Esophagus: Normal Anatomy, Function and Congenital Anomalies

7

Laura R. Carucci

The chapter will review the normal anatomy and function of the esophagus. The expected appearance of the esophagus on barium studies and computed tomography (CT) will be described. The technical details of performing these studies have been discussed elsewhere in this section. Congenital variants and anomalies affecting the esophagus are also included.

Normal Esophageal Anatomy

The esophagus is a hollow muscular tube that connects the pharynx to the stomach, extending for approximately 20–24 cm in length (Dodds 1989). The main function of the esophagus is to transport ingested material from the hypopharynx to the stomach and prevent involuntary regurgitation of gastric contents. The esophagus is bordered superiorly by the upper esophageal sphincter (UES, pharyngoesophageal segment) and inferiorly by the lower esophageal sphincter (LES, gastroesophageal junction) (Fig. 7.1) (Ott 2008).

The esophagus can be divided into three anatomic segments: cervical, thoracic, and abdominal. The cervical esophagus is posterior to the trachea and anterior to the cervical spine. The thoracic esophagus is in the posterior mediastinum adjacent to the trachea, heart, lungs, major vessels, and vertebrae (Chevallier et al. 1991) (Fig. 7.2). The superior thoracic esophagus is most closely associated with the trachea and the lower esophagus with the aorta. The abdominal esophagus extends for up to 3 cm in length and terminates at the gastroesophageal junction.

The esophageal layers include the mucosa, muscularis mucosa, submucosa, and the outer and inner muscularis propria. The esophagus does not contain a serosal layer and as a consequence malignant lesions can spread rapidly into adjacent structures. The esophageal mucosa consists of stratified squamous epithelium with a transition to columnar epithelium at the gastric cardia. The esophageal muscularis propria is composed of two different muscle types with striated muscle in the upper third and smooth muscle in approximately the lower two thirds of the esophagus. The transition from striated to smooth muscle is located in the vicinity of the aortic arch (Grishaw et al. 1996; Ott 2008). Minor motility alterations may be visible at fluoroscopy at this level, including proximal escape (see "Normal Esophageal Function" below). Motility disorders may selectively involve striated or smooth muscle with abnormal motility in primarily the proximal third or distal two thirds of the esophagus respectively (i.e., abnormal motility can be seen in the distal two thirds of the esophagus in scleroderma, a smooth muscle connective tissue disorder).

There are two esophageal sphincters that maintain a high resting pressure as compared with the adjacent luminal segment: the UES and LES. Normally, both sphincters are closed at rest and open when swallowing. The UES regulates the opening and closing of the upper esophagus. The UES is 1–3 cm in length, located at the transition between the pharynx and esophagus (i.e., the pharyngoesophageal junction), and formed primarily by the cricopharyngeal muscle. The distal esophagus is regulated by the LES.

L.R. Carucci
Department of Radiology, Virginia Commonwealth University Medical Center, Richmond, VA, USA

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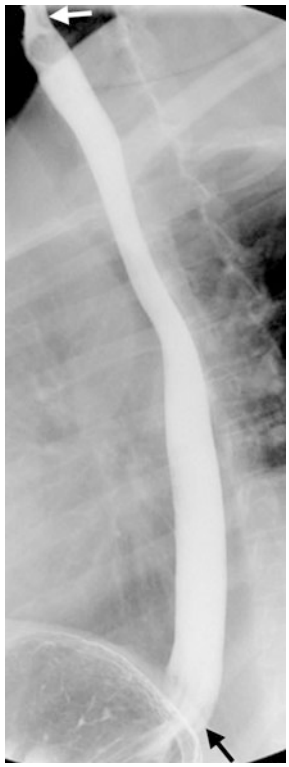

Fig. 7.1 Normal course of the esophagus on esophagography: A prone right anterior oblique image with the patient continuously drinking shows the normal course of the esophagus from the level of the pharyngoesophageal junction (*white arrow*) to the gastroesophageal junction (*black arrow*)

The LES is a physiologic sphincter rather than a distinct muscular entity (Mittal and Balaban 1997; Ott 2008). It is defined manometrically by a high-pressure zone extending for 1–4 cm in length between the esophagus and the stomach (Mittal and Balaban 1997). Although the esophagus is predominantly tubular and nondistensible, there is a normal expansion of the distal esophagus, termed "the phrenic ampulla." The phrenic ampulla corresponds to the location of the lower esophageal sphincter. The LES is located within the thorax and the abdomen, crossing the diaphragmatic hiatus. It separates positive intra-abdominal pressure from the negative pressure esophagus. Diaphragmatic crura and the phrenicoesophageal membrane contribute to the stability of the LES. At rest, the normal esophagus is collapsed with both the UES and LES closed to prevent retrograde flow of esophageal and gastric contents respectively (Dodds 1977).

Normal Esophageal Function

The function of the esophagus is to transport ingested contents from the oral cavity to the stomach. There are three components of esophageal functional activity: primary, secondary, and tertiary contractions (Summerton 2005). Primary and secondary contractions are propulsive peristalsis. Upon swallowing, primary esophageal contractions propel ingested contents toward the stomach and secondary contractions clear the residua. Tertiary contractions are nonperistaltic and have no known function.

Primary esophageal peristalsis is initiated by swallowing. Relaxation of the UES occurs within 0.2–0.3 s of the swallow and the LES relaxes seconds later (Dodds 1989; Ott 2008). Primary peristalsis consists of a major stripping wave that propels ingested material through the esophagus and into the stomach. The primary wave begins with an inhibitory impulse that passes down the esophagus and relaxes the lower esophageal sphincter before the bolus reaches it. The LES remains relaxed and the gastroesophageal junction opens as the bolus approaches. After the bolus enters the stomach the LES returns to its resting, contracted state. The primary peristaltic wave propagates through the esophagus within 6–8 s (Ott 2008).

Secondary peristalsis is initiated due to esophageal distension or irritation rather than by swallowing. Secondary peristaltic waves propagate similar to primary peristalsis; however, the peristaltic wave begins at the level of stimulation and propels the bolus distally. This clears the esophagus of residual material from a previous swallow or from refluxed gastric contents.

Tertiary (*nonperistaltic*) *peristalsis* is a result of uncoordinated, nonpropulsive esophageal contractions. Tertiary contractions are segmental, typically involving the smooth muscle segment of the esophagus, and may occur spontaneously or during swallowing. Tertiary contractions increase in incidence with age and can be nonspecific or related to structural or motility disorders of the esophagus (Stewart 1981). Tertiary contractions can be simultaneous or repetitive and weak or strong (Ott 2008). Severe and simultaneous nonperistaltic contractions can narrow or obliterate the esophageal lumen.

