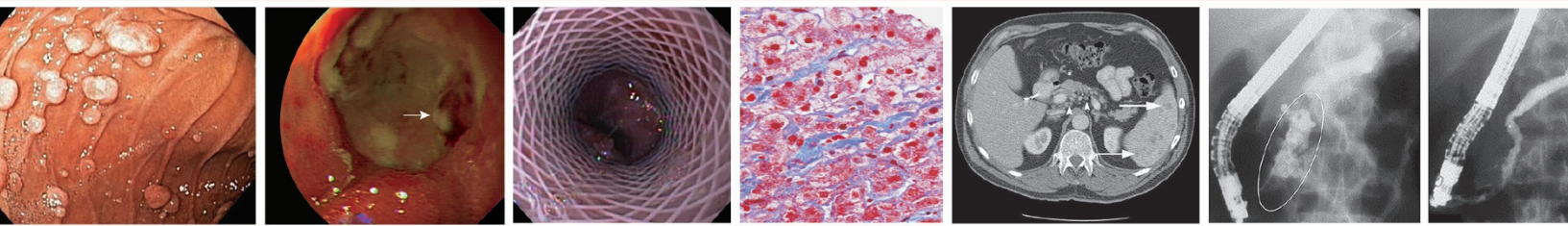


# YAMADA'S

Atlas of

# Gastroenterology



SIXTH EDITION

EDITED BY

**Timothy C. Wang, Michael Camilleri,  
Benjamin Lebwohl, Anna S. Lok, William J. Sandborn,  
Kenneth K. Wang, and Gary D. Wu**



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# **Yamada's Atlas of Gastroenterology**



# Yamada's Atlas of Gastroenterology

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# Preface

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Gastroenterology and hepatology remain highly visual specialties, in part because of the tremendous accessibility of the luminal gastrointestinal tract and the variety of other internal organs as well as advanced imaging modalities. Thus, in addition to standard views of cross-sectional imaging and histopathological analysis used in many disorders, the field encompasses multimodal endoscopic imaging, which has continued to advance over the last decade. This sixth edition of *Yamada's Atlas of Gastroenterology* continues to offer diverse images that provide an overview of the digestive field, but the objective is to achieve an overview through pictures and illustrations rather than through text. Consequently, this Atlas is designed to complement and accompany the primary textbook in the field, the seventh edition of *Yamada's Textbook of Gastroenterology*.

This sixth edition of the *Atlas* and the seventh edition of the *Textbook* represent the transition from the previous Editor-in-Chief, Dr Daniel K. Podolsky, to a new set of Editors-in-Chief and a new team of Associate Editors. This new editorial team has made changes to and updated both the main *Textbook* and the *Atlas*. Changes to the former have been much more substantial, and included a greater focus on the human microbiome, obesity, bariatric endoscopy and aging, along with consolidation of many older chapters. In contrast, changes to the *Atlas* have focused on updating chapters from prior editions, and the addition of several new *Atlas* chapters. Nevertheless, the *Atlas*, like the *Textbook*, continues to cover most aspects of gastroenterology and hepatology with a broad, global perspective.

This newest edition of the *Atlas*, a compendium to the *Textbook*, includes key figures as well as supplementary images designed to provide a highly informative snapshot of major gastrointestinal diseases covered in the *Textbook*. The *Atlas* starts with anatomy and development, then covers each of the key gastrointestinal

organs and their diseases, along with miscellaneous disorders, and ends with several chapters on diagnostic and therapeutic modalities in gastroenterology. Thus, it follows roughly the organizational structure of the newest edition of the *Textbook*, but with a much greater focus on images, including gross and microscopic anatomy, and the full range of imaging modalities, including endoscopy, spanning many newer technologies.

The *Atlas* is designed for a wide spectrum of readers, including students, residents, fellows and other trainees, practicing clinicians, professors, and related specialists. For a busy trainee or clinician seeking a quick introduction to the spectrum of GI diseases, the *Atlas* represents a practical and useful introduction to important disorders, building on the notion that in some instances, a “picture is worth a thousand words” and that images can often communicate complex ideas more effectively. We are hopeful that the pictorial reproductions and representations will bring to life the salient points accurately and comprehensively detailed in the *Textbook*.

This *Atlas*, as with the *Textbook*, would not have been possible without the effort of many individuals. We would like to thank all of our Editors, our teachers and mentors, our patients, and our partners and publishers at Wiley. In particular, we would like to thank our authors for procuring from their practice the many outstanding, supplementary images that have served as the foundation for this *Atlas*.

On behalf of all the Editors, we are excited to present to our readers this sixth edition of the *Atlas*, which we hope will be a resource for all students and practitioners of gastroenterology and hepatology.

