Esophageal introitus (with videos)  
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For GI endoscopists and otolaryngologists, esophageal introitus (EI) is an arbitrary yet overlapping boundary. Many times, the term EI is used interchangeably with pharyngoesophageal sphincter or upper esophageal sphincter.1-2 In this review, the authors broadened the definition of EI to cover the lower part of the hypopharynx posterior to the larynx, the esophageal opening posterior to the cricoid prominence, and the area between the bilateral pyriform fossae and the cervical esophagus (Fig. 1). Although much pathology can be observed within this region, careful and systemic examination of the EI often is not stressed enough during GI endoscopic training and practice. The authors demonstrate its anatomy, endoscopic findings, and pathologies within EI and their management. Dysphagia is a common symptom in patients with oropharyngeal or esophageal pathologies and is generally categorized into 2 types of dysphagia: oropharyngeal and esophageal.3 We prefer the term pharyngoesophageal dysphagia to describe dysphagia occurring within the EI,4 whereas oropharyngeal dysphagia more specifically refers to symptoms related to oropharyngeal pathologies, such as myasthenia and thyrotoxicosis. Therefore, dysphagia may be categorized into 3 types: oropharyngeal, pharyngoesophageal, and esophageal. Esophageal dysphagia refers to symptoms related to esophageal pathologies distal to the EI. The complete 1-hour digital video content pertaining to this review was published recently by the American Society for Gastrointestinal Endoscopy and is available at http://portal.asge.org/products/details.aspx?catid=3&prodid=408. With permission, selected video footages are included in this review.

ANATOMY AND ENDOSCOPIC EXAMINATION

The EI muscles include the inferior pharyngeal constrictor, cricopharyngeus, and cervical esophagus (Fig. 2).1-3,5-7 The length of EI is about 3 cm to 5 cm, and the cricopharyngeus is the main component of these closure muscles. The cricopharyngeus is a striated muscle and is responsible for the high pressure zone within the EI. The cricopharyngeus is innervated by branches of the vagus nerves and recurrent laryngeal nerve. Swallowing is accomplished when the cricopharyngeus muscle relaxes, and pharyngeal pressure is sufficient to propel a bolus through the open sphincter. EI contractions can be induced with either esophageal distension mediated by the vagovagal reflex or pressure on the pharyngeal mucosa mediated by a glossopharyngovagal reflex.1-2 Functional evaluation of the EI can be obtained with videofluoroscopic swallow examination, manometry, scintigraphy, and electromyography.2,5-8 However, a range of normal values remains controversial and their utility uncertain.2 The manometric evaluation of the upper esophageal sphincter is difficult considering the short zone of interest and the rapid movement with swallowing or any stimulation including that of the catheter. Videofluoroscopic evaluation is the most convenient of the currently available methods.

The authors recommend that endoscopic examination of the EI start from the oropharynx. Normal endoscopic examination and various postoperative anatomies of the EI are described in Video 1 (available online at www.giejournal.org). At the inlet of the oropharynx, the soft palate, uvula, bilateral palatine tonsils, and pharyngeopiglottic folds can be found (Fig. 3). At the root of the tongue, valleate papillae and lingual follicles can be observed as well. At the hypopharyngeal inlet, the endoscopist should see the pyriform fossa, aryepiglottic fold, cuneiform tubercle, and corniculate tubercle on each side (Fig. 4). From posterior to anterior direction, the midline anatomic landmarks are the posterior pharyngeal wall, posterior cricoid recess, interarytenoid notch, laryngeal inlet, vocal cords, epiglottis, and vallecula. The esophageal opening is located between the bilateral pyriform fossae and down in the postcricoid recess. The cricopharyngeus muscle is located at the level of the esophageal opening, which usually is

Abbreviations: EI, esophageal introitus; TEF, tracheoesophageal fistula.

DISCLOSURE: All authors disclosed no financial relationships relevant to this publication.