Assessing motility at fluoroscopy: Assessment of esophageal motility involves evaluation of the esophagus, UES, and LES. In order to optimally assess

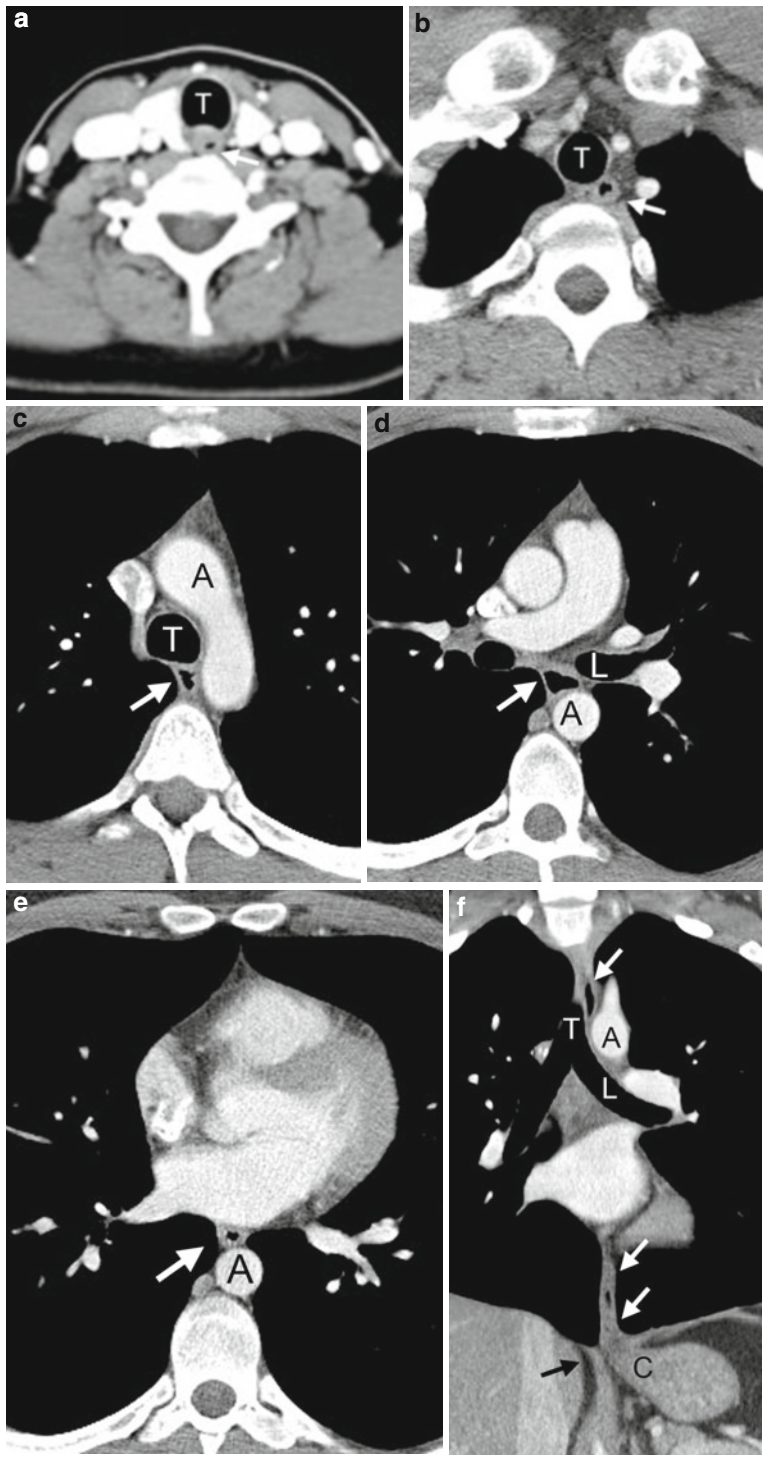

Fig. 7.2 Normal course of the esophagus on CT: Contrast-enhanced CT images show the course of the esophagus (*white arrows*). (**a**) An axial CT image in the neck shows the cervical esophagus posterior to the trachea (*T*) and anterior to the cervical spine. (**b**) At the thoracic inlet, the esophagus is posterior and slightly to the left of the trachea (*T*). (**c**) Slightly more inferiorly, there is mild mass effect on the esophagus by the normal left-sided aortic arch (*A*). (**d**) The esophagus then courses along the posterior aspect of the left main stem bronchus (*L*). (**e**) The lower thoracic esophagus is in the posterior mediastinum, posterior to the heart. (**f**) A coronal multiplanar reformatted image shows the course of the esophagus (*white arrows*) relative to the aorta (*A*), left main stem bronchus (*L*), and traversing the esophageal hiatus (*black arrow*) to the gastric cardia (*C*). *T* trachea, *A* aorta, *L* left main stem bronchus, *C* cardia of the stomach

motility, the patient is placed in the prone right anterior oblique (RAO) position. Prone positioning eliminates the affect of gravity and allows for a more accurate assessment of esophageal motility (Summerton 2005). The patient is instructed to take a single swallow of barium and individual swallows should be observed completely at fluoroscopy. A second swallow initiated before completion of a primary contractive wave

inhibits the propagating wave and may be mistaken for a peristaltic abnormality. Multiple or repeated swallows will inhibit peristalsis and preclude evaluation of esophageal motility. As many as five individual swallows may be required to adequately evaluate esophageal motility and LES relaxation (Ott et al. 1989; Ott 2008). Swallows should be separated by at least 20 s, as this is the refractory period for esophageal peristalsis (Low and Rubesin 1993). Recording techniques may aid in the assessment of esophageal motility abnormalities (Ott et al. 1989; Summerton 2005).

Upon swallowing, the UES relaxes and the pharyngoesophageal segment opens in response to the bolus. Normal primary peristalsis appears as a contractive wave that obliterates the esophageal lumen and progressively strips the barium bolus from the esophagus. At fluoroscopy, the primary peristaltic wave is seen as an inverted V configuration at the proximal end of the barium column. As the peristaltic wave progresses, the inverted V moves distally in the esophagus and should continue without interruption to the gastroesophageal junction (Ott et al. 1989; Ott 2008).

Proximal escape of barium can occur near the aortic arch due to the transition between esophageal striated and smooth muscle. Incomplete closure of the esophageal lumen in this location as the bolus passes allows retrograde flow of barium (Low and Rubesin 1993; Ott 2008). Although not considered a motility disorder, proximal escape increases in frequency with age and can mimic a peristaltic abnormality (Ott et al. 1989). Esophageal motility disorders demonstrate weakening or absent primary peristalsis and nonperistaltic contractions. Segmental nonperistaltic contractions can result in to-and-fro flow of barium in the esophagus.

In younger patients, the primary peristaltic wave typically strips barium completely from the esophagus and nonperistaltic contractions are rare (Ribeiro et al. 1998). There is increased variation of esophageal function with age so that elderly patients have more incomplete peristaltic waves, occasional LES dysfunction, and a higher prevalence of nonperistaltic contractions. The recorded amplitude of peristalsis at manometry also decreases with age (Grishaw et al. 1996; Ribeiro et al. 1998; Grande et al. 1999).

Abnormal esophageal sphincter function: The UES normally relaxes with swallowing and is not seen on esophagography. Abnormal UES relaxation results in an impression from the cricopharyngeus muscle that may be seen on esophagography as a smooth indentation on the posterior aspect of the pharyngoesophageal junction (Fig. 7.3). This can result in proximal trapping of barium in the pharynx or aspiration. Cricopharyngeal achalasia is the failure of peristalsis to coordinate with relaxation of the UES. True achalasia is due to incomplete relaxation of the LES and failure of the GE junction to open normally. This results in a smooth, tapered appearance of the distal esophagus and GE junction (Ott 2008).

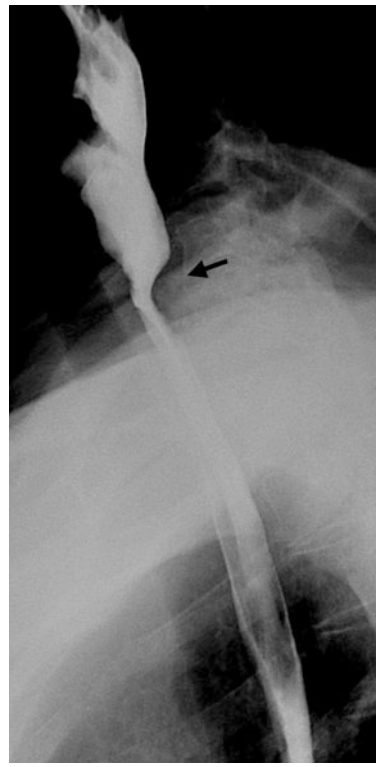

Fig. 7.3 Cricopharyngeal impression: a lateral fluoroscopic spot image acquired during a swallow shows posterior mass effect at the pharyngoesophageal junction due to incomplete relaxation of the cricopharyngeus muscle (*arrow*). There is mild proximal dilatation

Esophageal motility disorders are discussed elsewhere in this section (see Chap. "▶ Esophagus: Functional/Motility Abnormalities").