Timothy C. Wang, MD  
Michael Camilleri, MD

# About the companion website

This book is accompanied by a companion website:

**[www.yamadagastro.com/atlas6e](http://www.yamadagastro.com/atlas6e)**



The website includes:

- Videos
- Figures from the book as downloadable PowerPoint slides

## **PART 1**

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# **Anatomy and development**



## CHAPTER 1

# Esophagus: anatomy and structural anomalies

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An understanding of the normal and abnormal histology and structure is essential to the clinical care of patients with esophageal disorders. Esophageal biopsies obtained during endoscopy sample the squamous mucosa and, less commonly, the lamina propria of the esophageal wall. The histological evaluation of submucosal glands, Meissner and myenteric ganglia, and the muscularis propria depicted in Figure 1.1 traditionally requires surgical biopsy. Endoscopic ultrasonography (EUS) can evaluate the structural integrity and anomalies of deeper structures, including the muscularis propria. Endoscopic mucosal resection and per oral esophageal myotomy allow for histological evaluation of deeper mural structures. Extrinsic compression of the esophagus by adjacent mediastinal structures as shown in Figure 1.2 is better appreciated on radiographic barium examination, EUS, or cross-sectional imaging than endoscopy. Feline esophagus is depicted in Figure 1.3 and can be mistaken for esophageal rings in eosinophilic esophagitis. The feline pattern is transient plications visualized during retching and esophageal shortening and may represent contraction of the muscularis mucosa. Upon relaxation of the esophageal musculature and luminal distension with air insufflation, the plications disappear.

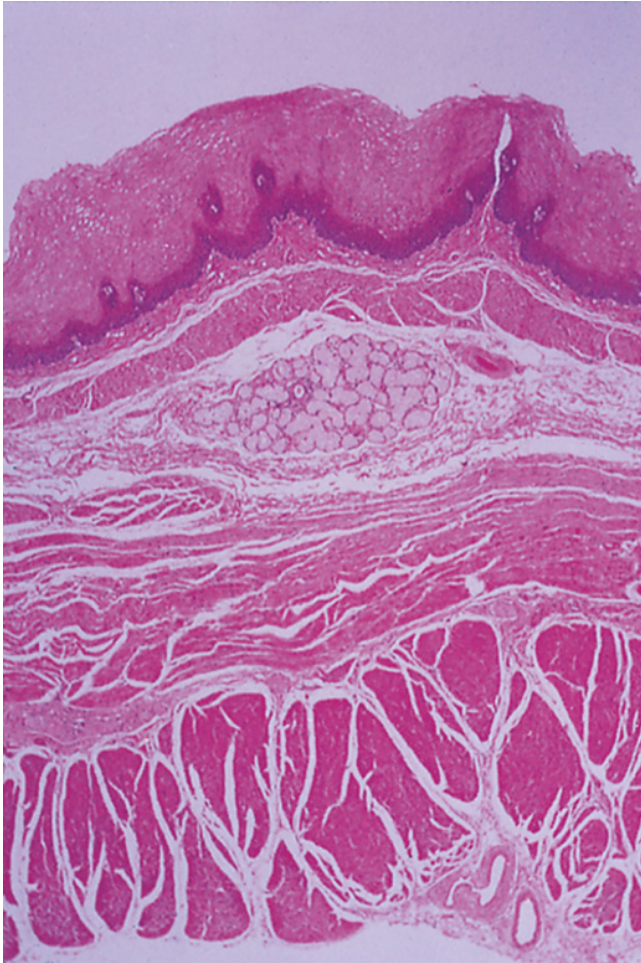
Esophageal developmental anomalies include vascular lesions, duplications, and heterotopic gastric mucosa. Congenital esophageal stenosis is caused by tracheobronchial cartilaginous remnants, fibromuscular wall hypertrophy, and membranous web formation. While typically presenting in childhood, delayed presentation in adults occurs as shown in Figure 1.4. Dysphagia lusoria is caused by extrinsic compression of the thoracic esophagus by congenital anomalies of the aortic arch, most commonly by an aberrant take-off of the right subclavian artery from the left side of the aortic arch (Figure 1.5). Congenital venous malformations illustrated in Figure 1.6 represent another vascular anomaly and are distinct from esophageal varices as vascular obstruction or portal hypertension is not present in the former. Congenital esophageal duplications assume both tubular (Figure 1.7) and cystic (Figures 1.8 and 1.9) forms. While most

are apparent before the age of 1 year, 25% can present in adults with symptoms of dysphagia. Heterotopic gastric mucosa (inlet patch) shown in Figure 1.10 is a common congenital anomaly detected with a prevalence of 4% based on an autopsy series. Infrequently, this anomaly is associated with cervical esophageal stricture (Figure 1.11) and web formation (Figure 1.12). Other uncommon developmental anomalies include esophageal atresia, congenital esophageal stenosis, and bronchopulmonary foregut malformations.

