Oesophagus

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2.1 Anatomy

The oesophagus is a tubular structure, approximately 25 cm long, extending from the laryngeal part of the pharynx at the level of the sixth cervical vertebra, passing through the diaphragm at the level of the tenth thoracic vertebra to join the stomach at the oesophagogastric (OG) junction (Fig. 2.1). For purposes of practicality during endoscopic procedures, the site of a lesion in the oesophagus is given as the distance from the upper incisor teeth. As it is approximately 16 cm from the upper incisor teeth to the proximal oesophageal limit, the OG junction is at approximately 40–41 cm. The oesophagus traverses the neck, thorax, and enters the abdominal cavity and so can be anatomically divided into three subsites:

1. Cervical oesophagus—2–3 cm long and extends from the proximal oesophageal limit (C6) to the thoracic inlet, which is marked by the surface landmark of the suprasternal notch of the sternum (breast bone).
2. Intrathoracic oesophagus—approximately 21 cm long and extends from the thoracic inlet to the oesophageal hiatus in the diaphragm. At 25 cm from the upper incisor teeth, the oesophagus is constricted by the aortic arch and the left main bronchus crossing its anterior surface.
3. Abdominal oesophagus—1–1.5 cm long and extends from the oesophageal hiatus in the diaphragm to the right side of the stomach. It is covered anterolaterally by the peritoneum and comes into close relationship with the left lobe of liver.

An internal landmark of relevance to determining the site of origin of an OG tumour is the OG junction where the pale oesophageal squamous mucosa meets the glandular mucosa of the gastric cardia. The OG junction can be somewhat irregular in outline (the Z line) and does not necessarily correspond to the lower physiological valve or sphincter. External landmarks are distal oesophagus orientated to adventitial fat while the junctional area and proximal stomach relate to a covering of serosa or peritoneum. Thus, a tumour of the distal oesophagus or OG junction can spread through the wall either to adventitial fat of the mediastinum or the abdominal peritoneum. Adventitial fat is disposed laterally, but absent anteriorly and posteriorly where the oesophagus

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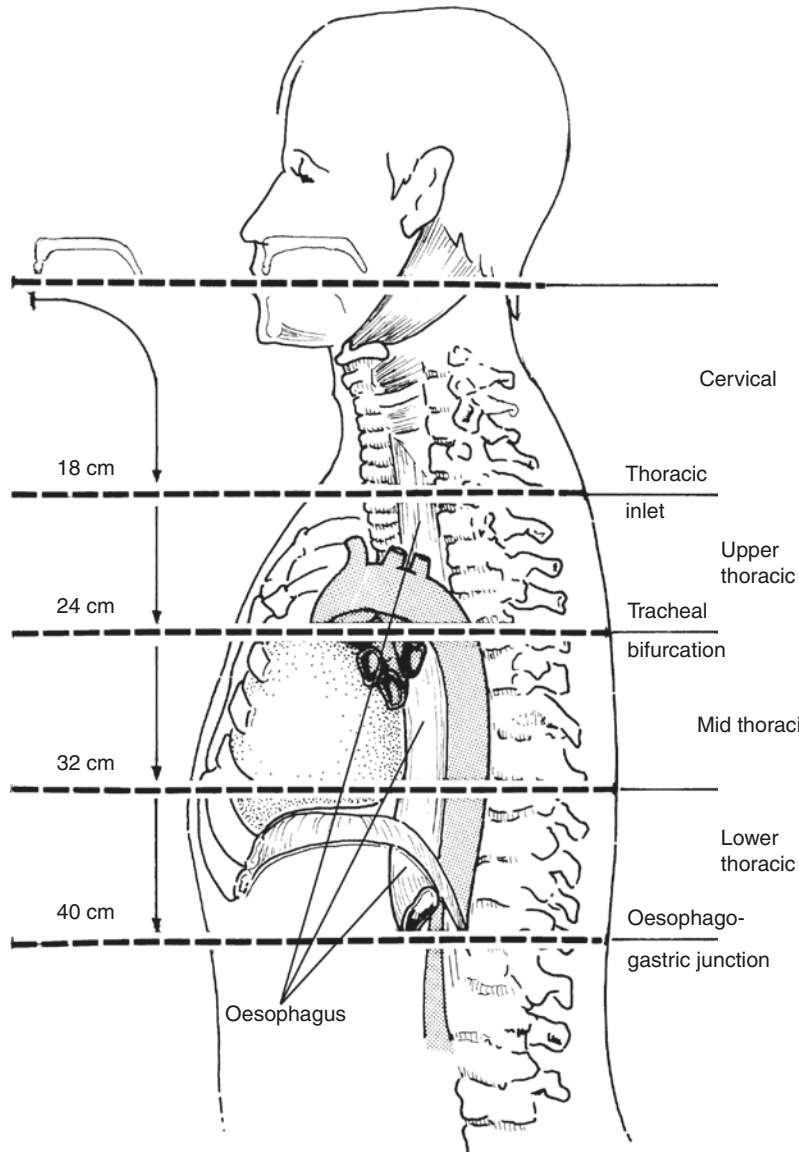

Fig. 2.1 Oesophagus (Used with the permission of the Union for International Cancer Control (UICC), Geneva, Switzerland. The original source for this material is from Wittekind et al. (2005))

is adjacent to the heart and vertebral column, respectively. Note that the adventitia of the mid-oesophagus may also relate to a serosal surface—that of resected mediastinal pleura.

