperative biopsy or EUS-guided fine-needle aspiration (FNA) is relatively contraindicated as the consequent scarring will increase the chance of breaching the mucosa during enucleation. Endoscopic resection is possible using submucosal tunnel- ling endoscopic resection (STER); this creates a mucosal open - ing a short distance from the leiomyoma (3–5 /uni00A0 cm proximally), allowing a submucosal tunnel to reach the lesion. The lesion is resected using ESD tec hniques and the specimen retrieved via the mouth. The mucosal opening is closed with clips (Figure 66.43). Because of the av ailability of STER, the threshold of removing smaller leiomyomas is reduced because of its minimal invasiveness. Larger

lesions (perhaps larger than 5 /uni00A0 cm) are technically challenging and are better removed thoracoscopically . Leiomyosarcoma is rare and resection o ff ers a chance of cure. Oesophageal gastrointestinal stromal tumours (GISTs) are uncommon and are usually found at the OGJ/proximal stomach. They should be managed along the same principles as GIST in the rest of the gastrointestinal tract.

Figure 66.41 Large tumour of the upper oesophagus showing as an opacity on chest radiograph homogeneous tumour causing tracheal compression (b) . The resected tumour was a Schwannoma. (a) . Computed tomography scan shows a

Figure 66.42 Leiomyoma of the oesophagus. (a) Endoscopic view of a submucosal lesion with intact mucosa. of a hypoechoic lesion arising from the muscularis propria layer (red arrows).

CARCINOMA OF THE OESOPHAGUS Epidemiology

CARCINOMA OF THE OESOPHAGUS Epidemiology

Oesophageal cancer is the eighth most common cancer worldwide and the sixth most common cause of cancer death. It most commonly presents in the sixth and seventh decades of life. Squamous cell carcinoma and adenocarcinoma are the most common cell types while other malignancies such as melanoma and small cell carcinomas are rare. Secondary malignancies are likewise rare; however, bronchogenic carcinoma or metastatic lymph nodes can invade the oesophagus. Squamous cell cancer is the predominant histological cell type but there are significant geographical and ethnic varia tions, with the incidence of oesophageal cancer 50–100-fold the world. higher in areas of high incidence than in the rest of Squamous cell carcinoma incidence r emains steady; however, among white populations, especially in developed Western countries, there has been an epidemiological shift since the 1990s from squamous cell carcinoma to adenocarcinoma such that the incidence of adenocarcinoma has surpassed that of squamous cell cancer.

(a) Mucosal incision (b) Submucosal tunnelling Figure 66.43 Submucosal tunnelling endoscopic resection (STER) technique to excise a leiomyoma. (b) Endoscopic ultrasound /f_i nding

CAUSTIC INJURY

CAUSTIC INJURY

Caustic injury to the oesophagus can be mild, but also is - potentially lethal. Most caustic ingestions occur in children, in whom it is usually accidental, or in adults with suicidal intent. The severity of the injury depends on the type, pH, quantity strong acid, causing coagulative necrosis with eschar forma tion, which may limit penetration to deeper layers, or strong alkali, leading to liquefactive necrosis. The latter potentially penetrates deeper into the oesophageal wall, producing a more se vere injury pattern. Diagnosis is usually not di ffi cult. When the injurious agent is identified, it should be tested for its pH, and suicidal attempt considered. Patients ma y have pain in the neck, throat, chest or even the abdomen. Drooling of saliva, dysphagia and ody nophagia can be present. Hoarseness of voice is an important sign to look for as it may signify laryngeal injury and potential airway obstruction. If the airway is judged to be compromised, careful assessment and emergency intubation using fibreoptic guidance, or even a surgical airway , are indicated. There is no role for gastric lavage or attempts to neutralise the acid or alkali. Initial treatment after securing an adequate airway is supportive, with intravenous fluid, oxygen supplementation and cardiorespiratory monitoring. Once the patient is stabilised, an endoscopy and CT scan with intravenous contrast should be considered. Care ful endoscopy allows assessment of the extent of the injury (Figure 66.34). The Zargar g rading can be used (Table 66.2 In general, the longer and more circumferential the injury , the more likely that strictur e will form. The stomach is assessed as well for injury , and a nasogastric/duodenal tube can be placed with endoscopic guidance. This can be used for ali mentary nutritional support; if a stricture forms, there is still a potential route of access through to the stomach. A CT scan can assess oesophageal oedema and also surrounding soft- tissue infiltration. Most caustic injuries can be managed conservatively with supportiv e measures. Deterioration requires surgical treat ment, with emergency oesophagectomy . T he oesophagus can be mobilised transhiatally or via a thoracoscopic approach. Immediate reconstruction is not recommended. A cervical oesophagostomy and a gastrostomy can be done and future reconstruction planned. A feeding jejunostomy is an alterna tiv e for nutritional support if the stomach also requires resec tion. Delayed complications include stricture and malignancy . A stricture can form early and may be resistant to dilatation (Figure 66.35). There is not enough evidence to support rou tine use of systemic steroids, intralesional injection of steroid or topical mitomycin C to reduce stricture formation. Endoscopic dilatation should be gradual, as the perforation rate is higher than in other forms of strictures . Long strictures are often resis tant to dilatation. Oesophagectomy or bypass surgery may be required. Oesophagectomy has the advantage of removing the oesophagus with its long-term risk of malignancy . However, surgery is di ffi cult because of scarring and adhesions to the mediastinum, thus a bypass operation may be preferable. The gastric conduit is placed in the retrosternal route to reach the neck. When it is also damaged and cannot be used, a colonic interposition is the alternative. The native oesophagus can be left in situ as the risk of dilatation and resultant mucocele is low . Showkat Ali Zargar , contemporary , gastroenterologist, Sher-i-Kashmir Institute of Medical Sciences, Srinagar, India. Burrill Bernard Crohn , 1884–1983, gastroenterologist, Mount Sinai Hospital, New Y ork, NY , USA,

described regional ileitis in 1932. - - -). - - - -

injury to the oesophagus. Zargar classi /f_i cation Description grade 0 Normal appearance 1 Oedema and hyperaemia 2a Super /f_i cial ulceration and friability 2b Deep ulceration or circumferential ulceration 3a Multiple deep ulceration ad scattered necrosis 3b Extensive necrosis 4 Perforation Figure 66.34 Endoscopy picture showing caustic burn to the oesophagus.

Clinical features

Clinical features

Symptoms of GORD can be classified into oesophageal or extraoesophageal. Typical oesophageal complaints include heartburn, which is defined as a burning sensation behind the sternum, and regurgitation, which is the perception of the flow of refluxed gastric content into the mouth or hypopharynx. Patients may also complain of epigastric pain, which can be a manifestation of erosive oesophagitis. Symptoms cannot accu rately predict the severity of the mucosal injury . Dysphagia can be related to large hiatus hernia, stricture or even oesophageal carcinoma. Bleeding from erosive oesophagitis, Cameron ulcers (gastric ulcers a t the level of the diaphragmatic constriction within large hiatus hernias) or tumours can present as co ff ee ground vomiting or frank haematemesis. Extraoesophageal symptoms may include chronic cough, laryngitis, asthma and dental erosions (especially on the lingual and palatal tooth surfaces). The causativ e relationship of extraoesophageal manifestations can sometimes be vague. These problems are usually multifactorial but can be aggravated by GORD. Other conditions with proposed association with GORD include sinusitis, pulmonary fibrosis, pharyngitis and recurrent otitis media. Symptoms are often provoked by food, particularly after a full meal with increased intragastric pressure or food that delays gastric emptying (e.g. oily , spicy food). T he refluxate can cause an unpleasant taste, often described as ‘acidic’ or ‘bitter’. Intensive exercise may sometimes induce GORD in healthy subjects owing to an increase in intra-abdominal pressure. Some patients may complain of nocturnal symptoms espe cially when lying supine, probably related to the gravitational e ff ect. This may significantly a ff ect patients’ sleep. Mild symp toms occurring two or more days a week or moderate/severe symptoms a ff ecting more than one day a week are considered troublesome.

Complex gastro-oesophageal reflux disease

Complex gastro-oesophageal reflux disease

Peptic strictures and dilatation Reflux-induced strictures are relatively rare in the era of PPIs as most patients will be treated empirically before long-term complications occur. These strictures generally respond well to dilatation and long-term treatment with a PPI. Antireflux surgery is an alternative to long-term PPI treatment, just as in uncomplicated GORD. Most patients do not require anything other than a standard operation. - Hiatus hernia and paraoesophageal (‘rolling’) hernia Hiatus hernia is a condition in which the abdominal contents migrate through the hiatal opening of the diaphragm into the mediastinum. There are four types of hiatus hernia (Figure 66.18): (i) the sliding hernia (type I), accounting for most hiatus hernias (85–95%), where the OGJ is herniated - upwards; (ii) the true paraoesophageal/rolling hernia (type II), where there is asymmetrical herniation of the stomach next to the oesophagus and the OGJ remains in its normal intra- abdominal position (this is relatively uncommon); (iii) the more common mixed sliding and paraoesophageal hernia (type III); and (iv) when abdominal viscera other than the stomach migrate into the hernia sac, it is classified as type IV . Hiatus hernia is closely related to advanced age and obesity . - A sliding hiatus hernia predisposes to GORD and is usually diagnosed in the presence of reflux symptoms. For asymptomatic patients, it can be an incidental finding on plain chest radiographs or CT as an intrathoracic gas bubble or fluid level (Figure 66.19). A paraoesophageal hernia, especially when large, presents more with obstructive symptoms. The term ‘giant paraoesophageal hernia’ is present when more than half of the stomach has herniated into the thoracic cavity (Figure 66.20). This may present as a surgical emergency if gastric volvulus occurs. It is more common for the stomach to rotate along its longitudinal axis, termed organoaxial volvulus. When the stomach rotates around the transverse axis, it is called mesentericoaxial volvulus. Gastric volvulus can produce symptoms such as dysphagia - and chest pain. In severe cases, it can cause obstruction, stran - gulation, ischaemia, perforation and compression on the lungs, leading to impaired lung function. Emergency presentation and operation with any of these complications carries a high morbidity rate on account of a combination of late diagnosis, advanced patient age, comorbidities and the complexity of surgery involved. Therefore, all symptomatic paraoesophageal hiatus hernias should be repaired. The decision to repair an asymptomatic paraoesophageal hernia needs to balance risk with the patient’s age and comorbidities, as the annual risk of developing acute symptoms requiring emergency surgery is probably less than 2%. Patients who present acutely should first be resuscitated, followed by nasogastric tube decompression. Immediate sur gery is needed if there is suspicion of ischaemia, perforation or unresolved obstruction. The sur gical principle is similar to that of sliding hiatus hernia repair but more technically any herniated demanding. The steps include reduction of organ, extensive mediastinal dissection to restore the intra- abdominal length of the oesophagus, excision of the hernia sac to prevent a recurrence, repair of the crura in a tension- free manner and some

form of fixation of the stomach in the abdomen. Summary box 66.3 - Hiatus hernia /uni25CF /uni25CF /uni25CF

(a) (c) Figure 66.18 Types of hiatus hernias. (a) Type I, sliding hiatus hernia. paraoesophageal hernia. (d) Type IV, giant hiatus hernia with herniation of another abdominal organ, e.g. colon. (b) (d) (b) Type II, true rolling/paraoesophageal hiatus hernia. (c) Type III, mixed Type I sliding hernia predisposes to GORD Types II/III/IV paraoesophageal hernia present mainly with obstructive symptoms Volvulus and strangulation require emergency surgical treatment

Figure 66.19 Chest radiograph showing a gastric bubble in the lower mediastinum behind the heart corresponding to a hiatus hernia.

