K. J. Lee: Essential Otolaryngology and Head and Neck Surgery (IIIrd Ed)

Chapter 16: Oral Cavity, Oropharynx, and Esophagus

Part 1: Oral Cavity

Anatomy: Special Points

Ducts in Oral Cavity

1. Parotid (Stenson's)
2. Submaxillary (Wharton's)
3. Sublingual (Rivinus')

Suprahyoid Muscle Innervation

1. Mylohyoid: V3
2. Anterior belly digastric: V3
3. Posterior belly digastric: VII
4. Stylohyoid: VII
5. Geniohyoid: XII

Papillae

Papillae cover the anterior two-thirds of the tongue and include: filiform (no tastebuds), fungiform, and foliate. Circumvallate papillae are a V-shaped group of large papillae that occurs at the junction of the anterior two-thirds and posterior one-third of the tongue.

Sulcus Terminalis

This is a bilateral groove that separates the anterior two-thirds from the posterior one-third of the tongue.

Foramen Cecum

The foramen cecum is a pit at the junction of the two grooves which form the sulcus terminalis from which the embryologic thyroid tissue begins its descent.
Frenulum

The frenulum is the anterior fold of mucous membrane that attaches the tongue to the floor of the mouth. Whartons' ducts open on either side of the frenulum.

Lingual Tonsil

The lymphoid tissue that extends over the base of the tongue (posterior one-third) is the lingual tonsil.

Vallecula

The valleculae are depressions (pockets) created by the glossoepiglottic folds at the base of the tongue and lingual surface of the epiglottis.

Extrinsic Muscles of the Tongue

These muscles include the genioglossus, hyoglossus, styloglossus, and palatoglossus. All are innervated by cranial nerve XII.

Intrinsic Muscles of the Tongue

The muscle bundles are separated by fibrous septa, the strongest of which lies in the midline (septum linguae). The four groups include the superior and inferior longitudinal, vertical, and transverse. All are innervated by cranial nerve XII.

The intrinsic muscles change the shape of the tongue which assists in speaking and swallowing.

Afferent Nerves from the Tongue

Innervation of the mucous membrane is from C1 and C2 that accompany the hypoglossal and lingual nerves, and via the glossopharyngeal and the superior laryngeal branch of X. Touch, pain, temperature, and taste are transmitted.

Blood Supply of the Tongue

1. Lingual artery: second branch of the external carotid

2. Lingual vein: does not travel with the lingual artery but with the hypoglossal nerve. Bleeding from this vein often leads to clamping of the hypoglossal nerve during surgery.

3. Lymphatics of the tongue drain into two separate systems. The anterior tongue is drained bilaterally by both a central and marginal system.

   The posterior drainage system runs through the pharyngeal wall to both deep cervical chains (jugulodigastric).
Taste

1. Base of tongue: IX (bitter)
2. Anterior two-thirds of tongue: (lingual nerve, chorda tympani); VII (sweet)
3. Palate and buccal mucosa: (sour).

Palate

The hard palate forms two-thirds of the palate and consists of the palatine processes of the maxilla and the horizontal plates of the palatine bones. The soft palate is a fibromuscular shelf. The muscles that are involved include: palatoglossus (anterior pillar), palatopharyngeus (posterior pillar), levator veli palatini, tensor veli palatini, and musculus uvulae.

- Palatoglossus --> Contracts lateral dimension
- Palatopharyngeus --> Contracts lateral dimension
- Musculus uvulae --> Alters the uvula
- Levator veli palatini --> Raises the soft palate to contact the posterior pharyngeal wall
- Tensor veli palatini --. Pulls the soft palate laterally to give rigidity and firmness.

Nerve Supply

Motor

The mandibular nerve (V3) innervates the tensor veli palatini. The remainder of the palatal muscles are innervated by the pharyngeal plexus, IX and X. Sensory innervation to the palate is from nerves V2, VII, IX, X, sympathetic chain.

Vascular Supply of the Palate

1. Maxillary artery --> descending palatine artery: greater and lesser palatine arteries.
2. Hard palate veins --> pterygoid plate --> internal jugular vein.
   Soft palate veins --> pharyngeal plexus --> internal jugular vein or
   --> external palatine vein --> tonsil fossa --> facial vein or pharyngeal vein.
3. Lymphatics
   Hard and soft palate: upper deep cervical nodes and retropharyngeal nodes.
Muscles of Mastication

All of the muscles of mastication are innervated by V3: masseter, temporalis, lateral pterygoid, medial pterygoid.

NB: The stylopharyngeus is the only muscle innervated by cranial nerve IX. Sensory innervation by the IX nerve includes base of tongue, tonsillar fossa, and soft palate.

Muscles of the Pharynx

1. Salpingopharyngeus
2. Stylopharyngeus
3. Palatopharyngeus.

These muscles are supplied by the pharyngeal plexus and the glossopharyngeal nerve. They elevate and dilate the pharynx during swallowing.

The external muscles run in an oblique direction and act as constrictors.

Superior Constrictor

The superior constrictor arises from the medial pterygoid plate, the base of the tongue, and mandible. It inserts, as do all constrictors, in the median raphe, and interdigitates with its corresponding muscle of the opposite side.

Medial Constrictor

This muscle arises from the hyoid bone and the stylohyoid ligament.

Inferior Constrictor

The inferior constrictor arises from the cricoid and thyroid cartilages. This constrictor may have additional innervation from the external branch of the superior laryngeal nerve.

Passavant's Ridge

Passavant's ridge represents the superior interdigitation of the superior constrictor muscle.

Pharyngeal Fascia

The pharyngeal fascia is not prominent except where there are areas of absent muscle. Two major fasciae are recognized:
1. Pharyngobasilar fascia: hangs the pharynx from the skull and the superior vertebrae.

2. Buccopharyngeal fascia: attaches posteriorly to the median raphe and the prevertebral fascia. This fascia prevents retropharyngeal abscess from crossing the midline.

**Teeth**

The child has 20 deciduous teeth; the adult has 32 permanent teeth.

**Deciduous**

Medial incisors (7 months)

Lateral Incisors (9 months)

First molar (15 months)

Canine (18 months)

Second molars (20-14 months)

**Permanent**

First molar (6 years)

Medial incisor (6-7 years)

Lateral Incisor (8-9 years)

First premolar (10-11 years)

Canine (10.5 to 11.5 years)

Second premolar (11-12 years)

Second molar (12-13 years)

Third molar (17-25 years).

**Saliva**

1. 1500 mL/day
2. pH: 6.2-7.4
3. 99.5% water, 0.5% organic/inorganic solids
4. Organic component: glycoprotein
5. Electrolytes:
   a. Na 10 mEq/L
   b. K 26 mEq/L
   c. Cl 10 mEq/L
   d. Bicarb 30 mEq/L.

Disorders of the Oral Cavity

Dental Plaque

Dental plaque is a soft amorphous deposit which accumulates on the surface of teeth consisting of bacteria, leukocytes, and desquamated epithelium.