As well as determining the position of the lesion within the oesophagus by its anatomical site, it can also be defined by its relative position in the upper, middle, or lower third of the oesophagus. This is of relevance clinically as the lymphovascular drainage is considered in these terms and is therefore important in cancer surgery.

Lymphovascular drainage (Fig. 2.2):

Upper third—deep cervical nodes

Middle third—superior and posterior mediastinal nodes

Lower third—nodes along the left gastric blood vessels and the coeliac nodes

Venous drainage from the middle third (azygos vein) into the lower third (gastric vein) leads to the formation of a porto-systemic anastomosis and, with raised portal venous pressure (e.g., as in liver cirrhosis), the possibility of the formation of oesophageal varices (dilatation of oesophageal veins).

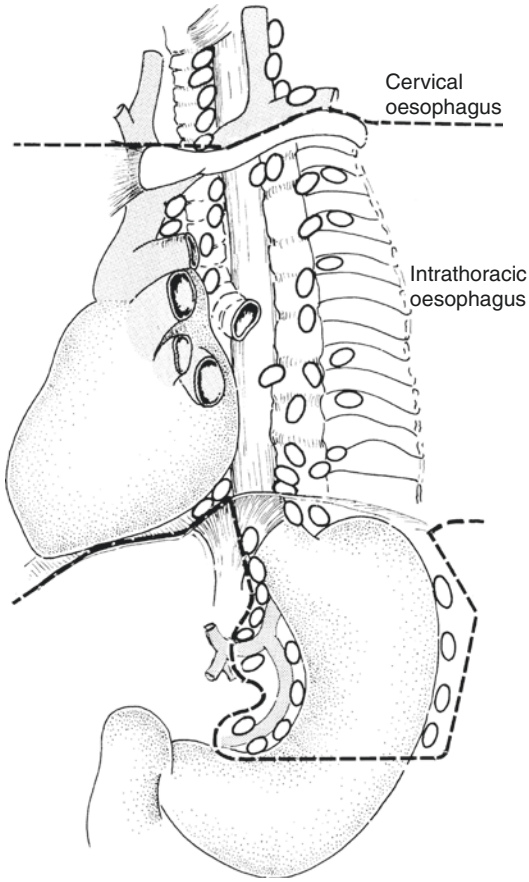

Fig. 2.2 The regional lymph nodes, irrespective of the site of the primary tumour, are those in the oesophageal drainage area including coeliac axis nodes and paraoesophageal nodes in the neck, but not supraclavicular nodes (Used with the permission of the Union for International Cancer Control (UICC), Geneva, Switzerland. The original source for this material is from Wittekind et al. (2005))

2.2 Clinical Presentation

Patients with oesophageal disease may be asymptomatic, but usually experience one or more of the following: chest pain, heartburn (a retrosternal burning sensation), reflux of acid/food, and dysphagia (difficulty swallowing). Dysphagia can be painful (odynophagia) and progressive due to benign or malignant strictures, i.e., initially for solid foods, e.g., meat, then soft foods, and ultimately liquids. Patient localization of the site of obstruction can be poor. Occult bleeding can lead to iron-deficiency anaemia, while haemorrhage (haematemesis) can be potentially life threatening (varices) or self-limiting due to linear tears of the OG junction mucous membrane after prolonged vomiting (Mallory-Weiss syndrome).

2.3 Clinical Investigations

- Endoscopy and biopsy.
- CXR to detect any enlargement of the heart, mediastinal lymph nodes, or pulmonary hilum that might extrinsically press on the oesophagus. Barium swallow to outline the contour of the oesophageal lumen and wall, and assess motility/swallowing.
- For biopsy-proven cancer—ELUS and PET CT scan chest and abdomen to determine the pretreatment tumour stage directed toward pT/pN and pM disease, respectively.
- Laparoscopy with peritoneal washings for OG junctional and select distal oesophageal adenocarcinoma cases.
- Twenty-four hour pH monitoring—has a high diagnostic sensitivity for reflux oesophagitis. Oesophageal manometry can assess the effectiveness of motility, e.g., achalasia, scleroderma.

