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.

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

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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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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 pharyngoepiglottic folds can be found (Fig. 3). At the root of the tongue, vallate 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, postcricoid 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

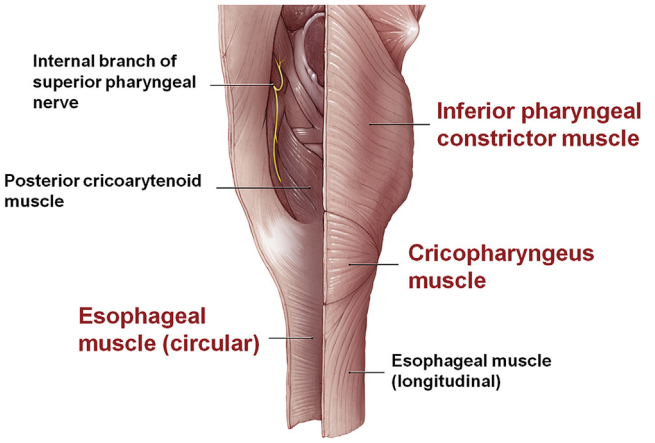

Figure 1. Oropharynx, hypopharynx, and cervical esophagus.

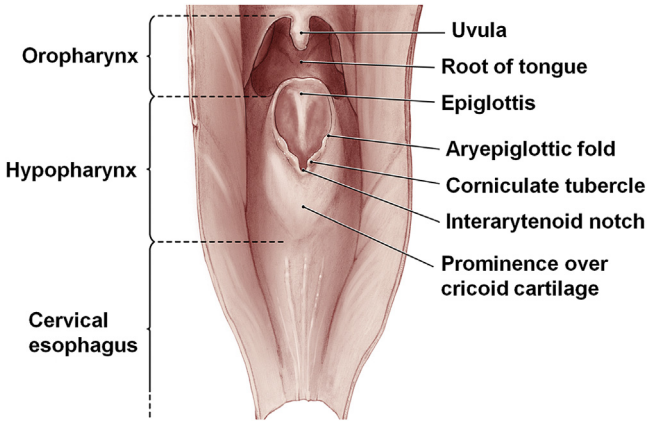

Figure 2. Closure muscles of the esophageal introitus or upper esophageal sphincter muscles: inferior pharyngeal constrictor, cricopharyngeus, and cervical esophagus. The cricopharyngeus is the main component of these closure muscles.

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 acanthosis[9-11] (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.[13] 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, mucosal findings 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]