Diagnosis

Diagnosis

In most cases, the diagnosis is assumed rather than proven, and treatment is empirical. Proton pump inhibitors (PPIs) are very e ff ective drugs in managing erosive oesophagitis and acid-related symptoms. They are often used empirically as a diagnostic trial. However, there is a significant placebo e ff ect. A positive treatment response with 2 weeks of a PPI can be observed in 69% of GORD patients and 51% of those without GORD (as defined by pH monitoring and endoscopic oesophagitis). This may create a problem of overdiagnosis of GORD and overuse of PPIs. Endoscopic examination provides anatomical assessment, screening for complications of GORD and potential alternative diagnoses such as eosinophilic oesophagitis (EOO), oesopha geal motility disorder or oesophageal cancer (Figure 66.14 practice, many patients will hav e received PPIs before referral Alan J Cameron , contemporary , gastroenterologist, Mayo Clinic, Rochester, MN, USA. - - - - to endoscopy and such information must be available before the procedure because PPIs can heal oesophagitis quickly (Figure 66.15). It is worth remembering that the correlation between symptoms and endoscopic appearances is poor. In patients with atypical or persistent symptoms despite PPI therapy , oesophageal manometry and ambulatory pH record - ing (with or without impedance measurement) are justified to establish the diagnosis and guide management. In patients considered f or surgery , it is also prudent to have objective proof of significant reflux before embarking on a potentially irrevers - ible invasive procedure. HRM is useful in (i) detecting major oesophageal motility disorder, e.g. achalasia, which can sometimes mimic GORD; (ii) defining the location of the LOS for accura te pH monitor - ing placement; (iii) assessing the function and morphology of the LOS, including the size of a hiatus hernia; and (iv) assessing oesophageal body motility to tailor intervention, especially the various antireflux procedures. - PPIs are usually stopped for 2 weeks befor e oesophageal). In pH recording. For patients with a strong pretest probability of

(c) (d) (e) (f) Figure 66.14 Endoscopic appearance of an oesophageal stricture with esophagitis (a) ; Los Angeles classi /f_i cation grade C oesophagitis (b) ; retrograde view of a hiatus hernia (c) ; end view of a hiatus hernia (d) ; Barrett’s oesophagus (e) ; normal oesophagogastric junction (f) .

GORD but refractory to PPI treatment, an ‘on-PPI’ assess ment can be performed to test for breakthrough acid expo sure despite PPI therapy . Wireless pH monitoring increases the duration of recording to 96 hours and could potentially increase the test sensitivity and diagnostic yield. A barium contrast study gives an objective and dynamic assessment of the oesophagogastric anatomy . This may be important in the conte xt of surgery for rolling or mixed hiatus hernias. It is also useful in assessing sur gical complications after antireflux surgery , such as a disrupted wrap, slipped fundopli cation or wrap herniation. However, it is not mandatory for ordinary and uncomplicated GORD patients.

(c) (d) Figure 66.15 Grading of oesophagitis according to the Los Angeles classi /f_i cation. Grade A, one (or more) mucosal breaks no longer than 5 mm that do not extend between the tops of the two mucosal folds (a) . Grade B, one (or more) mucosal breaks longer than 5 mm that do not extend between the tops of two mucosal folds (b) . Grade C, mucosal breaks that are continuous between the tops of two or more mucosal folds but that involve less than 75% of the circumference Grade D, mucosal break that involves at least 75% of the oesophageal circumference (d) .

diagnosis

Oesophageal cancer has a poor prognosis, which is in part related to late presentation; patients with early disease have no symptoms and the cancer tends to metastasise early . Both squamous cell and adenocarcinoma develop from dysplastic mucosa. Most adenocarcinomas are mucin producing with intestinal-type features. Squamous cell cancers tend to be found in the middle and upper part of the oesophagus, while adenocarcinoma predominantly a ff ects the lower oesophagus and OGJ. Both cell types may directly infiltrate adjacent organs and, depending on tumour location, can involve the trachea and bronchi (leading to haemoptysis, airway obstruction and even oesophagus–airway fistula), aorta (though aorto-oesophageal fistula is rare), diaphragmatic crura or pericardium. The oesophageal wall has a rich network of submucosal lymphatics, and therefore longitudinal submucosal spread of cancer is common. Lymph node metastasis can involve the cervical area, the mediastinum as well as the perigastric/ coeliac axis. Distant haematogenous spread can occur to non- regional lymph nodes, lungs, liver, brain and bones. Peritoneal metastasis is an important mode of spread of adenocarcinoma of the OGJ but is rare for squamous cell cancer. Progressive dysphagia is the most common symptom, related to increasing luminal obstruction by cancer. Early symptoms may include a mild hold-up sensation that, if ignored, will progress fr om dysphagia to a solid, soft and even - tually to a liquid diet. There may often be anorexia, weight loss, odynophagia and regurgitation symptoms. Hoarse - ness may indicate involvement of either recurrent lar yngeal nerve. Aspiration and choking symptoms may be related not - only to tumour obstruction but also to the presence of an oesophagus–airway fistula. Choking and cough on drinking wa ter are typical of fistulation and associated haemoptysis is - common. Blood loss is less common for squamous cell can - cer than adenocarcinoma. Chronic blood loss can lead to iron - deficiency anaemia. Acute gastrointestinal bleeding can occur, though it is rarely severe, except for aorto-oesophageal fistula, which usually presents with a smaller sentinel bleed followed pi - by massive bleeding that is invariably fatal. Early cancers are usually asymptomatic and are only picked up on endoscopy performed for other reasons, except in countries where a screening programme exists . For squa - mous cell dysplasia and cancer, chromoendoscopy using Lugol’s iodine is a useful adjunct (Figure 66.44); the normal squamous mucosa will be stained brown, while dysplastic and early cancer remains unstained. NBI light consists of only two wavelengths: 415-nm blue light and 540-nm green light. The di ff erential absorption and reflection of these spectra facilitates detection of mucosal abnormalities (see Chapter 9). A classi - fication of an intraepithelial papillary capillary loop (IPCL) system has been introduced to grade the severity of these early neoplastic changes (Figur e 66.45). For patients with advanced cancer, diagnosis is usually not di ffi cult. A barium contrast study may demonstrate the - tumour as an ulcerated or stenotic lesion with proximal plant. dilatation (Figure 66.46). Endoscopy will visualise the tumour (Figure 66.47), allowing biopsies or brush cytology to confirm the histology . Attempts can be made to traverse the tumour stenosis. If significant dysphagia and weight loss are present, a nasogastric tube can be placed for nutritional build-up. The stomach is assessed for other pathologies in case it

will be used to replace the oesophagus when oesophagectomy is performed. Care should also be taken to assess the rest of the oesophagus, pharynx and larynx to ensure no synchronous tumours are present. The mo vement of the vocal cords should be assessed. For cancers of the mid- or upper oesophagus in proximity to the airway , a bronchoscopic examination is required to ensure no airway involvement, in which case oesophagectomy will be contraindicated (Figure 66.48).

Figure 66.44 Early squamous cell cancer of the oesophagus. shown up on narrow-band imaging with magni /f_i cation (abnormal intraepithelial papillary capillary loops can be seen); by Lugol's iodine. IPC classi /f_i cation by H Inoue (2001) IPCL type I IPCL type II Tissue IPCL type III characterisation of /f_l at lesion IPCL type IV IPCL type V-1 m1 IPCL type V-2 m2 Cancer in /f_i ltration depth IPCL type V-3 m3, sm1 or deeper IPCL type V-N sm2 or deeper Indication for EMR/ESD Relative indication for EMR/ESD Surgical treatment Figure 66.45 Intraepithelial papillary capillary loop (IPCL) classi /f_i cation system. Pictorial diagram showing the appearance of the various types of IPCL correlating with the depth of invasion. EMR, endoscopic mucosal resection; ESD, endoscopic submucosal dissection. (a) A suspicious lesion under white light (marked by white arrows); (b) the area (c) the lesion is not stained Japanese Esophageal Society (JES) classi /f_i cation (2017) JES type A

- Normal IPCL or abnormal microvessels without severe irregularity JES type B1 Dilatation,
- Abnormal microvessels with meandering, severe irregularity or highly irregular calibre dilated abnormal vessels and form variation
- With loop-like formation Extension of IPCL of type V-1 JES type B2 Advanced
- Type B vessels without a destruction of IPCL loop-like formation Generation of new JES type B3 tumour vessel
- Highly dilated vessels with calibres that appear to be more than three times that of usual B2 vessels

Figure 66.46 Barium contrast study showing midoesophageal cancer. Mucosal irregularity with mild luminal narrowing at the lower third of the oesophagus (arrows). Figure 66.47 Endoscopic picture of an ulcerating cancer of the oesophagus.

Disease staging

Disease staging

Careful disease staging is essential to guide therapy . Current staging classification according to the American Joint Commit tee on Cancer (AJCC)/Union for International Cancer Control (UICC) (8th edition) is shown in Table 66.4 . The T stage advances as the tumour invades from mucosa deep to muscle, adventitia and beyond the oesophagus . Regional nodes -

Figure 66.48 Bronchoscopic picture of airway in /f_i ltration by oesophageal cancer. TABLE 66.4 TNM classi /f_i cation of oesophageal cancer. T: Primary tumour Tx Tumour cannot be assessed T0 No evidence of primary tumour Tis High-grade dysplasia, de /f_i ned as malignant cells con /f_i ned to the epithelium by the basement membrane T1 Tumour invades the lamina propria, muscularis mucosae or submucosa T1a: Tumour invades the lamina propria or muscularis mucosae T1b: Tumour invades submucosa T2 Tumour invades the muscularis propria T3 Tumour invades adventitia T4 Tumour invades the adjacent structures T4a: Tumour invades the pleura, pericardium, azygos vein, diaphragm or peritoneum T4b: Tumour invades other adjacent structures, such as the aorta, vertebral body or airway a N: Regional lymph nodes Nx Regional nodal status cannot be assessed N0 No regional lymph node metastasis N1 Metastasis in one or two regional lymph nodes N2 Metastasis in three to six regional lymph nodes N3 Metastasis in seven or more regional lymph nodes M: Distant metastases M0 No distant metastasis M1 Distant metastasis a Regional nodes extend from the paratracheal/oesophageal nodes in the neck to the coeliac nodes.

encompass the paratracheal nodes from the neck, through the mediastinum to the upper abdomen, including the coeliac nodes. The segregation of N1 to N3 is by the number of involved lymph nodes. Location is defined by the position of the epicentre of the tumour in the oesophagus (Figure 66.1 Stage groupings di ff er among squamous and adenocarcino mas. Separate groupings are assigned for clinical (cTNM), pathological (pTNM) and post-neoadjuvant (ypTNM) systems. Because of the complexity , these are not reproduced in this chapter b ut readers can refer to the staging manual. Controversy exists as to whether adenocarcinoma of the OGJ should be staged as oesophageal or gastric cancer. According to the latest staging definitions, a tumour involving the OGJ with its epicentre no more than 2 /uni00A0 cm into the gastric cardia is staged as adenocarcinoma of the oesophagus, while those with a centre located at more than 2 /uni00A0 cm distal to the anatomical OGJ are staged as gastric cancer. Endoscopic and percutaneous ultrasonography The cT stage and paraoesophageal nodes are best staged using EUS. EUS is the only imaging modality able to distinguish the various layers of the oesophageal wall, usually seen as five alter nating hyper- and hypoechoic layers using 12-MHz ultrasound (Figure 66.49). Infiltration to adjacent structures (cT4) is most accurately assessed. The accuracy of EUS for tumour and nodal staging averages 85% and 75%, respectively . T he draw back is that many advanced cancers do not permit passage of a conventional echoendoscope, though most non-traversable tumours are likely to be at least cT3. Miniaturised ultrasonog raphy catheter probes can

be used to pass through the working channel of a conventional endoscope. EUS-guided FNA can be used to obtain cytological proof of involved lymph nodes Bronchoscopic examination can assess airway involvement by the tumour. Percutaneous ultrasonography of cervical nodes is useful, as FNA can be obtained in the same setting. In diagnosing possible T4 cancer by CT scan, obliteration of the fat plane between the oesophagus and the aorta, trachea and bronchi, and pericardium is suggestive of invasion, but the paucity of fat in cachectic patients makes this criterion unreliable. Diagnosis of paraoesophageal nodes is less accurate than with EUS, but distant nodes are better assessed by CT scan. Fluorodeoxyglucose–positron emission tomography scans Squamous cell cancers are usually fluorodeoxyglucose (FDG) avid (Figure 66.50). Detection of the primary tumour is useful. Adenocarcinomas of the OGJ sometimes show limited or absent FDG accumulation regardless of tumour volume (FDG non-avidity). Positron emission tomography (PET) does not define the oesophageal wall and thus has no value in cT staging. Its spatial resolution is also insu ffi cient to separate the primary tumour with juxtatumoral nodes because of interfer - ence from the primary cancer. It is mostly used for detecting regional and non-regional nodes, as well as distant metastases. The uptake by the tumour may have some prognostic value, and c hange in uptake after neoadjuvant treatment is similarly useful in predicting histological response and outcome. Laparoscopy Laparoscopic staging is useful in adenocarcinomas, especially). those of the OGJ, but not for squamous cell cancer. Laparos - - copy should be reserved for patients in whom confirmation of metastatic disease that is not otherwise obtainable is essential in deciding on treatment.

Figure 66.49 Endoscopic ultrasonography (EUS) picture of an oesophageal tumour. The layers of the oesophageal wall on EUS are obliterated. The tumour appears eccentric with extraoesophageal invasion (6 o’clock to 9 o’clock).

