Chapter 4. Oral Cavity, Pharynx, and Esophagus

Oro-Naso-Hypopharynx

I. Anatomy. The pharynx is a musculomembranous tube posterior to the oral cavity that extends from the base of the skull to the larynx. Posteriorly, the pharynx is connected to the cervical vertebral column by loose areolar tissue; laterally, it is bordered by the constrictor muscles; superiorly, it is limited by the occipital bone and sphenoid sinus; inferiorly, it joins the oesophagus at the cricopharyngeus muscle level; and anteriorly, it is incomplete, with an opening communicating with the nasal cavities, the oral cavity, and the larynx. The pharynx is divided into the nasopharynx, the oropharynx, and the hypopharynx (laryngopharynx).

A. The nasopharynx is situated above the soft palate and communicates anteriorly with the nasal cavities via the choanae. The roof and posterior wall contain the adenoids, or pharyngeal tonsils, lymphatic tissue that often attains considerable size, especially in children. The paired eustachian tube orifices are situated on the lateral walls of the nasopharynx, behind the posterior ends of the inferior turbinates.

B. The oropharynx, visible directly by depressing the tongue, extends from the soft palate to the hyoid bone, opening anteriorly into the oral cavity and containing on its lateral wall the palatine, or faucial, tonsils.

C. The hypopharynx is inferior to the base of the tongue and extends from the hyoid bone to the lower border of the cricoid cartilage. It contains the triangular-shaped entrance to the larynx, the base of which is the epiglottis and the sides, the aryepiglottic folds. The oropharynx and hypopharynx are actually a muscular cone. Swallowing is made possible by the specialized constrictor muscles, and the attached accessory muscles protect the airway when gag and cough reflexes are initiated by unwanted material threatening the airway.

II. Physical examination. Most conditions of the pharynx are readily diagnosed from the history and physical examination.

A. Systematic examination of the oral cavity and oropharynx is imperative so that no abnormalities are overlooked. Dentures should be removed. Attention should be directed to the lips, buccal mucosa, teeth, gingiva, floor of mouth, tongue, hard and soft palates, and posterior pharyngeal wall. Effects of both local and systemic disease can be noted.

Palpation of lesions in the oral cavity and base of tongue is imperative in suspected neoplastic disease. The oropharynx can best be visualized with a tongue depressor. The palatine tonsils lie between the anterior and posterior tonsilar pillars, and are accessible to direct inspection and palpation. The tonsils are normally the same color as the surrounding oral mucosa and often contain visible crypts, in which exfoliated epithelium and debris can accumulate. Lymphoid nodules on the posterior pharyngeal wall appear as discrete submucosal masses. The lateral pharyngeal bands, also lymphoid-containing tissue, are located behind the posterior tonsilar pillar and descend from the nasopharynx toward the lingual tonsils at the base of the tongue. The adenoids, palatine, and lingual tonsils, along with the lateral pharyngeal bands, compose Waldeyer's ring.
B. Nasopharyngeal examination requires either proper use of the indirect mirror or direct visualization with rigid or flexible fiberoptic telescopes. An indirect, defogged No. 0 or No. 00 mirror is placed to one side of the uvula, nearly touching the posterior pharyngeal wall. A light beam from a head mirror or headlight is reflected from the nasopharyngeal mirror into the nasopharynx. The patient is instructed to breathe through the nose so that the palate will relax. The posterior ends of the nasal turbinates, the eustachian tube orifices with adjacent torus tubarius and fossa of Rosenmüller, the posterior end of the vomer, adenoid tissue, and the entire choanal circumference can be visualized. Direct nasopharyngoscopy is best performed after shrinking and anesthetizing the nasal mucous membranes. The scope is passed through one nasal chamber along its floor, into the nasopharynx, and manipulated so as to view the above structures.

C. Hypopharyngeal examination is performed with either a mirror or flexible fiberoptic telescope. The tongue is retracted anteriorly by grasping it with a gauze pad. A defogged No. 4 or No. 5 mirror is placed with the back touching the midline soft palate. A light is directed onto the mirror to visualize the base of tongue, valleculae, pharyngeal walls, piriform sinuses, and larynx.