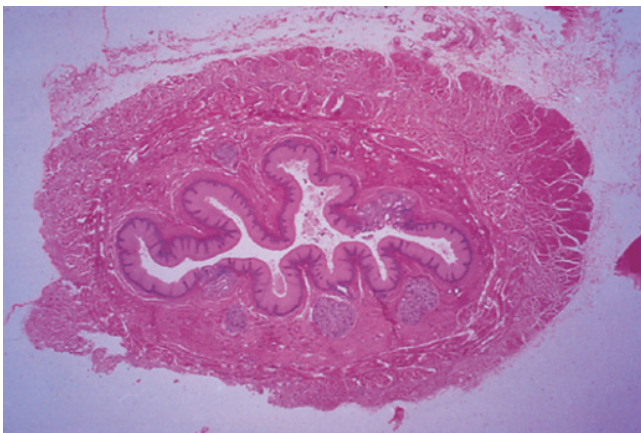
Structural esophageal anomalies include esophageal rings and webs, cricopharyngeal bar, pharyngoesophageal diverticula, and diffuse idiopathic skeletal hyperostosis of the cervical spine. The most widely recognized structural anomaly is the lower esophageal mucosal or Schatzki ring that is found in about 10% of adults. It is one of the most common causes of dysphagia and food impaction, although the majority of Schatzki rings are asymptomatic. The inner diameter of the ring is a critical determinant for dysphagia and can be assessed on endoscopic retroflexed view (Figure 1.13) or ingestion of a barium tablet of known diameter. A cricopharyngeal bar is found in 5–19% of radiographic studies of the pharynx (Figure 1.14). Like Schatzki rings, the majority are not associated with dysphagia.

Figure 1.15 illustrates progressive enlargement of a Zenker diverticulum with normalization of the pharyngeal outpouching following surgery. Therapeutic options for symptomatic cricopharyngeal bars and Zenker diverticula include both endoscopic and surgical approaches. Epiphrenic diverticula arise from the distal esophagus and are often associated with an underlying spastic esophageal motility disorder. With time, the diverticula can increase in size, resulting in food retention, bezoar formation, and symptoms of regurgitation (Figures 1.16–1.18).

Treatment for large or symptomatic epiphrenic diverticula is most commonly surgical and includes not only a diverticulectomy but also treatment of the underlying motility disorder. Intramural pseudodiverticulosis is a rare finding best appreciated on barium esophagram rather than upper endoscopy. The

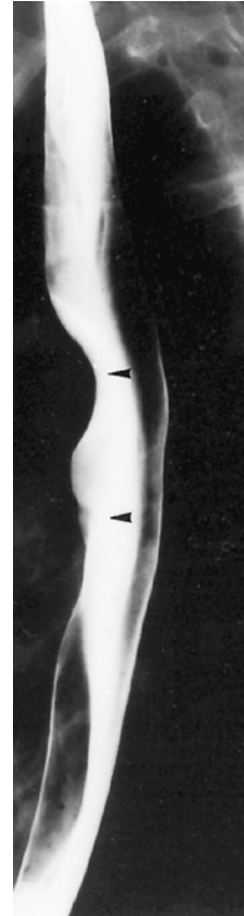


(a)



(b)

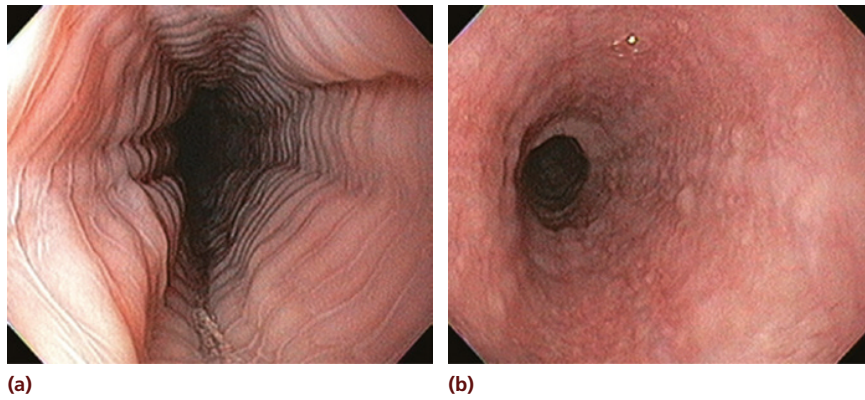
**Figure 1.1** (a) This cross-section ( $\times 2.5$ ) from the middle third of the esophagus has a mixture of skeletal and predominantly smooth muscle in the muscularis propria. The submucosal glands are clearly shown. An esophageal cardiac gland in which a small focus of glandular epithelium interrupts the squamous mucosa is a normal finding, seen in at least 1% of all esophagi. (b) Longitudinal section of esophageal wall ( $\times 10$ ). Source: Courtesy of Rodger C. Haggitt, MD, Seattle, WA.