EOSINOPHILIC OESOPHAGITIS

EOSINOPHILIC OESOPHAGITIS

- EOO is defined as a chronic, immune/antigen-mediated oesophageal disease, characterised clinically by symptoms related to oesophageal dysfunction and histologically by - eosinophil-predominant inflammation. However, there is over - lap with GORD-related eosinophilia and other pathologies that may also be associated with similar oesophageal eosino - philia, such as achalasia, hypereosinophilic syndrome, Crohn’s disease, coeliac disease, vasculitis, pemphigus , graft-versus-host disease and other connective tissue disorders. The incidence of EOO is increasing, ranging from 0.7/100 /uni00A0 000 to 10.7/100 /uni00A0 000 depending on the population studied. It is predominantly a disease among the white population in Western countries (white versus non-white ratio, 3:1). EOO is believed to commence with food antigen initia tion of cytokine-mediated signals that lead to eosinophilia, inflammation and subsequent remodelling by fibrosis. It is a progressive disease; thus, in infants or toddlers, it may present version and failure to thrive. In young with irritability , food a children vomiting and regurgitation predominate. In older children or early adolescents, it may present with heartburn and dyspeptic symptoms, while in adults dysphagia and food impaction become the most common symptoms. The peak age of presentation is around 20–30 years. There is a clear associa - tion with other atopic disorders such as asthma, atopic rhinitis or dermatitis and food allergies. Typical findings on endoscopy include the presence of rings, furrows, exudates, oedema, stricture , narrowing and ‘crepe paper mucosa’ (Figur e 66.36). Biopsies of the oesoph - agus show 15 or more eosinophils per high-power field (hpf). The eosinophilic infiltration is isolated to the oesophagus. Biopsies should be taken at a minimum of two separate levels of the oesophagus together with suspicious areas. Usually gas - and duodenal biopsies are also taken to exclude eosino - tric philia at these sites. Barium contrast study may demonstrate narrowing and assess the diameter of the oesophagus better than endoscopy . Treatment goals include reduction of oesophageal eosin - ophilia (to <15/hpf and preferably to <5/hpf), control of tion of endoscopic findings, although symptoms and normalisa these goals can only be completely achieved in a minority of patients. In children, the disease process is predominantly h as nausea, vomit - inflammatory , and associated symptoms suc - ing and abdominal pain are relieved with appropriate medical treatments. In adults, reduction in inflammation per se may e not relieve the fibrotic component (stricture and small-calibr oesophagus) and dilatation may be required. Topical corticosteroids are the mainstay medical treatment for EOO; swallowing topically acting corticosteroids such as

Figure 66.35 Contrast study showing a long undilatable stricture of the oesophagus secondary to caustic burn (a) . The patient underwent an oesophagogastrectomy and colonic interposition. Note that the oesophagus and the stomach were scarred (b). Figure 66.36 Endoscopic appearance of eosinophilic oesophagitis. The linear furrows and circular rings are evident.

budesonide or fluticasone is highly e ff ective in resolving symp toms. Systemic steroids should be avoided. PPIs are e ff ective in 40–50% of adults through blockage of cytokine release rather than acid suppression. Diet therapy is useful, eliminating glu ten, milk, soy , egg, nuts and seafood as the most likely antigens Endoscopy is used for diagnostic purposes, to monitor treatment progress and therapeutically to deal with strictures. Careful gradual dilatations should be performed, although perforation rates appear similar to other types of strictur

Figure 66.37 Open surgery was required to extract a broken denture stuck in the cervical oesophagus. The oesophagus was repaired in two layers.

Endoluminal functional lumen imaging planimetry

Endoluminal functional lumen imaging planimetry

Endoluminal functional lumen imaging planimetry (FLIP) is a volume-controlled distension balloon device. It utilises imped - ance planimetry to measure the cross-sectional areas along the length of the balloon, and one pressure sensor measures the - intra-balloon pressure. When placed inside the oesophagus spanning across the LOS, it gives a real-time assessment of LOS distensibility (cross-sectional area divided by intra-bag pressure) and oesophageal contractility in response to balloon distension. The device is mostly still investigational but is expected to gain wider clinical use. It can be used to guide the intraoperative end point for completeness of myotomy for achalasia (change in diameter and distensibility of the LOS) or in screening oesophageal function to assess the need for formal HRM (Figure 66.10).

200.0 UOS 180 Proximal 160 oesophagus 140 CFV tangent 120 100 80 60 DL 50 40 30 20 LOS relaxation 10 0 –10.0 Figure 66.9 High-resolution manometry picture of a typical swallow. The break at the upper oesophageal sphincter (UOS) signi /f_i es the beginning of a swallow. The oesophageal body contractility is represented by the distal contractile integral (DCI), calculated by multiplying the pressure (mmHg) and time (seconds) along the whole length (cm) of the oesophageal body. There is a re /f_l ex relaxation of the lower oesophageal sphincter (LOS) upon each swallow. The time between the start of a swallow to the contractile deceleration point (CDP) is the distal latency (DL). CFV, contractile front velocity; OGJ, oesophagogastric junction; PIP , pressure inversion point.

Endoscopy

Endoscopy

Endoscopy is an essential tool with both diagnostic and thera - peutic roles. A standard diagnostic upper endoscopy includes the examination of the pharynx, hypopharynx, laryngeal inlet, - oesophagus, stomach and part of the duodenum. The risk of the pr ocedure, depending on complexity , is generally low . Extra care should be taken when performing endoscopy for patients with achalasia or obstruction as the oesophagus can be fluid - filled and regurgitation may lead to aspiration. For patients with suspected oesophageal perforation or when the procedure is expected to be prolonged, carbon dioxide should be used for insu ffl ation since it is absorbed more quickly than air. Rigid oesophagoscopy is rarely used nowadays except for unusual circumstances, such as retrieving large or sharp foreign objects. Flexible endoscopy with the use of an overtube is an alternative. Jean Guillaume Auguste Lugol , 1786–1851, physician, Hôpital Saint Louis, Paris, France, suggested that his iodine solution could be used to treat tuberculosis. In patients with significant trismus or obstruction, an ultra-thin endoscope could be used, via either the oral or nasal route, to facilitate the diagnostic or therapeutic procedure. Increasingly ultra-thin endoscopy can be used in an outpatient clinic setting. Image-enhanced endoscopy improves the diagnostic yield and sensitivity of assessment. It should be performed with a high-definition upper gastrointestinal endoscope, equipped with digital image enhancement such as narrow-band imaging (NBI) and magnification. It can be further supplemented by c hromoendoscopy , using Lugol’s iodine (0.5–1%) to look for any suspicious unstained areas and pink colour sign in squamous neoplasia. Similarly , acetic acid (1–3%) is used to look for any loss of aceto-whitening of the mucosal surface of Barrett’s mucosa and neoplasia (Figure 66.7). Endocytoscopy is a novel ultra-high-magnification endoscopic in vivo assessment of lesions technique enabling high-quality with continuous zoom magnification up to 500 times. However, standardised staining methods and endocytoscopic classification are still lacking. With the aid of machine learning and deep learning by artificial intelligence, it is foreseeable that this technique of pattern recognition will greatly improve the sensitivity and specificity of early neoplasia detection and diagnosis.

Figure 66.4 A /f_l uid level (arrows) is apparent in a dilated oesophagus in a patient with achalasia. Figure 66.5 Chest

radiography showing pleural effusion (blue arrows), subcutaneous emphysema (red arrows) and pneumomediastinum (yellow arrows). Figure 66.6 Computed tomography scan showing perforation of the oesophagus secondary to Boerhaave’s syndrome. There is pneumo

mediastinum (red arrow), bilateral pleural effusion (blue arrows) and atelectasis of the left lung (orange arrow). A nasogastric tube is in the oesophagus (yellow arrow).

Endosonography

Endosonography

Endoscopic ultrasonography (EUS) relies on a high-frequency (5–30 /uni00A0 MHz) transducer to provide highly detailed images of the layers of the oesophageal wall and mediastinal structures close to the oesophagus. There are two types of EUS: radial echoendoscope, which has a rotating transducer that creates a circular image with the endoscope in the centre, and linear echoendoscope, which produces a sectoral image in the line of the endoscope. Mini-ultrasound probes that are around 2.0–2.9 /uni00A0 mm in diameter can be inserted through the biopsy channel of an ordinary endoscope to give a simple radial diagnostic assessment within a narrowed lumen. This is useful for obstructive tumour. The di ff erent layers of the oesophageal wall are charac terised by its alternating echogenicity (Figure 66.8). Di ff erent structures can be identified adjacent to the oesophagus and used as landmarks, such as the aorta, the azygos vein and the spine. EUS can also provide a Doppler signal to di ff erentiate index lesions from genuine vascular structures or abnormality bef ore attempting biopsy . Biopsy of submucosal oesophageal lesions or mediastinal masses such as lymph nodes can be per formed with linear echoendoscopes for histological diagnosis and staging.

Figure 66.7 Endoscopic pictures of the oesophagus. (a) High-grade dysplasia of the oesophagus stained by Lugol’s iodine solution. The unstained area is the abnormal area. (b) Early squamous cell cancer examined using narrow-band imaging. Abnormal intrapapillary capillary loops are seen. (c) Barrett’s oesophagus stained by acetic acid (black arrows). Top of gastric fold (green arrows).

FOREIGN BODIES IN OESOPHAGUS

FOREIGN BODIES IN OESOPHAGUS

Swallowed foreign bodies are common and tend to impact at the three narrow portions of the oesophagus; namely , the cricopharyngeus/pyriform fossa, the midoesophagus where the aorta/left main bronchus crosses the oesophagus and at the OGJ. It is a common problem in children; in adults, it is more prevalent among the elderly with swallowing di ffi culties, those with dementia, those with unhealthy alcohol use and those with mental health disorders (Figure 66.37). Bones from fish, pork and chicken are common o ff enders. In complete obstruction, patients may not even be able to swallow fluids or their saliva. A clear history may be volunteered, but in children, the elderly and those with mental health disorders the history may not be clear. Complaints should always be treated seriously even though they may sound implausible. A plain radiograph may reveal radio-opaque bodies and should be taken in two views. If in doubt, a CT scan is the best method to identify a foreign body . Flexible endoscopy is the mainstay of treatment to extract using forceps, nets, baskets or a balloon inflated distal to the object. Airway protection may be needed. Sharp objects should be retrieved with the sharp end pointing distally to lessen the chance of perforation. An overtube can be used as needed. Batteries should always be removed as they may cause injury by direct electrical burn or by liquefactive necrosis from leaked Richard Schatzki , 1901–1992, radiologist, Mount Auburn Hospital, Boston, MA, USA. - - battery content. A food bolus can usually be broken down and . either retrieved or pushed into the stomach. Occasionally open surgery is required for foreign body retrieval. Summary box 66.7 es. Foreign bodies /uni25CF /uni25CF /uni25CF

Figure 66.38 Pill-induced ulceration of the oesophagus. Note the ‘kissing’ ulcers on opposite sides. Swallowed foreign bodies tend to lodge at the three relative constrictions of the oesophagus: the cricopharyngeus, where the left main bronchus crosses the oesophagus and at the OGJ Beware of underlying pathology, such as re /f_l ux stricture, eosinophilic oesophagitis and Schatzki’s ring (see Miscellaneous conditions) Flexible endoscopy can remove most foreign bodies successfully

FURTHER READING

FURTHER READING

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GASTRO-OESOPHAGEAL REFLUX DISEASE Aetiology

GASTRO-OESOPHAGEAL REFLUX DISEASE Aetiology

GORD is defined by the ‘Montreal definition’ as a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications. The aetiology of GORD can be explained by the interaction between the reflux barrier and the pressure di ff erence between the thoracic and abdominal cavity . The reflux barrier consists of the crural diaphragm and the LOS. Physiological relaxation of the LOS in response to stretching of the gastric fundus, particularly after a meal to allow venting of swallowed air, is termed transient LOS relaxations (TLOSRs). An increased number of TLOSRs and a more compliant LOS would increase reflux. Delay in acid refluxate clearance from the oesophagus, as a result of defective oesophageal motility , also contributes to oesophageal exposure. Hiatus hernia is associated with GORD; it is formed when the weakened phreno-oesophageal ligament and widened cru ral opening allow the proximal stomach to herniate through the diaphragmatic hiatus. Ageing, connective tissue disease and elev ated intra-abdominal pressure (e.g. central obesity , ments, chronic straining) will further pregnancy , tight gar aggravate the hernia. An acid pocket is an area of unbu ff ered gastric acid that accumulates in the proximal stomach after meals and serves as a reservoir for acid reflux. Together with a hiatus hernia, it can exacerbate the severity and symptoms of GORD. The overall global incidence of GORD is increasing and has a predominant regional distribution in the Americas and ope; up to 33% of the population is a ff ected, compared with Eur - <10% in Asia. The increase in GORD incidence is attributed to the global increase in obesity and declining rates of Helico - bacter pylori infection. This increasing prevalence of GORD coincides with the increased mortality rates from oesophageal adenocarcinoma. Central obesity , independent of body mass index, is a risk factor for developing Barrett’s oesophagus and adenocarcinoma, while H. pylori may have a preventive role.

