Pathology of the oral cavity & esophagus
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Pathology of the oral cavity
Congenital anomalies

• Facial clefts:
  – Lateral – most common
    ➢ failure of fusion between first branchial arch (for maxilla) and frontal process
  • Cleft lip (cheiloschisis)(harelip)(Labium leporium)
  • Gnathoschisis - cleft of the upper jaw
  • Palatoschisis – cleft hard palate
  • Staphyloschisis – uvula (soft palate)
  – Medial
  – Oblique
  – Transverse
Congenital anomalies

- Other congenital anomalies of oral cavity:
  - Defect of vestibulum, accessory mouth, macro-, microstomia,
- Tongue:
  - Aglossia, microglossia, macroglossia (*commonly secondary!*),
  - ankyloglossia (short, lingual frenulum or a highly attached genioglossus muscle restricts tongue movement)
  - Lingua plicata – Fissured surface of the tongue (l.scrotalis, cerebriformis)
- Congenital anomalies of jaws
  - micrognathia,
  - **Prognathia (prognathism)** is a developmental anomaly of **maxilla** where it is positioned forward,
  - **Progenia** – is a developmental anomaly of **mandible** where it is positioned forward

- **Fordyce´s spots** - small, painless, raised, pale, yellow or white spots or bumps on buccal mucosa or on lips—ectopic sebaceous glands
Regressive and metabolic changes

- **Necrosis** (according to ETIOL):
  - Physical factors – Mechanical (incomplete teeth, dental prostheses) (decubital necrosis – ulceration – dif.dg.ca! ? RF of ca ?), termic, radiation
  - Chemical factors – dentistry(*arsenic trioxide*), Ingestion of acid solution, lye …
  - Oral Manifestantions of Systemic Diseases – scurvy, leucemia, …

- **Atrophy**
  - Senile a. – mucosa + salivary glands (+ tooth loss…)
  - Tongue – B-group vitamin deficiency, anemia, disorders of motor innervation
  - **Plummer-Vinson sy**
    - Glossitis, atrophy of tongue and pharyngeal musoca
    - dysphagia, **iron-deficiency** (hypochromic) anemia, koilonychia, achlorhydria, increased incidence of esophageal, pharyngeal, oral cavity cancer!!!
Regressive and metabolic changes

• **Dystrophy**
  – **Amyloidosis**
    • sec. or prim.,
    • commonly tongue – sec. macroglossia, or tumoriform amyloid deposits

• **Pigmentation**
  • **Endogenous**
    – Melanotic pigmentation! (Addison disease, Peutz-Jegher syndrome)
    – Hemosiderin
    – Icterus
  • **Exogenous**
    – heavy metals, …
Circulatory changes

• Usually local manifestation of systemic disorders

• **Anemia**

• **Hyperemia**
  – Inflammation – Erythema, Scarlet fever - raspberry tongue

• **Cyanosis**
  – Chronic heart failure
  – congenital heart defects
  – Polycytemia

• **Edema**
  – Mainly tongue and lips
  – Insect bite, Quincke's edema
  – Inflammations

• **Hemorrhage**
  – hemorrhagic diathesis – petechiae, ecchymosis …
  – Hemangioma, Hereditary hemorrhagic telangiectasia
  – scurvy
Inflammatory processes in the oral cavity

• Terminology – according to location:
  – Cheilitis
  – Gingivitis
  – Stomatitis

• Etiology
  – local
  – systemic
Inflammations in the oral cavity

Nonspecific

Acute
- Serous
- Purulent
- Vesicular
- Pseudomembranous
- Ulcerous

Chronic
- Chronic gingivitis
- Stomatitis nicotinica
- Anguli infectiosi
- Atrophic glossitis
- Cheilitis granulomatosa
- Candidiasis (soor, thrush)

Specific
- TB
- SYPHILIS
Progressive changes

Tumors and tumor-like lesions

- Leukoplakia
- Erythroplakia
Oral cavity

- **Tumor-like lesions** - fibroproliferative lesions
  (pyogenic granuloma, irritation fibroma, peripheral ossifying fibroma, peripheral giant cell granuloma), epulis, cysts

- **Precancerous lesions** - leukoplakia, erythroplakia

- **Tumors** - *benign* (epithelial, mesenchymal)
  - *malignant*
    squamous cell carcinoma
    adenocarcinoma
    malignant melanoma

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**Leukoplakia** - clinical term used to describe *patches* of keratosis.
!!! can be *premalignant*.
hyperkeratosis and hyperplasia of the squamous epithelium
in some cases dysplastic changes - herald the onset of malignant change!!!