III. Infectious pharyngitis. Sore throat is the outstanding symptom of acute pharyngitis, no matter what the etiology.

A. Acute bacterial pharyngotonsillitis

1. Signs and symptoms

   a. Bacterial tonsillitis or pharyngitis, usually caused by group A beta-hemolytic streptococci, presents with the sudden onset of sore throat, malaise, fever, anorexia, and odynophagia. Temperature may rise to 102.2-104°F over the first 24-48 hours, often to 104-105°F in children. Dehydration may result from the odynophagia and fever. Myalgias, arthralgias, headache, and vomiting may occur. Cough and rhinorrhea make a viral etiology more likely.

   b. On physical examination, the patient appears ill with pharyngeal erythema (sometimes deeply red-purple), patchy yellow-white exudate, and hypertrophy of all local lymphoid tissue. Edema of the soft palate, as well as tender cervical adenopathy, may be evident. There may be a patchy yellow-white exudate, which easily can be wiped away. The presence of exudate does not establish a specific etiology and may be seen in infections due to *S. pyogenes*, *S. pneumoniae*, *H. influenzae*, *H. parainfluenzae* (children), and *Corynebacterium diphtheriae*, as well as some viruses (adenovirus and mononucleosis).

2. Diagnosis is made by throat culture (on 5% sheep blood agar), which will usually grow group A beta-hemolytic streptococci in this clinical picture. Culture accuracy varies with the aggressiveness of the technique. Sampling both sides of the pharynx yields a higher percentage of positive growth. A single, representative throat culture has approximately 95% of the accuracy of serial cultures. New rapid tests for group A streptococci can provide information in hours. However, false-negatives occur, and follow-up traditional cultures
should be performed when the clinical picture suggests streptococci. A leukocytosis and negative mononucleosis spot test aid in making the diagnosis.

3. Therapy

   a. Treatment consists of bed rest, hydration, warm saline pharyngeal irrigations hourly while awake until pain subsides, antipyretics, and an antibiotic.

   b. Penicillin is the drug of choice unless allergy precludes it. Two regimens should be considered:

   (1) Oral penicillin V, 250 mg PO tid or qid for 10 days; 500.000 units/kg/day PO tid or qid for 10 days in children.

   (2) IM benzathine penicillin, a single dose, 600.000 units for children less than 6 years of age; 900.000 units between 6 and 9 years old; and 1.2 million units for those over 9 years old. Higher initial levels can be achieved with the addition of IM procaine (IM Bicillin R, 1.2 million units) or oral penicillin along with benzathine penicillin.

      Benzathine, 900.000 units, plus procaine penicillin G, 300.000 units, may be better tolerated than plain benzathine penicillin G in the pediatric patient. In cases of penicillin allergy, erythromycin estolate, in a dose of 10-20 mg/kg/day for 10 days, is effective therapy. For erythromycin intolerance, clindamycin should be considered.

   c. An occasional patient needs hospitalization for IV hydration because of dehydration secondary to severe odynophagia.

   d. Recurrent acute or chronic tonsillitis may require tonsillectomy. The indications are seven episodes in 1 year, five episodes per year for 2 years, or three episodes per year for 3 years.

   e. For symptomatic bacteriologic failure, rifampin 20 mg/kg, not exceeding 600 mg/day, administered as a single daily dose for the last 4 days of a new primary regimen (cephalosporin), is frequently effective.

B. Diphtheria is a rare disease today, but it must be considered in any pharyngeal infection, especially in those demonstrating a membranous exudate. Required immunization has decreased the incidence during the past 40 years, but nonimmunized patients are common and endemic. Occasional epidemics (eg, San Antonio, 1970) are still seen. Immediate and specific treatment is necessary to prevent the serious complications of paralysis or myocarditis.

1. Signs and symptoms

   a. Onset is more insidious than streptococcal pharyngitis, with several days of sore throat, low-grade fever, and increasing swelling of the anterior cervical nodes. Associated symptoms are headache, malaise, nausea, and increasing toxemia.
b. The patient presents with a characteristic, adherent dirty gray membrane over the tonsils, extending over the pillars and sometimes onto the soft palate; when removed with a culture swab, the underlying area bleeds. The mucosa of the nose, larynx, and hypopharynx may also be involved. Ulcerations are not common. Complications can include airway obstruction and cardiac toxicity.

2. Diagnosis. Direct culture should always be obtained for smear (Gram) and culture (on Klebs-Löffler and tellurite agar media). Patients treated with penicillin or erythromycin before being seen may have less marked symptoms and eradicated organisms, but antibiotics are totally ineffective against circulating toxin.