Normal Appearance of the Esophagus: Barium Studies

Fluoroscopic techniques to evaluate the esophagus are described in detail separately in this section. Briefly, double contrast technique should be performed if at all

possible due to its superior evaluation of mucosal detail as compared with single contrast technique (see Chap. "▶ Pharynx and Esophagus: Barium Studies and Fluoroscopic Evaluation" ▶ Fig. 1.5). A double contrast esophagram is ideally a biphasic study incorporating both double and single contrast techniques, with double contrast evaluation of mucosal detail and single contrast assessment of luminal caliber, contour, and function. Single contrast prone RAO images should be acquired while the patient continuously drinks barium to optimally distend the distal esophagus and gastroesophageal junction. This allows for detection of hiatal hernias, distal esophageal strictures, or rings that may not be well seen on other views (Chen et al. 1985; Ott et al. 1986; Smith et al. 1998). In elderly, debilitated, obese, and postoperative patients, it may be necessary to use single contrast technique alone (Frederick et al. 1997).

On double contrast images, there is a smooth featureless appearance of the mucosal surface of the distended esophagus (Fig. 7.4) (Levine and Laufer 2000). Although the esophagus is a predominantly tubular structure, there is a saccular distal segment that communicates with the stomach. This saccular segment is referred to as the phrenic ampulla or vestibule (Fig. 7.5). The phrenic ampulla corresponds to the location of the LES, a relatively high-pressure zone above the gastroesophageal junction that prevents reflux of gastric contents into the esophagus (Wolf et al. 1968; Dodds 1977). The phrenic ampulla extends inferiorly through the esophageal hiatus of the diaphragm to join the gastric cardia. The relaxed gastric cardia may be seen on upright double contrast studies as an arch-shape at the gastroesophageal junction (Fig. 7.6) (Levine and Laufer 2000).

The anatomy of the distal esophagus and GE junction includes the A line, B line, and the Z line. The A line is a subtle indentation at the proximal extent of the phrenic ampulla (Fig. 7.7). The B line is a subtle indentation at the lower boundary of the phrenic ampulla. The Z line is an irregular, serrated line that corresponds histologically to the squamocolumnar mucosal junction, located between the esophagus and stomach. The Z line may rarely be seen on double contrast studies in patients with sliding-type hiatal hernias and appears as a thin, zigzagging, relatively radiolucent line along the distal esophagus near the GE junction (Fig. 7.8) (Friedland 1978; Ott et al. 1984). The Z line should not be mistaken for pathologic ulceration.

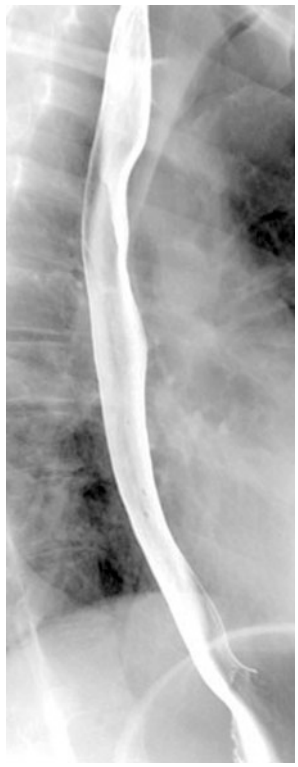

Fig. 7.4 Double contrast esophagram: normal esophagus. Upright left posterior oblique image obtained following administration of an effervescent agent and acquired while the patient drinks high-density barium shows the normal, smooth, featureless appearance of the esophageal mucosa

Mucosal Ring

A lower esophageal mucosal ring is a membranous ridge covered by squamous epithelium superiorly and columnar epithelium inferiorly (Goyal and Spiro 1970). Lower esophageal mucosal rings are fixed and reproducible. However, mucosal rings may not be apparent on double contrast images and may only be detected with optimal distension above and below the diameter of the ring (Chen et al. 1985). Single contrast technique with the patient continuously drinking barium in the prone right anterior oblique position most often allows for maximal distension and the sensitivity for detection of mucosal rings approaches 100% with this technique (Chen et al. 1985). More than 50% of lower esophageal rings seen on prone single contrast views are not visualized with double contrast images alone (Chen et al. 1985; Ott et al. 1986).

The lower esophageal mucosal ring, or B ring, is located at the inferior margin of the esophageal phrenic

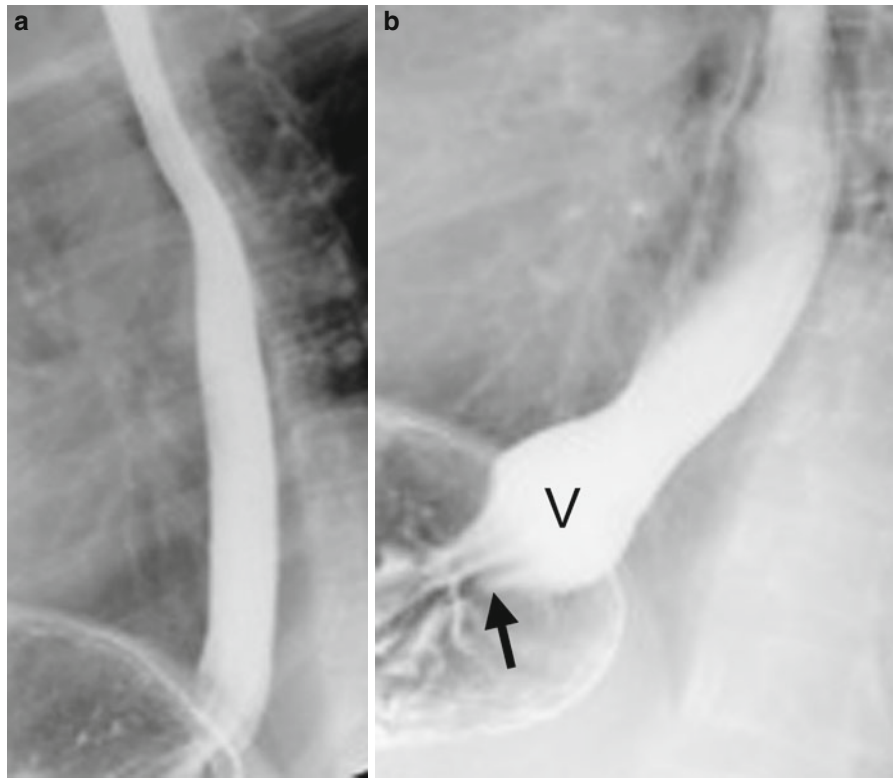

Fig. 7.5 Esophagus: tubular and vestibular components. (**a**) A prone right anterior oblique image with the patient continuously drinking thin barium shows the normal tubular appearance of the majority of the esophagus. (**b**) Further distension of the distal esophagus demonstrates the normal saccular distal esophageal segment known as the phrenic ampulla or vestibule (*V*). This should not be mistaken for a hiatal hernia. Note the gastric folds more inferiorly (*arrow*)

ampulla (B line) (Goyal and Spiro 1970; Ott et al. 1984). On barium studies, this appears as a thin, smooth, circumferential narrowing at the gastroesophageal junction extending for no more than 2–4 mm in length (Fig. 7.9). The focal, fixed, short segment, and symmetric nature of a mucosal ring allows for distinction from other entities such as a peptic stricture (Goyal and Spiro 1970; Ott et al. 1984). Mucosal ring diameters exceeding 20 mm rarely cause symptoms (Ott et al. 1984).