2.4 Pathological Conditions

2.4.1 Non-neoplastic Conditions

Reflux oesophagitis: usually due to hiatus hernia (slippage of the OG junction into the thorax) resulting in gastro-oesophageal reflux (GOR) of acid and bile; there is poor correlation with symptoms, endoscopy and biopsy being normal in 20–30% of cases. Otherwise well-orientated biopsies show basal zone hyperplasia and prominent vascularized connective tissue papillae. This is superseded by inflammatory infiltrates of neutrophils and eosinophils, surface erosion, full thickness ulceration, and, ultimately, fibrous stricture formation (10% of cases). It may require operative dilatation (often repeatedly) to relieve

dysphagia and, although it usually has a smooth outline, it may be difficult to distinguish endoscopically from a malignant growth. Prior to this, treatment of GOR is either medical (weight loss, antacids) or occasionally surgical. This is usually done laparoscopically by wrapping the fundus of the stomach around the distal oesophagus (Nissen fundoplication) to maintain lower oesophageal tone and retain it in the abdominal cavity.

Eosinophilic oesophagitis: may be associated with trachealization of the oesophagus or a "feline" or striped appearance at endoscopy particularly in younger males. It is characterised by increased numbers of eosinophils (>15 per hpf), transmucosal distribution, eosinophilic microabscess formation and involvement of mid oesophagus on biopsy.

Infective oesophagitis: may be seen in otherwise healthy individuals but is more commonly encountered where there is alteration of either local or systemic immunity (e.g., HIV-AIDS). Underlying ulceration, broad-spectrum antibiotics, diabetes, corticosteroid therapy, and immunosuppressive drugs can all alter the local gut flora resulting in superimposed infection. Causative agents are candidal fungus, herpes simplex virus (HSV 1 and 2), cytomegalovirus (CMV), and atypical mycobacteria.

Miscellaneous: Other causes of oesophagitis, ulceration, and/or stricture are drugs (e.g., NSAIDs, aspirin), mediastinal radiotherapy, motility disorders (e.g., achalasia), Crohn's disease and direct injury (foreign body, prolonged nasogastric intubation, corrosive ingestion).

Incidental endoscopic findings are inflammatory or fibrovascular polyps of the OG junction.

2.4.2 Neoplastic Conditions

Benign tumours: These are rare in surgical material, e.g., squamous papilloma, leiomyoma, or granular cell tumour.

Oesophageal carcinoma: Predisposing conditions to oesophageal cancer include GOR, obesity, diverticula, achalasia, and Plummer–Vinson syndrome (elderly females, iron-deficiency anaemia, upper oesophageal web). Predisposing lesions to oesophageal cancer are squamous cell dysplasia and Barrett's metaplasia/dysplasia.

Squamous cell dysplasia/carcinoma in situ: Macroscopically often inapparent but seen histologically adjacent to, overlying or distant from squamous cell carcinoma.

Barrett's metaplasia or columnar epithelium lined lower o*esophagus* (*CLO*): Seen in about 10% of patients with hiatus hernia and/or GOR. It arises from erosion with differentiation of multipotential stem cells to metaplastic small intestinal or gastric glandular epithelia. The Barrett's segment appears as a velvety area proximal to the OG junction surrounded by pale squamous mucosa. It can be multifocal or continuous. The segment is either classical/long (≥3 cm) or short (<3 cm).

Whilst Barrett's oeosophagus is associated with an increased risk of developing oesophageal adenocarcinoma, population based studies have shown lower rates of progression (0.5% per patient per year) than previously suggested. Surveillance for Barrett's oeosphagus is currently recommended by many groups, despite the lack of formal evidence from randomized controlled trials.

Dysplastic change within Barrett's mucosa is associated with an increased risk of progression. Higher rates of progression have been reported if the diagnosis of dysplastic change is made infrequently, is made by a specialist (rather than community based) pathologist, is confirmed by two or more independent pathologists, or if accompanied by aberrant p53 staining or abnormal DNA content.

Dysplastic Barrett's mucosa may be treated endoscopically by radiofrequency ablation (RFA) with relatively low complication rates and high rates of complete eradication reducing the risk of disease progression.

The appearances of Barrett's metaplasia may be significantly altered by its treatment with antacid medication, ablative techniques, or photodynamic therapy.

Squamous cell carcinoma: Forms 30–40% of oesophageal cancers and is typically seen in the mid-oesophagus of elderly patients. It is usually moderately differentiated and keratinizing, ulcerates or strictures with rolled, irregular margins,

involves a long segment of oesophagus, and has spread through the full thickness of the wall at presentation. Palliation can be achieved by chemoradiation, ablative laser therapy, or the insertion of an expanding metal stent or tube to relieve obstruction. Primary treatment in a medically fit patient with a locally confined lesion <5–10 cm in length may entail radical chemoradiotherapy alone or in the UK, neoaduvant chemotherapy with subsequent surgery. Preoperative chemoradiotherapy produces signs of tumour regression (degeneration, necrosis, fibrosis, keratin granulomas) in some 50–60% of cases, but often makes identification of tumour on gross inspection of the specimen difficult. Perforation with potentially fatal mediastinitis is a possible complication of preoperative therapy and endoscopy of malignant strictures. Depending on the CT chest findings, bronchoscopy is sometimes done to exclude the possibility of a primary lung cancer invading oesophagus, which would preclude primary resection as do haematogenous and distant nodal metastases or invasion of mediastinal vessels and main structures.