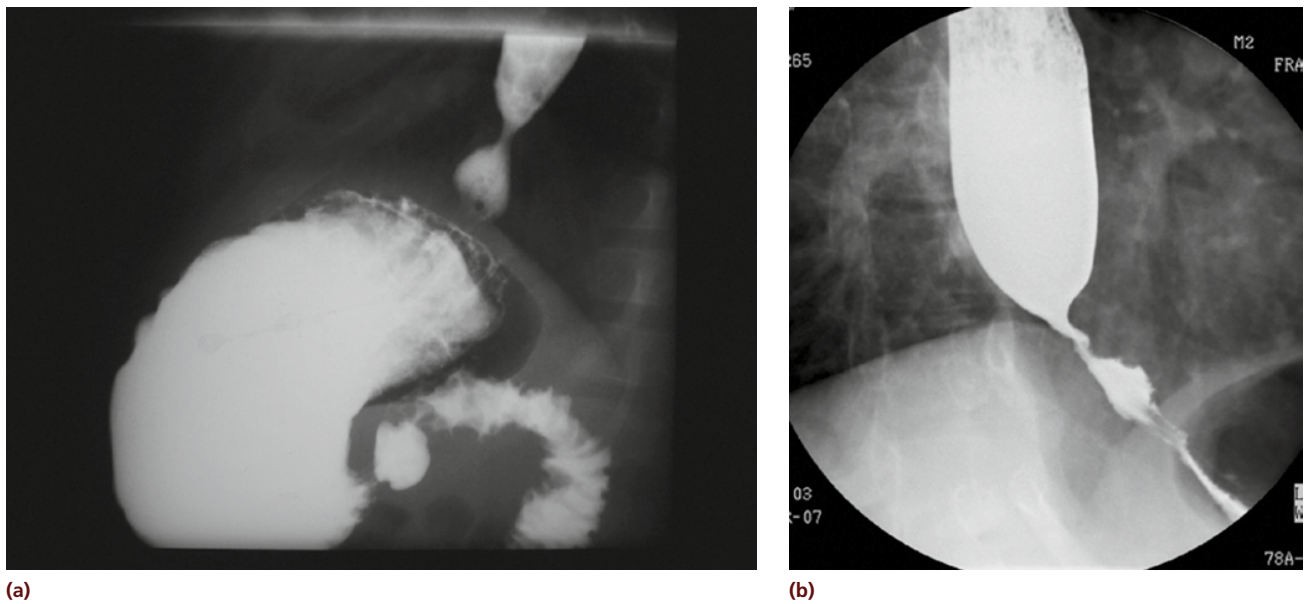
**Figure 1.2** Barium esophagram shows normal indentation of the esophageal lumen by the aorta (top arrow) and left mainstem bronchus (bottom arrow).

disorder results from dilation of the excretory ducts of submucosal esophageal glands and is associated with proximal esophageal strictures and esophageal candidiasis. Diffuse idiopathic skeletal hyperostosis (DISH) of the cervical spine leads to ossification of the anterolateral ligaments and entheses. Dysphagia may result from extrinsic compression of the cervical esophagus (Figure 1.19).