A Schatzki ring is described as an annular narrowing at the gastroesophageal junction, or B line. Although often used synonymously with a lower esophageal ring, the Schatzki ring was originally described as a pathologically stenotic ring with associated dysphagia (Schatzki and Gary 1953). The diameter of the mucosal ring is the most important factor for determining patient symptoms. Almost all rings less than 13 mm in diameter cause dysphagia and rings 13–20 mm in diameter may cause symptoms as well (Schatzki 1963; Ott et al. 1984). The term "Schatzki ring" may be reserved for symptomatic patients with narrow rings at the GE junction. On esophagography, a Schatzki ring has a similar appearance and location as an asymptomatic mucosal ring (Schatzki and Gary 1953; Schatzki 1963; Goyal and Spiro 1970; Ott et al. 1984). A hiatal hernia is almost always seen below the ring.

Muscular Ring

A muscular contractile ring, or A ring, is a dynamic, changeable ring-like narrowing in the distal esophagus along the proximal aspect of the phrenic ampulla (A line) (Dodds 1977). Unlike a fixed mucosal ring, a muscular ring is transient, due to muscular contractions of the distal esophagus, and it is lined with squamous epithelium. On esophagography, a muscular ring appears on as a broad, smooth area of narrowing that changes caliber and configuration during the study and often disappears with increased esophageal distension (Fig. 7.10) (Wolf et al. 1968; Goyal and Spiro 1970; Ott et al. 1984). It can be distinguished from a mucosal ring by its dynamic or transient nature, broader configuration, and location.

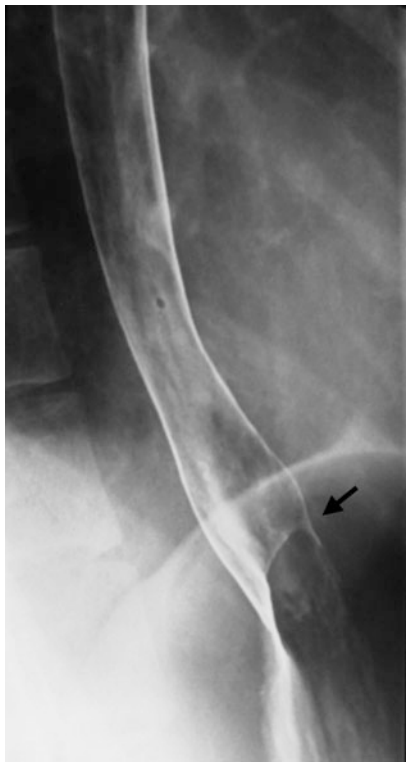

Fig. 7.6 Double contrast gastroesophageal junction. An upright left posterior oblique double contrast image shows the relaxed gastric cardia with an arch-shaped appearance of the gastroesophageal junction (*arrow*)

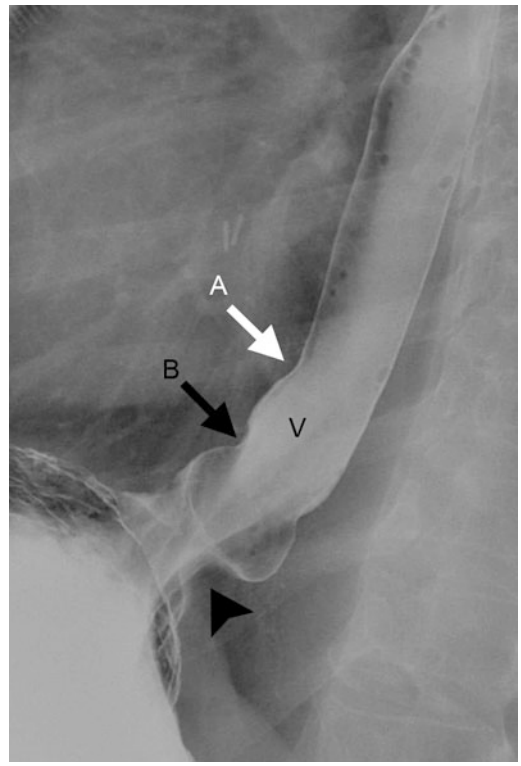

Fig. 7.7 Anatomy of the distal esophagus and gastroesophageal junction on a prone right anterior oblique image. The A line (*A*) is an indentation that may be seen at the proximal extent of the phrenic ampulla or vestibule (*V*). The B line (*B*) is an indentation at the distal aspect of the vestibule. This is seen at the superior margin of a small hiatal hernia. Note the mild pinching where the gastric folds cross the diaphragm (*arrowhead*)

Esophageal Folds

With collapse of the esophagus, normal longitudinal folds may be seen. Normal longitudinal esophageal folds appear as thin, straight, and parallel lines, measuring only several millimeters in width (Fig. 7.11).

Focal spiculation of the upper thoracic esophagus may be seen as a normal variant with double contrast technique and should not be mistaken for a focal area of esophagitis (Levine et al. 1992). This is a transient finding that can be seen near the aortic arch, likely as a consequence of focal weakening of peristalsis or localized contraction at the junction of the esophageal striated and smooth muscle.

Fine transverse folds in the esophagus may be seen intermittently in some patients. This is referred to as the *feline esophagus*. Although a normal finding in felines, this occurs transiently in humans due to contraction of the longitudinal fibers of the muscularis mucosae and this is associated with gastroesophageal reflux (Gohel et al. 1978; Williams et al. 1983). Fine transverse folds are 1–2 mm in height and extend without interruption across the esophageal lumen (Fig. 7.12) (Williams et al. 1983). This should be distinguished on esophagography from thicker transient transverse bands of nonperistaltic contractions and from fixed transverse scarring.

Cervical esophagus: A small anterior indentation on the cervical esophagus can be seen due to mucosal redundancy over the submucosal pharyngeal venous plexus at approximately the C6 level (Fig. 7.13). This is a normal finding and the appearance may vary during the study. This should not be mistaken for a cervical esophageal web or neoplasm (Friedland and Filly 1975). A prominent cricopharyngeal impression can be seen posteriorly (Fig. 7.3) related to failure of the cricopharyngeus muscle to relax and/or for early closure.

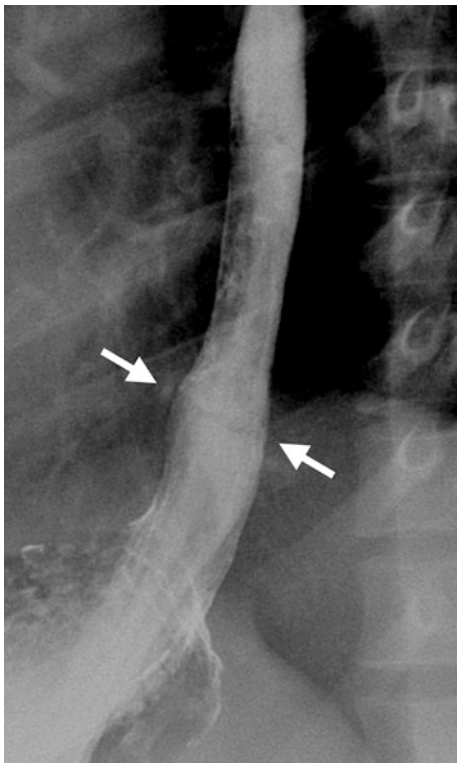

Fig. 7.8 The Z line. A right anterior oblique image of the distal esophagus and gastroesophageal junction shows a small hiatal hernia with a visible thin and subtle zigzagging radiolucent line (*arrows*) between the esophagus and stomach denoting the histologic squamocolumnar junction (i.e., Z line)