Variants of squamous carcinoma are verrucous carcinoma (warty, slow growth), basaloid carcinoma (aggressive), and spindle cell/polypoid carcinoma (carcinosarcoma—intermediate prognosis).

Adenocarcinoma: Forms 50–60% of oesophageal cancers and arises in the distal oesophagus/OG junction, often secondary to intestinal-type Barrett's metaplasia and dysplasia. Over 90% of oesophageal adenocarcinoma patients present symptomatically with established malignancy, often late stage disease. Less than 10% of patients will have had a previous endoscopic biopsy diagnosis of Barrett's oesophagus. The incidence of this tumour has greatly increased in the last 20 years due in part to antibiotic eradication of helicobacter pylori with loss of its gastric acid suppressor effect, resulting in more GOR disease. As well as extensive radial spread through the wall out to the circumferential radial margin (CRM), it can spread upward, undermining the oesophageal squamous mucosa and downward to the proximal stomach where clear distinction from a primary gastric carcinoma can be difficult. Clues as to site of origin are both anatomical and histological in the adjacent mucosa (oesophagus—Barrett's metaplasia/dysplasia; stomach—gastritis/intestinal metaplasia/dysplasia). TNM 8 includes as an oesophageal cancer any tumour of the proximal stomach where its epicentre is within 2 cm of the OG junction and involves the oesophagus (Siewert 3). Adenocarcinoma is usually ulcerated with irregular rolled margins or polypoid, and histologically tubular or papillary with an intestinal glandular pattern but sometimes of diffuse signet ring cell type.

Intramucosal adenocarcinoma may be treated by endoscopic mucosal resection (EMR) of visible nodules/plaques and RFA to the remaining flat Barrett's mucosa. Submucosal invasion (pT1b) is associated with a markedly increased risk of nodal metastasis and is generally an indication for radical surgical resection. More advanced disease is optimally managed by perioperative chemotherapy combined with surgical resection.

Other features: Oesophageal cancer tends to show multifocality (15–20%). Examination of specimen proximal and distal surgical margins is therefore important. "Early" or superficial squamous carcinoma is confined to the mucosa or submucosa with or without regional lymph node involvement and is of better prognosis than "advanced" or deep muscle invasive carcinoma. Involvement of the perioesophageal CRM is partly dependent on individual patient anatomy but is also an indicator of extent of tumour spread, adequacy of surgical resection, and potential local recurrence due to residual mediastinal disease.

Other cancers: Rare but can include small cell carcinoma, malignant melanoma, leukaemia/malignant lymphoma, metastatic cancer (e.g., lung or breast), leiomyosarcoma, and Kaposi's sarcoma (HIV-AIDS).

Prognosis: Prognosis of oesophageal cancer is poor (5-year survival 5–15%) relating mainly to depth of spread and lymph node involvement, i.e., tumour stage, and involvement of longitudinal and circumferential excision margins. Early or superficial carcinoma does significantly better—55% → 88% 5-year survival depending on the depth of mucous membrane invasion.

2.5 Surgical Pathology Specimens: Clinical Aspects

2.5.1 Biopsy Specimens

Two main types of oesophageal endoscopy exist, namely rigid and flexible. Rigid oesophagoscopy is only occasionally used to provide larger biopsies when previous flexible endoscopy (OGD—oesophagogastroduodenoscopy) samples have proven non-diagnostic. Specific lesions such as polyps or ulcers necessitate multiple targeted biopsies that may be supplemented by brush cytology of the mucosal surface. Mapping and annual/biennial surveillance of flat mucosa for Barrett's metaplasia and dysplasia is achieved by multiple segmental (every 2 cm) and quadrantic biopsies. The clinical extent of the Barrett's is described according to its length of circumferential disposition (cm) and total length (cm) of the metaplastic segment respectively, e.g., C2M6. The basis of an oesophageal stricture may be easier to demonstrate if malignant in nature because of carcinoma ulcerating the squamous epithelium, whereas a benign peptic stricture due to submucosal or mural fibrosis is often not accessible to mucosal biopsy. Endoscopic biopsy of achalasia or oesophageal webs is often unrewarding as it provides intact surface mucosa only.

2.5.2 EMR Specimens

EMR ("big biopsy") specimens can be both diagnostic, staging and therapeutic, e.g., in dysplastic Barrett's to diagnose and remove any "early" nodular areas of carcinoma confined to the mucosa.