This video can be viewed directly from the GIE website or by using the QR code and your mobile device. Download a free QR code scanner by searching “QR Scanner” in your mobile device’s app store.

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located at 14 to 15 cm as measured from the incisor teeth, at about the level of the C-6 vertebra.

On endoscopy, the normal mucosa lining of the EI is squamous and appears whitish pink or silver-red in color, smooth, and with fine vascular patterns (Fig. 5). The vascular patterns and mucosal pathology can be better visualized and demarcated under digital chromoendoscopy, such as narrow-band imaging. Normally, we should not observe much mucus accumulation within the EI.

**Glycogenic acanthosis**

In about 3% to 4% of patients undergoing upper endoscopy, we see glycogenic acanthosis9-11 (Fig. 6). Glycogenic acanthosis appears as focal or multifocal whitish smooth plaques a few millimeters in size, and they are hyperplastic squamous epithelium with abundant intracellular glycogen deposits. Mild glycogenic acanthosis is a normal benign finding and is not associated with inflammation. Extensive glycogenic acanthosis can be associated with Cowden’s syndrome.11

**Gastric mucosal heterotopia**

Gastric mucosa heterotopia in the cervical esophagus is frequently underdiagnosed, and its prevalence rate is about 2%.12-14 The heterotopic mucosa is mostly consists of cardiac-fundic gland–type gastric mucosa.12-13 Under white light endoscopy, it appears as a flat or slightly raised salmon-colored patch, also called the inlet patch. The inlet patch can be singular or multiple. Gastric mucosal heterotopia is best observed under digital chromoendoscopy (Fig. 7). Occasionally, circumferential gastric heterotopia, or inlet segment, can be found.14 In one study, the prevalence of Helicobacter pylori infection in the patch was 25%.12 Gastric column heterotopia and metaplasia (as in Barrett’s esophagus) are different. Heterotopia generally means displacement of tissue in an abnormal location during embryologic development, whereas metaplasia represents conversion or transformation of one type of adult tissue into another after birth.15 In contrast to Barrett’s esophagus, gastric mucosa heterotopia should not be regarded as a precancerous lesion.12 Malignant transformation to adenocarcinoma is exceedingly rare.12 Inlet patch and inlet segment generally do not cause symptoms and are simply incidental findings. It is unknown why the majority of these patients with inlet patch are asymptomatic. Occasionally, symptoms may arise and are regarded as a result of the acid produced by the parietal cells.13,15 The symptoms include retrosternal burning sensation, pharyngoesophageal dysphagia and/or odynophagia, globus sensation, throat irritation, hoarseness, and coughing. Esophageal stricture and web can develop within the inlet segment. There are no clinical guidelines on whether and when to obtain endoscopic biopsy specimens for heterotopic tissue confirmation. Most inlet patches are in fact asymptomatic, and routine biopsies to confirm the presence are unnecessary. Endoscopic biopsy should be performed in adult patients with throat or introital symptoms suspicious of squamous dysplasia or other neoplasm, infection, ulceration, stricture, fistula, or mass lesion. Asymptomatic inlet patch or inlet segment requires neither specific therapy nor endoscopic surveillance. In patients with throat or introital symptoms suspicious of a causal relationship with acid secretion from the heterotopic mucosa, histologic confirmation by biopsy and proximal pH measurements can aid diagnosis and guide therapy.15 Because the proposed therapy is minimally invasive, it is reasonable to skip pH testing if the suspicion is strong and the patient agrees. Management of symptomatic lesions can start with proton pump inhibitor therapy. If optimal symptomatic control cannot be achieved, endoscopic ablation with bipolar coagulation, argon plasma coagulation, or a radiofrequency ablation device can be attempted. One study suggests that the globus sensation caused by an inlet patch can respond to endoscopic argon plasma coagulation ablation therapy.16
Cricopharyngeal bar