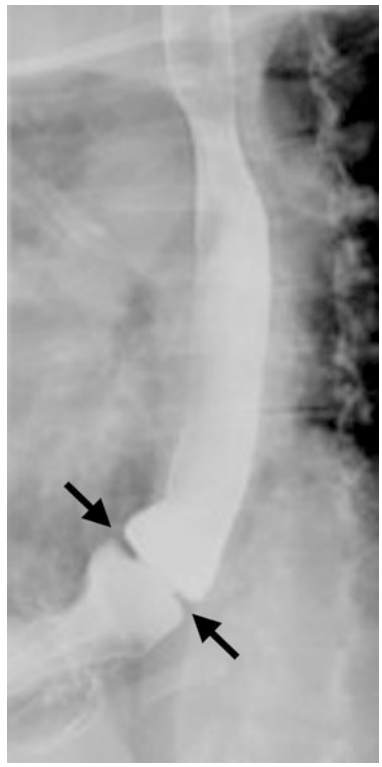

Fig. 7.9 Lower esophageal B ring or Schatzki ring. A prone right anterior oblique image with the patient continuously drinking barium demonstrates maximum distension of the distal esophagus and gastroesophageal junction. There is a small hiatal hernia with a fixed and reproducible short segment concentric luminal narrowing along the gastroesophageal junction (*arrows*) consistent with a mucosal ring. No luminal narrowing was seen on the upright LPO double contrast images obtained on this patient

Normal and Normal Variant Extrinsic Impressions on the Esophagus

Mass effect on the esophagus may be seen on barium studies as a normal finding, due to normal anatomic variants or due to pathologic processes.

Structures that normally produce an extrinsic impression on the thoracic esophagus can be routinely identified on barium studies. There are normal extrinsic impressions upon the esophagus due to the aortic arch, left main stem bronchus, and the heart. Indentations due to the aortic arch and left main stem bronchus are seen in the middle third of the thoracic esophagus (Fig. 7.14). There is a normal impression superiorly on the left due to the transverse arch of the aorta. This becomes more prominent with dilatation and tortuosity of the aorta. More caudally, there is a normal impression caused by the left main stem bronchus (Levine and Laufer 2000). In approximately 10% of patients, the left inferior pulmonary vein or the confluence of the left pulmonary veins near the left atrium produces extrinsic indentation on the left anterior wall of the esophagus 4–5 cm below the carina. A mild smooth indentation may be seen along the right posterolateral wall of the upper thoracic esophagus in up to 10% of patients due to a prominent right inferior supra-azygous recess abutting the esophagus (Sam et al. 1998).

Extrinsic compression of the esophagus on barium studies may also indicate normal variant vascular anatomy including a right-sided aortic arch, an aberrant subclavian artery, vascular rings, or slings. With a right-sided aortic arch, the superior thoracic esophagus is indented on the right rather than the left.

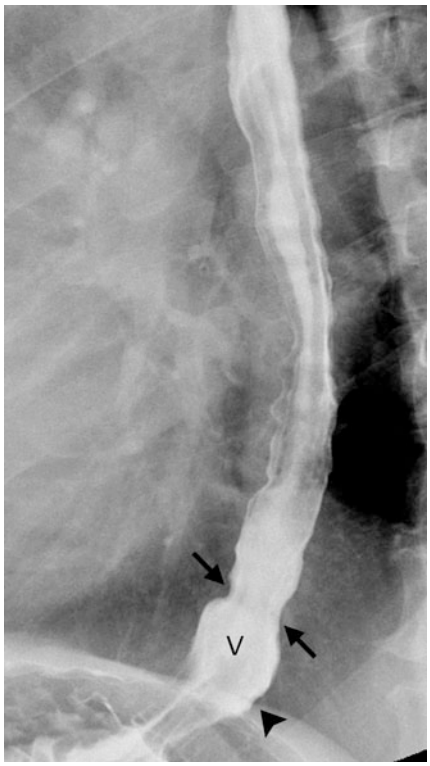

Fig. 7.10 Muscular ring. A prone right anterior oblique image with the patient drinking barium demonstrates a broad, smooth concentric narrowing of the distal esophagus at the A line, just above the vestibule (*V*). This disappears with increased distension and represents a changeable muscular ring. Note mild pinching at the level of the diaphragm more inferiorly (*arrowhead*)

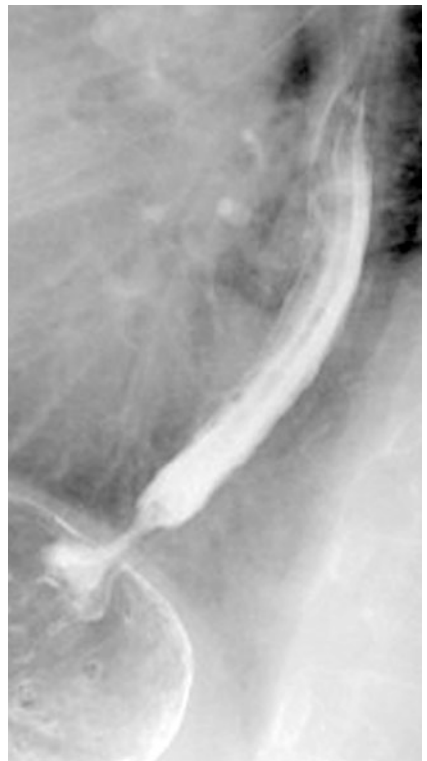

Fig. 7.11 Normal longitudinal esophageal folds. A prone right anterior oblique image of the mid-to-distal esophagus partially collapsed (mucosal relief) reveals normal, thin, parallel, longitudinal folds, each measuring several millimeters in width

An aberrant right subclavian artery arises just distal to the left subclavian artery with a left aortic arch and crosses posterior to the esophagus to produce a posterior and obliquely oriented indentation on the esophagus, best seen in the straight lateral and frontal projections (Fig. 7.15). On the frontal view, the esophageal impression courses obliquely upward to the right. This may be an incidental finding on esophagography and is not associated with congenital heart disease. Many additional congenital heart and vascular anomalies may produce mass effect on the esophagus. Abnormal impressions on the esophagus may also occur as a consequence of enlargement of expected adjacent structures, including the heart and aorta, or from abnormal adjacent structures such as enlarged lymph nodes or mediastinal masses.

Normal Appearance of the Esophagus: Computed Tomography

Although barium studies are useful to evaluate the mucosal surface of the esophagus, little information can be gained about the extent of esophageal disease relative to adjacent structures or about the etiology of processes displacing or compressing the esophagus. This information is better assessed with computed tomography (CT). CT can assess the esophageal wall and the extramural extent of disease (Ba-Ssalamah et al. 2009). In patients with known esophageal neoplasm, CT can demonstrate focal esophageal wall thickening, mediastinal involvement, adjacent lymphadenopathy, and distant spread.

Multidetector CT allows for imaging a large volume with a very short scan time, so that the entire course of the esophagus can be imaged in a single breath hold.