2.5.3 Resection Specimens

The surgical techniques for resecting oesophageal tumours fall into two broad categories—those which employ a chest incision (thoracotomy) and those which do not (transdiaphragmatic hiatal procedures). The type of procedure used depends on the general level of health of the patient, any previous operations, the preference of the operating surgeon, the size and position of the tumour in the oesophagus (see Table 2.1), and the choice of oesophageal substitute, i.e., stomach, jejunum, or colonic interposition. Ideally the surgeon should strive for a 5 cm longitudinal margin of clearance with adenocarcinoma and 10 cm for squamous carcinoma, with an appropriate lymphadenectomy. There is currently no evidence-based favoured method of resection.

Table 2.1 Choice of surgical procedure in oesophageal neoplasia

Proximal 1/3 tumours	Pharyngo-oesophagectomy
Middle 1/3 tumours	Ivor Lewis technique
	Thoracoabdominal oesophagectomy
	Two-field transhiatal oesophagectomy
Lower 1/3 tumours	Ivor Lewis technique
	Thoracoabdominal oesophagectomy
	Transhiatal oesophagectomy
Barrett's	Transhiatal oesophagectomy

2.5.3.1 Procedures Employing a Thoracotomy

(a) *Ivor Lewis technique*—in this operation, upper abdominal and right thoracotomy incisions are made. The proximal stomach is divided and the oesophagus is transected proximal to the tumour. The distal stomach is then raised into the chest and an OG anastomosis is fashioned.

(b) *Thoracoabdominal oesophagectomy*—a continuous incision extending from the midline of the upper abdomen running obliquely across the rib margin and posterolateral aspect of the chest wall. The left diaphragm is divided and this gives access for potential en bloc resection of the oesophagus, stomach, gastric nodes and, if required, the spleen and distal pancreas. An oesophagojejunal or OG anastomosis is fashioned in the neck.

2.5.3.2 Transhiatal Oesophagectomy

Depending on whether a total or distal oesophagectomy is to be performed, two variations of this procedure are used.

(a) "Two-field approach"—the entire oesophagus and stomach is mobilized via upper abdomi-

nal and oblique neck incisions. The cervical oesophagus is divided and anastomosed to stomach which had been mobilized and raised high into the posterior mediastinum.

(b) Distal oesophagectomy with proximal gastrectomy (for distal oesophageal/junctional tumours)—only an upper abdominal incision is used, with the distal oesophagus being mobilized and an OG anastomosis fashioned in the chest.

Although transhiatal resection for diseases of the thoracic oesophagus used to be uncommon, it is now more commonly used, reducing the physiological insult experienced with a thoracotomy. Minimally invasive oesophagectomy (MIO) procedures are being developed using combined laparoscopic and thoracoscopic techniques.

Whenever possible the stomach should be used in the anastomosis and with appropriate mobilization the stomach will reach the neck in virtually all patients. If the tumour is limited to the OG junction, the entire greater curvature of the gastric fundus (shaded area in Fig. 2.3), including the point which usually reaches most cephalad to the neck (*in Fig. 2.3), may be preserved while still obtaining a 4–6 cm gastric margin distal to the malignancy.

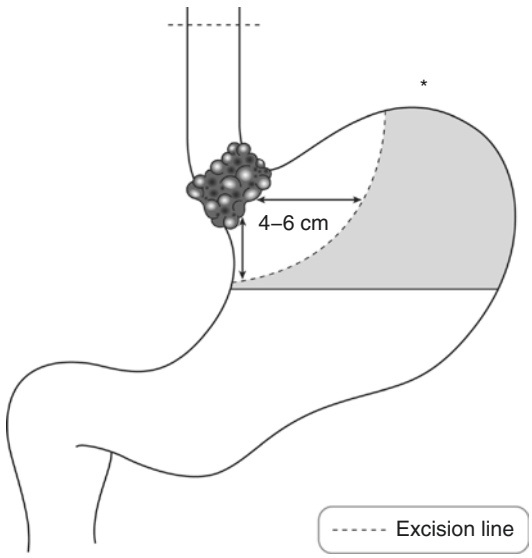

Fig. 2.3 Transhiatal oesophagectomy with limited proximal gastrectomy (Reproduced, with permission, from Allen and Cameron (2013))

There are several benefits in performing a total thoracic oesophagectomy with cervical anastomosis: maximum clearance of surgical margins is obtained while the risk of mediastinitis, sepsis, and GOR that can be seen with an intrathoracic anastomosis is diminished.

2.6 Surgical Pathology Specimens: Laboratory Protocols

2.6.1 Biopsy Specimens

See Chap. 1.

2.6.2 EMR Specimens

The majority of EMR specimens are submitted "piecemeal" to the laboratory in contrast to Endoscopic Submucosal Dissection (ESD) which is more likely to produce a single disc shaped specimen. EMRs should be examined to identify the mucosal surface and the cauterised deep margin which may be inked. Smaller specimens without a macroscopically visible surface lesion should be serially sectioned and all tissue processed for histological examination through levels, with the judicious application of special stains to facilitate identification of lymphovascular/venous invasion. The use of sponges within specimen casettes may help prevent artefactual curling up and twisting of the specimens.