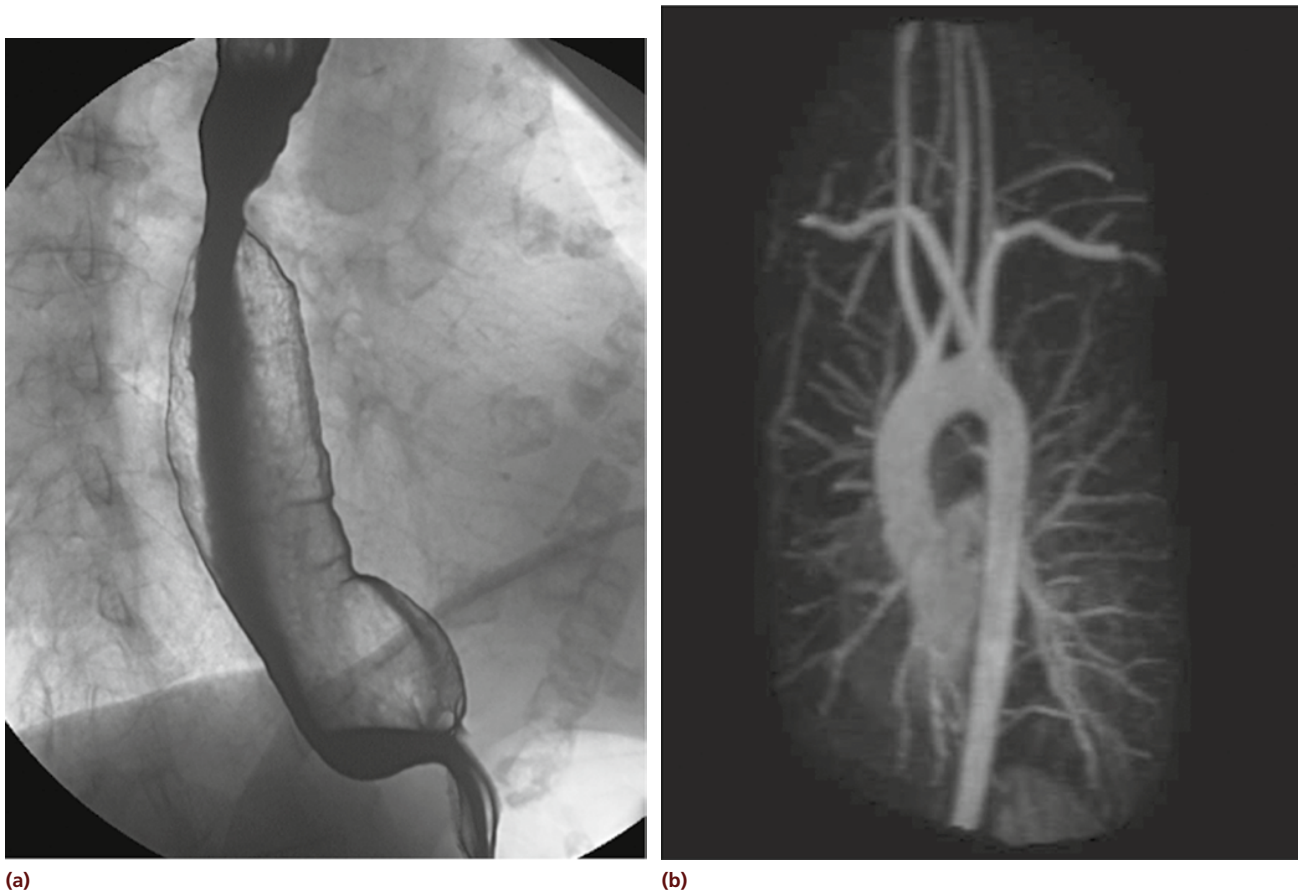




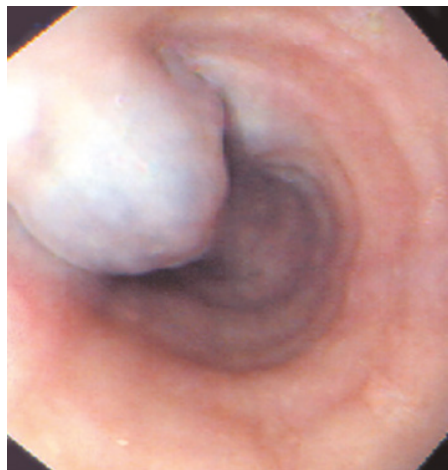
**Figure 1.3** (a) Feline esophagus demonstrating rippling or plications of the esophageal mucosa. This is a transient occurrence and disappears with continued observation as shown in the left panel. (b) Eosinophilic esophagitis can present with a similar appearance but the rings persist with air insufflation and are less tightly spaced apart.