**ETI**: heavy cigarette smoking, excessive alcohol consumption and poor dental hygiene
( India and Sri Lanka - chewing betel quids)
Tumors of the oral cavity

**Mesenchymal**
- Benign
  - Fibroma
  - Giant cell granuloma
  - Lipoma
  - Hemangioma
  - Lymphangioma
  - Leiomyoma
  - Granular cell tumor
  - Exostosis
- Malignant
  - Fibrosarcoma
  - Kaposi sarcoma

**Epithelial**
- Benign
  - Papilloma
- Malignant
  - Squamous cell ca

*Location*
- Verrucous ca

**Other**
- Lymphomas
- Neuroectodermal

- Location!
Squamous cell carcinoma SCC

**EPI:**
- the *most common* malignant tumor of the oral mucosa
- may occur at any site
- most often involving the tongue,
- then, in descending order the floor of the mouth, alveolar mucosa, palate and buccal mucosa.
- The male:female ratio is 2:1 for the gums but 10:1 for the lip

**RF:**
- tobacco, alcohol, betel
- HPV infections (tonsils, tongue)
- Radiation
- genetic predisposition
SCC

• MACRO:
  – early stages - raised, firm, pearly plaques or irregular, roughened, or verrucous areas of mucosal thickening, (possibly mistaken for leukoplakia)
  – Either pattern may be superimposed on a background of apparent leukoplakia or erythroplakia.
  – As these lesions enlarge, they typically create **ulcerated and protruding masses** that have irregular and indurated (rolled) borders.

• HISTOL:
  – *similar to the same tumor in other sites and is generally preceded by carcinoma in situ. SCC ranges*
  – from well to poorly differentiated, including undifferentiated and sarcomatoid variants.
  – Well-differentiated, or grade I, tumors are frequently keratinizing
  – At the other end of the spectrum, tumors may be so poorly differentiated that their origin is difficult to determine

• PROGN:
  – Nearly all patients with lip cancer survive five years;
  – by contrast, of those with carcinoma of the **floor of the mouth**, only about one third survive.
Teeth

• Congenital anomalies

• Caries

• Odontogenic cysts and tumors (ameloblastoma)
Congenital anomalies of teeth

- **Disturbances in Size**
  - microdontia
  - macrodontia

- **Disturbances in Shape**
  - Supernumerary cusps
  - Gemination
  - Dens in dente

- **Disturbances in Number**
  - anodontia
  - hypodontia
  - hyperdontia

- **Structural anomalies**
  - Amelogenesis imperfecta
  - Dentinogenesis imperfecta
  - Other factors important in **Enamel** production

- **Disturbances in Eruption**
CARIES (TOOTH DECAY):

• **ETIOPATOG:** interactions of several factors:
  - Bakteria (lactobacillus acidophilus a nonhemolytic streptoc.)
  - saliva
  - Dietary factors
  - Fluoride

• disintegration of enamel prisms after decalcification of the interprismatic substance

• The development of caries proceeds in a stepwise fashion:
  - disintegration of the enamel by bacterial action
  - formation of a small pit that ultimately extends to the dentinoenamel junction,
  - lateral spread of the lesion along the dentin
  - ultimately invasion of the dental pulp, which produces pain.