Pathology includes:
1. Caries
2. Periodontal disease.

Dental Development Abnormalities

1. Anodontia (partial or complete); hereditary.
2. Dilaceration: The tooth root, as a result of trauma, fails to develop normally resulting in an angular malformation of the root. Associated diseases include rickets and cretinism.
3. Supernumerary teeth.
4. Enamel hypoplasia.

Periapical Disease

1. Granuloma (asymptomatic).
2. Alveolar abscess may lead to:
   a. Sinusitis
   b. Osteomyelitis
   c. Ludwig's angina
   d. Bacteremia.
3. Radicular cyst.

**Inflammation of Oral Mucosa**

**Stomatitis**

Stomatitis is generalized inflammatory involvement of the oral mucosa and includes:

1. Gingivitis
2. Periodontitis (pyorrhea)
3. Periodontosis
   a. Chronic degenerative destruction of the periodontal tissue
   b. Papillon-Lefevre syndrome: periodontosis, hyperkeratosis of the soles and palms with calcification of the dura.
4. ANUG (acute necrotizing ulcerative gingivitis), Vincent's angina, trench mouth
   a. Fetid odor to breath
   b. Excessive salivation
   c. Bleeding gingiva
   d. Organism: Borrelia vincenti (fusiform bacillus)
   e. Treatment: good oral hygiene, penicillin.
5. Herpetic gingivostomatitis
6. Herpes labials (most common viral infection of the mouth)
7. Herpes zoster
8. Herpangina (group A coxsackievirus)
9. Infectious mononucleosis
10. Noma (fusospirochetal and streptococcal or staphylococcal) (cancrum oris)
    a. Usually in debilitated children
    b. Children more frequent than adults
    c. Acute gangrenous process with high mortality.
11. Bacterial
   a. Streptococcal
   b. Staphylococcal
   c. Gonoccal

12. Thrush (Candida albicans)

13. Actinomyces

14. Blastomycosis

15. Histoplasmosis (Histoplasma capsulatum)

16. Pyogenic granuloma.

   Noninfectious Lesions

1. Recurrent aphthous ulcer
   a. Sutton's disease: variant with multiple, large deep ulcers which cause extensive scarring of the oral cavity

2. Erythema multiforme

3. Pemphigus vulgaris (intraepidermoid bullae)

4. Pemphigoid (subepidermoid bullae)

5. Lichen planus


   Oral Mucosal Manifestations of Systemic Processes

1. Pernicious anemia: Mucosa and lips are pale yellow gray and susceptible to ulcerations. The tongue is shiny, smooth, and red.

2. Iron deficiency anemia: Oral mucosa is ash gray (Plummer-Vinson syndrome).

3. Sickle cell anemia: Stepladder alignment of the trabeculae of the interdental septum. Pallor, yellow discoloration to mucosa.


5. Polycythemia: Bright blue red with gingival bleeding.


8. Thrombocytopenic purpura: Petechia and hemorrhagic vesicles.

9. Menopausal (senile atrophy). Gingivostomatitis (dry, burning sensation), diffuse erythema, shininess, and occasional fissuring in the mucobuccal fold.

10. Nutritional pathology (deficiency):
   b. Pyridoxine: Angular cheilosis.
   c. Nicotinic acid: Angular cheilosis, glossopyrosis with burning.
   d. Vitamin C: Gingivitis and "bleeding gums".

**Pigmentation Changes to Oral Cavity**

1. Melanosis (physiologic pigmentation).
4. Arsenic: Black.
5. Lead: Blue/gray.
7. Silver: Violet/blue/gray.
10. Xanthomatous diseases: Yellow/gray.
Common Childhood Oral Cavity Disease Manifestations

1. Measles (rubeola): Koplik's spots
2. Chickenpox (varicella): vesicles
3. Scarlet fever: strawberry tongue
4. Congenital heart disease: gingivitis, cyanotic gums
5. Kawasaki's disease: strawberry tongue (this is due to protuberance of the tongue papillae).

Leukoplakia (White Plaque)

1. Whitish appearance which is due to hyperkeratosis of the epithelium

Median Rhomboid Glossitis

This is a developmental anomaly of the tongue in which the tuberculum impar persists in the midline. The incidence is less than 1%.

Fordyce Granules

These are painless, pinpoint yellow elevations which usually occur bilaterally in the posterior buccal spaces. They represent ectopic sebaceous glands.

NB: They frequently accompany tongue atrophy in syphilis.

Macroglossia

Etiologies:
1. Hemangioma
2. Myxedema
3. Acromegaly
4. Amyloidosis
5. Cysts
6. Actinomycosis
7. Pierre Robin syndrome (relative macroglossia)
8. Tertiary syphilis


**Tumors of the Mandible (Excluding Carcinoma)**

A. Odontogenic fibroma
   1. Circumscribed radiolucency
   2. Area of impacted canine or molar
   3. Differential: dentigerous cyst
   4. Excision: no tendency toward recurrence.

B. Ameloblastoma
   1. True neoplasm of enamel origin
   2. Third decade
   3. Majority in mandible (especially molar region)
   4. Slow, expanding, painless growth
   5. X-ray demonstrates a multilocular cyst (septa or spokes of bone).

C. Cementoma
   1. Arise around apex of mandibular incisor
   2. More common in women (second decade)
   3. Rarely needs excision.

D. Odontoma
   1. Composed of more than one kind of odontogenic tissue

E. Adenoameloblastoma
   1. Well encapsulated follicular cyst
   2. More common in adolescent girls
   3. Frequency associated with impacted teeth.
F. Ameloblastic fibroma
1. Slow-growing, painless
2. Adolescents
3. Molar area of mandible

G. Ameloblastic sarcoma
1. Poor prognosis

H. Ameloblastic odontoma (rare)

I. Ewing's sarcoma (diffuse endothelioma of Ewing)
1. Commonly occurs between ages 10-25 years
2. Males outnumber females
3. Morphologic appearance
   a. Hemorrhage and necrosis common
   b. Fifty percent show a concentric (onion skin) layered appearance
4. Histologically: highly differentiated
5. Clinically:
   a. Rapid growth
   b. Painful
   c. Metastasis
   d. Less than 15%, 5-year survival.

**Odontogenic Cysts**

A. Radicular
1. Most common
2. Commonly due to an infected tooth apex
3. Usually asymptomatic
4. No transformation or malignant degeneration potential

B. Dentigerous (follicular)

1. Due to alteration of the enamel epithelium in the crown of a permanent tooth

2. Common in mandibular third molar or maxillary cuspid as these are the most commonly impacted teeth

3. Ameloblastoma formation has occurred in cyst wall.

C. Primordial

1. Uncommon

2. Found to normal tooth location without evidence of different tooth elements.

**Other Oral Cavity Lesions**

A. Erythroplasia of Queyrat

1. Historically associated with syphilis

2. Single white plaque (well-defined) which readily changes to a red velvety surface without inflammation

3. Histologically: similar to carcinoma in situ

B. White hairy tongue

1. Accumulation of keratin on the surface of filiform papillae.

C. Epulis.

1. Least common is the congenital type which looks much like the granular cell myoblastoma. It has no malignant potential.