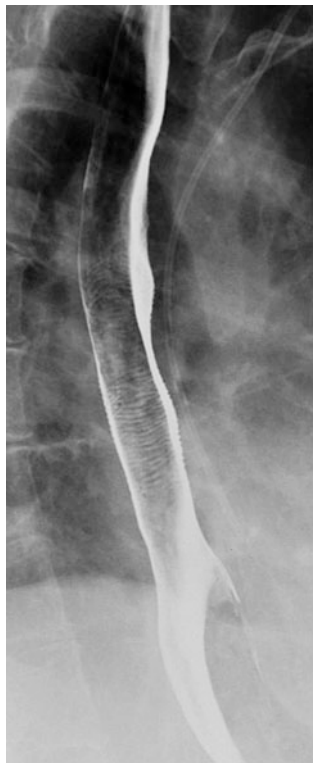

Fig. 7.12 Fine transverse folds or "feline esophagus." A well-distended double contrast upright left posterior oblique image of the esophagus shows transient fine transverse folds in the esophagus, 1–2 mm in height and extending transversely across the lumen without interruption

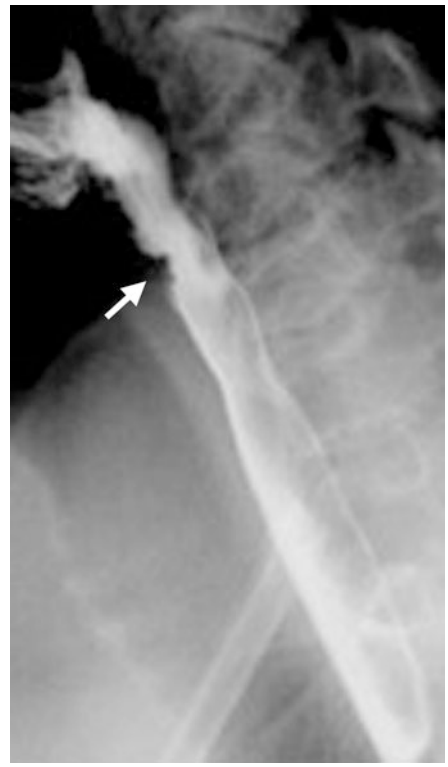

Fig. 7.13 Normal mucosal redundancy along the anterior cervical esophagus. A slightly oblique lateral rapid sequence image of the cervical esophagus acquired during swallowing demonstrates minimal luminal narrowing with a small anterior indentation along the cervical esophagus (*arrow*) corresponding to normal mucosal redundancy over a venous plexus. This occurs at approximately the C6 level and should not be mistaken for pathology

The esophagus can be depicted with high-quality multiplanar reformation and 3D reconstruction. Techniques for imaging the esophagus with CT are reviewed elsewhere in this section (see Chapter: Pharynx and Esophagus: CT/MRI). However, CT is ideally performed with administration of both oral and intravenous contrast material. On routine CT examinations of the thorax, the normal esophagus is collapsed, limiting its evaluation. For evaluation of the esophagus, the patient should be administered additional oral contrast, barium paste, or an effervescent agent just prior to the examination to attempt to distend the esophagus (Noh et al. 1995). Distension of the esophagus enhances detection and characterization of esophageal processes.

The esophagus is usually well visualized throughout its course on CT images (Fig. 7.2). The cervical esophagus is midline, posterior to the trachea (Fig. 7.2a). At the thoracic inlet, the esophagus is posterior and slightly to the left of the trachea (Fig. 7.2b). The thoracic esophagus courses between the azygous vein and the aortic arch (Fig. 7.2c) and then along the posterior aspect of the left main stem bronchus (Fig. 7.2d). The esophagus more inferiorly extends to the right of the descending aorta in the posterior mediastinum, posterior to the heart and left pulmonary veins (Fig. 7.2e) (Chevallier et al. 1991). Paraesophageal fat can be seen in most patients between the esophagus and adjacent structures including the airway, aorta, and pericardium. The esophagus traverses the esophageal diaphragmatic hiatus to enter the abdomen at approximately T10, anterior to the aorta and slightly to the left of midline. Deviation of the esophageal course can occur due to enlargement of adjacent organs or due to adjacent pathology.

A small amount of gas can be seen in the normal thoracic esophagus, however, a fluid-filled lumen or

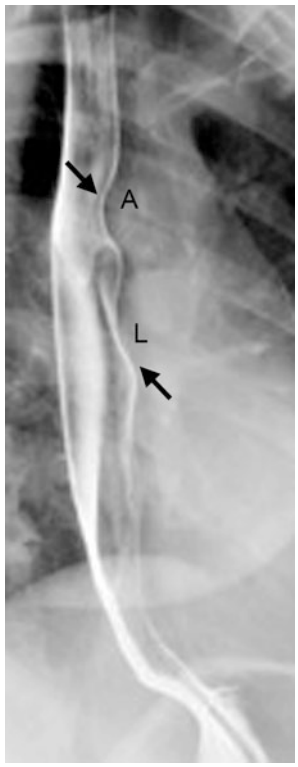

Fig. 7.14 Normal impressions on the esophagus. A double contrast upright left posterior oblique spot image shows normal impressions on the esophagus (*arrows*) due to the aortic arch (*A*) and due to the left main stem bronchus (*L*)

a diameter greater than 10 mm can indicate an esophageal motility abnormality or obstruction (Halber et al. 1979). The thickness of the normal esophageal wall varies with the degree of luminal distension so that with incomplete distension the normal esophageal wall can measure up to 5 mm. However, with optimal distension, wall thickness should not exceed 3 mm (Desai et al. 1991).

Congenital Anomalies

Ectopic Gastric Mucosa/Inlet Patch

Ectopic gastric mucosa is a congenital anomaly with a reported incidence of 4–10% at endoscopy (Jabbari et al. 1985; Schroeder et al. 1987; Borhan-Manesh and Farnum 1991). A rest of ectopic gastric mucosa in the esophagus is typically located superiorly at or just above the thoracic inlet and may be referred to as the "inlet patch" (Jabbari et al. 1985; Takeji et al. 1995). Patients are most often asymptomatic, however, inflamed or ulcerated ectopic gastric mucosa can rarely lead to dysphagia and/or odynophagia (Schroeder et al. 1987). On esophagography, ectopic gastric mucosa may appear as a broad, shallow depression with small indentations along its margins, most commonly seen on the right lateral wall of the thoracic esophagus at or near the thoracic inlet (Fig. 7.16) (Takeji et al. 1995). The appearance and location are characteristic and this should not be mistaken for ulceration or intramural dissection (Lee et al. 1999).

Congenital Esophageal Web

Congenital esophageal webs are smooth, thin, membranes covered with normal mucosa, most often located in the cervical esophagus within 2 cm of the pharyngoesophageal junction (Fig. 7.17). A web is typically transversely oriented, protruding into the esophageal lumen and arising from the anterior wall of the esophagus (Clements et al. 1974). Much less often, a web may appear circumferential with a thin radiolucent band concentrically narrowing the lumen. Webs are best seen at esophagography with maximal luminal distension.

Intraluminal Diverticulum

An intraluminal diverticulum is a focal outpouching within the esophageal lumen that is attached to the inner wall of the esophagus. It is open proximally and closed distally and has a thin wall that is covered on both sides with esophageal mucosa. An intraluminal diverticulum can be caused by downward intraluminal ballooning of a congenital esophageal web. Increased intraluminal pressure may permit a small mucosal outpouching through a web or weakness in the esophageal wall with subsequent ballooning into a diverticulum. On esophagography, this appears as a thin radiolucent line separating barium within the intraluminal diverticulum from barium in the remainder of the esophageal lumen and this can create a double-barreled appearance of the esophagus (Schreiber and Davis 1977).