3. Therapy

a. Prompt treatment with specific diphtheria antitoxin should be initiated if the smears are questionable and if strong clinical suspicion prevails. After appropriate skin sensitivity testing, 20,000-50,000 units of antitoxin are given IM or IV (100-200 mL saline diluted over 30 minutes). This should be given within the first 48 hours of the disease onset.

b. Isolation until three consecutive daily nose and throat cultures are negative, strict bed rest, and antibiotics are ordered while carefully observing the patient's airway for signs of obstruction. Tracheotomy may be necessary.

c. Antibiotic therapy for the pediatric patient should be procaine penicillin, 25,000-50,000 units/kg/day IM q12h, or erythromycin, 50 mg/kg/day PO for 7-10 days. In the adult, procaine penicillin, 1.2 millions units IM, or erythromycin, 250-500 mg PO qid, is used.

C. Infectious mononucleosis is an acute disease, occurring most commonly among adolescents and young adults; it is caused by the Epstein-Barr virus (EBV). It is characterized by fever, pharyngitis, lymphadenopathy, lymphocytosis with atypical cells, a positive heterophile antibody titer, and persistent EBV-antibody responses. CMC and Toxoplasma gondii infections can mimic mononucleosis, presenting with atypical circulating lymphocytes and peripheral lymphocytosis.

1. Signs and symptoms

a. Sore throat is the most common symptom. Over 80% of patients demonstrate sore throat, fever, and anterior and posterior cervical lymphadenopathy.

b. Other symptoms are malaise, persistent fever, and headache. These symptoms can be present in spite of previous treatment with antibiotics, further aiding the diagnosis.

c. Physical examination reveals a febrile (101-103°F) patient (although children often have no fever), with marked hyperplasia, inflammation, and edema of the pharyngeal lymphoid tissue. In 50% of cases, a gray-white membrane covers the tonsils for 7-10 days. There is anterior and posterior cervical adenopathy. Palatal petechiae are seen in about one-thirds of patients, splenomegaly occurs in about one-half, and a transient faint maculopapular rash is present on the trunk and extremities of about 10% of patients. Fifty percent of patients
develop hepatomegaly, and 70-75% develop splenomegaly. There is anterior and posterior cervical adenopathy.

2. **Diagnosis.** Laboratory findings include a leukocytosis of 10,000-20,000 with 40-60% lymphocytes and monocytes, including 10-20% atypical forms. A positive Monospot test and high or rising heterophil antibody titers support the clinical diagnosis. Eighty percent of patients have abnormal liver function tests. EBV seroconversion makes the diagnosis.

3. **Therapy**

   a. Treatment of infectious mononucleosis includes penicillin for secondary bacterial infection if throat cultures are indicative of such; symptomatic measures for fever, pharyngitis, and headache (analgesics, warm saline irrigations); and, in patients with severe pharyngotonsillitis with edema, steroids.

   b. Prednisone, 40-80 mg the first day and decreasing this dosage daily over 7-10 days, is very effective in reducing inflammation.

4. **Complications** of mononucleosis include splenic rupture (often from vigorous palpation), hepatitis, myocarditis, ascending paralysis, hemolytic anemia, and airway obstruction.

   D. **Vincent's angina (gangrenous pharyngitis).** Vincent's angina is usually found in older patients with poor oral hygiene and general lowered resistance to infection. Tissue destruction seems to be caused by the synergistic interaction between several endogenous mouth organisms under anaerobic conditions. The lesions contain large numbers of fusiform (Borrelia vincenti), spirochetes, and other normal mouth flora.

1. **Signs and symptoms**

   a. **Symptoms of sore throat,** dysphagia, salivation, fetid breath, bad taste in the mouth, and tender gingiva are typical.

   b. **Physical findings** include low-grade fever, submandibular lymphadenopathy, ulcerative lesions on the pharyngeal mucous membranes, with a dirty, gray-yellow membrane covering the tonsils and occasionally the tonsillar pillars and soft palate. As opposed to the diphtheric membrane, this membrane peels off easily. The gingiva and interdental papillae are ulcerated, bleed easily, and are also covered with a membrane.

2. **Diagnosis** by laboratory depends on demonstrating typical organisms in cultures and on biopsy sections of the necrotic lesions.