2. More common is the giant cell type (giant cell reparative granuloma).
   a. Histology shows reticular and fibrous connective tissue with numerous giant cells.
   b. X-ray of the bone shows cuffing or sclerotic margins.

D. Ranula.

1. Small, painless, floor-of-mouth lesion.

2. Histology: Retention cyst.
3. Excision should include sublingual gland to prevent recurrence.

E. Torus palatinus.
1. Arise from persistent bone growth. 2. Benign.

F. Torus mandibularis.
1. Excessive bone growth.
2. Growth occurs past puberty.
3. Occasionally due to denture irritation.

**Deglutition**

A. Oral phase.
1. Voluntary.
2. Solids are masticated.
3. Saliva mixes with food bolus.
4. Bolus is molded by tongue and teeth.
5. Bolus is collected on tongue dorsum.
6. Anterior tongue elevates/base of tongue elevates.
7. Mylohyoid muscle elevates floor of mouth.
8. Hyoid is pulled up an forward.
9. Bolus is propelled into the pharynx.
10. For liquids the mouth and contents form an anatomic funnel.

B. Pharyngeal phase
1. Reflex response due to wall receptors.
2. Nasopharynx closed off (levator/tensor palati).
3. Contraction of the lingual muscles keeps the tongue against the palate.
4. Larynx closed off:
   a. Elevated and moved forward.
   c. Epiglottis.

5. Three sphincters (true vocal cords, false vocal cords, aryepiglottic folds).

6. Vertical height of pharynx is reduced by: Palatopharyngeus, stylopharyngeus, and elevation of the hyoid.

7. Circular and horizontal fibers in synchrony, move food inferiorly.

8. Cricopharyngeus opens.

9. Peristaltic waves pick up in esophagus, not pharynx.

C. Esophageal phase

1. Fluid movement is passive.

2. Peristalsis effect solids and semisolids.

3. The upper one-third of the esophagus has voluntary muscle: rapid peristaltic movement.

4. Passage in the upper one-third is less than 1 second.

5. Lower one-third is smooth muscle, with approximately 3 second passage.

6. Gravity plays only a small role in food passage in the esophagus.

7. Changes in position play a minimal or no role.

8. Reverse peristalsis is not normal.

9. There are bare nerve endings that will cause spasm if the esophagus is overly distended.

10. Highly coordinated feedback pathway controls gastroesophageal bolus transfer.
Swallowing Nerve Map

Sensory Receptors

1. Soft palate.
2. Mucosa at tongue base.
3. Tonsillar pillars.
4. Posterior pharyngeal wall.

Central Reception

1. (V) Gasserian ganglion.
3. (X) Inferior (jugular). Superior (nodose) ganglions.

Efferent Pathway

1. Teeth/jaw (V).
2. Palate (V, X).
3. Lips (VII).
4. Buccal space (VIII).
5. Pharynx (IX, X).
7. Esophagus (X).
8. Tongue (XII).
Part 2: Tonsils/Neck Infections/Spaces

Parapharyngeal Space

The parapharyngeal space is a potential space filled with loose connective tissue. It is inversely pyramidal in shape with the following boundaries:

Superior: Skull base.
Inferior: Lesser cornua of the hyoid.
Lateral: Ascending ramus of the mandible, medial pterygoid, interpterygoid fascia.
Posterior lateral: Fascia surrounding the parotid gland.
Medial: Visceral fascia on the superior constrictor. Fascia on the tensor veli palatini/levator veli palatini, and fascia of the styloglossus muscle.
Anterior: Fascia directed to the lateral angle of the mandible and above to the pterygoid fascia of the fascia on the buccinator muscle.
Posterior: Fascia about the carotid sheath (some authors include the styloid muscles and vertebrae).

Parapharyngeal Space Compartments

1. Prestyloid (anterior).
2. Retrostyloid (posterior).
3. Retropharyngeal (medial).

Compartment Contents

A. Prestyloid.
1. Internal maxillary artery.
2. Lingual nerve.
3. Inferior alveolar nerve.
4. Auriculotemporal nerve.
B. Poststyloid.
1. Internal carotid artery.
3. Cranial nerves IX, X, XI, XII.
4. Nodes.
C. Retropharyngeal.

Nodes (node of Rouviere).

**Pharyngeal Communication to Other Compartments**

1. Paralingual.
2. Parotid.
3. Carotid.
4. Masticator.
5. Retropharyngeal.

**Tumors Involving the Parapharyngeal Space**

1. Parotid.
   a. Only 5% enter the parapharyngeal space but 50% of the tumors of the space are salivary.
   b. Neurogenous.
   a. Thirty percent of parapharyngeal space tumors.
   b. Paragangliomas, glomus jugulare, chemodectomas, glomus intravagale.
4. Lymphomas.

The Mayo series of 100 parapharyngeal space tumors revealed:

1. Mixed tumor: 43%.
2. Malignant lymphoma: 25%.
3. Schwannoma: 16%.
4. Paraganglioma: 12%.
5. Hemangiopericytoma: 2%.
6. Hemangioendothelioma: 1%.
7. Lipoma: 1%.

**Fascial Planes**

1. The cervical fascia lies in two planes: Superficial and deep.

2. Superficial.
   a. Encloses the neck and is continuous with the platysma anteriorly.
   b. The superior boundary is the zygoma while inferiorly attaches to the clavicle.

   a. Superficial layer encloses the muscle compartment, the parotid, and submaxillary glands.
   b. Middle layer encloses the visceral organs.
   c. Deep layer encloses the vertebrae.
   d. All three layers enclose the carotid system.

   The fascia that encloses the paraspinoius muscles and the vertebrae attaches to the transverse processes laterally then splits into a prevertebral and alay layer. The "potential" space thus formed is the prevertebral space, running from the skull base to the diaphragm.

   Directly posterior to the posterior pharyngeal wall and anterior to the alar fascia is the retropharyngeal space, extending from the base of the skull to the tracheal bifurcation.

   NB: A midline raphe is formed where the superior constrictor attaches to the prevertebral fascia.

**Submandibular Space**

The submandibular space is bounded by:

1. Inferior: Mylohyoid muscle.

2. Lateral: Body of mandible.

3. Superior: Mucosa of the floor of the mouth.

   The submandibular space is in continuity with the sublingual space along the posterior margin of the mylohyoid muscle and is bordered laterally by the anterior belly of the digastric muscle.
The visceral space lies adjacent to the esophagus and trachea. It is limited superiorly by the hyoid and readily can be contaminated by injury to either structures.

Deep Neck Infections

1. The oral cavity may be benign on examination.

2. Fluctuations of the neck is almost never noted.

Specific Infection Locations

Retropharyngeal Space

NB: Physical examination reveals swollen, boggy, inflamed mucosa on the posterior pharyngeal wall with unilateral swelling due to the midline raphe.