The cricopharyngeal bar is a radiologic description of the posterior bar-like impression at or below the esophageal opening, corresponding to the cricopharyngeus muscle.\(^{17-19}\) One study suggested that the underlying pathogenesis of the cricopharyngeal bar as reduced cricopharyngeus muscle compliance.\(^{17}\) In about 5% to 19% of the asymptomatic population, it can be seen on the lateral view of a barium esophagram (Fig. 8) and on videofluoroscopic swallowing studies.\(^{18}\) The bar usually is an incidental finding and does not cause symptoms. In some patients with a prominent bar, pharyngoesophageal dysphagia is present. Treatment options of symptomatic cricopharyngeal bar or spasm include endoscopic dilation by using savory dilators or injection of botulinum toxin into the muscle.\(^{19-20}\) Cricopharyngeal bar also can be seen in Zenker’s diverticulum and is described under the diverticulum section. Injection of botulinum toxin is ineffective in this setting. Cricopharyngeal achalasia is a term generally reserved for pharyngoesophageal dysphagia caused by cricopharyngeus muscle dysfunction.\(^{17,18}\) Cricopharyngeal myotomy and botulinum toxin injection can be used to treat this condition.

Esophageal rings and webs

In the esophagus, although the terms rings and webs often are used interchangeably, in fact they are different entities. An esophageal ring is circumferential or concentric with mucosal and submucosal involvement, whereas a web is partial or eccentric and involves the mucosa.\(^{21}\) A web generally appears as a membrane-like constriction in the cervical esophagus, referred to as an esophageal or postcricoid web (Fig. 9). On the other hand, an esophageal ring usually is located at the squamocolumnar junction in the distal esophagus. When multiple rings are present throughout the esophagus, the terms ringed esophagus, corrugated esophagus, or feline esophagus sometimes are used, and they can be caused by eosinophilic esophagitis, reflux and non-reflux esophagitis such as graft-versus-host disease, and some autoimmune conditions. The barium esophagram defines a web better than endoscopy and generally reveals a single, thin indentation. Congenital esophageal webs are rare and can present as dysphagia or regurgitation in early childhood.\(^{22}\) In adults, most esophageal webs are idiopathic and respond to endoscopic dilation if they cause obstructive dysphagia. Esophageal biopsy around the web should be considered to rule out eosinophilic esophagitis and other forms of esophagitis. In addition, the web or ring can be disrupted by performing biopsy on the lesion, obviating the need for endoscopic dilatation. Plummer-Vinson syndrome, also called Peterson-Brown-Kelly syndrome or sideropenic dysphagia, consists of a triad of pharyngoesophageal dysphagia, esophageal webs, and iron deficiency anemia.\(^{23}\) Other coexisting symptoms and signs may include glossitis, angular cheilitis, and koilonychia. The primary therapy is iron repletion, which usually reverses these symptoms. In case of significant obstruction of the esophageal lumen by esophageal web or persistent dysphagia despite iron supplementation, dilation or disruption of the web by biopsy is indicated. These patients should be followed closely for their increased risk of squamous cell carcinoma of the pharynx and the upper esophagus.\(^{24}\) Although surveillance endoscopy is recommended,\(^{25}\) the optimal
surveillance interval is unknown because of the uncommon nature of this syndrome.

**Stenoses**

Etiologically, stenoses or strictures within the EI can be categorized into two types: (1) intrinsic stenoses caused by inflammation, infection, fibrosis, neoplasia, and anastomosis; or (2) extrinsic stenoses from external neoplastic invasion or compression from surrounding lymph node enlargement or vertebral osteophytes. Inflammatory conditions include peptic injury, caustic substance ingestion, medication, radiation, iatrogenic, autoimmune, and idiopathic disease processes. Although GERD causes a majority of the esophageal strictures, the stenosis usually is in the distal to mid esophagus. In a recent review, the overall risk of pharyngoesophageal stricture after treatment for head and neck cancer was 7.2%: 5.7% after conventional radiation and 16.7% after intensity modulated radiation therapy. For complex stenoses involving dysmotility, a multidisciplinary approach is recommended, with a team involving a GI endoscopist, speech pathologist, nutritionist, and, at times, a surgeon.