3. **Therapy.** Treatment is with penicillin (250 mg PO qid) or tetracycline (1.0 gm PO qid), along with frequent gargles with oxidizing agents (half-strength hydrogen peroxide or sodium perborate, 1 tsp : 1 glass of warm water) or sodium perborate pastes applied several times a day.
E. **Candidiasis (moniliasis).** *Candida albicans* is a yeast found in 15-25% of normal mucous membrane surfaces. The normally saprophytic organism becomes pathogenic in states of impaired tissue resistance (debilitated, diabetic, immunocompromised patients) or in those patients on prolonged antibiotic regimens or receiving radiation therapy. A mild form is seen in normal infants and is commonly known as "thrush".

1. **Signs and symptoms**

   a. Sore mouth and throat and odynophagia can occur.

   b. **Examination** reveals a red, edematous mucous membrane with a soft, white mucoid exudate present on the tonsils, buccal, and gingival mucosa. These patches are easily scraped off and reveal a reddened, slightly ulcerated surface beneath. Cervical adenopathy and fever are usually absent.

2. **Diagnosis** is confirmed by a potassium hydroxide preparation of the scrapings from the lesion or by culture on Sabouraud’s agar.

3. **Therapy.** Nystatin oral suspension (200,000 units/mL), 2-3 mL swished in the mouth and then swallowed q4h until inflammation is controlled, is an appropriate treatment regimen. If candida esophagitis is present, systemic treatment with oral ketoconazole can be given.

F. **Syphilis**

1. **Signs and symptoms.** Syphilis, caused by *Treponema pallidum*, may produce pharyngeal lesions that are relatively asymptomatic in any of the three states of the disease.

   a. **Primary stage.** The primary chancre is seen as a firm, indurated, painless lesion with superficial ulceration, usually on the lip, tongue, tonsil, or soft palate. Regional adenopathy accompanies the primary chancre. The nodes are firm and painless and may persist long after the chancre heals (in 4-6 weeks).

   b. **Secondary stage.** Superficial mucosal ulcerations, called mucous patches, are characteristic of secondary stage syphilis. These lesions vary and appear as painless silver-gray erosions, surrounded by a red periphery, white papules or macules, or as a large, deep, ulcerated unilateral tonsillitis. Posterior cervical lymphadenopathy, a macular rash, and other mucocutaneous lesions may accompany the oropharyngeal findings.

   c. **Tertiary stage.** These lesions, called gumma, may affect the maxilla, mandible, or oral cavity. The tongue lesions can mimic carcinoma.

2. **Diagnosis**

   a. Except for gummatous lesions, darkfield examination is positive. Care must be taken to differentiate *Treponema microdentium*, which is part of the normal oral flora, from *Treponema pallidum*. 


b. The serologic tests for syphilis are positive, with the fluorescent treponemal antibody absorption test (FTA-ABS) being the confirmatory test of choice. All patients being tested for syphilis should be HIV tested.

c. The mucous membrane lesions must be differentiated from follicular tonsillitis, diphtheria, Vincent's angina, pemphigus, herpes simplex, minor trauma, and infectious mononucleosis. Infectious mononucleosis poses a special problem in that false-positive serologic tests for syphilis can occur.

3. Therapy. Treatment entails benzathine penicillin G, 2.4 million units IM biweekly to total 4.8 million units. Doxycycline 100 mg bid for 2 weeks or tetracycline 500 mg qid for 2 weeks should be used in penicillin-allergic patients. Erythromycin must be used with caution, because increasing resistance is being noted.

G. Gonococcal pharyngitis. Gonorrhea is the most prevalent of all the venereal diseases and may be transmitted both heterosexually and homosexually. Pharyngitis is seen following oral-genital transmission of organisms, often in male homosexuals.

1. Signs and symptoms

a. Symptoms are sore throat and perhaps mild temperature elevation.

b. Examination of the oropharynx may reveal erythema and edema, with or without exudate of the uvula, soft palate, and tonsillar pillars.

2. Diagnosis. Culture of the areas on Thayer-Martin medium reveals typical gram-negative, oxidase-positive colonies, and smear of the mucous membranes demonstrates gram-negative intracellular cocci.

3. Therapy. Treatment is with ceftriaxone 250 mg IM in a single dose. Ofloxacine 500 mg PO as a single dose may be appropriate. Tetracycline resistance is widespread in the United States, and tetracycline therefore is no longer considered adequate.

H. Tuberculosis. The pharyngeal mucosa is not a common site of involvement in extrapulmonary tuberculosis but must be considered in a patient with active cavitary pulmonary tuberculosis and indolent, punched-out ulcers of the gingivae and pharyngeal mucosa.