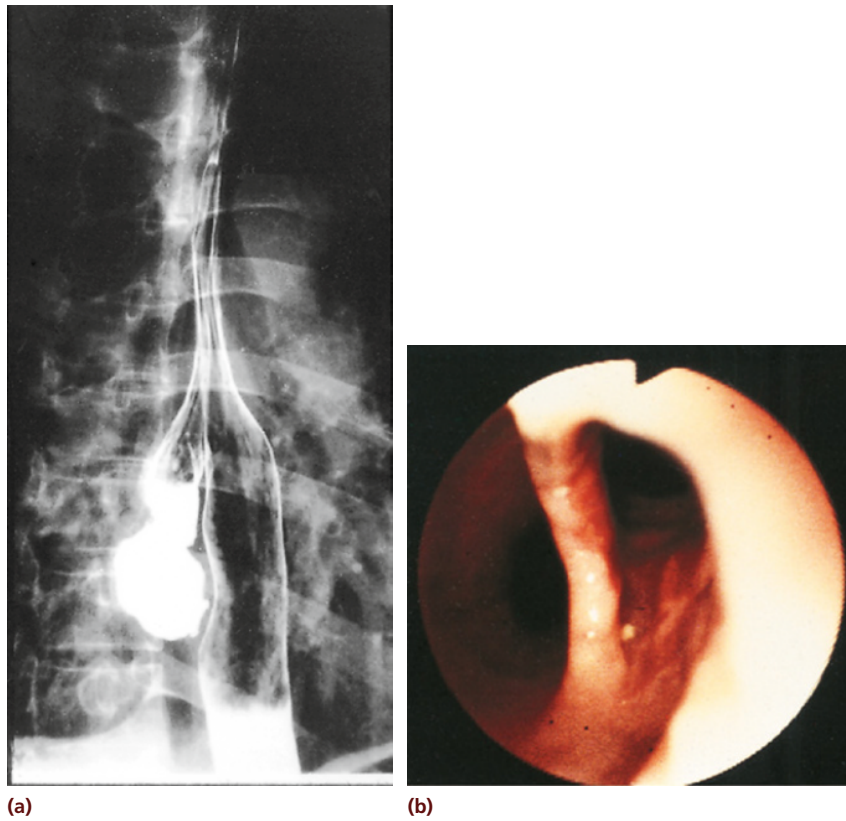
**Figure 1.4** Barium esophagram of a patient with congenital esophageal stenosis. Initial study at age 1 (a) with subsequent image at age 20 (b) showing a persistent distal esophageal stricture.



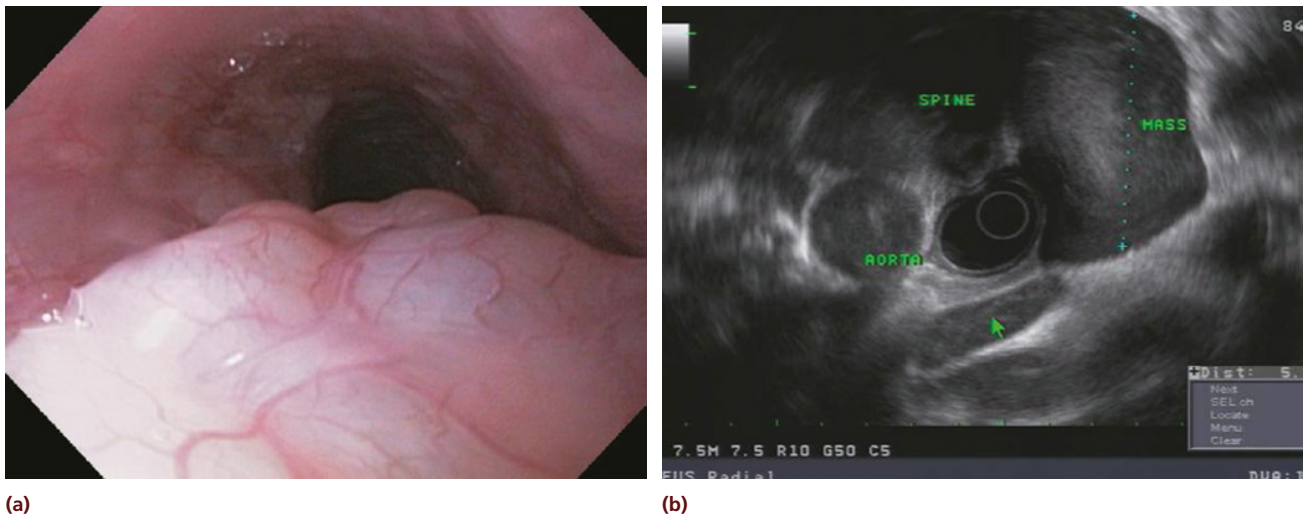
**Figure 1.5** Dysphagia lusoria represents symptomatic esophageal compression by a vascular anomaly of the aortic arch, most commonly by an aberrant right subclavian artery. **(a)** Barium esophagram in a patient reveals thoracic esophageal compression by an aberrant right subclavian artery posterior to the esophagus. **(b)** Magnetic resonance angiography reveals an aberrant right subclavian artery arising from the aortic arch.



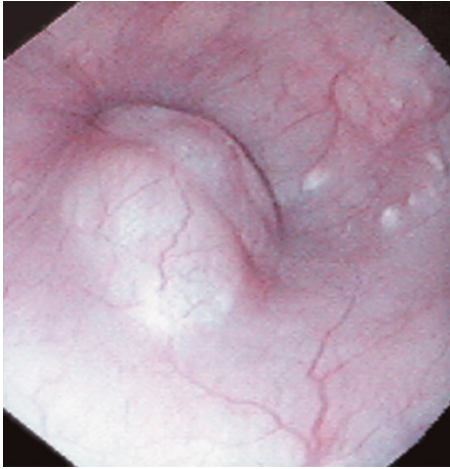
**Figure 1.6** Congenital venous malformations as depicted may also be referred to as primary esophageal varices when no secondary cause such as portal hypertension can be identified. These venous structures rarely bleed spontaneously. Endosonography confirmed a conglomerate of venous channels in this case.