(a) Figure 66.10 Endoluminal functional lumen imaging planimetry reading before with achalasia. The column of numbers on the right-hand side of each panel shows the diameter of each 0.5-cm segment along the narrowest segment is indicated by the green circle). This is used to calculate the cross-sectional area. The distensibility index (DI) (red circle) is 3 calculated by dividing the cross-sectional area (mm) by the intra-balloon pressure (mmHg) (blue circle). The DI improves from 0.8 mm 3 to 4.2 mm /mmHg after POEM, indicating that the lower oesophageal sphincter has become more ‘compliant’. (a) Figure 66.11 (a) Wireless capsule used to measure pH data in pH monitoring, with the capsule in the delivery catheter before deployment. (b) Catheter used to measure pH as well as impedance. The catheter is placed through the nostril. The most distal sensor is placed 5 cm above the upper border of the lower oesophageal sphincter measured on high-resolution manometry. (b) (a) and after (b) peroral endoscopic myotomy (POEM) in a patient catheter (the 3 /mmHg (b)

8.0 A pH1 12:00AM 12:00PM 12:00AM 0.0 Interpretation / Findings DeMeester Score Acid exposure time 2 h 42 min 40.2 Day1 off PPI 1 h 17 min 16.9 Day2 off PPI 15 min 4.3 Day3 on PPI 0.3 Day4 on PPI Overall • DeMeester Score = 15.9 Figure 66.12 A wireless pH monitoring trace over 4 days. A re /f_l ux episode is de /f_i ned when the pH drops to less than pH 4 (below the horizontal blue line). This patient had stopped taking a proton pump inhibitor (PPI) 2 weeks before the study. He was then given a daily dose of PPI starting on day 3 of the examination. The DeMeester scores were abnormal (>14.7) on days 1 and 2 but returned to the normal range after acid suppression by the PPI. The symptom association probability (SAP) was 100% on days 1 and 2, meaning that every time there was acid re /f_l ux the patient experienced symptoms. SI, symptom index; SSI, symptom sensitivity index. LOSd, lower oesophageal sphincter, distal; LOSp, lower oesophageal sphincter, proximal; PIP , pressure inversion point; UOS, upper oesophageal sphincter. Auto Range 11:06:20.915 k /uni03A9 7.00 Z1 2.05 0.00 k /uni03A9 7.00 Z2 1.50 0.00 k /uni03A9 7.00 Z3 5.90 0.00 7.00 k /uni03A9 1.73 Z4 0.00 A 7.00 k /uni03A9 Z5 0.53 0.00 7.00 k /uni03A9 Z6 0.41 0.00 A 9.0 pH 119 pH1 3.4 11:06:20 11:06:30 0.0 Display Mode Play 15:00 18:00 21:00 00:00 03:00 06:00 09:00 12:00 Figure 66.13 pH impedance data tracing. A drop in impedance signi /f_i es liquid within the oesophageal lumen while a belch (with air) results in a rise in impedance. In this /f_i gure, the x-axis represents time. There is a sequential drop of impedance from the distal sensor (Z6) to the more proximal sensor (Z3) (indicated by the red arrow and the shaded boxes), signifying a liquid re /f_l ux episode. At the same time, the pH sensor detected pH < 4 (lowest row), indicating that the re /f_l ux episode is an acid re /f_l ux. LOSd, lower oesophageal sphincter, distal; LOSp, lower oesophageal sphincter, proximal; PIP , pressure inversion point; UOS, upper oesophageal sphincter. UOS 19.0 Oesophagus 36.1 LOSp 41.1 PIP 42.2 Diaphragm LOSd 44.8 Stomach 12:00PM 12:00AM 12:00PM 12:00AM 12:00PM No. of total reflux/post-prandial/supine SI, SSI, SAP 77.8, 12.3, 100.0 57/46/1 85.7, 11.1, 100.0 54/48/0 0 10/9/0 0 0 0 Pharynx 53 UOS 20.0 26.2 28.2 30.2 Oesophagus 34.2 36.2 38.2 39.2 40.2 42.2 LOSp 44.2 PIP 45.6 LOSd 47.1 120 Stomach 11:06:40 11:06:50 11:07 pH = 0.0 pH 9.0 Z = 0.00 k /uni03A9 7.00 Meal Supine Mode Anatomy Range Med Symptom Anatomy Source:

as a result of H. pylori -related corpus gastritis or gastric atrophy would lead to decreased oesophageal acid exposure.

INVESTIGATIONS OF

INVESTIGATIONS OF

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Introduction

Introduction

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Learning objectives

Learning objectives

To understand: The anatomy and physiology of the oesophagus and their benign and malignant disease with particular reference to • relationship to disease common adult disorders

MALLORY–WEISS SYNDROME AND INTRAMURAL OESOPHAGEAL

MALLORY–WEISS SYNDROME AND INTRAMURAL OESOPHAGEAL HAEMATOMA/DISSECTION

Forceful vomiting may lead to a tear at the OGJ, mostly imme diately below the squamocolumnar junction. Patients present with haematemesis. Bleeding is rarely severe, and the diagnosis George Kenneth Mallory , 1900–1986, pathologist, Boston University , Boston, MA, USA. Soma Weiss , 1898–1942, Physician in Chief, Peter Bent Brigham Hospital, Boston, MA, USA. is readily made with endoscopy . Endoscopically the bleeding can be stopped by adrenaline (epinephrine) injection or endo - - scopic clips to stop bleeding and close the mucosal defect. Intramural oesophageal dissection is characterised by the separation of the mucosa and/or submucosa from deeper m us - cular layers. This most commonly occurs in elderly patients taking anticoagulants or patients with coagulation disorders. It is often precipitated by vomiting. A break in the oesoph - ageal mucosa is followed by an incr ease in intraoesophageal pressure that causes separation of mucosa and/or submucosa from the muscle layers. The mucosal break can also be caused by trauma such as foreign body impaction or air insu ffl ation during endoscopy . Patients present with acute onset of chest discomfort or odynophagia. If the haematoma ruptures into the oesoph - ageal lumen haematemesis ensues. When the dissection or haematoma is confined to the oesophageal wall, treatment is conservative. Anticoagulation is corr ected and the haematoma usually resolves in 7–14 days.

Stent for oesophageal perforation. (a) Leakage of oral contrast outside the (b) contrast /f_l owing through the stent, no leakage is seen.

MISCELLANEOUS CONDITIONS

MISCELLANEOUS CONDITIONS

Plummer–Vinson (in the USA) or Paterson–Brown-Kelly syndrome (UK) refers to the findings of a cervical oesophageal web, iron deficiency anaemia and dysphagia. It is also known as sideropenic dysphagia. The pathophysiology remains elusive, and the association of iron deficiency is also controversial. Possible mechanisms include iron and nutritional deficiencies, genetic predisposition and autoimmunity . It is a rare disease, mainly a ff ecting middle-aged women. There is a predispo sition to postcricoid, cervical oesophageal cancer. A web is distinguished from a Schatzki's ring in that both the proximal and distal surfaces of the web are lined by squamous mucosa; in a Schatzki's ring, the pro ximal side is lined with squamous mucosa and the distal side by columnar mucosa, coinciding with its location at the squamocolumnar junction. Treatment is with iron therapy , diet modification and, if necessary , endo scopic dilatation of the cervical web. A ring is located a few centimetres proximal to the B ring, if found) is a mucosal band found at the squamocolumnar junction and is not an uncommon cause of food impaction in adults (Figure 66.58). Radiological studies using barium contrast by Scha tzki demonstrated that, in individuals with a ring size of less than 13 /uni00A0 mm, the symptom of dysphagia may occur. The exact aetiology of Schatzki's ring is uncertain but is believed to be related to GORD; hiatus hernia is more common. Treatment is by endoscopic dilatation if symptomatic with dysphagia.

Figure 66.58 Schatzki's ring found on endoscopy. Note there is also oesophagitis.

MOTILITY DISORDERS AND DIVERTICULA Oesophageal mot

MOTILITY DISORDERS AND DIVERTICULA Oesophageal motility disorders

Oesophageal motility disorders are a spectrum of diseases that involve the diminished, overaction or desynchronised neuro muscular function of the oesophageal body or sphincters. The most common presenting symptom is dysphagia. Chest pain, with or without swallowing di ffi culty , is another frequent complaint. Patients often undergo extensive investigations befor e the oesophagus is considered the responsible organ. It is important to have a proper diagnostic work-up before treatment is considered.

Management of uncomplicated GORD

Management of uncomplicated GORD

Lifestyle modification Patients are recommended to have a healthy diet, avoid over eating and avoid dietary items (e.g. carbonated drinks, alcohol, tea or co ff ee) or activities that in the patient’s experience would provoke the symptoms. Patients with nocturnal symptoms should have early dinner and avoid r ecumbence after meals. Elevation of the head of the bed may also help. Smoking cessation reduces severe reflux symptoms in normal-weight individuals on medical treatment. Weight management is recommended for overweight patients. Medical management Most patients with GORD self-medicate with over-the counter medicines such as simple antacids, antacid–alginate Gastro-oesophageal reflux disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF preparations and H -receptor antagonists. Consultation is 2 more likely when symptoms are severe, prolonged and unre - sponsive to simple measures and treatments. Pharmacological treatments mainly target acid reduction or neutralisation. With the development of PPIs in the 1980s, they have quickly become the first-line tr eatment for symptomatic GORD. Given an adequate dose for 8 weeks, most patients have a rapid improvement in symptoms (within a few days), and more than 90% can expect full mucosal healing of oesophagitis (if present) at the end of this time. A policy of ‘step-down’ medical treatment is advocated after the initial 8 weeks of treatment to a dose that keeps the patient free of symptoms, and this might even mean the cessation of PPI. Most patients do not make sustained major lifestyle changes and because PPIs are so e ff ective many remain on long-term treatment. Those patients who have an inadequate - treatment response may benefit from changing to another - PPI, an increased dosage of the same PPI, a twice-a-day regi - -receptor antagonist. PPI is also men or the addition of an H 2 important in patients with reflux-induced strictures, resulting in significant prolongation of the intervals between endoscopic dilatations. There have been numerous reports on the associ - ation between chronic PPI use and a myriad of side e ff ects. Most could not demonstrate a causal relationship except some enteric infections and fundic gland polyps. However, patients - ar e still advised to use the lowest e ff ective dose for symptom control. Prokinetic agents, e.g. metoclopramide and domperidone, are not particularly useful and have potential safety issues. Other TLOSR inhibitors were also disappointing. Antacid–alginate pre parations target the acid pockets and form a polysaccha - ride barrier at the proximal stomach. A more recent develop - ment are the potassium-competitive acid blockers (P-CABs). - Compared with PPIs, P-CABs have a more rapid, competitive, reversible inhibition of proton pumps. How ever, they are avail - able in only limited regions. With pH monitoring, it is possible to identify patients with di ff erent phenotypes, especially distin - guishing those having pathological versus physiological reflux, and positive v ersus negative symptom correlations. For patients with discordant reflux activity and symptom association, as in oesophageal hypersensitivity or functional disorder, antireflux therapy is likely to fail. Other treatment options include per - ception modulators, e.g. tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs), or

alternative therapies, - e.g. hypnosis and beha vioural therapy .

GORD is common but symptomatology may be confused with other disorders, such as achalasia; both may present with regurgitation Sliding hiatus hernia predisposes to GORD Heartburn and regurgitation are typical GORD symptoms Beware of extraoesophageal manifestations of GORD A PPI is the most effective medical treatment, but regurgitation is not well controlled by PPIs (c) .

(c) (d) Figure 66.16 Examples of various types of fundoplication. (a) Normal anatomy. (b) Nissen 360° fundoplication. (c) Dor anterior fundoplica tion. (d) Toupet posterior fundoplication.