• **COMPLIC:**
  - *Pulpitis:* inflammation of the pulp by bacteria in dental caries that is associated with pain and may be accompanied by abscesses
  - *Apical (or periapical) granuloma:* most common sequel of pulpitis; composed of chronically inflamed periapical granulation
tissue surrounded by a fibrous capsule that is attached to the root
  - *Radicular cyst* (apical periodontal cyst): proliferation of the squamous epithelium of an apical granuloma to form a cyst lined by stratified squamous epithelium
  - *Periapical abscess:* abscess around the root of a tooth
  - *Osteomyelitis:* uncommon, but may develop by extension of a periapical abscess into the adjacent bone
FIGURE 25-10. Dental caries. A. A large cavity close to the gingival margin is illustrated. Arrows indicate band of secondary dentin that lines the pulp chamber. This newly formed dentin is opposite the area of tooth destruction and was produced by the stimulated odontoblasts. B. Deposits of debris cover the surface. Bacterial colonies (dark purple) have extended into dentinal canals.
Cysts in jaws and in head and neck location

- Odontogenic cysts
  - Radicular cyst
  - Folicular (dentigerous) cyst
- Non-odontogenic cysts
  - Branchial cleft cysts
  - Thyreoglossal duct cyst
Odontogenic tumors

- **Epithelial** - benign (ameloblastoma, Pinborg, squamous)
  - malignant (ameloblastic carcinoma, malignant ameloblastoma, clear cell CA)
- **Ectomesenchymal** (fibroma, myxoma, cementoblastoma)
- **Mixed** - benign (fibro-odontoma, complex, compound o.)
  - malignant (ameloblastic fibrosarcoma)
Odontogenic tumors

Ameloblastoma

- Tumor of odontogenic epithelia
- **EPI:** the most common clinically significant odontogenic tumor
- Locally invasive tumors
- Generally follow a **benign** clinical course, but can be locally destructive

**MACRO:**
- Most arise in the mandibular ramus or molar area, maxilla or floor of the nasal cavity.
- Grow slowly as a central lesion of bone
- Often characteristic “soap bubble” radiographic appearance

**HISTOL:**
- These tumors resemble the enamel organ in its various stages of differentiation, and a single tumor may show several histologic patterns
- Tumor cells resemble ameloblasts at the edges of epithelial nests or cords, where columnar cells are oriented perpendicularly to the basement membrane

**Prognosis**
- Favorable.
- Incompletely excised tumors recur.
- Some may metastasize and yet remain histologically benign (**metastasizing ameloblastoma**). (**Ameloblastic carcinomas**)

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**Ameloblastoma.**
A common histologic pattern is characterized by islands of odontogenic epithelium with a central stellate reticulum-like area, surrounded by basal cells with a “picket fence” appearance, due to subnuclear vacuoles.
Salivary glands
Inflammations of Salivary glands

• **ETI** – bact., viral, autoimmunity
• Most commonly - gl. parotis

• **Acute bacterial sialadenitis**
  – Route of infection – ascending, rarely hematogenous
  – Predisposing factor - reduced secretion / stagnation of saliva,
  – **ETI** – st.aureus, str. viridans, …
  – **MORPHOL** – catarrhal, purulent, absceding,
  – **COMPLIC** – fistulas, phlegmone of adjacent soft tissues, facial vein thrombophlebitis, peripheral sepsis!!!

• **Chronic interstitial sialadenitis**
  – Primary or transition from acute infl.
  – Often associated with sialolithiasis
  – **MORPHOL** – mixed inflammatory infiltrate, fibrosis,
    • *sometimes pseudotumorous appearance* – „Küttner tumor“
Inflammations of Salivary glands

- **Viral sialoadenitis:**

  - **Parotitis epidemica** (*Mumps*)
    - **ETI**
      - paramyxoviruses
    - **MACRO**
      - usually bilateral parotid swelling, pain, pasty consistency,
    - **MICRO**
      - hyperemia, edema, dense lymphocytic and macrophage infiltrates, epithelial degeneration and necrosis
    - **COMPLICATIONS**
      - !!!may also cause: zánět pancreatitis, orchitis, oophoritis, serous leptomeningitis, myocarditis

  - **Cytomegalovirus sialadenitis**
    - **EPI:**
      - *Newborns* - intrauterine infection
      - *Adults* - immunosuppression (AIDS)
    - **MORPHOL**
      - Eosinophilic intranuclear inclusions
Inflammations of Salivary glands