**Foreign body impaction**

Although the majority of ingested foreign bodies will pass spontaneously, impaction of foreign bodies in the esophageal introitus is a medical and endoscopic emergency. If the foreign body is suspected in the hypopharynx, an otolaryngologist is generally called by the emergency department staff. A GI endoscopist is called if the foreign body cannot be located in the hypopharynx or is suspected in the esophagus from the beginning. Commonly found objects include fish bones, chicken bones, pieces of glass, dental prostheses, coins, and needles. The aims of the initial patient assessment are to identify the type of object, its location, and the presence of any associated adverse events. Radiographic evaluation is helpful to confirm the location of foreign bodies and associated adverse events. Plain films of the neck and chest commonly will show the location of radiopaque objects. Both anteroposterior and lateral views are necessary, because some radiopaque objects overlying the vertebral column may be visible only on the lateral view.
Multidetector row CT is superior to plain radiographs for the detection of pharyngoesophageal foreign bodies and provides additional information for the management of complicated cases (Fig. 10). In cases of pointed or sharp object ingestion, symptoms and signs suggestive of perforation, infection, abscess formation, or other coexisting conditions, CT is recommended. CT also is useful in excluding potential vascular structures and evaluating extraluminal conditions near the object. Emergent endoscopy is indicated in (1) foreign-body impaction within the esophageal introitus (Fig. 11), (2) esophageal obstruction when the patient is unable to swallow secretions, and (3) disk batteries and sharp-pointed foreign bodies in the esophagus. In general, esophageal foreign bodies and food impactions without complete obstruction should be removed within 24 hours (urgent endoscopy). For sharp or pointed objects, commonly used over-the-scope devices for mucosa and/or airway protection include foreign body hood protectors, banding device caps, and friction-fit caps. In the authors’ experiences, foreign body hood protectors and banding device caps are inexpensive and versatile devices for removing sharp or pointed objects. For most foreign bodies in the EI, rigid endoscopic removal by an otolaryngologist is equally effective and safe as a flexible endoscopic approach. For foreign bodies impacted in the cervical esophagus, flexible endoscopy is probably more convenient.

**Diverticula**

Diverticula are uncommon in the EI, and they generally are of 4 types: lateral pharyngeal or laryngopharyngeal diverticulum, Zenker’s diverticulum, posttraumatic

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**Figure 7.** Endoscopic images of the gastric mucosa heterotopia or inlet patch in the cervical esophagus under digital chromoendoscopy.

**Figure 8.** On barium esophagram, the cricopharyngeal bar (arrow) appears as a post bar-like impression at or below the esophageal opening, corresponding to the cricopharyngeal muscle.