Management

Management

In stable patients with a clear history and contained perfo ration, sometimes conservative expectant treatment can be successful. This usually applies to cervical/pharyngeal perfo ration when patients are much less septic. Antibiotics should be given; patients are kept nil by mouth and should wait for the perf oration to heal by itself. In intrathoracic perforations, patients are usually sicker. T hey should be resuscitated with intravenous fluid and given antibiotics and oxygen supplement. Electrolyte disturbances are corrected if present. Septic shock - is treated appropriately . The objectives of treatment are (i) seal the perforation if possible, (ii) adequate drainage, and (iii) supportive measures, including nutrition (alimentary preferred over par enteral), cardiorespiratory support and sepsis control. In patients with significant pleural fluid and pneumothorax that result in respiratory compromise, a wide-bore chest tube - is inserted to the appropriate side for drainage while waiting for more definitiv e investigations such as a CT scan. Endos - - copy can be both diagnostic and therapeutic. The location and size of the perforation site should be ascertained. Foreign bodies are retrieved. Endoscopic sealing of the perforation site with clips and self-e xpanding metallic stents may be possible (Figure 66.33). The stent is usually removed around 4–6 weeks later. Healing is expected to have occurred. A nasogastric tube can be placed at the same time for nutritional support. Surgical intervention is indicated in the presence of sig - nificant sepsis when drainage is not a ff ected by other means (such as interventional radiology), and no e ff ective closure of the perforation can be done otherwise. These conditions are usually present when the perforation is large, when the per - foration is in the intrathoracic oesophagus, when the pleura - is breached, when there is a large septic load and when the presentation is delayed. - When the diagnosis is delayed, closure of the perforation is unlikely to succeed; con version of the perforation into a controlled fistula is another option. A simple way would be to place a T-tube through the defect and repair around it, in addi - tion to adjacent drains. With moder n supportive treatment, oesophageal diversion (cervical oesophagostomy; often an end

(b) Figure 66.32 Epiphrenic diverticulum on a barium contrast study (a) . Endoscopic picture (b) showing the diverticulum (green arrows) and true lumen (red arrow).

stoma is required for e ff ective diversion and OGJ ligation) with later staged reconstruction is rarely needed. Oesophagec tomy is even more uncommonly indicated, perhaps except for extensive caustic burn with perforation when the oesophagus is necrotic. Summary box 66.6 Oesophageal perforation /uni25CF /uni25CF /uni25CF /uni25CF

(b) Figure 66.33 oesophagus; A potentially lethal condition due to sepsis Surgical emphysema, chest pain and vomiting constitute the classic triad of Boerhaave’s syndrome Treatment aims at adequate drainage, closure of the perforation site if possible and supportive measures Delayed diagnosis and management lead to high morbidity and mortality rates

Manometric classification

Manometric classification

Oesophageal motility disorders are classified on HRM under the Chicago classification. A hierarchy diagnostic algorithm is utilised (Figure 66.23). Broadly speaking, these disorders can be classified as disorders of OGJ outflow and disorders of peristalsis. Disorders of OGJ outflow are characterised by an elevated integrated relaxation pressure (IRP), which is the relaxation pressure across the OGJ in response to a swallow . Diagnoses include the three types of achalasia and OGJ outflow obstruction (OGJOO). Disorders of peristalsis include absent contractility , distal oesophageal spasm, hyper contractile oesophagus and ine ff ective oesophageal motility . All motility disorders have to be associated with symptoms or other supporting tests to make them clinically relevant. Various metrics have been developed in HRM; the details are beyond the scope of this chapter and the reader is encouraged to consult the relevant publications.

Multimodality treatment strategies

Multimodality treatment strategies

Results from surgical resection alone have improved. Mortality from surgery is less than 5% in dedicated centres. The long term prognosis remains suboptimal. Since the 1990s, multi- modality treatment has gained impetus and is now routine for advanced disease. Neoadjuvant therapy is more popular than postoperative therapy , in the form of either neoadjuvant chemotherapy or chemoradiotherapy . In OGJ cancers, peri operative (pre- and postoperative chemotherapy) has advocates. The optimal regimen remains controversial. Recent advances in immunotherapy add to the armamentarium. Overall, after treatment in a multimodality prog ramme, a 5-year survival rate of 40–50% is expected. The disease stage and the ability to perform an R0 resection are the most powerful predictors of outcome.

NEOPLASMS OF THE OESOPHAGUS

NEOPLASMS OF THE OESOPHAGUS

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OESOPHAGEAL DISEASES Radiography

OESOPHAGEAL DISEASES Radiography

- As a posterior mediastinal structure, the oesophagus is normally - obscured on plain radiographs by other structures such as the - spine, major vessels, airway and heart. However, this simple imaging test often gives clues of major pathologies, such as a dilated oesophagus with a fluid level in advanced achalasia (Figure 66.4) or pneumomediastinum and pleural e ff usion in oesophageal perforation (Figure 66.5). Radio-opaque foreign - bodies can also be seen. A barium contrast swallow can - demonstrate narrowing, anatomical distortion or abnormal oesophageal motility . It is however inaccurate in the diagnosis of GORD and should not be used for this purpose. Computed tomography (CT) scanning is important in the staging of a malignant neoplasm, delineating the anatomical relationship with other mediastinal structures, or detecting surgical site - infection and extraluminal gas densities (Figure 66.6). When used in conjunction with oral contrast, CT is sensitive in iden - tifying perforation and leakage. -

OESOPHAGEAL INVOL VEMENT IN SYSTEMIC DISEASE

OESOPHAGEAL INVOL VEMENT IN SYSTEMIC DISEASE

The oesophagus can be a ff ected by a variety of systemic diseases; examples include systemic sclerosis/scleroderma, polymyositis, dermatomyositis, systemic lupus erythematosus and polyarteritis nodosa. Scleroderma most frequently The oesophagus is the most commonly a ff ected part of the gastrointestinal tract, characterised by the excessive deposit of collagen, resulting in fibrosis. Symptoms are related to GORD (heartb ur n and regurgitation) as well as dysmotility (dysphagia and chest discomfort). On HRM the classical findings are poor oesophageal motility or even distal oesophageal aperistalsis (smooth muscle portion), with a hypotensive LOS. GORD can be severe because of the combination of dysmotility and hypotensive LOS, and complications such as peptic strictures and Barrett’s oesophagus can be found in about one-third of patients. Management is directed at the control of GORD. PPIs remains the mainstay of treatment but standard doses may not be enough. Other medicines such as prokinetics and alginic acid may be added, though e ffi cacy is limited. The results of surgical fundoplication are suboptimal because of poor oesophageal motility . Strictures are treated by standard endoscopic therapy .

(a) Figure 66.40 Inlet patch or heterotopic gastric mucosa in the upper oesophagus. (b) Using narrow-band imaging.

OESOPHAGEAL PERFORATION

OESOPHAGEAL PERFORATION

Oesophageal perforation is associated with high morbidity and mortality rates. It is an emergency and prompt treatment should be instituted because delayed diagnosis and treatment are associated with a marked increase in mortality rate. Baron Carl von Rokitansky , 1804–1878, pathologist, philosopher and politician, Vienna, Austria, classified oesophageal diverticula as either traction or pulsion in 1840. Louis Hamman , 1877–1946, physician, Johns Hopkins Hospital, Baltimore, MD, USA. - - - - -

Figure 66.31 A linear stapler is introduced orally and the ‘ridge’ including the muscle is divided.

OESOPHAGEAL ULCERATION INFECTIONS

OESOPHAGEAL ULCERATION/ INFECTIONS

GORD is the most common cause of oesophageal ulceration but there are a variety of other reasons, including iatrogenic related to endoscopic procedures, the presence of a naso - gastric tube and medications such as tetracyclines, potassium chloride tablets, non-steroidal anti-inflammatory drugs and bisphosphonates. Typically , when a medication is lodged in the oesophagus there may be ‘kissing’ ulcers with ulcera tion on opposite sides of the oesophagus. Patients may present with odynophagia (Figure 66.38). Infection of the oesophagus typically occurs in immuno - compromised, elderly , debilitated or steroid-dependent patients. Candidiasis is the most common fungal infection, characteristically seen as adherent white pseudo-membranes (Figure 66.39). Viral infections include herpes simplex virus (HSV) or cytomegalovirus (CMV). HSV gives rise to punched- out ulcers with edges that appear vesicular, while CMV ulcers are more shallow . CMV inclusions can be found histologically . Special immunohistochemical stains are required to make the diagnosis. Tuberculous infection can also occur, such as bovine tuberculosis when infected unpasteurised milk is consumed. Treatment would depend on individual aetiology and under lying predisposing conditions.

Figure 66.39 Oesophageal candidiasis.

Oesophageal manometry

Oesophageal manometry

Manometry is used to diagnose oesophageal motility disorders and to assess the oesophageal body and LOS function before surgery , such as antireflux operations. Conventional manome try was developed in the 1950s with water-perfused catheters. Recordings were made by passing a multilumen catheter (usually with only eight channels) down the oesophagus and into the stomac h. The catheter is withdrawn progressively up the oesophagus and recordings are taken at intervals of 0.5–1.0 /uni00A0 cm to measure the length and pressure of the LOS and assess motility in the body of the oesophagus during swallowing water boluses. With the introduction of the colour contour plot by Ray Clouse in 1995, conventional manometry is gradually being replaced by high-resolution manometry (HRM) with solid-state pressure catheters. A typical HRM catheter has 36 circumferential sensors along its length, each spaced 1 /uni00A0 cm apart. HRM defines important anatomical land marks and abnormality of the UOS, LOS and hiatus hernia. Christian Johann Doppler , 1803–1853, Professor of Experimental Physics, Vienna, Austria, enunciated the Doppler principle in 1842. Ray E Clouse , 1951–2007, gastroenterologist, Washington University , St Louis, MO, USA. - - It also measures the contractility of the oesophageal body (Figure 66.9). Various parameters are measured in response to a standardised protocol of drinking a small volume of water. Other optional evaluations include solid test swallows and/or - pharmacological provocation tests. Oesophageal peristalsis that is triggered by the swallow centre in the brain is called primary peristalsis. A hierarchical analysis is established under the Chicago classification to diagnose various oesophageal motility disorders.

Mucosa Muscularis mucosae Muscularis propria Adventitia Submucosa Figure 66.8 Endosonographic picture of an oesophagus. Five layers of the oesophageal wall can be seen.

Oesophageal varices

Oesophageal varices

Oesophageal varices usually present with sudden, large-volume haematemesis secondary to portal hypertension, which is most commonly due to hepatic cirrhosis. Details of presentation and management can be found in Chapter 69 .

Other oesophageal motility disorders

Other oesophageal motility disorders

Hypercontractile motility disorders Distal oesophageal spasm is a condition in which there are incoordinate, premature and rapidly propagated contractions of the oesophagus, causing dysphagia and/or chest pain. The condition may be dramatic, with marked hypertrophy of the circular muscle and a corkscrew oesophagus on the barium oesophagogram (Figure 66.29). These abnor mal contractions are more common in the distal two-thirds of the oesophageal body . Hypercontractile (jackhammer) oesophagus is characterised by high-amplitude contractions and should be di ff erentiated from contractility disorder secondary to outflow obstruction. Patients may present with dysphagia or pain. There is no well-proven treatment strategy for hypercon - tractile motility disorders. Patients should avoid any identifi - able triggering factors (e.g. dietary or GORD related). Similar to achalasia, medical therapy such as calcium channel blockers, nitrates, 5 /uni2032 -phosphodiesterase inhibitors and pain modulators hav e been used with limited e ffi cacy . Botulinum toxin injection in the oesophageal body may be useful. Long-segment surgical myotomy has been attempted with good results. POEM with extended myotomy is also advocated as a minimally invasive approach to treat these disorders.

(a) (b) Figure 66.28 gus (a) Creation of a mucosal opening. (b) A tunnel is created between the (d) Myotomy is carried out End-stage achalasia. A grossly dilated sigmoidal-shaped oesopha- (a) . Transected oesophagus (b) .

Functional oesophageal disorders According to the Rome IV classification, functional oesoph ageal disorders include a variety of oesophageal symptoms (heartburn, chest pain, dysphagia, globus) that are not explained by mechanical obstruction (stricture, tumour, EOO), major motor disorders (achalasia, OGJOO, absent contractility , distal oesophageal spasm, jackhammer oesophagus) or GORD. The mechanisms responsible are unclear but are likely to be more related to visceral hypersensitivity and hypervigilance. The diagnosis is generally by exclusion. Physiological and psycho logical factors should be considered. Among all the oesophageal disorders defined under the Rome diagnostic criteria, functional heartburn and reflux hypersensitivity contribute to most diagnostic confusion with genuine GORD. Therefor e, patients have to be carefully assessed before antireflux surgery is o ff ered. Pharmacological agents such as PPIs, tricyclic antidepressants, SSRIs and other pain modulators can be part of the treatment strategy . Surgery has a very limited role in the treatment and usually results in a poor outcome.

Figure 66.29 Barium contrast study showing a corkscrew oesopha gus in a patient with diffuse oesophageal spasm.