A. Diagnosis.

1. History.
   a. Ethmoid or sphenoid sinusitis.
   b. Associated with lateral pharyngeal abscess.

2. Physical examination.
   a. Hyponasal speech.
   b. Anterior displacement of posterior pharyngeal wall.
   c. Posterior pharyngeal wall is soft.

3. Lateral neck film.
   a. Air in the retropharyngeal space.
   b. Widened soft tissue shadow overlying C-2 (average 4 mm), greater than 7 mm is pathologic.
   c. Retrocochlear tissue at C-6 is abnormal in children (under 5 years) if greater than 14 mm, or in the adult if greater than 22 mm.
B. Treatment.

1. Control of the airway.

2. Trendelenburg's position.

3. Peroral drainage in uncomplicated infection, i.e. other spaces are not violated.

4. Antibiotics.

**Lateral Pharyngeal Space**

A. Diagnosis.

1. Trismus.

2. Drooling.

3. Dysphagia.

4. Odynophagia.

5. Physical examination: Medial displacement of the lateral pharyngeal wall and fullness in the retromandibular region.


B. Treatment.

1. Control airway.

2. Drainage procedure.

a. Incision parallel to the anterior sternocleidomastoid.

b. Horizontal submandibular incision.

NB: The lateral pharyngeal space should never be drained from an intraoral route.

Submandibular Space Infection

A. Diagnosis.
1. Pain.
2. Dysphagia.
4. Swollen, red, inflamed oral (sublingual) mucosa.
5. Limited tongue mobility (see Ludwig's angina).
6. Respiratory distress.

B. Treatment.
1. Airway control.
2. Antibiotics (ampicillin, oxicillin).
3. Submental incision (horizontal). If space involved is sublingual only, it may be drained intraorally.

C. Microbiology.
1. Anaerobic streptococci (common).
2. Staphylococcus aureus (common).
3. Hemolytic streptococci (common).
5. Mycobacterium tuberculosis (rare).
6. Atypical mycobacteria (rare).
7. Actinomyces (rare).
8. Nocardia (rare).

NB: Prevertebral abscess is commonly due to tuberculosis (Pott's disease).

NB: In over 50% of deep neck infections no specific source of infection is identified.
**Ludwig's Angina**

Ludwig's angina is an acute inflammatory process beginning in the floor of the mouth that invades the mylohyoid and rapidly spreads in the submaxillary space.

1. Rapid spread of infection (phlegmon, not an abscess).
2. Extension by continuity not lymphatics.
3. High spiking temperatures with chills and shakes.
4. Posterior two-thirds of tongue, then anterior one-third is pushed superiorly and posteriorly in direction.
5. Hard, tense woody cellulitis is palpated.
6. Inability to handle secretions becomes pronounced.
7. Respiratory embarrassment may be acute.
8. Drainage produces a serosanguinous foul discharge, but rarely pus.
9. Commonly found in the debilitated or immunocompromised host.

**Sources of Deep Neck Infection and Their Pattern of Spread**

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NB: Petrositis: With the base (roof) of the lateral pharyngeal space in contact. Infection may spread in a caudal direction from a temporal bone primary infection.

| Nasal cavity                  | Retropharyngeal space     |
| Paranasal sinus               | Retropharyngeal space     |
| Nasopharynx                   | Retropharyngeal space     |
| Eustachian tube               | Retropharyngeal space     |
| Soft palate                   | Retropharyngeal space     |
| Tongue tip                    | Submental space           |
| Skin of chin                  | Submental space           |
| Lower lip                     | Submental space           |
Complications of Deep Neck Infections

1. Airway obstruction.
2. Aspiration.
3. Pneumonia.
5. Peritonitis (prevertebral space tract).
6. Carotid artery rupture.
8. Paralysis of cranial nerves IX, X, XI, XII.
10. Retrograde thrombophlebitis (see no. 7).
11. Emboli: Bacterial endocarditis.
12. Bran abscess (secondary to no. 11).
13. Parotitis.
15. Spinal cord/vertebrae involvement.

Tonsil

1. Lateral extension of the pharyngeal pouch is largely absorbed (second pharyngeal pouch). Dorsal remnants of the pouch persist to become epithelium of the palatine tonsil.
2. Tonsillar pillars originate from second and third branchial arches.
3. Tonsillar crypts are noted during the third to sixth month of embryologic life.
4. Tonsillar capsule begins to form in the fifth embryologic month.
Palatine Tonsile

1. 20-25 mm length.
2. 15-20 mm width.
3. 12 mm thickness.
4. Average weight of adult tonsil 1-5 g.

Anatomy

Lingual Tonsil

1. Lymphoid follicles vary in number from 30-100; irregular in size and shape.
2. Supplied by lingual branch of external carotid artery.
3. Drained by lingual veins to the internal jugular vein.
4. Gerlach's tonsil: Lymphoid tissue within the lip of the fossa of Rösenmuller. Actually goes into the eustachian tube.

Palatine Tonsil Blood Supply

1. Ascending pharyngeal.
2. Ascending palatine.
3. Dorsal lingual.
4. Facial (primary blood supply).
5. Descending palatine.

Palatine Tonsil Venous Drainage

Lingual or pharyngeal vein to internal jugular vein.

Immunology

1. The role of the tonsils remains controversial.
2. Tonsils produce secretory IgA.
3. Tonsils do not produce IgE.
4. Interferon and lymphotoxin have been isolated from tonsil tissue.
5. Infection often results from imbalance between host resistance and normal flora.

**The Microbiologic Environment of the Adult Mouth**

1. Staphylococci (skin contaminants earliest in newborn).
2. Nonhemolytic streptococci.
3. Lactobacilli.
4. Actinomyces.
5. Leptothrix.
8. Spirochetes.
10. Virus.
   a. Myxovirus.
   b. Adenovirus.
   c. Picornavirus.
   d. Coronavirus.

**Acute Tonsillitis**

1. Beta streptococci.
2. Staphylococci.
3. Streptococcus pneumoniae (Diplococcus pneumoniae).
4. Haemophilus.
Differential Diagnosis

1. Diphtheria.
2. Scarlet fever.
4. Infectious mononucleosus.
5. Leukemia.
6. Agranulocytosis.
7. Malignancy.
8. Pemphigus.

Complications of Peritonsillar Abscess

1. Airway obstruction.
2. Local venous thrombosis.
3. Phlebitis.
4. Endocarditis.
5. Lateral pharyngeal abscess.
8. Peritonitis.
10. Perichondritis of thyroid cartilage.
11. Aspiration pneumonia.
Tonsillectomy

1. Procedure referred to by Celsus in De Medicina (10 A.D.)
2. Documented surgery by Cague of Rheins (1757)
3. Mortality: 0.5-1:10,000 (reported)

Indications for Tonsillectomy

1. Recurrent episodes of acute/chronic tonsillitis (over six per year)
2. Tonsillitis causing febrile convulsions
3. Diphtheria carrier
4. Hypertrophy involving airway obstruction
5. Hypertrophy involving deglutition problems
6. Possibility of malignancy (biopsy)
7. Peritonsillar abscess.