Autoimmune sialoadenitis

• **Sjögren syndrome**
  – = clinicopathologic entity
  – Xerostomia + keratoconjunctivitis sicca
  – Autoimmune destruction of salivary and lacrimal glands

• **FORMS**
  • Primary
  • Secondary

• **MORPHOL:**
  • Swelling/enlargement of large salivary glands
  • Periductal a perivascular lymphocytic infiltration

**FIGURE 6-35** Sjögren syndrome. A, Enlargement of the salivary gland. (Courtesy of Dr. Richard Sontheimer, Department of Pathology, Southwestern Medical School, Dallas, TX.) B, Intense lymphocytic and plasma cell infiltration with ductal epithelial hyperplasia. Dennis Burns, Department of Pathology, University of Texas Southwestern Medical School, Dallas, TX.)
Tumors and tumor-like lesions of the salivary glands
CYSTS of salivary glands and other dysontogenetic cysts of head and neck region

• salivary glands
  – mucocele
  – Mucophagic granuloma
  – Ranula – retention cyst of sublingual gland

• Other cysts
  – Cystis ductus thyreoglossi (median cervical cyst)
  – Branchiogenic (lymphoepithelial) cysts (lateral cervical cyst)
  – Dermoid cyst
Tumors of the salivary glands

- **EPI**
  - Relatively rare (2%?)
  - 75% parotis
  - 10% submandibular
  - 15% small salivary gl.

- Malignancy is more common in smaller glands: minor (buccal mucosa) > sublingual > submandibular > parotid.

- About 30 different histol. types of tumors !!!

- Larger salivary glands can contain lymph nodes – reactive enlargement, lymphoma, metastatic ca
Tumors of the salivary glands

Primary

Epithelial

Benign
- Pleomorphic adenoma
- Warthin tu (cystic adenolymphoma)
- Myoepithelioma
- Basal cell adenoma
- Canalicular adenoma
- Oncocytoma
- Ductal papilloma

Malignant
- Carcinoma ex pleomorphic adenoma ("malignant mixed tu")
- Acinic cell carcinoma
- Mucoepidermoid ca (MEC)
- Adenoid cystic carcinoma
- Ductal ca (salivary duct ca)

Secondary

Other ("non-epithelial")
- Hemangioma, lipoma, neurilemmoma, neurofibroma
- Lymphomas

Meta - ca, m.m.
- Lymphomas /leukemia
Pleomorphic adenoma

- the Most Common Tumor of Salivary Glands (major and Minor)
- EPI:
  - Middle-aged people and women are most affected
- also called „mixed tumors“
- benign proliferations
- admixed epithelial and stromal elements
- MACRO:
  - Slowly growing, painless, movable, firm masses with smooth surfaces
- HIST:
  - epithelial tissue intermingled with myxoid, mucoid or chondroid areas hence the older term mixed tumor.
  - now considered to be of epithelial origin.

The tumor contains characteristic myxoid and chondroid portions.
The tumor is partly encapsulated, but a nodule protruding into the parotid gland lacks a capsule. If such nodules are not included in the resection, the tumor will recur.
Cellular components of pleomorphic adenomas include an admixture of glands and myoepithelial cells within a chondromyxoid stroma.
Warthin tumor

- benign parotid gland neoplasms composed of cystic glandular spaces within dense lymphoid tissue
- it may be **bilateral** (15%) or **multifocal** within one gland.
- **EPI:**
  - the only salivary gland tumors that are more common in men than in women.
  - after age 30, with most arising after age 50.
- **PATHOLOGY:**
  - glandular spaces that tend to become cystic and show papillary projections.
  - lined by characteristic eosinophilic epithelial cells (**oncocyes**)
  - embedded in dense **lymphoid tissue** with germinal centers
Mucoepidermoid carcinoma

- These tumors derive from ductal epithelium, which has a considerable potential for metaplasia.
- They account for 5% to 10% of major salivary gland tumors (parotid gland) and 10% of those in the minor salivary glands.
- **EPI:**
  - most in adults
  - more common in women.
- **MACRO:**
  - slowly growing
  - firm, painless masses.
- **HIST:**
  - low-grade (well-differentiated) tumors form irregular solid, ductlike and cystic spaces that include squamous cells, mucus-secreting cells and intermediate cells
  - Intermediate-grade
  - High-grade (poorly differentiated) carcinomas - markedly pleomorphic, without evidence of differentiation save, perhaps, for scattered mucus-secreting cells.