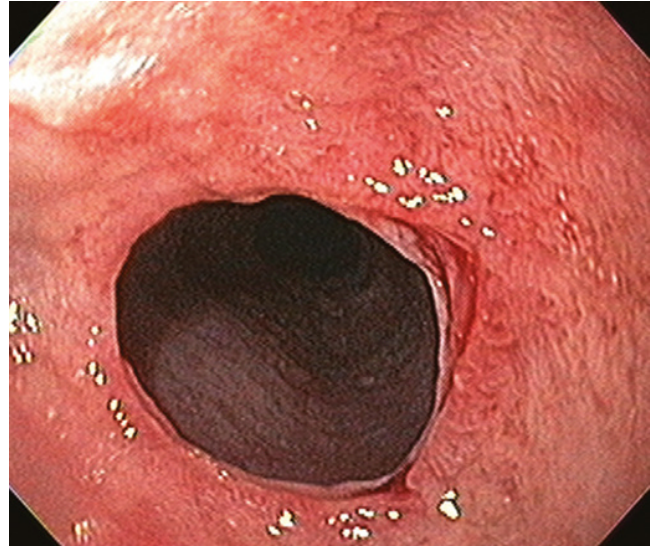
**Figure 1.7** (a) Radiograph showing a large, congenital, tubular duplication of the esophagus. (b) Endoscopic view showing the opening to the tubular duplication (right) and esophageal lumen (left). Congenital esophageal duplications may be tubular or cystic.



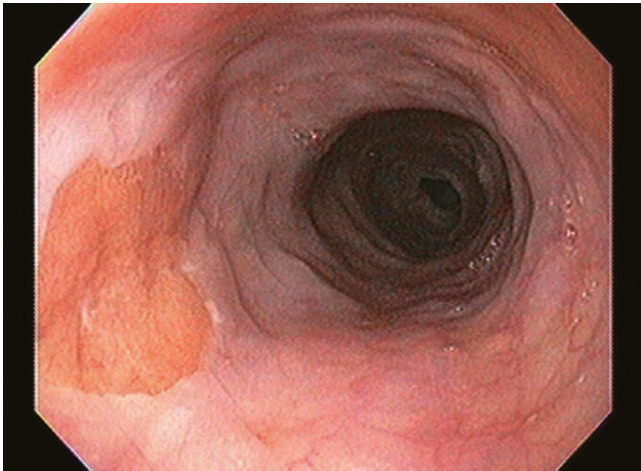
**Figure 1.8** Congenital esophageal duplication cysts may be present as submucosal lesions on upper endoscopy (a). (b) Endoscopic ultrasonographic image of a large duplication cyst. Duplication cysts are the second most common benign esophageal submucosal lesion with stromal tumors being more common. Source: Images courtesy of Sri Komanduri, MD.



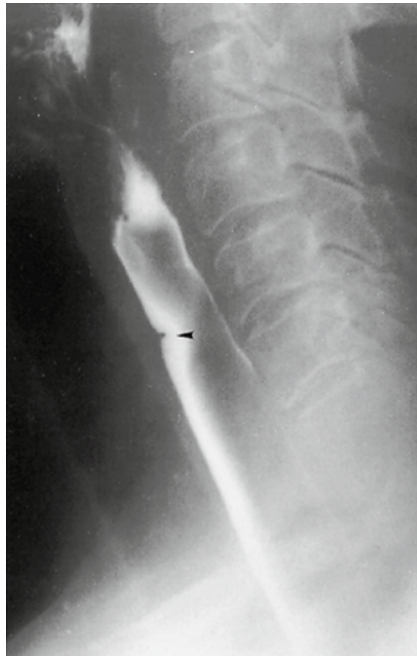
**Figure 1.9** Small intramural cysts such as the bilobate type shown here are not symptomatic and are typically identified on barium esophagram or endoscopy for another indication. The cystic nature of the lesion can be confirmed using endoscopic ultrasonography. The differential diagnosis includes submucosal esophageal lesions and esophageal varices.