Part 3: Esophagus

Dysphagia

Difficulty in swallowing may be due to oral, pharyngeal, or esophageal dysfunction.

Symptoms May Include

1. Regurgitation
2. Aspiration
3. Pain
4. Tongue mobility dysfunction
5. Substernal fullness
6. Retrosternal fullness
7. Sensation of "stuck" bolus.
Radiologic Findings May Include

1. Dilatation of pyriform sinus or atony.
2. Aspiration of contrast into trachea.
3. Regurgitation of contrast material.
4. Obstruction.
5. Narrowing of the lumen.
6. Absent peristalsis.

Diseases with Dysphagia

1. Inflammatory lesions of the pharynx associated with viral infections.
2. Vincent's angina.
3. Thrush.
4. Tonsillitis (peritonsillar abscess).
5. Retropharyngeal abscess.
6. Plummer-Vinson syndrome.
7. Polio.
8. Pseudobulbar palsy.
10. Acute myelogenous leukemia (AML).
11. Multiple sclerosis (MS).
12. Myasthenia gravis.
13. Polyneuritis.
15. Myotonia congenita.
17. Muscular dystrophy.
18. Primary muscular tumors.
19. Primary muscular invasion due to tumor.
22. Adenocarcinoma.
23. Laryngeal carcinoma.
24. Thyroid mass.
25. Achalasia.
27. Scleroderma.
28. Raynaud's phenomenon.
29. Esophageal webs.
30. Esophageal spasm.
31. Psychologic.
32. Schatzki's ring (lower esophageal).
33. Burns.
34. Dysphagia lusoria.
35. Leiomyoma (benign tumors).

**Physioanatomical Considerations**

1. The esophagus is a mobile structure fixed only at the carotid level.
2. From the teeth to cardia of stomach is 40 cm.
3. The left vagus gradually runs onto anterior surface.
4. The right vagus gradually runs onto posterior surface.
5. The esophagus has no serosal layer.

6. The esophagus has minimal secretory functions.

7. The squamous epithelium is poor for absorption.

8. The oblique angle of entry of the esophagus into the stomach (angle of His) is important. When the angle is lost, reflux occurs. In infants the angle is almost nonexistent; hence, the baby readily refluxes.

9. Reflux is further reduced by the lower esophageal sphincter (LES) which is readily demonstrated manometrically.

10. In most adults the LES extends 1-2 cm below and above the diaphragm.

11. The LES is controlled by the interplay of acetylcholine and gastrin.

**Esophageal Symptoms**

Odynophagia: Painful swallowing.

A. Heartburn: Vague term usually implying acid reflux (brackish test in back of throat, need to clear throat continually, burning in throat).

1. Peptic esophagitis.

2. Reflux secondary to stomach distension or intra-abdominal pressure increase (pregnancy, bending over, etc).

3. Hiatus hernia.

B. Chest pain.

1. Duration greater than angina.

2. Associated with eating or stress.

Aerophagia: Belching is rarely organic. However, in children this may be a source of dysphagia.

Rumination: Rare symptom in which the patient regurgitates one mouthful of food from the stomach into the mouth - chews - and then swallows.

Hypersalivation: Rarely organic.

Lump in Throat: Globus hystericus.
**Workup for Esophageal Dysfunction/Pathology**

1. Barium swallow with follow-through in stomach.

2. Cine study.

NB: A table or barium marshmallow can be used more often to highlight esophageal lumen to bring out minor strictures.

3. Cytologic studies: Study of the exfoliated cells in the over 40-year-age group is a valuable diagnostic tool. Saline lavage is the only requirement to obtain cells.

   a. Levine tube aspirates stomach contents and is withdrawn to 35 cm from nostrils.
   b. Control infusion of 0.9% sodium chloride, 125 drops/min x 15 min.
   c. Followed by 0.1 N HCl up to 30 min.

5. Acid reflux test.


7. Motility studies (manometrics).
   a. Study three locations: LES, body of esophagus, cricopharyngeus.
   b. Three conditions in which motility studies have proved valuable include:
      1. Achalasia (high pressure).
      2. Esophageal spasm (high pressure).
      3. Scleroderma (low pressure).

8. Esophagoscopy.
   a. Rigid:
      1. Foreign body.
      2. Lesions of cervical esophagus.
      3. Obstruction with debris or fluid.
      4. Evaluation of stenosis.
b. Flexible.

1. Evaluate lesions.
2. Evaluate esophagitis.
3. Evaluate hemorrhage.
4. Obtain biopsy.

**Congenital Lesions**

A. Tracheoesophageal fistulae.

1. 1:1000 live births.

2. Types:
   a. Dilated upper esophagus ends as a blind pouch with lower esophageal segment attached to the trachea in 87% of the cases.
   b. A blind upper and lower esophagus without a true fistula to the trachea is present in 8% of cases.
   c. The "H" deformity is a true fistula without atresia. This occurs in 4%.
   d. Less than 1% have the upper esophageal segment open directly into the trachea.
   e. Less than 1% have the upper and lower esophageal segments open independently into the trachea.

3. Sixteen percent of the infants had hydramnios.

4. Clinical features:
   a. Drooling.
   b. Coughing.
   c. Abdominal distension.
   d. Vomiting.
   e. Cyanosis and rarely asphyxia.
   f. Poor feeding.

5. Diagnosis.
a. Chest x-ray - right upper lobe pneumonia (aspiration pneumonitis).

b. Flat plate of abdomen - marked air filling.

c. Radiopaque fluoroscopic evaluation of upper aerodigestive tract.

6. Sixty to eighty percent survive and do well. However if concomitant genitourinary tract or cardiac malformations are present only 22% survive.

B. Dysphagia lusoria.

This an uncommon condition of symptomatic compression of the esophagus by the anomalous location of the right subclavian artery (RSA)(Bayford's syndrome).

1. Normally the RSA passes:

a. Eighty percent posterior to the esophagus.

b. Fifteen percent between the trachea and the esophagus.

c. Five percent anterior to the trachea and the esophagus.

2. Clinical features.

a. Intermittent dysphagia.

b. Weight loss over long period.

3. Diagnosis.

a. Barium study (cine).

b. Esophageal motility evaluation.

c. Upper gastrointestinal endoscopy.

d. Angiography.

4. Associated entities.

a. Aneurysm.

b. Fibrosis secondary to prior surgery around the RSA and/or esophagus.

c. Vascular congenital anomaly. The RSA is the most common of arch anomalies and occurs in 0.5-1.8% of the population.

d. Aging associated with changes in vessel diameter and thoracic cage dimension.
changes.

e. Atherosclerosis.

5. Treatment.
a. Surgery.

C. Chalasia.

Occurrence of reflux in infants (by definition there is no hiatus hernia).

1. Clinical features.
a. Vomiting or "spitting up" within 3-10 days of birth.
b. Belching.
c. Weight loss or failure to gain.