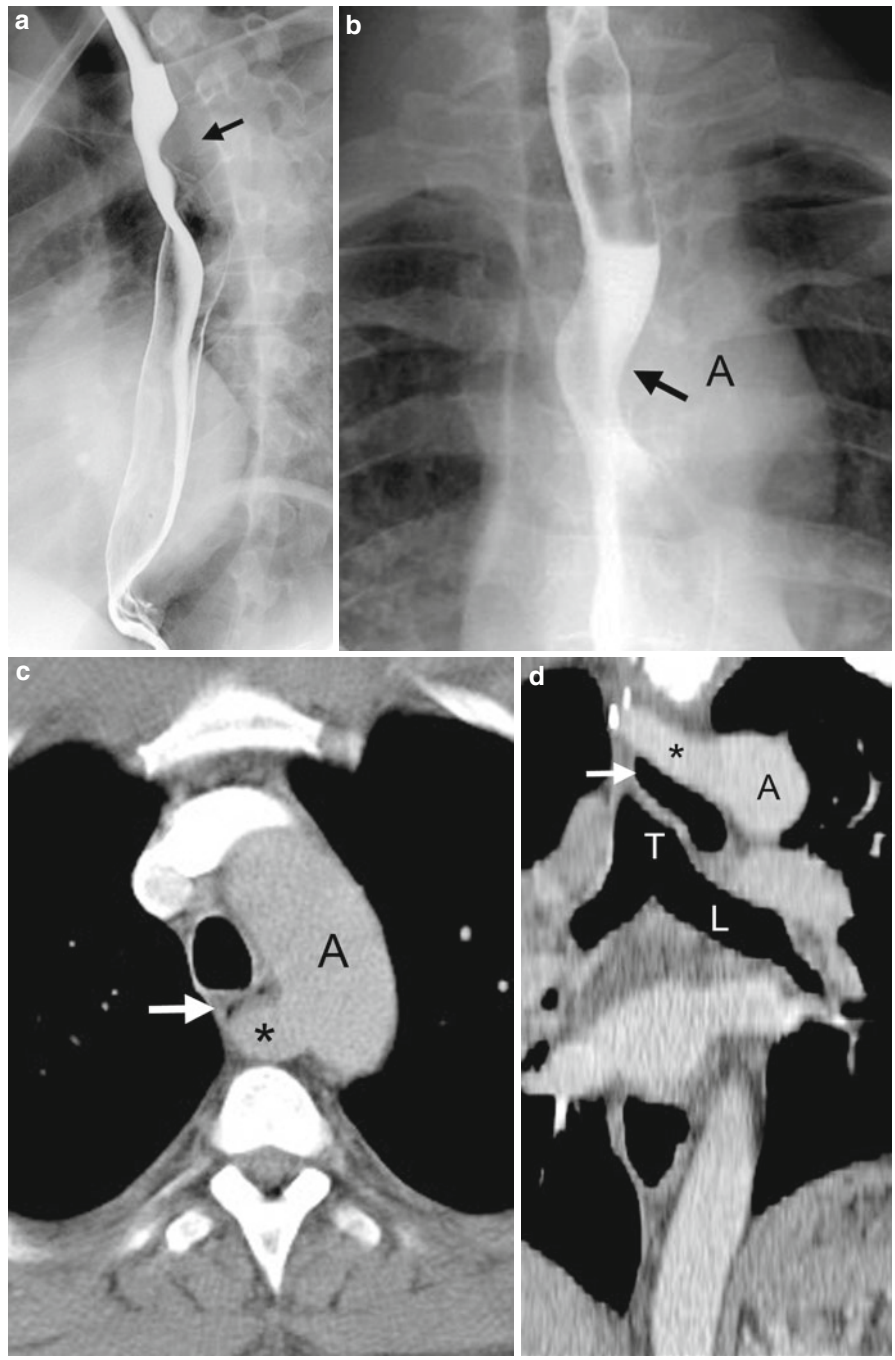

Fig. 7.15 Abnormal impression on the esophagus due to an aberrant right subclavian artery. (**a**) A right lateral upright double contrast image shows an indentation along the posterior aspect of the upper thoracic esophagus (*arrow*). (**b**) A frontal image shows a normal left-sided aortic arch (*A*) and an obliquely oriented impression on the esophagus extending superiorly to the right (*arrow*). This is the classic impression due to an aberrant right subclavian artery. (**c**) Axial and (**d**) coronal contrast-enhanced CT images show significant impression upon and displacement of the esophagus (*white arrows*) due to a large aberrant right subclavian artery (*) arising from the left aortic arch, crossing posterior to the esophagus and then obliquely upward toward the right. *A* aortic arch, *L* left main stem bronchus, *T* trachea

Esophageal Duplication Cyst

Esophageal duplication cysts represent approximately 20% of all gastrointestinal tract duplications (Macpherson 1993). An esophageal duplication cyst may result from a diverticulum of the dorsal bud of the primitive foregut or from aberrant recanalization of the gut (Fitch et al. 1986). Esophageal duplication cysts are attached to or located within the esophageal wall and 60% are located in the posterior mediastinum inferiorly, often extending to the right of the distal esophagus (Fig. 7.18) (Fitch et al. 1986; Macpherson 1993). Duplication cysts contain mucosa, submucosa, and muscularis propria. In up to 50% of cases, ectopic

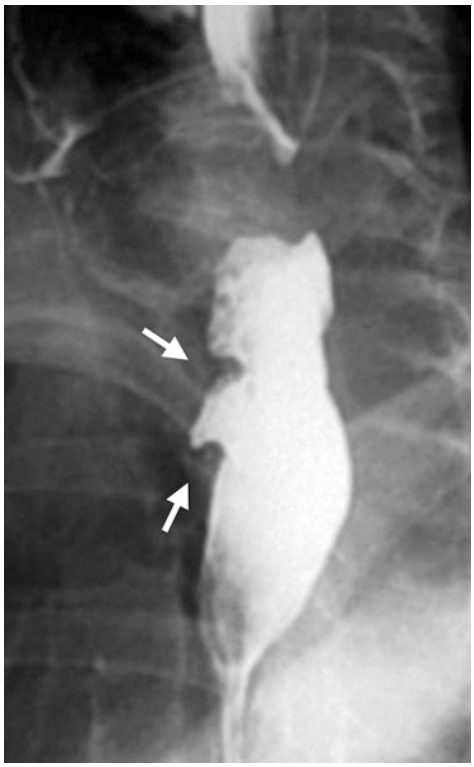

Fig. 7.16 Ectopic gastric mucosa or "inlet patch." An oblique lateral image while swallowing shows a broad, shallow depression along the right anterolateral wall of the upper thoracic esophagus near the thoracic inlet due to ectopic gastric mucosa (i.e., inlet patch). Small indentations can be seen along the cephalad and caudal margins of the inlet patch (*arrows*)

gastric mucosa is found within the cyst (Fitch et al. 1986; Macpherson 1993). Duplications may be classified as cystic or tubular. Most duplications are cystic and do not communicate with the esophageal lumen. Tubular duplications occasionally communicate with the esophageal lumen. Esophageal duplication cysts may occur as an isolated finding, or may be associated with other congenital anomalies including vertebral anomalies and esophageal atresia (Macpherson 1993).

Neonates with esophageal duplications may present with respiratory or feeding problems. Adult patients are typically asymptomatic; however, they may become symptomatic due to obstruction or infection of the cyst. As the cyst dilates with retained material it can compress the esophagus and other adjacent structures and produce pain, dysphagia, and/or respiratory distress. Bleeding or perforation may occur and is more likely in cysts containing ectopic gastric mucosa.

At esophagography, a duplication cyst most often appears as extrinsic compression or as a submucosal mass that may be indistinguishable from a solid intramural or mediastinal mass (Fig. 7.18) (Fitch et al. 1986). In most cases, there is no communication with the esophageal lumen so that contrast material does not opacify the cyst. Less often, communicating duplications appear as a tubular outpouching arising from the esophagus which can result in a double-barreled appearance of the esophagus. CT can better characterize the lesion with respect to its shape, extent, internal components, and relationship to adjacent structures. On CT, duplication cysts may appear as well-defined, fluid-filled structures contiguous with or abutting the esophagus. The lesions may appear smooth and spherical or less commonly tubular. Internal components can have complex fluid density with measurements ranging from 20 to 35 Hounsfield units and do not enhance with intravenous contrast (Fitch et al. 1986). An air-fluid level may be present, especially with communicating cysts. Endoscopic ultrasound may confirm the cystic nature and further establish the location of the cyst relative to surrounding tissues.