2.6.3 Resection Specimens

Specimen

- Most oesophageal resections are for neoplastic conditions: partial oesophagectomy, total thoracic oesophagectomy (TTO), oesophagectomy with limited gastrectomy, oesophagogastrectomy
- The oesophagus can easily lose 25–33% of its length after removal from the patient and fixation in formalin. The length of tumour and distances from longitudinal resection margins

will be potentially affected and a strong case can be made for pinning the specimen out fresh onto a board as soon as possible prior to immersion in formalin.

Initial procedure

- By palpation and with the index finger locate the luminal position of the tumour.
- Paint the overlying external CRM comprising adventitial fatty connective tissue and any related serosa.
- Most authorities (and clinical trial protocols) recommend leaving the tubular oesophagus unopened and serial transverse slicing after the painted margin has dried (Fig. 2.4). This method facilitates assessment of the CRM in T3 disease and is also useful where perioperative chemotherapy makes the macroscopic identification/assessment of tumour/tumour bed difficult.
- Opening the specimen longitudinally (after inking) may be considered in cases of dysplastic Barrett's oesophagus or T1/T2 disease (e.g., post EMR) where identification of suspicious ulcerated or nodular areas of mucosa can greatly facilitate appropriate block selection and where the CRM is not likely to be threatened by the primary tumour (Fig. 2.5).
- Measurements:
  - Oesophagus—length (cm), width (cm)
  - Proximal stomach—lengths (cm) along lesser and greater curvatures
  - Tumour—length × width × depth (cm) or maximum dimension (cm)
  - Distances (cm) to the proximal and distal limits of resection
  - Distance (cm) to the OG junction if the tumour is mid-oesophageal in location
  - Relationship to the OG junction: distal oesophageal tumour involving the junction (Siewert 1), tumour straddling the junction (Siewert 2), proximal gastric tumour involving the junction (Siewert 3).
  - Note that the junction may be obscured by tumour, and external landmarks (oesophagus—adventitia; stomach—serosa) should also be used in determining the location.
  - Barrett's mucosa—location/length (cm).
- Photograph.
- Fixation by immersion in 10% formalin for 48 h preferably pinned out on a corkboard in the opened position but not placed under tension (to avoid splitting).

Description

- Tumour
  - Polypoid: spindle cell carcinoma/carcinosarcoma
  - Warty/verrucous: verrucous carcinoma
  - Nodular/plaque: superficial carcinoma
  - Fungating/strictured/ulcerated/infiltrative edge: usual carcinoma
  - Multifocal
  - Regression and scarring
- Mucosa
  - Barrett's mucosa (velvety appearance)
- Wall
  - Tumour confined to mucous membrane, in the wall or through the wall
- Other
  - Achalasia, diverticulum, mucosal web, perforation.

Blocks for histology (Figs. 2.4 and 2.5)

- Sample the proximal and distal limits of surgical resection—complete circumferential transverse section (oesophagus) or multiple circumferential blocks (proximal stomach).
- Alternatively, if separate anastomotic doughnuts are submitted, take one complete circumferential transverse section of each.
- Serially section the bulk of the tumour transversely at 3–4 mm intervals.
- Lay the slices out in sequence and photograph.
- Sample a minimum of four blocks of tumour and wall to show the deepest point of circumferential invasion (Fig. 2.4).
- Sample one block of oesophagus proximal to the tumour and one block of oesophagus (or proximal stomach) distal to the tumour.
- Sample any abnormal background mucosa, e.g., multiple sequential blocks may be required to map the extent of Barrett's metaplasia.