2. Diagnostic by radiographic study.

3. Prognosis: Excellent, most children can tolerate feeding while lying on back by 6 weeks.

D. Duplication of the esophagus.

E. Esophageal rings.

F. Esophageal webs.

Esophageal Burn (Corrosive)

A. History.

1. Identify agent: Reference to toxicology screen.

2. Try to determine if material was actually ingested.

B. Physical examination.

1. Obvious burn.

2. Dysphagia.

3. Odynophagia.

C. Burn suspected.
1. IV fluids.
2. Steroids.
3. Penicillin.
4. NPO.
5. Esophagoscopy within 24 hours: ASAP.
6. Do not induce vomiting.
7. Patient may have pharyngeal-esophageal burn without evidence of burn in oral cavity.

D. Findings on esophagoscopy.
1. No burn.
   a. Follow-up in 2 weeks - asymptomatic - discharge.
   b. Symptomatic.
      1. Barium swallow.
      2. Esophagoscopy (?)
2. Burn.
   a. Do not proceed beyond point of burn.
   b. Antibiotics.
   c. Steroids (2-3 weeks taper).
   d. Serial esophagoscopies done after 2 weeks to decide if burn has healed.
   e. Dilatation if stricture forms.

NB:
1. Early, close observation for airway compromise.
2. Intubation (NG) is controversial. If done it must be within 24 hours and optimally should be under direct visualization.
3. May place string into esophagus to act as guide for esophagoscopy.
E. Pathologic Sequence.

1. 0-24 hours: Dusky cyanotic edematous mucosa.
2. 2-5 days: Grey white coat of coagulated protein. Fibroblasts appear.
3. 4-7 days: Slough with demarcation of burn depth. Weakest from days 5-8.
4. 8-12 days: Collagen appears.
5. 6 weeks: Scar formation/stricture evident.

**Motor Disturbance in Esophagus**

1. Esophageal spasm: Simultaneous repetitive nonperistaltic and often powerful contractions of the esophagus.
2. Presbyesophagus: Associated with age. Mild symptoms to increased dysphagia, substernal pain.
4. Irritant-induced: Gastroesophageal reflux, corrosive irritation.
5. Obstruction of the cardia: Carcinoma, benign stricture.
7. Idiopathic: Often very severe with young patients.
8. Spasm: Symptoms include curling, tertiary contractions, corkscrew esophagus, rosary bead esophagus.

**Diagnosis**

1. Laboratory studies are normal.
2. Barium swallow may be normal.
3. Cine fluoroscopy.
   a. Tertiary contractions trap barium in little segments.
   b. Retrograde displacement of barium into the mouth.
4. Motility studies.
   a. Spontaneous waves not preceded by a swallow.
b. Three to five repetitive waves that follow each other after one swallow.
c. Simultaneous high-peaked waves occurring in all three leads.

**Cricopharyngeal Dysphagia**

1. Presumably analogous to achalasia in the failure of a sphincter to relax.
2. Diseases.
   a. Idiopathic.
   b. Polio.
   c. Thyrotoxic myopathy.
   d. Left or right recurrent nerve paralysis.
   e. S/P pharyngectomy with hypertrophied cricopharyngeus muscle.
3. Diagnosis.
   a. Barium swallow.
   b. Cine studies.
   c. Manometrics.

**Achalasia**

A. Megaesophagus

1. A disorder (cardiospasm) of esophageal motility characterized primarily by failure of the LES to relax normally.
2. Diseases.
   a. Status postvagotomy.
   b. Psychologic.
   c. Chagas' disease.
   d. Neutrotrophic virus.
   e. Destruction of myenteric plexus.
   f. Drugs.
g. Gastroesophageal tumor invasion.

h. Hereditary component (?)..

Table 16-1. Differential Comparison of Esophageal Spasm and Achalasia

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Esophageal Spasm</th>
<th>Achalasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphagia</td>
<td>Midsternum</td>
<td>Xyphoid or suprasternal notch</td>
</tr>
<tr>
<td>Pain</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Belching</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Emotional</td>
<td>Common</td>
<td>Common</td>
</tr>
</tbody>
</table>

Motility

<table>
<thead>
<tr>
<th>Waves</th>
<th>Simultaneous</th>
<th>Weak, ineffective</th>
</tr>
</thead>
<tbody>
<tr>
<td>LES (relax)</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Methacholine (Mechlolyl stimulation)</td>
<td>Slight</td>
<td>Hyperactive</td>
</tr>
</tbody>
</table>

Radiographic

<table>
<thead>
<tr>
<th>Esophageal contraction</th>
<th>Active</th>
<th>Weak</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophageal emptying</td>
<td>Efficient</td>
<td>Poor</td>
</tr>
</tbody>
</table>

Response to Therapy

<table>
<thead>
<tr>
<th>Bougienage</th>
<th>Good</th>
<th>Fair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumostatic Dilatation</td>
<td>Not indicated</td>
<td>Good</td>
</tr>
<tr>
<td>Surgical Rx</td>
<td>Long myotomy</td>
<td>Low cardioesophageal myotomy.</td>
</tr>
</tbody>
</table>

&NB: Both achalasia and spasm can mimic angina pectoris.

Table 16-1 compares the observations that differentiate between spasm and achalasia.

**Structural Disorders of the Esophagus**

A. Four classic anatomic compression locations.

1. Cricoid.

2. Left mainstem bronchus.

3. Diaphragm.
4. Aorta.

B. Three neck compression sites include:

1. Thyroid.
2. Parathyroid.
3. Thymus.

NB: The cervical spine may impinge especially in the elderly.

C. In the chest, compression may come from:

1. Large nodes (associated with histoplasmosis, sarcoid, tuberculosis).
3. Duplication cysts.
4. Enlargement of the heart especially secondary to mitral valve.
5. Aneurysms.
6. Poststenotic dilatation secondary to coarctation of the aorta.
7. Massive enlargement of the liver.

Diverticulum

A diverticulum is generally due to anatomic weakness including:

1. Killian's dehiscence: Between the cricopharyngeus and thyropharyngeus muscles.
2. Lamier-Hackeman space: Between the circular and longitudinal fibers of the esophagus.
3. Killian-Jameison space: Between the cricopharyngeus and the circular fibers of the esophagus.

A. Zenker's.

This diverticulum is related to a developmental weakness of the muscular coat of the posterior portion of the pharynx between the oblique fibers of the inferior constrictor muscle and the horizontal fibers of the cricopharyngeus. It represents 80% of all diverticula.
1. Symptoms.
   a. Delayed regurgitation (as late as 24 hours).
   b. Aspiration.
   c. Dysphagia (a late symptom).
   d. Foul breath.
   e. Bleeding and perforation are very rare.
   f. Gurgling or fluid slosh.

2. Diagnosis.
   a. Barium swallow.
   b. Esophagoscopy.

3. Treatment.
   a. Excision of sac and cricopharyngeal myotomy (Dohlman's procedure is not recommended).

   B. Midesophageal.
   1. Most commonly at the level of the pulmonary hilum.
   2. Accounts for 7% of all diverticula.

   C. Epiphrenic.
   1. Lies just above the cardioesophageal junction.
   2. Often on the right side.
   3. Very minimal symptoms.
   4. Accounts for 13% of all diverticula.