Congenital Tracheoesophageal Fistula and Esophageal Atresia

Congenital tracheoesophageal fistulas (TEF) result from failure of the esophageal lumen to completely separate from the trachea. In development, the trachea and upper alimentary tract share a common origin with subsequent division so that the esophagus assumes a more dorsal position and the trachea and lung buds are located ventrally. Failure of complete separation leads to tracheoesophageal fistulas. Esophageal atresia is associated with TEF and in about 10% of patients atresia is complete. Esophageal atresia and TEF are associated with congenital malformations involving other systems including skeletal, cardiovascular, and gastrointestinal anomalies.

Congenital Esophageal Stenosis and Cartilaginous Rings (Tracheobronchial Rests)

Congenital esophageal stenosis (CES) is a rare anomaly occurring in 1 of 25,000 or 50,000 live births due to

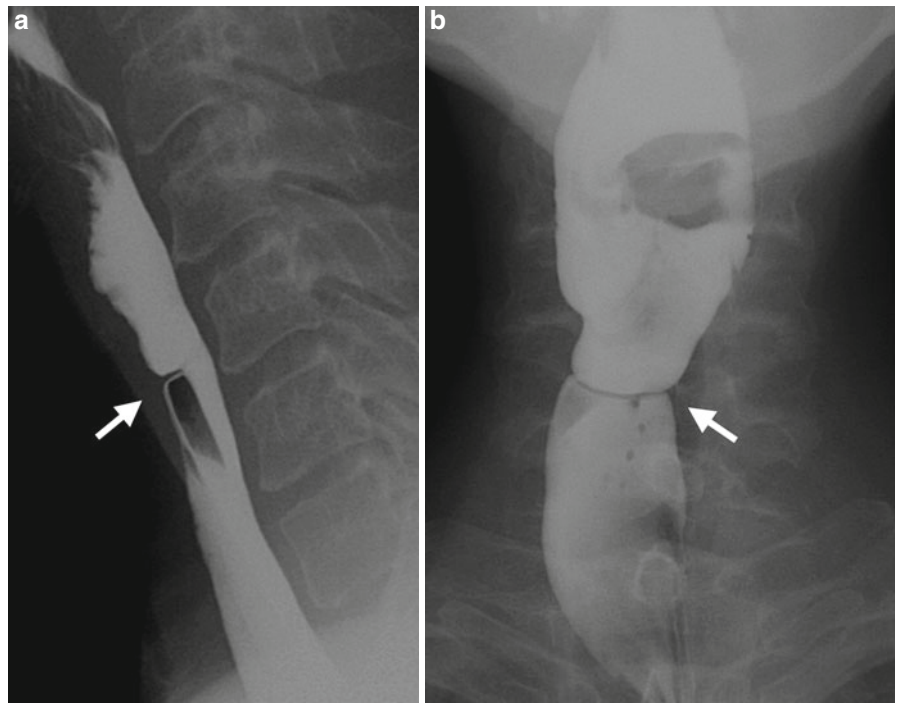

Fig. 7.17 Anterior cervical esophageal web. Rapid sequence imaging acquired during swallowing in the lateral (**a**) and frontal (**b**) projections demonstrates a thin radiolucent transversely oriented web arising from the anterior wall of the cervical esophagus and protruding into the lumen (*arrow*)

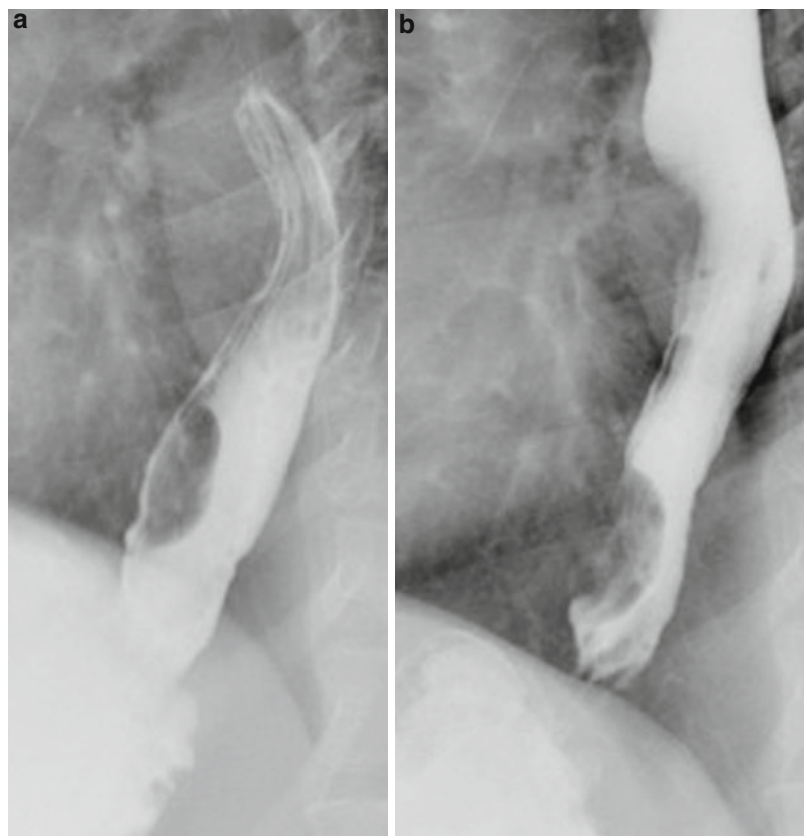

Fig. 7.18 Esophageal duplication cyst. (**a**) A prone right anterior oblique image while drinking barium shows a filling defect protruding into the lumen of the right anterolateral distal esophagus. (**b**) On a second image with a slightly different degree of distension, the lesion appears smooth and submucosal. Endoscopic ultrasound confirmed that this represented a noncommunicating cystic esophageal duplication

defective canalization of the esophagus during embryologic development (Murphy et al. 1995). There are three forms of CES: membranous diaphragm, fibrosis of the submucosa and muscularis mucosa, or focal tracheobronchial remnants, including cartilage, tracheal glands, and respiratory epithelium (Murphy et al. 1995). Defective separation of the primitive foregut from the respiratory tract embryologically can result in tracheobronchial precursor cell sequestration in the esophageal wall (Oh et al. 2001). Cartilaginous rings consisting of tracheobronchial rests may be found in the esophagus (Ishida et al. 1969; Murphy et al. 1995; Oh et al. 2001).

CES can result in a variable degree of esophageal obstruction. In infancy or early childhood, patients may present with recurrent vomiting, failure to thrive, and/or aspiration pneumonia. Patients with a lesser degree of stenosis may present in adolescence or early adulthood with intermitted dysphagia, chest pain, and occasional food impaction (Katzka et al. 2000; Oh et al. 2001).

Esophagography may reveal a web or a smooth, tapered stricture in the thoracic esophagus. Alternatively, strictures can contain multiple ring-like constrictions on double contrast studies (Oh et al. 2001). Linear tracts of barium representing ducts of tracheal bronchial glands may be seen extending from the area of narrowing. An esophageal stricture with ring-like constrictions on esophagography may suggest a diagnosis of congenital esophageal stenosis. However, idiopathic eosinophilic esophagitis may also appear as a stricture with ring-like constrictions and this diagnosis may be distinguished clinically (Zimmerman et al. 2005). Ring-like indentations have also been described in fixed transverse folds in patients with reflux esophagitis and an associated peptic stricture (Oh et al. 2001).

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