Palliation

Palliation

In the presence of distant metastases, palliation is the aim. Dysphagia is the main symptom to relieve. Placement of a self-expanding metallic stent is simple and e ff ective and allows immediate relief of dysphagia (Figure 66.57). The risks are stent migration, tumour ingrowth, airway compression and tracheal erosion if placed in the mid- and upper oesophagus. Other endoscopic methods, such as dilatation, laser treatment and photodynamic therapy , can be used. Chemotherapy/radiotherapy/brachytherapy can help restore luminal patency; in case of bleeding from OGJ tumours, radiotherapy can be haemostatic. Immunotherapy has promise in selected patients. Henry Stanley Plummer , 1874–1937, physician, Mayo Clinic, Rochester, MN, USA. Porter Paisley Vinson , 1890–1959, surgeon, Mayo Clinic, Rochester, MN, USA. Donald Ross Paterson , 1863–1939, ENT surgeon, Cardi ff Royal Infirmary , Cardi ff , UK. Adam Brown-Kelly , 1865–1941, ENT surgeon, Victoria Infirmary , Glasgow , UK. - - - Summary box 66.8 Oesophageal cancer /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 66.57 Self-expanding metallic stent in palliation of oesopha

geal cancer. Squamous cell cancer still predominates in the East, while Barrett’s adenocarcinoma is more common in the West Late presentation and early spread are reasons for poor prognosis Early diagnosis has the best chance of cure Lymphatic spread can be widespread, from the neck to the mediastinum and coeliac axis Adenocarcinomas around the OGJ are sometimes regarded as proximal gastric cancer and treatment is particularly controversial

Pharyngeal and oesophageal diverticula

Pharyngeal and oesophageal diverticula

Oesophageal diverticula can be classified as true diverticula, which involve a full-thickness oesophageal wall, and false diverticula, which involve mucosal outpouching only . Diver ticula are usually described by their location as pharyngeal, Gustav Killian , 1860–1921, Professor of Laryngology , Freiburg and later Berlin, Germany . - - midoesophageal and epiphrenic. Diverticula alone seldom produce troublesome symptoms unless large or secondary to an underlying oesophageal motility disorder. The most common symptoms are dysphagia, regurgitation, halitosis and recurrent aspiration. Pharyngeal pouch (Zenker’s diverticulum) Zenker’s diverticulum is a false pulsion diverticulum as it protrudes posteriorly above the cricopharyngeal sphincter through the natural weak point (the dehiscence of Killian) between the oblique and horizontal fibres of the inferior pharyngeal constrictor. The pathophysiology is believed to involve loss of coordination between pharyngeal contraction - and opening of the upper sphincter (Figure 66.30).

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Figure 66.30 Barium contrast study showing a Zenker’s diverticulum.

desynchronisation of swallowing with predominantly pha ryngeal dysphagia. As the pouch enlarges, it tends to fill with food on eating, and the fundus descends into the mediasti num. Regurgitation of trapped food can occur and lead to aspira tion. Another symptom is halitosis. Conventional sur gical treatment involves an open left cervical incision (most diverticula point towar ds the left side) with diverticulectomy and cricopharyngeus myotomy . Another option is diverticular suspension, whereby the diverticulum is dissected and inverted with its ape x pointing cranially . This will stop food from enter ing the pouch. The absence of a suture line lessens the chance of a postoperative leak. A cricopharyngeus myotomy is also an integral part of the surgery . Newer techniques include tran soral introduction of a linear stapler to divide the se ptum in between the diverticulum and the true oesophageal lumen. This creates a common channel, and the myotomy is in e ff ect performed by the stapler transection (Figure 66.31). Midoesophageal diverticula (Rokitansky diverticulum) Midoesophageal diverticula are usually small traction divertic ula of no particular consequence. In granulomatous diseases with chronic inflammation, fibrosis or lymphadenopathy in the mediastinum can exert traction force onto the oesophageal wall and cause full-thickness outpouching. Rarely it may cause fistula tion into the airway in uncontrolled pulmonary tuber culosis. Asymptomatic midoesophageal diverticulum does not warrant any treatment. HRM may be indicated in symptomatic patients to exclude pulsion diverticulum due to oesophageal motility disorder, e.g. hypercontractile oesophagus . Epiphrenic diverticula Epiphrenic diverticula are pulsion diverticula typically situated at the distal 10 /uni00A0 cm of the oesophagus. They are commonly associated with oesophageal motility disorders, e.g. achalasia, or other causes

of oesophageal outflow obstruction. Barium oesophagogram is a useful investigation depicting the size and anatomical relationship of the diver ticulum and, at the same time, screening for oesophageal motility disorder (Figure 66.32). Large diverticula should be excised combined with a myotomy from the neck of the diverticulum to the cardia to reliev e the functional obstruc tion. Concurrent fundoplication or repair of hiatus hernia may be necessary , depending on the size of the diverticulum or associated conditions. A laparoscopic approach is the preferred option to reduce morbidity .

Presentation and diagnosis

Presentation and diagnosis

Sometimes the history is obvious, such as after instrumenta - - tion or foreign body ingestion. At other times there may not have been any precipitating cause. Patients with Boerhaave’s syndrome may have the classic triad of vomiting , chest pain and subcutaneous emphysema. There may or may not be asso - - ciated haematemesis. Typically , the site of perforation is the lower oesophagus towards the left pleural cavity . Gastric juice as well as ingested food is forcefully ejected into the left chest. A left pleural e ff usion rapidly accumulates. Physical examination r eveals subcutaneous emphysema on the chest wall, sometimes extending to the cervical region as well. In the presence of sepsis, the patient will run a fever, has tachycardia and appears tachypnoeic. Hamman’s sign refers to a crunching sound on auscultation of the heart owing to surgical emphysema. Di ff erential diagnoses usually include pneumonia, myocardial ischaemia or other intra-abdominal pathologies such as a perforated viscus when the pain is referred to the epigastrium. Patients with cervical oesophageal or pharyngeal perfora tion are usually much less septic than those with intrathoracic perforations and mediastinitis. A typical example would be foreign body perforation, such as by a shar p fishbone, having lodged at the postcricoid area and perforated the oesophagus. There may be a history of foreign body ingestion and neck pain and physical examination may reveal tenderness and sub cutaneous emphysema. Subcutaneous and mediastinal air, pneumothorax, hydro pneumothorax and a widened mediastinum may be seen on the chest radiograph (Figure 66.5). Contrast swallow study using Gastrografin or a non-ionic contrast usually re veals the site of perforation. However, sick patients may not be able to tolerate oral contrast. A CT scan (preferably with intravenous as well as oral contrast) will be able to demonstrate the site (and aetiology) of perforation and the extent of mediastinitis, e ff usion and collections (Figure 66.6).

Screening and surveillance

Screening and surveillance

The risk factors for Barrett’s oesophagus and related neoplasm include chronic (>5 years) GORD symptoms, advanced age (>50 years), smoking, central obesity and male gender. For non-dysplastic Barrett’s, the risk of progression to cancer is around 0.2–0.5% per year. This increases to around 0.7% per year for low-grade dysplasia. For high-grade dysplasia, the risk of cancer progression can be as high as 7%. Therefore, screen ing and surveillance protocols should be tailored to individuals - - according to the potential benefit of cancer prevention and the risk and cost-e ff ectiveness of such intervention (Figure 66.22). Screening or surveillance aims to identify premalignant lesions - and to treat them early .

Figure 66.20 Computed tomography scan showing a large type IV paraoesophageal hernia with stomach and intestine in the mediasti

num and left thoracic cavity. Mediastinal shift towards the right side is evident. Depth of endoscope insertion (cm) 28 Squamocolumnar junction 30 32 Columnar metaplasia 34 Oesophagogastric junction 36 36 Hiatus hernia Stomach Figure 66.21 Prague C&M criteria to report endoscopic Barrett’s oesophagus. The location of the oesophagogastric junction is de /f_i ned by the top of the gastric folds (36 cm). Prague criteria of Barrett’s oesophagus are expressed in C (circumferential), in this case 33–36 /uni00A0 cm (3 cm), and M (maximum extent), in this case 28–36 cm (8 /uni00A0 cm). This patient therefore has Prague C3 M8 Barrett’s oesophagus.

The vast majority of patients with Barrett’s oesophagus in the community are asymptomatic; a general population screening programme is considered not to be cost-e ff ective. High-definition white light endoscopy including NBI is used to assess the mucosal and vascular patterns of the Barrett’s segment. Additional chromoendoscopy using di ff erent agents, e.g. methylene blue, acetic acid or indigo carmine, could aid diagnosis. The histological specimen obtained should be examined by an experienced gastrointestinal pathologist. The sensitivity of the assessment can be

improved by increasing the number of biopsies and, w hen in doubt, endoscopy and biopsy should be repeated. The Seattle biopsy protocol, which includes four-quadrant random biopsies every 2 /uni00A0 cm in addi tion to targeted biopsies on macroscopically visible lesions, is recommended at the time of diagnosis and subsequent sur veillance.

Repeat OGD every – 3 5 years No dysplasia Maximum length < 3 cm Maximum length < 3 cm Maximum length Gastric metaplasia Intestinal metaplasia ≥ 3 cm Repeat OGD every Repeat OGD every Repeat OGD 3–5 years 2–3 years Length < 3 cm Gastric metaplasia Consider discharging Figure 66.22 Algorithm for managing Barrett’s oesophagus, including dysplasia. Summary of guidelines from the American College of Gastro enterology (orange) and the British Society of Gastroenterology (blue). BO, Barrett’s oesophagus; HGD, high-grade dysplasia; LGD, low-grade dysplasia; MDT, multidisciplinary team; OAC, oesophageal adenocarcinoma; OGD, oesophagogastroduodenoscopy.

Surgical management

Surgical management

While most patients’ symptoms are satisfactorily controlled with PPIs and other medications, surgery remains an import ant option. The indications for surgery include (i) incomplete symptom control with medical management, (ii) intolerance of, or unwillingness to comply with, long-term medical therapy , (iii) regurgitation despite medication (less well amenable to PPI), (iv) presence of a large hiatus hernia, (v) complica arising from GORD, and (vi) extraoesophageal symptoms. The predictors of good surgical outcome include typical GORD symptoms, PPI responders, presence of hiatus hernia and pres ence of GORD complications, e.g. reflux oesophagitis (grade B or above) and non-dysplastic Barrett’s oesophagus. Factors leading to poor surgical outcomes are normal preoperativ monitoring when performed o ff PPI, functional heartburn, EOO, connective tissue diseases and extreme obesity . Careful preoperative counselling is essential. Risks of anti reflux surgery include a small mortality rate (0.1–0.5%), failed operation (5–10%) and side e ff ects such as dysphagia, gas bloat or abdominal discomfort (10%). When perfor med well in appropriately selected patients, 80–90% of patients should be satisfied with the result of the operation. Wilhelm His , 1831–1904, Professor of Anatomy , Leipzig, Germany . Rudolph Nissen , 1896–1981, Professor of Surgery , Istanbul, Turkey , and later Basel, Switzerland. André M T oupet , 1915–2015, surgeon, St Cloud Hospital, Senior Consultant, University of Paris, France, proposed his technique in 1963. Jacques Dor , 1904–1997, thoracic surgeon, Marseilles, France. Anthony Watson , contemporary , surgeon, Royal Free Hospital, London, UK. Antireflux operations have three essential components: (i) restoration of an intra-abdominal segment of the oesoph - agus, (ii) crural repair, and (iii) some form of reinforcement of the LOS by the upper stomach (fundoplication) or by a prosthesis placed around the intra-abdominal oesophagus. The major types of antireflux operations were all devel - oped in the 1950s. For many years, the relative merits of tho - racic and abdominal approaches were hotly debated. With the introduction of laparoscopy , laparoscopic fundoplication with hiatal reconstruction is the standard approach. The mech - anism of fundoplication is to create a ‘floppy’ valve around the OGJ and to restore the angle of His. It has the e ff ect of increasing LOS basal pressure, lessening TLOSR and r educ - ing the capacity of the gastric fundus, thereby enhancing gas - tric emptying. The di ff erent types of fundoplication have been compared extensively in clinical trials but the superiority of one over the others could not be shown. Complete fundoplication (Nissen) is associated with a higher incidence of short-term dysphagia but is most durable in reflux control. P artial fundoplication, whether performed posteriorly (Toupet) or anteriorly (Dor, Wa tson), has fewer short-term side e ff ects, although this is at the expense of a slightly higher long - term failure rate (Figure 66.16). The most common side e ff ect of fundoplication is short- term dysphagia, related presumably to tissue oedema and inflammation. It usually resolves within 3 months of surgery . Some patients may experience ‘gas-b loat syndrome’, especially after a complete fundoplication. Typically , the patient would complain of gaseous distension of the abdomen and failure to belch or vomit, together with an increase in flatulence. In the last decade, a magnetic prosthesis has become available to reinforce the LOS after hiatal reconstruction (Figur e 66.17). This has a similar e ffi cacy to fundoplication in - the mid- to long

term and has fewer gas-bloat side e ff ects. The magnetic ring prosthesis consists of titanium-coated magnetic beads, connected by titanium wire. The physics of the magnets allows a lower attraction force when the beads are separated. This property of ‘relaxation’ is a more physiological sphincter tions to allow food passage and is less likely to create oesophageal outflow obstruction. This device is contraindicated in patients with major motility disorders, ine ff ective oesophageal motility - or connective tissue disease. e pH Summary box 66.2 Laparoscopic fundoplication /uni25CF - /uni25CF /uni25CF