Mucoepidermoid carcinoma is characterized by an admixture of mucocytes (straight arrows), epidermoid cells (curved arrows) and intermediate cells. The mucocytes are clustered and have a clear cytoplasm with eccentrically situated nuclei. Epidermoid cells are squamouslike cells but lack keratinization and intercellular bridges. Intermediate cells (best seen at lower left) are smaller than epidermoid cells.
**Acinocellular carcinoma**

- uncommon parotid tumors (10% of all salivary gland tumors). (occasionally in other salivary glands)
- principally in young men between the ages of 20 and 30.

**MACRO:**
- They are encapsulated, round masses, usually less than 3 cm and may be cystic.

**HIST:**
- uniform cells with a small central nucleus and abundant basophilic cytoplasm, similar to the secretory (acinic) cells of normal salivary glands

**PROGN:**
- may spread to the regional lymph nodes
- After surgery most (90%) patients survive for 5 years, but local recurrence may be expected in one third of patients.
- Only half survive for 20 years.
Adenoid cystic carcinoma

- Slowly growing salivary gland malignancies.
- 5% of major salivary gland tumors and 20% of those of the minor salivary glands.
- One third arise in the major salivary glands and two thirds in the minor ones. (also in lacrimal glands, nasopharynx, nasal cavity, paranasal sinuses and lower respiratory tract)
- EPI: 40 to 60 years
- HIST:
  - Variable
  - The tumor cells are small, scant cytoplasm, in solid sheets or as small groups, strands or columns
  - Tumor cells interconnect to enclose cystic spaces, resulting in a solid, tubular or cribriform (sievellite) arrangement
- CLIN
  - Tend to infiltrate perineural spaces → often painful.
- PROGN:
  - Difficult to eradicate completely
  - Poor long-term prognosis.
„cribriform“

perforated like a sieve; having “Swiss cheese”-like spaces.
Etymology: Latin *cribrum*, sieve.
Pathology of the pharynx

... a tonsils
Inflammatory processes of the pharynx

- **Tonsilitis and pharyngitis** = inflammation of the lymphatic apparatus of pharynx

- Classification:
  - Location
  - Pathogenesis
  - Primary X associated with other disease = symptomatic or secondary

- Most important - **tonsillitis** (*amygdalitis*)
Tumors of the pharynx

• Nazopharyngeal angiofibroma

• Nazopharyngeal carcinoma
  – All types of NPC are considered variants of squamous cell carcinoma
    – keratinizing SCC
    – nonkeratinizing SCC
      • Differentiated
      • Undifferentiated
        – obsolete (and misleading) term: „lymphoepithelioma“

• Oropharyngeal carcinomas
  – nonkeratinizing SCC, basaloid type
  – Often associated with high risk HPV infection
  – Most commonly arising in lingual and palatine tonsil
  – Called: HPV-associated head and neck squamous cell carcinoma (HPV-HNSCC)

• Lymphoma/leucaemia
  – NHL – predominantly B-lymphomas, DLBCL
  – CLL
  – Hodgkin lymphoma – rare
Nasopharyngeal angiofibroma
(“juvenile nasopharyngeal angiofibroma”)

- uncommon, highly vascular neoplasm of the nasopharynx.
- **EPI**: most commonly arise in **adolescent males**. (However, as they are not restricted to this age group, the designation of “juvenile” is no longer encouraged)
- It is histologically benign but locally aggressive.
- These tumors are multinodular, lobulated or smooth pink-white masses, which may show surface ulceration and obvious blood vessels
- They typically arise submucosally in the **posterolateral nasal wall posterior to the sphenopalatine foramen**

- **HIST**: vascular and stromal components
- Blood vessels vary in size and shape; their walls lack a smooth muscle layer and show irregularly arranged smooth muscle.

