GIT practical 1
Figure 16-1 Aphthous ulcer. Single ulceration with an erythematous halo surrounding a yellowish fibrinopurulent membrane.
Figure 16-2 Fibroma. Smooth pink exophytic nodule on the buccal mucosa.
Irritation fibroma
Figure 16-5  Leukoplakia. A, Clinical appearance of leukoplakias is highly variable. In this example, the lesion is relatively smooth and thin with well-demarcated borders. B, The histologic appearance of a leukoplakia showing severe dysplasia that is characterized by nuclear and cellular pleomorphism, numerous mitotic figures, and a loss of normal maturation.
Figure 16-4 Erythroplakia. A, Lesion of the maxillary gingiva. B, Red lesion of the mandibular alveolar ridge. Biopsy of both lesions revealed carcinoma in situ.
At low power, there is normal squamous mucosa at the lower right, but a squamous cell carcinoma is infiltrating the submucosa and muscularis of the tongue. Risk factors for oral cancers include: use of tobacco, alcoholism, prolonged irritation from ill-fitting dentures or irregular teeth, and chewing betel nut.
At medium power, there is normal squamous mucosa at the right, with a squamous cell carcinoma is infiltrating the submucosa of the tongue at the left. Oral cancers can develop and spread quickly, but many can be detected early and excised.
mucocele
mucocele
mucocele
sialolithiasis
sialadenitis

The slide shows a classic focal lymphocytic infiltration in a minor salivary gland section stained with hematoxylin and eosin. These findings are typical of Sjogren's syndrome.

Courtesy of NIH/NIDCR.
Lip biopsy for Sjögren syndrome
Figure 16-15 Pleomorphic adenoma. A, Slowly enlarging neoplasm in the parotid gland of many years duration. B, The bisected, sharply circumscribed, yellow-white tumor can be seen surrounded by normal salivary gland tissue.
Figure 16-16 Pleomorphic adenoma. A, Low-power view showing a well-demarcated tumor with adjacent normal salivary gland parenchyma. B, High-power view showing epithelial cells and myoepithelial cells within a chondroid matrix material.
Warthin tumor
Figure 16-17 Warthin tumor. A, Low-power view showing epithelial and lymphoid elements. Note the follicular germinal center beneath the epithelium. 
B, Cystic spaces separate lobules of neoplastic epithelium consisting of a double layer of eosinophilic epithelial cells based on a reactive lymphoid stroma.
Figure 16-18 A, Mucoepidermoid carcinoma growing in nests composed of squamous cells as well as clear vacuolated cells containing mucin. B, Mucicarmine stains the mucin reddish pink.
Mucoepidermoid carcinoma
HSV esophagitis
HSV esophagitis
Candidal esophagitis
Candidal esophagitis
Candidal esophagitis
Esophageal Histology: useful

Normal esophagus

GER

Eosinophilic esophagitis

Small number of intraepithelial eosinophils
Basal cell thickening
Lengthening of stromal papillae
Figure 17-5 Esophagitis. A, Reflux esophagitis with scattered intraepithelial eosinophils and mild basal zone expansion. B, Eosinophilic esophagitis is characterized by numerous intraepithelial eosinophils. Abnormal squamous maturation is also apparent.
Barett esophagus
Figure 17-7 Barrett esophagus. A, Normal gastroesophageal junction. B, Barrett esophagus. Note the small islands of residual pale squamous mucosa within the Barrett mucosa. C, Histologic appearance of the gastroesophageal junction in Barrett esophagus. Note the transition between esophageal squamous mucosa (left) and Barrett metaplasia, with abundant metaplastic goblet cells (right).
Another cause for inflammation is a so-called "Barrett esophagus" in which there is gastric-type mucosa above the gastroesophageal junction. The metaplasia results from chronic gastroesophageal reflux disease (GERD). Note the **columnar epithelium** to the left and the **squamous epithelium** at the right. This is "typical" Barrett mucosa, because there is intestinal metaplasia as well (note the goblet cells in the columnar mucosa).
This is Barrett esophagus associated with gastroesophageal reflux disease (GERD) with some dysplasia of the columnar epithelium. There is a long-term risk for adenocarcinoma. The short term problem is inflammation and/or ulceration.
Figure 17-8 Dysplasia in Barrett esophagus. A, Abrupt transition from Barrett metaplasia to low-grade dysplasia (arrow). Note the nuclear stratification and hyperchromasia. B, Architectural irregularities, including gland-within-gland, or cribriform, profiles in high-grade dysplasia.
Figure 17-9 Esophageal cancer. A, Adenocarcinoma usually occurs distally and, as in this case, often involves the gastric cardia. B, Squamous cell carcinoma is most frequently found in the mid-esophagus, where it commonly causes strictures.
SCC
SCC
SCC
Adenocarcinoma, esophagus
Figure 1 Large esophageal varices at EGD.
Varices
Chronic gastritis
Chronic Pan-Gastritis
HELICOBACTER PYLORI
H. Pylori gastritis
Acute gastritis
Atrophic gastritis, intestinal metaplasia
The inside of the stomach where a peptic ulcer has formed.

Diagram shows Pepti ulcer and duodeum ulcer.
Malignant gastric ulcer
Figure 2).

- Four zones of active peptic ulcer.
- The necrotic fibrinoid debris and nonspecific inflammatory infiltrate are labeled by arrowhead.

- Beneath the necrotic and inflammatory zones, there is granulation tissue (arrow).
- Below the granulation tissue, fibrotic tissue is seen (F).

Figure 3). Necrotic fibrinoid debris and inflammatory infiltrate in the ulcer base.
Gastric polyp
Hyperplastic polyp
Fundic gland polyp
Gastric adenocarcinoma, intestinal type
Gastric adenocarcinoma, diffuse type (signet ring cells)
Lymphoepithelial lesion in MALT lymphoma
Gastric MALT lymphoma
GIST

PathPedia.com
GIST
Carcinoid tumor
Carcinoid tumor