**Pathophysiology**

A. Pulsion diverticula often are seen concomitant with other esophageal disorders such as spasm and hiatal hernia. As the diverticulum enlarges, its sac lies lower in the neck so that it traps food and liquids.
B. Traction diverticula are pulled out by an inflammatory process adjacent to the esophagus. They usually occur in the midesophageal area at T-4, T-5 region where the esophagus lies closest to the nodes of the tracheobronchial area. They tend to be on the left side and are rare.

NB: Traction diverticula are associated commonly with tuberculosis.

C. Symptoms from diverticula of the esophagus do not always intensify (25% get worse).

D. Complications include:

1. Aspiration pneumonia.
2. Bronchoesophageal fistula.
4. Abscess.

NB: Intraluminal diverticulosis of the esophagus is associated with Candida albicans (1-3 mm size; multiple).

**Hiatal Hernia**

The presence in the chest, above the diaphragm, of a portion of a stomach that has passed up through the normal esophageal hiatus is referred to as a hiatal hernia.

1. Incidence: 0.8-29.6%.
2. Incidence is age related: under 40, 9%; over 70, 69%.
3. Found in women more frequently than men.
4. Loss of muscle tone is a common factor in pathogenesis.
5. Associated disease or pathologic processes include:
   a. Pregnancy.
   b. Obesity.
   c. Tight girdles.
   d. Ascites.
   e. Esophageal tumors.
f. Exercise tension.

g. Prolonged nasogastric tube placement.

h. Constipation.

i. Intra-abdominal tumors.

j. Kyphoscoliosis (high association).

k. Status post-Heller's procedure.

l. Status post partial gastrectomy.

m. Trauma.

6. Types.

a. Paraesophageal (rare).

b. Sliding (common).


7. Symptoms.

a. Reflux esophagitis (postprandial).

b. Spasm.

c. Dysphagia.

d. Bleeding.

e. Vomiting (incarceration).

f. Aspiration.

8. Sandifer's Syndrome.

Bizarre contortions of the neck, apparently in an effort to reduce discomfort in the lower esophageal region, have been reported as symptoms of unrecognized hiatus hernia in some children.

9. Plummer-Vinson syndrome often is associated with hiatus hernia. The sudden onset of projectile vomiting, severe pain or spasm (cramps), and complete aphagia heralds acute incarceration and is a surgical emergency.
NB: The physical examination of a patient with a hiatus hernia is often negative except for chest (anterior and posterior) wall auscultation revealing borborygmi.

**Diagnosis**

1. Laboratory: Occult blood in stool.

2. Hypochromic microcytic anemia.


5. Esophagoscopy is not indicated unless additional information is desired regarding other possible pathology. On examination the gastroesophageal rosette is at 34-38 cm instead of the usual 40 cm.

Saint's Triad: Gallstones, diverticular colonic disease, hiatus hernia.

10. Dermatomyositis.

A diffuse inflammatory disorder of striated muscle which causes symmetrical weakness and muscular atrophy.

a. Stomatitis is present.

b. Weakness of facial muscles may hamper eating.

c. Peristalsis is diminished and poorly coordinated.

d. The esophagus may be dilated.

e. Manometric evaluation:

1) Decreased upper esophageal sphincter pressure.

2) Low multiple contractions of the pharynx and the esophagus.

f. In contrast to scleroderma, the striated muscles of the hypopharynx and esophagus are involved.

g. Hiatus hernia and the reflex esophagitis commonly associated with scleroderma are absent.


a. Sixty percent of the patients have symptomatic dysphagia.
b. Reflux and esophagitis is not uncommon.

c. Aperistalsis of the lower two-thirds of the esophagus is present.

d. Marked decrease in lower esophageal sphincter pressure is demonstrated by manometrics.

e. Normal peristalsis can be shown in the upper esophagus.

f. Barium studies show: Aperistalsis, dilatation, and gastroesophageal reflux.

NB: Both scleroderma and achalasia show distension in the supine position by barium study. However, when the patient is standing an air/fluid level is often seen in achalasia while free passage to the stomach is noted in the patient with scleroderma.

**Lower Esophageal Ring (Schatzki's Ring)**

This is a concentric ring or a weblike narrowing occurring at the junction of the esophageal and gastric mucosa.

A. Incidence.

1. Is 6-14% of routine barium swallows, but only one-third are symptomatic.

2. Symptoms are rare under 40 years of age.

B. Pathology.


3. Dyphagia is likely to present when the ring reduces the esophageal lumen to less than 13 mm in diameter.

C. Symptoms.

1. Intermittent dysphagia is the salient feature. The most characteristic feature is the patient's ability to bring up food or to force it down when stuck, and the continue to ear.

2. Absence of heartburn.

D. Diagnosis.

1. Laboratory studies are of no value.

2. Manometry is of no value.
3. Cine barium swallow (patient should be recumbent for study).

4. Flexible fiberoptic esophagoscopy.

5. The ring is usually noted 4-5.5 cm above the apparent diaphragmatic shadow, above the pinched-off segment of barium, moving upward away from the diaphragm as the segment below the ring distends with barium.

**Esophageal Webs**

A web is an aberrant structure consisting of squamous mucosa located anywhere along the esophagus.

1. Dysphagia due to a web is slowly progressing.

2. Cervical webs are associated with iron deficiency.

3. There is a significant association between webs and hypopharyngeal carcinoma (see Plummer-Vinson syndrome).

4. Webs usually are noted on the anterior wall of the esophagus and require better lateral or oblique films for demonstration. A ring on the other hand is best noted when the esophagus is distended.

5. Webs are asymmetric whereas rings are symmetric.

E. Differential.

1. Plummer-Vinson syndrome.

2. Carcinoma.

3. Achalasia.


5. Leiomyoma.

6. Ring of cartilaginous tissue.

F. Therapy.

1. Bougienage.

2. Inflatable bag.

Boerhaave's Syndrome

Boerhaave's syndrome results from a tear through all three layers of the wall of the esophagus just above the diaphragm produced by a sudden increase in esophageal pressure.

A. Occurrence.
1. Rare in both adults and children.

B. Pathology.
1. Short, linear, 1 to 4 cm long tear.
2. Located on the left side 90% of the time.
3. Five times more frequent in men than women.

C. Clinical picture.
1. Vomiting.
2. Abrupt, sharp (knifelike) pain in epigastrium.
3. Radiation of pain to left shoulder.
4. "Something gives way inside".
5. Shock.
6. Respiratory difficulty.
7. Occasional hoarseness.
8. Subcutaneous emphysema.

10. Diagnosis.
1. History and physical (note subcutaneous emphysema).
2. Chest x-ray (left effusion).
   a. If tapped check for amylase to rule out pancreatitis.
   b. Culture and Gram stain.
3. Barium should not be used for contrast study, Hypaque is more benign and does not cause granulomas. (Some radiologists feel barium is best and not dangerous.)
E. Differential.