**Figure 9.** On barium esophagram, an esophageal web (arrow) appears as a thin indentation in the cervical esophagus.
diverticulum or pseudodiverticulum,40 and Killian-Jamieson diverticulum41-43 (Video 2, available online at www.giejournal.org). Diverticula in the EI and esophagus are best studied by barium esophagram. Endoscopy can miss small lesions. In addition, a video fluoroscopic study can reveal the dynamics of the diverticulum in relationship to the EI during observed swallowing. Lateral pharyngeal diverticula or pharyngoceles are rare (Fig. 12). It is a protrusion in the piriform recess or in the vallecula. It can be unilateral or bilateral. The presumed etiology is pharyngeal overpressure from dysphagia. The treatment is surgical or endoscopic.33-34 Zenker’s diverticulum is the protrusion of pharyngeal mucosa through the posterior wall of the pharynx through the Killian triangle, limited inferiorly by the cricopharyngeal muscle and laterally by the thyropharyngeal muscle (Fig. 13).35 Bilateral Zenker’s diverticulum is rare.39 Posttraumatic diverticulum can mimic Zenker’s diverticulum.40 The primary etiology of Zenker’s diverticulum is thought to be the increased intraluminal pressure secondary to cricopharyngeal spasm. Zenker’s diverticulum can cause dysphagia, regurgitation of undigested food, post-swallow hypopharyngeal reflex, cough, and aspiration. Zenker’s diverticula often can create difficulty in esophageal intubation during endoscopy. Iatrogenic perforation and submucosal dissection within the esophageal introitus have been reported.41 Rigid and flexible endoscopic treatment for Zenker’s diverticula focuses on releasing the cricopharyngeal spasm by performing diverticulotomy on the septum.35-38 Flexible endoscopic diverticulotomy is safe and effective37-38 (Fig. 14). Killian-Jamieson diverticulum, also called lateral cervical esophageal diverticulum, protrudes through a muscular gap (Killian-Jamieson triangle) in the anterolateral wall of the cervical esophagus inferior to the cricopharyngeus, superior to the circular muscle of the esophagus, and lateral to the longitudinal muscle of the esophagus (Fig. 15).41-42 Killian-Jamieson diverticulum arises inferior and lateral to the Zenker’s diverticulum. Unlike Zenker’s diverticulum, Killian-Jamieson diverticulum does not have a cricopharyngeal bar or septum. The pathogenesis of Killian-Jamieson diverticulum is unclear. It is likely to be acquired. The treatment is surgical or endoscopic for symptomatic Killian-Jamieson diverticulum.42-43

Ischemia
Because of the rich vascular supply of EI, ischemia or necrosis of EI is not common. Acute esophageal necrosis, also called black esophagus, can be observed in patients with systemic hypotension or shock, severe caustic substance injury, local infection, or inflammation leading to reduction in esophageal blood perfusion (Fig. 16). Elderly patients are particularly susceptible.35 Diffuse ulcerations with tissue friability and dark pigmentation of the esophagus are seen on upper endoscopy, and there are histopathologic findings of mucosal necrosis. The most pronounced endoscopic findings usually are present in the distal third of the esophagus, likely because of superimposed acid reflux injury. The most common presenting symptom is acute upper GI bleeding. The treatment is supportive care, hemodynamic stabilization, and aggressive acid suppression.35 The mortality associated with acute esophageal necrosis usually is related to coexisting illnesses rather than further esophageal adverse events such as perforation.
Postoperative esophageal conduit ischemia or necrosis is an uncommon but serious adverse event of esophageal surgery. Unexplained tachycardia, respiratory failure, leukocytosis, or any evidence for graft or anastomotic leakage should prompt a search for conduit ischemia. The diagnosis is made by contrast study, endoscopy, or direct operative inspection.

**Proximal esophageal (downhill) varices**

Proximal esophageal varices, often called *downhill varices*, generally develop in patients with superior vena cava obstruction and/or superior vena cava syndrome, with reversal of the normal blood flow direction in the upper esophageal plexus. The etiology of superior vena cava syndrome in reported cases includes intravenous catheter–related thrombosis, systemic venulitis, mediastinal fibrosis, carcinoma of the lung or thyroid gland, intrathoracic goiter, surgical ligation of the superior vena cava, and metastatic carcinoma or mediastinal mass of unknown origin. Bleeding from the downhill varices is rare. Endoscopic band ligation and injection sclerotherapy have been reported for proximal variceal bleeding. Because of its hemodynamics, band ligation can be started from the proximal end within the introitus (*Video 3*, available online at [www.giejournal.org](http://www.giejournal.org)).

**Inflammation and infection**

The symptoms associated with the inflammation and infection within the EI can include pharyngoesophageal dysphagia, odynophagia, sore throat, throat irritation,
cough, GI bleeding, or spitting of blood. Common noninfectious etiologies include GERD, recurrent vomiting, acute radiation mucositis, chronic radiation injury, esophagitis desiccans superficialis,49 eosinophilic esophagitis, caustic substance ingestion, toxic fume inhalation, graft-versus-host disease,50 pemphigus vulgaris, 51-52 benign mucous membrane (cicatricial) pemphigoid,53-54 epidermolysis bullosa dystrophica, 55-56 Crohn’s disease, and drug-induced mucositis.57

EI can be involved in graft-versus-host disease after allelogeneic bone marrow transplantation.59 On endoscopy, inflammation, erosion, and esophageal webs can be seen. The diagnosis is confirmed by random and targeted biopsy.