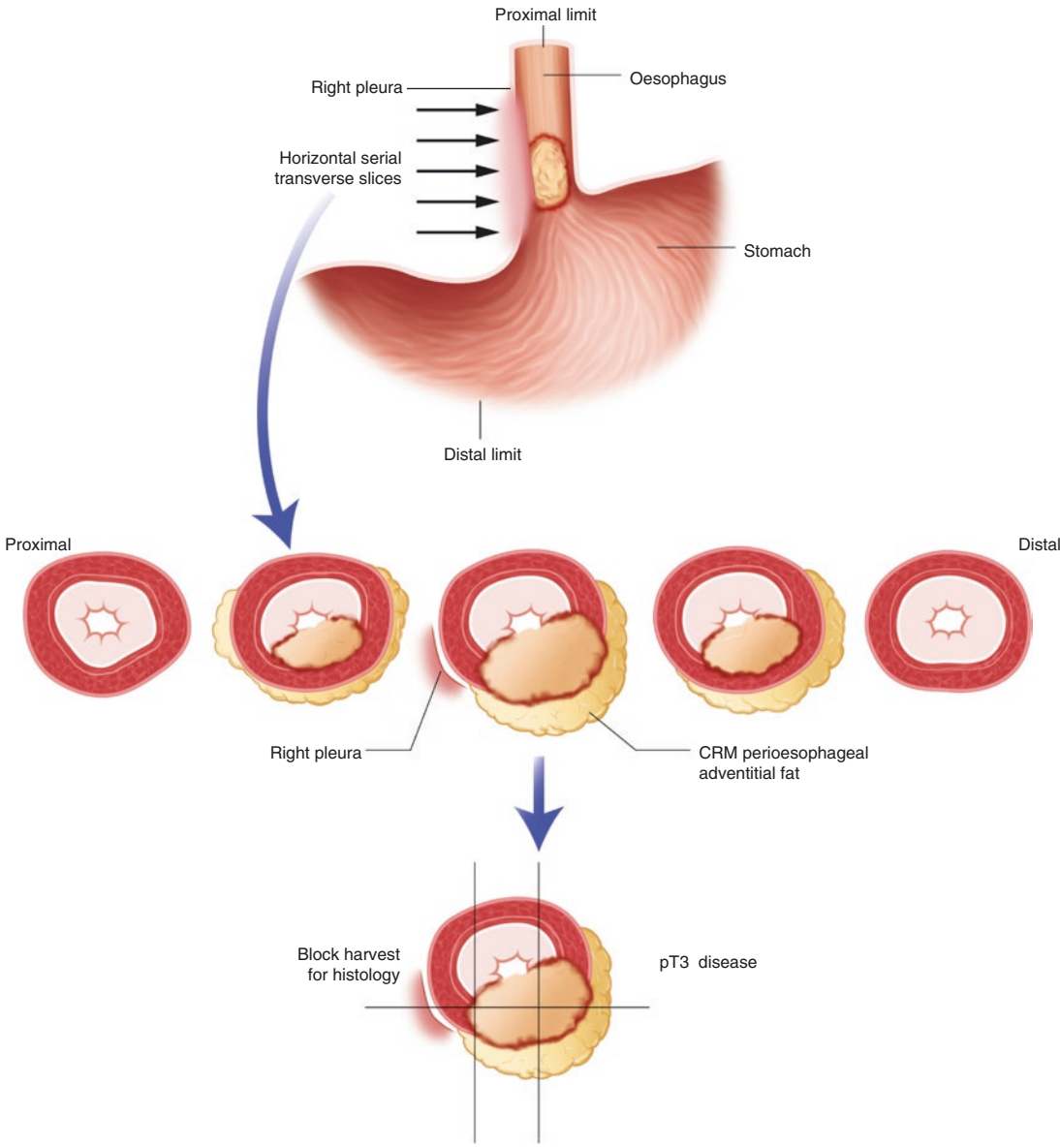

Fig. 2.4 Oesophageal carcinoma: unopened, painted (CRM) oesophagogastrectomy specimen cut into horizontal, serial transverse slices for histology block harvest

- If tumour is not seen grossly, sequentially sample and correspondingly label unremarkable and abnormal areas of mucosa.
- Count and sample all lymph nodes.
- Sample the midpoint and proximal surgical limit (as marked by the surgeon) of any separate proximal segment of normal oesophagus excised to facilitate pull-through of the OG anastomosis to the neck.
- *Histopathology report*
- Tumour type—adenocarcinoma/squamous carcinoma/other

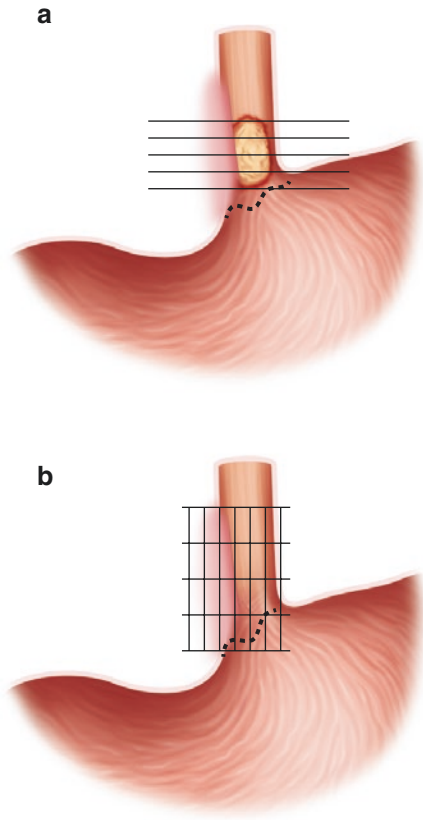

Fig. 2.5 Oesophageal carcinoma (T1/T2 disease) or Barrett's dysplasia: opened oesophago-gastrectomy specimen for (**a**) targeted or (**b**) sequential grid blocks

- Tumour differentiation

	Adenocarcinoma	Squamous carcinoma
Well	>95% glands	Keratinization/ intercellular bridges
Moderate	50–95% glands	
Poor	<50% glands	No keratinization/ intercellular bridges

- Tumour edge—pushing/infiltrative/lymphoid response
- *Extent of local tumour spread: TNM 8: for carcinoma*

pTis	Carcinoma in situ/high-grade dysplasia
pT1	Tumour invades pT1a. lamina propria or muscularis mucosae, or, pT1b. submucosa
pT2	Tumour invades muscularis propria
pT3	Tumour invades adventitia
pT4	Tumour invades adjacent structures (pT4a: pleura/pericardium/diaphragm/peritoneum, pT4b: aorta/vertebral body /trachea)

Note also any invasion of the proximal gastric serosa.