- **Diagnosis** is usually made on clinical examination and imaging studies.
- Vessel wall defects preclude vasoconstriction, leading to brisk bleeding after trauma!!!
- **Biopsies may thus be dangerous, and are contraindicated.**
<table>
<thead>
<tr>
<th>Syndrome (other names)</th>
<th>Inheritance</th>
<th>Gene Protein</th>
<th>Chromosome</th>
<th>Pathology and main clinical features</th>
</tr>
</thead>
</table>
| Familial adenomatous polyposis (FAP) | AD | APC (adenomatous polyposis coli) | 5q21 | **Intestine**: early onset of 100s to 1000s of adenomas. Virtually 100% will develop colorectal carcinoma if not treated with total colectomy. Small intestinal adenomas (particularly of proximal duodenum and peri-ampullary); periampullary adenocarcinoma is the major cause of death following colectomy; fundic gland polyps (25–40% of fundic gland polyps have dysplasia although these polyps are biologically inert).  
**Soft tissue tumors** *(Gardner’s)*: Fibromatosis (desmoid tumor), Osteomas, Nuchal fibroma and Gardner fibroma  
**Skin Lesions** *(Gardner’s)*: Epidermoid cysts, pilomatricomas  
**Dental abnormalities** *(Gardner’s)*: Unerupted teeth, supernumerary teeth  
**Brain tumors** *(Turcot’s)*: Medulloblastomas  
**Other**: Thyroid cancers (1–2% of young women with FAP) essentially pathognomonic for FAP is the cribriform-morular variant of papillary thyroid CA; Juvenile nasopharyngeal angiofibromas *(adolescent males, 25x risk)*  

*“Attenuated FAP”*: far fewer adenomas (~ 30 adenoma) and cancer develops ~ 10 years later  

*In both syndromic and sporadic setting, many FAP-associated tumors (tubular adenoma, fibromatosis, JNA, fundic gland polyps) can be identified by IHC for nuclear β-catenin.*
Nasopharyngeal carcinoma (NPC)

- malignancy of the nasopharynx
- Subclassified:
  - Keratinizing type
  - nonkeratinizing type (associated with EBV infection)
  - differentiated
  - Undifferentiated
- EPI:
  - Undifferentiated nonkeratinizing carcinomas are particularly common in southeast Asia and parts of Africa
- HISTOL:
  - undifferentiated tumors, clusters of poorly delimited or syncytial cells have large oval nuclei and scant eosinophilic cytoplasm
  - A lymphoid infiltrate may be prominent in the undifferentiated variety, accounting for the obsolete (and misleading) term “lymphoepithelioma”
  - immunoreactive with cytokeratin
Pathology of the esophagus
Congenital malformations of the esophagus

- **EPI** – relatively rare
- **CLIN**
  - breast-feeding $\rightarrow$ regurgitation / aspiration of milk

- **Atresia**
  - Most commonly associated with **fistula**
    coomunicating with trachea or bronchus
    (Tracheoesophageal fistula)

- **Stenosis**
  - Congenital
  - X Secondary!
    - more frequently (strictures, tumors, compression)
Congenital tracheoesophageal fistulas.
A. The most common type (85% of cases) is a communication between the trachea and the lower portion of the esophagus. The upper segment of the esophagus ends in a blind sac.
B. In a few cases, the proximal esophagus communicates with the trachea.
C. H-type fistula without esophageal atresia.
D. Tracheal fistulas to both a proximal esophageal pouch and distal esophagus.
Functional disorders of the esophagus

- Diverticula
- Achalasia
- Hiatal hernia
- Laceration
Esophageal Diverticula

- ("true") **Diverticulum** = outpouching of the wall that contains all layers of the esophagus
- **Pseudodiverticulum** ("false" d.) sac has no muscular layer

- **Zenker ("pulsion") diverticulum**
  - Adjacent to the cricopharyngeus muscle – above the upper esophageal sphincter, disordered function of cricopharyngeal musculature – accumulation of food, aspiration

- **Traction d.**
  - mainly in the middle of the esophagus, they attach to adjacent mediastinal lymph nodes and are usually associated with tuberculous lymphadenitis or motor dysfunction, stagnation - mediastinitis