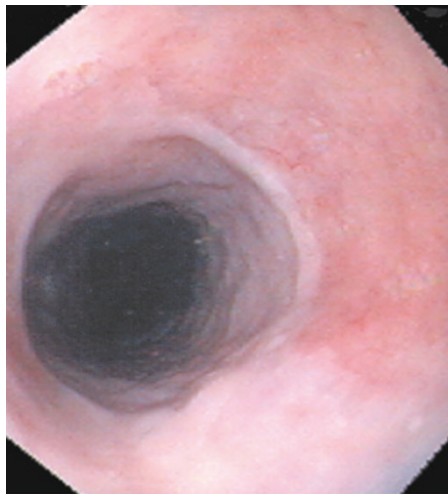
**Figure 1.11** A large, circumferential focus of heterotopic gastric mucosa in the cervical esophagus associated with a circumferential mucosal web immediately distally. The web in this case likely represents a form of peptic stricture related to acid secretion from parietal cells within the inlet patch.



**Figure 1.10** Heterotopic gastric mucosa (inlet patch) in the cervical esophagus. The reported prevalence approximates 4%. The lesions can be unifocal, as in the case illustrated, multifocal or circumferential.

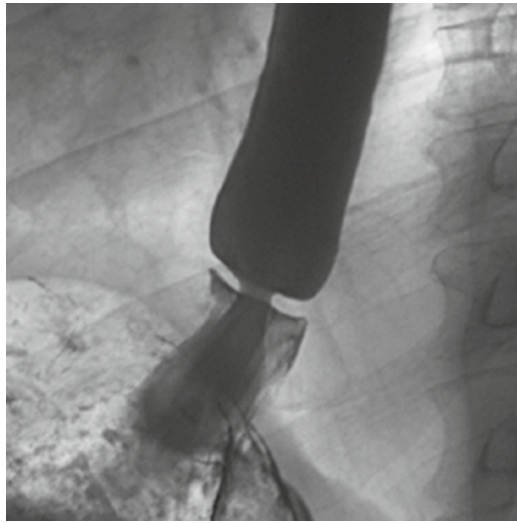


(a)

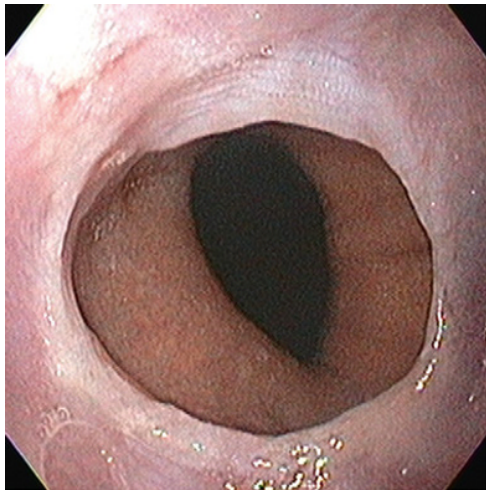


(b)

**Figure 1.12** (a) Barium contrast radiograph showing a mucosal web in the cervical esophagus, often an incidental finding. (b) Corresponding endoscopic view of the cervical web from (a) demonstrates a proximal gastric inlet patch with web creating a shelf or lip at the distal aspect of the heterotopic gastric mucosa.

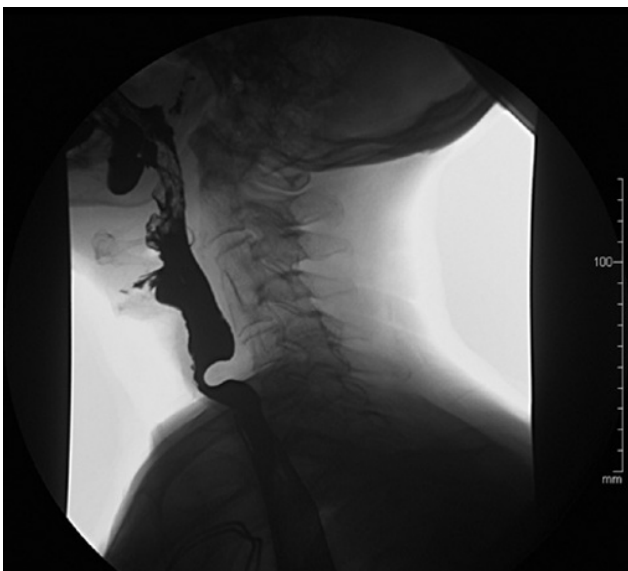


(a)



(b)

**Figure 1.13** (a) A high-grade stenosis from a Schatzki ring located at the esophagogastric junction on a barium esophagram. (b) Anterograde and retrograde endoscopic views of a Schatzki ring. Schatzki rings are almost invariably seen in association with hiatal hernia as in the cases here. The inner ring diameter of a Schatzki ring is an important determinant of whether the ring is associated with dysphagia.



**Figure 1.14** Barium esophagram depicting a cricopharyngeal bar in an elderly patient presenting with dysphagia. The bar is a posterior indentation arising from the cricopharyngeus muscle.