EI can be associated with certain skin diseases, such as pemphigus vulgaris, benign mucous membrane (cicatrical) pemphigoid, and epidermolysis bullosa dystrophica. Pemphigus is characterized by cutaneous and mucosal blistering. Pemphigus vulgaris, the most common and important variant, is an autoimmune blistering disease characterized by circulating pathogenic immunoglobulin G antibodies against desmoglein 3.51-52 Vesiculobullous lesions and ruptured bullae with mucosal denudation can be seen within the EI (Fig. 17). The role of endoscopy is to confirm EI involvement and to rule out other conditions. Current treatment is largely based on systemic immunosuppression by using systemic corticosteroids with other immunomodulators. Cicatricial pemphigoid is a rare systemic autoimmune subepidermal bullous disease primarily involving mucous membranes.53-54 Histologically, there are subepidermal blisters and erosions that heal with scarring. EI involvement is uncommon, and it occurs usually in patients with established ocular disease. The classic endoscopic findings include erosions and scarring, esophageal webs or strictures, and blister formations after dilation or trauma (Fig. 18). Despite immunosuppressive drugs, some patients need endoscopic dilatation to treat the symptomatic dominant esophageal strictures. Through-the-scope endoscopic balloon dilatation is preferred by us in this situation in order to avoid or minimize extensive blister formations after dilatation with a Savary dilator or a bougie. Epidermolysis bullosa acquisita is a subepidermal blistering disorder associated with autoimmunity to type VII collagen, which is the collagen localized to anchoring fibrils within the dermoeipidermal junction of skin.55-56 Inflammation, bullous eruption, ulceration, scars, webs, and strictures can be observed in EI.

Medication-induced stricture (pill mucositis) often occurs at the anatomic narrowing: esophageal opening, middle one third behind the left atrium, and the gastroesophageal junction. The most common culprit medications are nonsteroidal anti-inflammatory drugs, iron supplements, tetracycline, doxycycline, alendronate, potassium chloride, ascorbic acid, and quinidine.57

Infectious esophagitis generally develops in immunocompromised patients, with candidiasis being the most common infection.58-62 Less-common infections include herpes simplex and cytomegalovirus esophagitis.63 When obtaining tissue diagnosis, physicians should ensure that biopsy specimens are taken from both the ulcer edge and base. In herpes infection, the viropathic changes tend to be present at the ulcer margins, whereas viropathic
findings are present within the ulcer base during cytomegalovirus infection. Other infectious agents or infections include HIV, actinomycesis, aspergillosis, histoplasmosis, and tuberculosis. Immunocompromised patients also can develop phlegmonous esophagitis and infectious necrotizing esophagitis. They are rare and associated with high mortality. These are suppurative bacterial infections, and the most common pathogens are *Streptococcus*, *Staphylococcus*, *Escherichia coli*, *Haemophilus influenzae*, *Proteus*, and *Clostridia*.

**Fistulas**

Fistulas within the EI include pharyngocutaneous, pharyngo-respiratory, esophagopleural or esophagomediastinal, and esophagorespiratory or tracheoesophageal fistulas (TEF). The development of a pharyngocutaneous fistula after oncologic head and neck surgery is a serious adverse event and is common after major hypopharyngeal and laryngeal ablative surgery. It was recommended that these patients are best treated with regional mucocutaneous flaps or free tissue transfers at expert centers. TEF is a congenital or acquired communication between the trachea and esophagus. Most patients with congenital TEFs are diagnosed immediately after birth or during infancy. Acquired TEF occurs secondary to malignant disease (neoplasms of the esophagus, lung, and trachea), infection (tuberculosis, aspergillosis), ruptured diverticula, and trauma. Post-intubation TEF infrequently develops after prolonged mechanical ventilation with an endotracheal or tracheostomy tube. Infrequently, traumatic TEF develops after tracheostomy and is related to improper tracheal incision (Fig. 19). Improperly positioned tracheal tubes exert posterior pressure against the esophagus, resulting in a TEF. The prognosis is generally poor in these patients. Endoscopic interventions with mechanical tissue approximation (such as the over-the-scope clipping devices), tissue adhesive application, and temporary or permanent stenting have been reported with variable success rates.