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HRM and pH monitoring are recommended investigations before consideration of surgical treatment pH monitoring con /f_i rms GORD and HRM assesses oesophageal body function and LOS characteristics Surgery should be tailored to oesophageal motility

Complications of antireflux surgery and revisional surgery Structurally , a wrap can be too tight or too loose. It can also be partially or completely disrupted, herniated or slipped. Struc tural laxity can give rise to recurrent or persistent GORD. Too long or too tight a fundoplication can give rise to dysphagia and gas-bloat syndrome. Endoscopy , contrast radiography and functional testing can assess the anatomy responsible and guide further management. Management strategies include a PPI for recurrent acid reflux, endoscopic dilatation of stenosis and surgical revision as a last resort. A tight complete fundoplication can be rem edied by conversion to a partial fundoplication. For patients with anatomical failure and refractory symptoms, revisional surgery carries a lower chance of success; in some patients, local re vision is technically impossible, as often there will be adhesion formation and altered anatomy . Transient dysphagia is common after both fundoplication and magnetic sphincter augmentation. For the latter, there may be more prolonged dysphagia requiring dilatation or, rarely , migration and ero sion (0.15%). Removal of the device is required in 2.7% of patients; the majority can be accomplished endoscopically or laparoscopically . Endoscopic treatment Several endoscopic treatments have been tested that attempt to augment a failing LOS. Transoral incisionless fundoplication mimics classic fundoplication by recreating the dynamics of the angle of His using an endoscopic stapling device. Meta-analysis demonstrates improvement in clinical response compared with PPIs; however, oesophageal acid exposure time and reflux episodes are not significantly improved and PPI usage increases with time. Radiofrequency ablation (RFA) is another strategy to remodel the LOS by reducing compli ance. Again, it partially improves quality of life but does not normalise pH exposure time and almost 50% of patients still require PPI at follow-up. Antireflux mucosectomy makes use of the endoscopic mucosal resection (EMR) tec hnique to remove subcardiac mucosa while preserving a 1-cm gap at the lesser and greater curves. The contraction and scarring shown improvement in both quality of life and acid reflux. A significant proportion of patients require balloon dilatation for stenosis. These procedur es have been applied to patients with only small hiatus hernias or none at all, so only a small proportion of patients are suitable. Recently argon plasma coagulation has been used to accomplish the same subcardiac mucosal injury instead of EMR. Technically this is much less demanding than EMR.

Oesophagus Right crus Figure 66.17 Magnetic sphincter augmentation. Intraoperative photograph following hiatus hernia repair. The magnetic sphincter is implanted around the lower oesophagus, in between the posterior vagus nerve (white arrows) and the oesophageal wall.

Treatment

Treatment

When Barrett’s oesophagus is discovered, the treatment is that of the underlying GORD. Pharmacological therapy generally is the same as treatment of symptomatic GORD patients. Antireflux surgery is indicated if it is associated with GORD symptoms. A randomised trial suggested that aspirin, as a chemoprevention agent, in combination with a high-dose PPI, may improve outcomes in patients with Barrett’s oesophagus measured by progression to cancer and mortality . In patients with dysplastic Barrett’s oesophagus without suspicion of invasive cancer, the epithelium can be ablated or resected. Indication for such procedures in non-dysplastic Barr ett’s oesophagus is controversial. Ablative therapy aims to completely eradicate all intestinal metaplasia. When the mucosa regenerates after ablation in a non-acidic environ - ment (when a high-dose PPI is prescribed), a ‘neosquamous’ lining is formed. Ablative approaches that are supported by evidence include photodynamic therapy , RFA and cryother - ap y . Among these methods, RFA is most popular because there is evidence of its e ff ectiveness, cost and side-e ff ect pro - file. EMR by the cap method or multiband technique can be done to remove the whole segment of the mucosa. When this is applied to cir cumferential Barrett’s oesophagus, the stric - ture rate is high when healing occurs . The procedure can be performed in stages, allowing mucosal healing to occur first in one half of the oesophagus before a second stage to remove - the other half, thus lessening the chance of stenosis. In con - trast, the incidence of stricture formation is low following - RFA, because the depth of ablation extends to the muscularis mucosae only . Endoscopic ablation should only be applied to flat lesions without nodularity , ulceration or irregular contour. Suc h features are suggestive of invasive neoplasm that should be inv estigated and treated by EMR or endoscopic submucosal dissection (ESD). ESD, though more technically demanding, provides en bloc resection of the index lesion with better mar - gins for histological diagnosis. EMR is easier and large areas can be resected in a piecemeal manner. If histological exam - ination of the resected tissue demonstrates the absence of inva - sive cancer, or T1a tumour, only the ‘biopsies’ can be reg arded as curative. When T1b lesions are found on histology , or when the resection margins (lateral or deep) are in volved, additional therapy including oesophagectomy should be considered. Regardless of treatment performed, the patient should enter a surveillance programme to detect recurrent or persistent Bar - rett’s oesophagus or neoplasia.

mucosa Systematic cold biopsy Con /f_i rmed dysplasia by two

independent pathologists Inde /f_i nite for HGD or LGD dysplasia T1a OAC MDT Repeat OGD with OGD every 6 discussion maximal acid months until: suppression 2 consecutive Therapeutic evidence of non- intervention dysplastic BO De /f_i nite dysplasia Follow non- Follow LGD Endoscopic dysplasia or HGD eradication /f_l owchart /f_l owchart therapy

Barrett’s oesophagus /uni25CF /uni25CF /uni25CF /uni25CF

Endoscopic examination and biopsies are crucial in the diagnosis of Barrett’s oesophagus Dysplasia should be con /f_i rmed by at least two experienced pathologists Surveillance or ablation are options for low-grade dysplasia In patients with high-grade dysplastic Barrett’s oesophagus, ablative, endoscopic resection and oesophagectomy should be considered

Treatment

Stage-directed therapy Treatment principles depend on the disease stage and phys - iological reserve of patients. Patients should be discussed in a multidisciplinary team to decide on the best course of management. When distant metastatic disease is identified palliation is the aim. Endoscopic treatment The chance of nodal metastasis depends on the depth of infiltration of the primary tumour. Cancers that are confined - to the mucosa (T1a) rarely metastasise, but squamous

tumours that have infiltrated the submucosa (T1b) have a substantial risk of nodal spread. In adenocarcinoma, the corresponding risk is less. Such early cancers may be amenable to curative - endoscopic treatment (Figure 66.51). EMR involves the injection of saline (or other solutions such as glycerol or hyaluronic acid) into the submucosal plane to raise the mucosal - lesion; it is then sucked into a cap fitted onto the tip of the endoscope, looped by a snare wire and cut by electrocautery . The limitation of this method is the size of the cap, so it is . generally recommended for smaller lesions. For larger lesions, if resected by EMR, piecemeal resection is required; therefore, it is associated with higher incomplete resection and recurrence rates. ESD is more complex. It involves first marking the margins of the lesion, then submucosal injection, cutting the mucosal edges along the line of marking, submucosal dissection of the tumour from its bed (superfi cial to the muscle layer) and lastly haemostasis. There are various ‘knives’ that can be inserted via the biopsy channel of the endoscope to carry out these procedures. Technically ESD is more demanding than EMR but is not limited by the size of the lesion for en bloc resection. There is an increased risk of postresection stricture formation if too much of the circumference of the mucosa (such as over two-thirds) is removed. This chance is somewhat reduced by steroid treatment (often endoscopic injection at the time of EMR/ESD, combined with oral medication for some time). For early Barrett’s cancer, EMR and ESD are options, and additional circumferential resection or ablation of the whole length of Barrett’s mucosa can be done. For ablative therap y , RFA is most commonly used. RFA energy is delivered by the bipolar electrode and the energy causes frictional heating of cellular water molecules. After ablation, in the presence of a non-acid milieu (suppressed by a high-dose PPI), the epi - thelium would regenerate to be squamous cell mucosa. The advantage of RFA is that it is technically easy to operate but the drawback is that no surgical specimen for detailed histopatho - logical examination is available. Other examples of ablative technique include cryotherapy or photodynamic therapy .

(b) Figure 66.50 Positron emission tomography/computed tomography (CT) scan for staging of oesophageal cancer and assessment of response to neoadjuvant therapy. The patient underwent neoadjuvant chemoradiotherapy. The tumour had high /f_l uorodeoxyglucose uptake (SUV before treatment (b) . After chemoradiotherapy the SUV dropped to 4.1 with a corresponding reduction in size of the cancer seen on CT max scan (a) . 23.5) max

Because the pretreatment distinction of T1a and T1b dis ease may not be accurate, it may be prudent to perform endo scopic resection first in case of uncertain diagnosis. Should the resected specimen be found to be T1a with clear margins and without lymphovascular permeation in the pathological examination, the endoscopic treatment is deemed curative. If the tumour is found to be deeper than e xpected or if resection margins are not clear, further therapy can be planned. Surgery The primary indication for surgical resection is for potential cure, which can be achieved in patients whose tumours are confined to its wall and only limited local/regional disease is found. One should aim to maximise the chance of an R0 resection (macroscopic and microscopic clearance of proximal, distal and lateral margins), a parameter that has consistently been shown to result in the best long-term survival. Surgical resection alone is generally indicated for more advanced cancers when endoscopic treatment is unlikely to be curative (T1b, T2, N0). For patients with more advanced disease (≥ T3, N+), multimodality treatment is usually preferred. Patient selection and preparation Oesophagectomy is a major procedure; patients should be assessed carefully for operative risk and their physiological status optimised. Cardiorespiratory assessments are essential. Patients must stop smoking and alcohol intake. Chest physio therapy is instituted,

and incentive spirometry is a good preop erative exercise. Patients with high-grade oesophageal tumour stenosis may have lost a substantial amount of weight. A fine-bore nasogastric tube can be placed for nutritional support while work-up is performed. Feeding jejunostomy is an alter native. Enhanced recovery after surgery (ERAS) programmes entail preoperative ‘pre-habilitation’ as well (Table 66.5 measures are aimed to optimise patients for surgery . Immedia preoperative preparations include prophylactic antibiotics and Ivor Lewis , 1895–1982, surgeon, North Middlesex Hospital, London, later Rhyl, UK. Norman Cecil Tanner , 1906–1982, surgeon, Charing Cross Hospital, London, UK. - deep vein thrombosis prophylaxis. Bowel preparation is not - necessary unless a colonic interposition is intended. Surgical techniques Choice of surgical approach The choice of the appropriate technique depends mainly on: (i) the location of the tumour, (ii) the intended extent of lymphadenectomy , and (iii) the reconstructive technique. The surgeon should be well versed in the methods adopted to di ff er - ent clinical situations. For ease of description, the following sections discuss the surgical approach by tumour location. Cervical oesophageal cancer Surgery involves removing the pharynx, larynx and oesoph - agus (pharyngo-laryngo-oesophagectomy); a gastric pull-up is used to anastomose with the neo-pharynx. In cases where involvement of the cervical oesophagus is limited, pharyngo-laryngo-cervical oesophagectomy can be carried out without the need for total oesophageal resection. The resultant gap can be bridged using either a free jejunal graft, or various musculocutaneous flaps. Definitive chemoradiotherapy has become the preferred alternative treatment to preserve the larynx. Surgery is therefore mostly reserved for salvage, when there is an incomplete response or for recurrent disease. Intrathoracic oesophageal cancer - The surgical procedures usually performed are: - /uni25CF Left thoracoabdominal incision. Via a large incision tra - versing the chest and upper abdomen, the whole left upper quadrant of the abdomen and left thoracic cavity are accessed at the same time for oesophagectomy , gastro - - plasty and anastomosis (Figure 66.52a). /uni25CF Lewis–Tanner (or Ivor Lewis) procedure. This is a two-). All phase oesophagectomy consisting of laparotomy for gas - te tric mobilisation and tubularisation, followed by a right

(a) (b) Figure 66.51 Schematic diagrams showing technique of (a) endoscopic mucosal resection – cap with submucosal injection and snare excision; (b) endoscopic submucosal dissection.

thoracotomy for oesophageal resection. The gastroplasty is delivered into the right thoracic cavity for an oesophago gastrostomy near the apex of the chest (Figure 66.52b /uni25CF McKeown or three-stage oesophagectomy . This con sists of the mobilisation of the thoracic oesophagus and lymphadenectomy via a thoracotomy (usually right side), followed by abdominal and neck incisions for preparation of the oesophageal substitute (usually the stomach) and its delivery to the neck for a cervical anastomosis. /uni25CF Left thoracic resection (Sweet oesophagectomy). Via a single posterolateral incision on the left chest wall (usually fifth to sixth intercostal space), the oesophagus is mobilised. The diaphragm is opened and the gastroplasty prepared from this opening. The stomach is delivered to the left tho racic cavity for anastomosis. Kenneth Charles McKeown , 1912–1995, surgeon, Darlington Memorial Hospital, Durham, UK. Richard H Sweet , 1901–1962, surgeon, Massachusetts General Hospital, Boston, MA, USA. /uni25CF Transhiatal oesophagectomy . Through a cervical and ab - - dominal approach, the oesophagus is mobilised via both). directions, being stripped out bluntly from its mediastinal - bed. The gastric conduit is delivered to the neck for cervi - cal anastomosis (Figure 66.53). /uni25CF Minimally invasive surgical approaches. Traditional open procedures (described above) are increasingly replaced by minimally invasive methods, by a combination of video- assisted thoracoscopy (V ATS) and

laparoscopy or robotic techniques (Figure 66.54). Both thoracic and abdominal phases can be performed via minimally invasive tech - niques, or one phase can be minimally invasive and the other by open surgery (hybrid procedures). The anastomo - - sis can be constructed in the chest or the neck.