Siewert 1–3 tumours are staged as oesophageal under TNM 8.

- Lymphovascular invasion—present/not present. Note perineural invasion.
- Regional lymph nodes
- Perioesophageal, including coeliac axis nodes and paraoesophageal nodes in the neck, but not supraclavicular nodes. A regional lymphadenectomy will ordinarily include 7 or more lymph nodes.

pN0	No regional lymph node metastasis
pN1	Metastasis in 1–2 regional lymph node(s)
pN2	Metastasis in 3–6 regional lymph nodes
pN3	Metastasis in 7 or more regional lymph nodes

- Excision margins
  Proximal and distal limits of tumour clearance (cm)
  Separate proximal oesophageal and distal gastric anastomotic doughnuts—involved/not involved
  Deep CRM of clearance (mm)
- Other pathology:
- Squamous dysplasia, Barrett's metaplasia/dysplasia, radio−/chemotherapy necrosis and Tumour Regression Grade (Mandard score: TRG1 no residual tumour—TRG5 absence of regressive changes), perforation, achalasia, oesophageal web, diverticulum

Bibliography

Allen DC. Histopathology reporting. Guidelines for surgical cancer. 3rd ed. London: Springer; 2013.

Allen DC, Cameron RI. Histopathology specimens: clinical, pathological and laboratory aspects. 2nd ed. Berlin: Springer; 2013.

Allum WH, Blazeby JM, Griffin SM, Cunningham D, Jankowski JA, Wong R. On behalf of the Association of Upper Gastrointestinal Surgeons of Great

Britain and Northern Ireland, the British Society of Gastroenterology and the British Association of Surgical Oncology. Guidelines for the management of oesophageal and gastric cancer. Gut. 2011;60:1449–72.

Bejerano PA, Berho M. Examination of surgical specimens of the esophagus. Arch Pathol Lab Med. 2015;139:1446–54.

Bosman FT, Carneiro F. WHO classification of tumours of the digestive system. 4th ed. Lyon: IARC Press; 2010.

Brierley JD, Gospodarowicz MK, Wittekind C, editors. TNM classification of malignant tumours. 8th ed. Oxford: Wiley-Blackwell; 2017.

Cotton P, Williams C. Practical gastrointestinal endoscopy. 4th ed. London: Blackwell Science; 1996.

Ibrahim NBN. Guidelines for handling oesophageal biopsies and resection specimens and their reporting. J Clin Pathol. 2000;53:89–94.

Lewin KJ, Appelman HD. Tumors of the esophagus and stomach, Atlas of tumor pathology, vol. 3rd series. Fascicle 18. Washington, DC: AFIP; 1996.

Logan RPH, Harris A, Misciewicz JJ, Baron JH, editors. ABC of the upper gastrointestinal tract. London: BMJ Books; 2002.

Mandard AM, Dalibard F, Mandard JC, Marnay J, Henry-Amar M, Petiot JF, Roussel A, et al. Pathologic assessment of tumour regression after preoperative chemoradiotherapy of oesophageal carcinoma. Cancer. 1994;73:2680–96.

Odze RD, Goldblum JR, editors. Odze and Goldblum Surgical pathology of the GI tract, liver, biliary tract, and pancreas. 3rd ed. Philadelphia: Elsevier Saunders; 2015.

Schlemper RJ, Riddell RH, Kato Y. The Vienna classification of gastrointestinal epithelial neoplasia. Gut. 2000;47:251–5.

Shepherd NA, Warren BF, Williams GT, Greenson JK, Lauwers GY, Novelli MR, editors. Morson and Dawson's gastrointestinal pathology. 5th ed. Oxford: Wiley-Blackwell; 2013.

The Royal College of Pathologists. Cancer datasets (oesophageal carcinoma, gastric carcinoma, carcinomas of the pancreas, ampulla of vater and common bile duct, colorectal cancer, gastrointestinal stromal tumours (GISTs), liver resection specimens and liver biopsies for primary and metastatic carcinoma, endocrine tumours of the gastrointestinal tract including pancreas) and tissue pathways (gastrointestinal and pancreatobiliary pathology, liver biopsies for the investigation of medical disease and for focal liver lesions). https://www.rcpath.org/profession/publications/cancer-datasets.html. Accessed Sept 2016.

Wittekind C, Greene L, Hutter RVP, Klimfinger M, Sobin LH. TNM atlas: illustrated guide to the TNM/pTNM classification of malignant tumours. 5th ed. Berlin: Springer; 2005.