**Neoplasms**

Over 95% of hypopharyngeal cancers are squamous carcinomas, which usually present as infiltrating ulcerative lesions. The most common site for hypopharyngeal cancer is the piriform sinus, followed by the postcricoid region and the posterior pharyngeal wall (Fig. 20). The
incidence of poorly differentiated cancer is higher in the hypopharynx than in other regions. Hypopharyngeal cancers are difficult to catch in their earliest stages and have one of the highest mortality rates of any head and neck cancer. Symptoms of hypopharyngeal cancer may include swollen lymph nodes in the neck, sore throat, radiating pain from the throat to the ears, voice change, pharyngoesophageal dysphagia, and odynophagia. Postcricoid cancers are frequently circumferential and cause dysphagia, whereas cancers in the piriform sinus tend to remain asymptomatic for some time. The risk factors of hypopharyngeal cancer are smoking, chewing tobacco, heavy alcohol use, poor diet, and Plummer-Vinson syndrome. It should be noted that there is an increased risk of esophageal cancer in patients with otolaryngeal cancers as either synchronous or metachronous lesions or secondary to radiation exposure. Small, early cancers of the piriform sinuses are curable with radiation therapy. Small, early cancers of the posterior pharyngeal wall can be treated with larynx-preserving local excision. There is growing evidence and interest that the detection and resection of early pharyngeal and hypopharyngeal dysplasia and cancers can be achieved with flexible endoscopy under digital chromoendoscopic guidance.\(^7\) If a cancer extends to or arises in the cervical esophagus, laryngopharyngectomy and esophagectomy may be required. A gastric pull-up procedure may be needed. Other neoplasms found in the EI include papilloma and papillomatosis, lipoma, fibrolipoma, adenocarcinoma, leiomyoma, granular cell tumor, spindle-cell carcinoma, neuroendocrine tumor, and lymphoma.

**REFERENCES**


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1. Inlet patch
   a. It is composed of metaplastic gastric epithelium
   b. Can be colonized with H. pylori
   c. Should always be biopsied to be sure there is no malignancy
   d. If symptomatic, ablation with APC may be effective

2. True statements regarding the cricopharyngeus include
   a. It is composed mainly of smooth muscle
   b. Is innervated by the vagus nerve and the recurrent laryngeal nerve
   c. Relaxes when the esophageal lumen is distended
   d. Esophageal manometry is an accurate way of assessing cricopharyngeus function

True or False

3. Esophageal webs are visualized better by barium studies than by EGD.

4. Whitish smooth plaques in the esophagus are caused by hyperplastic squamous epithelium, is known as glycogenic acanthosis and are considered premalignant lesions.

5. Pemphigus vulgaris can affect the proximal esophagus with ruptured bullae and mucosa denudation, if dilation is needed, balloons are preferred to Savary dilators

6. Barium studies are more sensitive than endoscopy to detect smaller upper esophageal diverticuli.

7. A cricopharyngeal bar noted on barium esophagram indicates cricopharyngeal sphincter dysfunction and usually accounts for symptoms of dysphagia

8. Iron replacement in Plummer-Vinson syndrome may be sufficient to resolve the esophageal web in some cases

9. A web only contains mucosa; a ring contains mucosal and submucosal tissue

10. Downhill esophageal varices may be a complication of superior vena cava thrombosis