Preoperative Preoperative counselling Nutritional assessment Nasogastric tube feeding for those with signi /f_i cant stenosis of the oesophagus, and oral supplement in those at risk of malnutrition Preoperative exercise General and incentive spirometry + pre-habilitation programme Stop smoking and alcohol intake Chest physiotherapy Carbohydrate loading on day of surgery No solid food 6 hours before and /f_l uid 2 hours before surgery. Carbohydrate loading night before and /f_i nishes 2 hours before surgery No need for bowel preparation unless colonic interposition is planned Intraoperative Prophylactic antibiotics DVT prophylaxis Mechanical +/– pharmacological Judicious use of intraoperative /f_l uids Avoid hypothermia Minimally invasive surgery if possible Epidural analgesia Postoperative Nutrition POD1 carbohydrate drink, gradual advancement to soft diet by POD5 (if no vocal cord palsy and assessment by speech therapist shows no risk of aspiration) PPN/TPN/feeding via jejunostomy in those at nutritional risk and oral intake insuf /f_i cient Nasogastric tube Removal on POD1 (if no vocal cord palsy and assessment by speech therapist shows no risk of aspiration) Analgesia Epidural analgesia/patient-controlled analgesia/multimodal analgesia Chest drain Single closed small-calibre drain (19Fr Blake drain), removal POD3–4 when output <200–300 /uni00A0 mL/day Early mobilisation From POD1, supervised by physiotherapist Urinary catheter Early removal as soon as close monitoring of urine output is not essential Intravenous /f_l uid Balanced intravenous /f_l uid to avoid over- and underhydration DVT prophylaxis DVT, deep vein thrombosis; POD, postoperative day; PPN, peripheral parenteral nutrition; TPN, total parenteral nutrition.

(b) Oesophagogastric junction cancer The options detailed above for intrathoracic cancers also apply to cancers of the OGJ. Suitability depends in part on the extent of oesophageal and gastric involvement by cancer and the intended extent of resection and lymphadenectomy . In addition, an extended total radical gastrectomy can be performed. The whole stomach and the lower oesophagus (accessed via the oesophageal hiatus from the abdomen) are resected and intestinal continuity is restored with a jejunal Roux loop (Roux-en-y reconstruction). In selected patients with early disease, a proximal gastrectomy can be performed as nodal spread to the distal stomach is rare. César Roux , 1857–1934, Professor of Surgery and Gynaecology , Lausanne, Switzerland, described this method of forming a jejunal conduit in 1908.

Figure 66.52 The common open approaches for surgery of the oesophagus: (a) left thoracoabdominal; (b) two-stage Lewis–Tanner (Ivor Lewis) approach. In the McKeown approach a third incision in the neck is made to allow anastomosis to the cervical oesophagus. (a) 5 mm 4 5 cm 5 6 10 mm 7 10 mm Figure 66.54 Port sites for video-assisted thoracoscopic oesophagectomy in the left lateral position (b) . Port sites can vary depending on the surgeon’s preference. Figure 66.53 Transhiatal oesophagectomy whereby the oesophagus is mobilised blindly using /f_i ngers from the neck and hand inserted from the abdomen. (b) (a) and laparoscopic gastric mobilisation

Lymphadenectomy ensures adequate nodal sampling for staging, improves local disease control and increasingly there is evidence to show the prognostic impact of extended lymphadenectomy . The most appropriate extent of lymph adenectomy remains somewhat controversial. Transhiatal

oesophagectomy does not allow adequate mediastinal nodal dissection (for the mid- and upper thoracic part oesophageal mobilisation is mostly a ‘blind’ procedure) and thus is often chosen by surgeons who perform only a limited lower medi astinal dissection f or OGJ adenocarcinoma. Squamous cell cancers are mostly more proximally located and the transhiatal approach may be dangerous except in early cancers. T he extent of lymphadenectomy can be defined as ‘fields’. Two-field dissection refers to lymphadenectomy of the mediastinum and upper abdomen around the coeliac trifur cation. The mediastinal ‘field’ is further classified as (i) stan dard: lymphadenectomy below the tracheal bifurcation, (ii) e xtended: standard lymphadenectomy plus right paratracheal nodal dissection including those around the right recurrent laryngeal nerve, and (iii) total: extended lymphadenectom plus nodal dissection along the left recurrent laryngeal nerve chain (Figure 66.55). The third field refers to bilateral cervical lymphadenectomy , including those in the paratracheal as well as supraclavicular fossae. The most appropriate extent of lymphadenectomy remains a contentious issue. For patients with squamous cell cancers, most surgeons would perform at least a total two-field showed significant nodal metastases, especially around the bilateral recurrent laryngeal nerves. In selected patients and in particular those with upper thoracic cancers, additional third - field nodal dissection is perfor med (three-field lymphadenec - - tomy). For oesophageal adenocarcinoma, most surgeons perform an infracarinal two-field lymphadenectomy . For OGJ tumours (in particular those with limited oesophageal extent and centre on the OGJ), surgeons are divided among those who prefer oesophagectomy and those who perform e xtended - total gastrectomy with limited lower oesophageal resection and lymphadenectomy . The issue is unsettled. The extent of resection (and lymphadenectomy) has to be balanced against associated morbidities and physiological reserve of the indi - vidual patient. - Reconstruction - Restoration of intestinal continuity after oesophageal extirpa - tion is mostly done using a gastric conduit. The right gastro - epiploic vessels are its main blood supply . A pyloric drainage y procedure is optional, with some surgeons advocating its use to facilitate gastric emptying, after the inevitable vagotomy . In the case of a previous gastr ectomy , or if concomitant pathology (such as gastric cancer) requires its removal, the colon (right ileocolon, left or transverse colon) can be used. The surgery is more extensive and three anastomoses are required. The conduit can be placed in the right thoracic cavity (as in after a Lewis–Tanner oesophagectomy) or the neck for cervical

(a) (b) Figure 66.55 The lymph node station nomenclature according to the Japanese classi /f_i cation. Extent of mediastinal lymphadenectomy. /uni00A0 (a) Standard mediastinal lymphadenectomy includes stations below the tracheal bifurcation. includes standard lymphadenectomy + right paratracheal nodal dissection including those around the right recurrent laryngeal nerve. mediastinal lymphadenectomy includes extended lymphadenectomy + left paratracheal area and nodes along the left recurrent laryngeal nerve. Two- /f_i eld lymphadenectomy includes mediastinal dissection plus nodal dissection around the coeliac axis and three- /f_i eld dissection includes cervical lymphadenectomy. (c) (b) Extended mediastinal lymphadenectomy (c) Total

choices of routes of reconstruction exist: posterior mediastinal, retrosternal or subcutaneous. Perioperative care For most patients, a standardised clinical pathway is helpful, along the lines of the ERAS protocol (Table 66.5). ERAS is a global perioperative quality improvement initiative based on attenuation of the stress response to surgical injury . The gastrointestinal system is

central to many of the core ERAS elements, including carbohydrate loading, no prolonged fasting, avoidance of mechanical bowel preparation, avoid ance of nasogastric intubation, maintaining fluid balance and early feeding. Employing these ERAS care practices leads to improved clinical outcome. Management of complications Complications are common as patients are often elderly with pre-existing morbidities and surgery is extensive. Atelectasis and pneumonia are managed by chest physiotherapy , adequate pain relief, avoidance of fluid overload, appropriate antibiotics and, if needed, sputum suction by bronchoscopy . Atrial fibrilla tion occurs in around 15–20% of patients; it is benign in most and is treated by antiarrhythmic medication. In some patients, it is a reflection of underlying serious complication, such as bronchopneumonia, or more importantly surgical morbidities such as anastomotic leak or ischaemia of the conduit. Its occurrence should prompt appropriate investigations, such as endoscopy . Recurrent laryngeal nerve injury is not uncommon when superior mediastinal lymphadenectomy or neck nodal dissection is carried out. Postoperatively the patient will expe rience hoarseness of the voice, coughing becomes less e ff ective and aspiration may be a problem when the diet is introduced. Active chest therapy and delay of oral intake may be necessary . and coughing e ff ort and lower the chance of aspiration. More definitive therapy may be needed if vocal cord function does not return. Gross ischaemia of the conduit usually presents within the first 2–3 days after the operation and dictates taking down of the conduit, adequate drainage and staged reconstruction later once sepsis is under control (Figure 66.56). In selected cases immediate reanastomosis is an option if the patient is haemo - dynamically stable and an adequate length of healthy stomach remains. Clinically apparent thoracic anastomotic leaks usually - occur within the first week. Signs of sepsis and excessive out - put from the chest drain, which may be turbid in colour, may lead to the diagnosis. The location and magnitude of the leak can be visualised by a water-soluble contrast study . A carefully performed flexible endoscopic examination is also helpful and will not worsen the leak. For small, contained leaks, CT-guided drainage or use of a luminal vacuum Endo-Sponge™ may suf - fice. In septic patients with a sizeable leak, exploration is war - ranted to establish drainage. Direct repair is seldom possible. For cervical anastomosis, leakage is suspected when there is inflammation and pain of the neck wound. Turbid infected - discharge is found when the skin stitches are removed. Leaks that are truly confined to the neck are simply trea ted by lay - ing the wound open with daily washing and frequent changes of dressing. Leaks that communicate with the mediastinum , may require for mal exploration and placement of mediastinal drains. In all leaks, treatment with broad-spectrum antibiotics is required, guided by microbial culture and sensitivity . Nutri - tional support is essential. With an intrathoracic stomach, care - - ful endoscopic placement of a fine-bore feeding tube into the duodenum for enteral feeding is useful. Injection of fibrin g lue, placement of intraluminal stents, use of a luminal vacuum

(a) (b) Figure 66.56 (a) Cervical wound with erythema, swelling and discharge of purulent material,

typical of a cervical anastomotic leak. (b) An ischaemic gas

tric conduit; 5 cm of the stomach appeared unhealthy and required resection.

increasingly used to treat leaks; sealing of the leak allows early control of sepsis and resumption of oral alimentation. The stent can be removed afterwards, depending on the severity of the leak in the first place. Usually , 4–6 weeks will su ffi ce for adequate healing. A chylous leak is suspected when there is excessive chest drainage. A milk challenge, looking at the colour of the e ffl uent before and after taking milk by mouth or via the nasogastric tube, will usually be obvious. This can be aided by biochemical testing of the drain fluid, measuring triglyceride level or chylo microns. In a low-output fistula of less than 0.5–1 litre per day , conservative management with total parenteral nutrition or a mid-chain triglyceride diet may su ffi ce. In case of persistence or if the output is more than 1 litre per day , prolonged conservative treatment is not recommended and early re-exploration is warranted. A lymphangiogram preoperatively will help locate the site of leakage, and intraoperative milk feed will also serve the same purpose. The site of the leak can then be clipped or sutured. Increasingly , however, the interventional radiological method of percutaneous embolisation has gained success and has reduced the need for surgical re-exploration.