1. Myocardial infarction.
2. Pulmonary embolus.
3. Ruptured gastric ulcer.
4. Ruptured duodenal ulcer.
5. Acute pancreatitis.
6. Aortic aneurysm rupture.

F. Treatment.

1. Thoracotomy and mediastinum drainage.
2. Systemic antibiotics.

**Mallory-Weiss Syndrome**

This syndrome is manifest by bleeding from the cardia of the stomach (lacerations).

1. Massive upper gastrointestinal bleeding.
2. Seen most often in alcoholics.
3. Seen most often in males over 40.

**Iatrogenic Perforation**

This complication is largely due to an inexperienced endoscopist.

A. Clinical picture.

1. Sore throat immediately after procedure.
2. Tachycardia out of proportion to fever.
3. Temperature spike, chills.
4. Subcutaneous emphysema.
B. Chest x-ray.

1. Effusion.
2. Widened mediastinum.
3. Maybe pneumoperitonitics.

C. Therapy.

1. Antibiotics.
2. Drainage.
3. Closure.

**Foreign Bodies in Esophagus**

History and physical examination. Foreign bodies commonly lodge at or just below the criopharyngeus.

A. Evaluation.

1. Auscultation.

2. Barium swallow is best avoided since, in a symptomatic patient, treatment will be instituted regardless, and if the patient needs surgery barium will obscure the field. Furthermore in awake intubations the patient may aspirate.

3. Occasionally a cotton pledget or marshmallow coater with barium may of assistance in localizing a bone if there is a question of a foreign body.

B. Treatment.

1. Assure the patient that his life is not in danger.

2. Never attempt to push a foreign body into the stomach by bougienage.

3. Attempt relaxation with:
   a. Meperidine (Demerol)(mg/kg) and atropine.
   b. Amyl nitrite (young healthy patients only).
   c. Diazepam (Valium).
   d. Glucagon (1 mg/amp) IV, followed by water; wait 20 minutes and repeat.
4. The use of papain to dissolve meat that is stuck in the esophagus is contraindicated, and its use is currently not recommended although products such as meat tenderizer have been reported to be successful. A tendency toward perforation is the feared sequela.

5. Esophagoscopy and removal.
   a. Positioning of the patient is 90% of the knack.
   b. Many instruments designed for foreign body removal are sharp and by themselves dangerous in inexperienced hands.
   c. Follow Jackson's dictum for pointed objects "don't look for the foreign body, look for its point".

4. Never use force - bougienage is not indicated.

5. Perhaps most important is to have complete and safe control of the airway before beginning any manipulation.

**Congenital Diaphragmatic Hernias**
1. Pleuroperitoneal (Bochdalek's): Posterior.
2. Retrosternal (Morgagni's): Anterior.

**Inflammatory Disorders**

**Plummer-Vinson Syndrome (Patterson Kelly)(Sideropenic Dysphagia)**
1. Incidence: 90% female, 10% male.
2. Predominant occurrence in Northern hemisphere.
3. Hypothyroidism is commonly associated.
   A. Clinical features.
   1. Esophageal webs.
   2. Iron deficiency anemia.
   3. Dysphagia.
   4. Lower esophagus is usually normal.
   5. Tongue and oropharynx are usually atrophic.
   6. Painless. However, a choking sensation is felt.
7. Ninety percent of the patients have absence of teeth.
9. Achlorhydria (30-40%).
10. Pernicious anemia.
11. Atrophic gastritis (40%).
12. Multiple circulatory autoantibodies (50%).
13. Splenomegaly may be present.
14. Cheilosis is notable.

B. Pathology.
1. The esophageal and pharyngeal mucosa may be atrophic at autopsy.
2. Striated muscle at the upper end of the esophagus is often atrophic and replaced by cartilaginous tissue.
3. The lower portion of the esophagus is normal.
4. Biopsy of the associated webs reveals only chronic inflammation with normal squamous epithelium.

C. Diagnosis.
1. Cinefluoroscope is essential.
2. Esophagoscopy.
   a. A web is viewed as a smooth, grey, diaphragmatic opening with eccentric lumen.

D. Treatment.
1. Treat underlying anemia.
2. Dilate.
3. NB: Over 50% of women with PV syndrome develop carcinoma of the upper gastrointestinal tract or hypopharynx.
Esophagitis

A. Diagnosis.
1. Sphincter incompetence.
   a. pH monitoring.
   b. Radiographic scanning (cine studies).
   c. Esophageal manometry.
2. Esophagitis by acid infusion test (Bernstein's test).
3. Esophagoscopy.
4. Esophageal biopsy.

B. Treatment.
1. Position.
2. Correct underlying problem (nasogastric tube, hiatus hernia, etc).
3. Cimetidine (300 mg PO every 4 hours, PC and HS).
4. Antacids.
5. Surgical closure of hiatus hernia/vagotomy.

Barrett's Esophagus

The lower portion of the esophagus is lined with columnar (gastric) rather than squamous epithelium.

Barrett's Ulcer

The term Barrett's ulcer is used to describe the sharply punched out, deep peptic ulcer occurring in such epithelium.

Delahanty's Syndrome

Hoarseness, chronic laryngitis, and arytenoid inflammation due to acid spillover.
Moncreiff's Syndrome
Hiatus hernia associated with sucrosuria and mental retardation.

Mendelsohn's Syndrome
Aspiration pneumonia from gastric reflux (70% R, 30% L).

Sandifer's Syndrome

Neoplasms

Benign Tumor

1. Most common is leiomyoma.
2. Followed by:
   a. Fibroma.
   b. Lipoma.
   c. Hemangioma.
   d. Neurofibroma.

Carcinoma

1. Four percent of cancer deaths.
3. No genetic predisposition.
4. Associated with chronic alcohol use and tobacco.
   a. Twenty-five percent more common in heavy drinkers.
5. May increase risk:
   a. Lye stricture.
   b. Plummer-Vinson syndrome.
   c. Esophagitis.
6. Location.
a. Forty to fifty percent occur in the lower one-third.

b. Thirty to forty percent occur in the middle one-third.

c. Ten to thirty percent occur in the upper one-third.

7. Other malignant neoplasms include:

a. Sarcoma.

b. Leiomyosarcoma.

c. Fibrosarcoma.

**Esophagostomy**

1. Advantages.

a. Tube may be left out.

b. Easy skin care.

c. Feeding in upright position.

d. Less risk of postoperative ileus/atelectasis.

2. Disadvantages.

a. Cellulitis.

b. Infection.

c. Hemorrhage.

d. Recurrent nerve injury.

e. Aspiration pneumonia.

3. Contraindications.

a. Superior vena cava syndrome.

b. Complete esophageal obstruction.

c. Anterior laryngo-pharyngoscopy or neck carcinoma.

d. Irradiation to neck (greater than 5000 rads).
e. Severe gastric reflux.

f. Uncontrolled aspiration.