- **Epiphrenic**
  - located immediately above the diaphragm (lower e.sphincter), motor disturbances of the esophagus
Hiatal hernia

- herniation of the stomach through an enlarged diaphragmatic opening into posterior mediastinum

- **PATHOG**: laxity of the circumferential connective tissue, obesity, trauma, functional and organic shortening of esophagus

- **Sliding** hernia
- **Paraesophageal** h.
- **Mixed** h.
- **Sliding hernia**
  - Most common
  - Herniated proximal stomach – dilated above diaphragm
  - Often asymptomatic
  - Incompetence of the lower esophageal sphincter – g/e reflux – *reflux esophagitis*
Paraesophageal hernia

- uncommon
- portion of gastric fundus herniates through a defect in the diaphragmatic connective tissue that defines the esophageal hiatus and lies beside the esophagus

- CLIN
  - dysphagia,
  - less commonly reflux esophagitis
Achalasia

- Failure of the lower esophageal sphincter to relax with swallowing
- Absence of peristalsis in the body of the esophagus
- Food retention, dilation, hypertrophy
- Loss or absence of myenteric ganglion cells

- ETI
  - Chagas disease,
  - Other etiol. - ?
  - Autoimmunity?

- CLIN
  - Dysphagia, odynophagia, reflux
  - Chronic condition – RF for SCC!!!
Esophagitis

- **Esophagitis** *(not associated with reflux)*
  - ETI
    - Chemical, mech. factors, radiation, hot beverages/food, systemic diseases – uremia, GVHD
    - Infections – bakteriemia, viral – HSV, CMV, mycotic – candida albicans (mucor, aspergilóza)

- **Reflux esophagitis**
  - Chemicaly induced inflammation, resulting from pathologic g-e reflux
Reflux Esophagitis

- ETI of reflux:
  - Agents that decrease the pressure of the lower esophageal sphincter
  - Anatomical changes of g/e junction
  - ...
TUMORS AND PRECANCEROUS LESIONS OF THE ESOPHAGUS

• Precancerous lesions - Barrett’s esophagus

• Benign (epithelial, mesenchymal)

• Malignant
  - adenocarcinoma
  - squamous cell ca
  - malignant melanoma
  - metastases/direct invasion of bronchogenic cancer
Barrett’s esophagus

- is a complication of chronic GERD (gastroesophageal reflux disease) that is characterized by intestinal metaplasia within the esophageal squamous mucosa.
  - pre-malignant condition
  - confers an increased risk of esophageal adenocarcinoma!!!

- Diagnosis requires both
  - endoscopic evidence of abnormal mucosa above the gastroesophageal junction and
  - histologically documented intestinal metaplasia.

  - Goblet cells, which have distinct mucous vacuoles that stain pale blue by H&E and impart the shape of a wine goblet to the remaining cytoplasm, define intestinal metaplasia and are necessary for diagnosis of Barrett esophagus
Figure 15-9 Barrett esophagus. Normal esophagus is lined by layers of flat, squamous epithelium. Normal stomach is lined by tall, columnar cells. Chronic reflux of gastric acid evokes change (metaplasia) of normal esophageal squamous epithelium into gastric epithelium (Barrett metaplasia). Esophageal ulcers and adenocarcinoma may arise in areas of Barrett metaplasia.
Barrett’s mucosa

- Normal Lining
- Barrett’s Esophagus
  - with low-grade dysplasia
  - with high-grade dysplasia
- Invasive carcinoma
Adenocarcinoma

RF: GERD/ Barrett’s mucosa (+ radiation, tobacco, obesity)  
    gender (M:F=7:1),  
    Caucasians

Morphology:
    adenocarcinoma!!!
    (intestinal-type, signet ring cells, NOS,...)
Squamous cell carcinoma

**RF:** male gender (4x), rural areas (Iran, China, Brazil,...) alcohol, tobacco, poverty, caustic injury, diet, achalasia,...

**Morphology:** middle 1/3, dysplasia, in-situ metastases – LN cervical mediastinal, paratracheal, tracheobronchial, gastric, celiac