1. Signs and symptoms. Hoarseness, dysphagia, and extreme odynophagia suggest laryngeal tuberculosis in this setting.

2. Diagnosis. Confirmational biopsy, smear, and culture identification of the tubercle bacilli are necessary prior to treatment.

3. Treatment is with isoniazid and rifampin for 6 months, with pyrazinamide added during the first 2 months. In patients exposed to drug-resistant organisms, treatment is with isoniazid, rifampin, and ethambutol, with pyrazinamide added during the first 2 months.
I. Viral pharyngitis. Most cases of mild pharyngitis are due to viruses and are manifested by mild pharyngeal erythema associated with cough, rhinorrhea, myalgias, headache, and fever. Also favoring a viral origin are a normal white blood count, hoarseness, and absence of lymphadenopathy, which is present usually in bacterial pharyngitis or mononucleosis. Exudative pharyngitis may be caused by adenoviruses, EBV, as well as other viruses, so the presence of exudate does not rule out viral etiology. Two characteristic, viral-caused diseases are listed below.

1. Herpangina is a benign, infectious disease of childhood, less common in young adults, caused by various coxsackie and ECHO viruses. It occurs in epidemic form, usually during the fall and summer months.

   a. Signs and symptoms. Characteristic symptoms include sudden onset of high fever (104°F), severe sore throat, odynophagia, nausea and vomiting, and malaise with absence of rhinorrhea and other respiratory tract symptoms. The tonsillar pillars, tonsils, and soft palate demonstrate marked erythema, injection, and numerous 1- to 2-mm vesicles, which soon rupture and enlarge into 3- to 4-mm, punched-out, shallow ulcers with gray craters surrounded by deep red areolas. These ulcers heal in 4-5 days with regression of local symptoms, and total recovery is within 8-10 days.

   b. The diagnosis is usually made clinically. Clinically these lesions are for the most part difficult to differentiate from herpes simplex virus (HSV). The site can help, with HSV involving the gingiva more frequently. Throat cultures and acute and convalescent antibody titers confirm the diagnosis.

   c. Therapy. Treatment consists of local and symptomatic measures. Topical anesthetics may be used if the patient has difficulty eating or drinking. Saline gargles and irrigations, a soft diet, and antipyretics are also helpful.

2. Herpes (primary herpetic gingivostomatitis). Either the type 1 or type 2 herpes simplex virus can cause primary infection of the oral cavity and pharynx. Transmission is via close personal contact. Children between ages 2 and 5 years are usually affected.

   a. Signs and symptoms. Symptoms include a prodrome of fever, headache, malaise, nausea and vomiting, irritability, oral fetor, and tender submaxillary lymphadenopathy. Physical examination reveals erythema and edema of the gingival and anterior oropharyngeal mucous membranes, with multiple vesicles present. These vesicles quickly rupture and form ulcers that heal in 1-2 weeks. An important diagnostic sign is the generalized acute marginal gingivitis. Systemic symptoms cease within 3-5 days. As opposed to herpangina, the vesicles are usually confined to the anterior portion of the mouth, and the disease does not occur in epidemic form.

   b. Diagnosis. Impression smears (Giemsa stain) of the clear liquid from an opened vesicles reveals syncytial giant cells with intranuclear inclusions. Viral culture and immunofluorescence can confirm the diagnosis.
c. **Therapy.** Symptomatic therapy as outlined for herpangina (see 1.c.) is usually the only treatment necessary. Antibiotics and steroids are of no help in primary herpes infections. Acyclovir is given only to immunosuppressed patients with chronic oropharyngeal herpes.

J. **Lingual tonsillitis.** Although commonly occurring with tonsillitis, lingual tonsillitis can be an isolated infection. It is characterized by a mild temperature elevation, odynophagia, and a burning or painful sensation localized deep in the thorax. The patient is tender over the hyoid bone and may have altered phonation, the typical "hot potato" voice. Mirror examination demonstrates pooled secretions, red and edematous lingual tonsils with patchy exudate, and a normal epiglottis. Treatment is essentially the same as that for acute bacterial pharyngitis (see A.).

K. **Nasopharyngitis.** The adenoid tissue is usually simultaneously inflamed in a bacterial or viral pharyngitis, especially in children. Symptoms include those of acute pharyngitis along with nasal obstruction or pain, rhinorrhea, and burning behind the nose and above the palate with respiration or swallowing. Indirect nasopharyngoscopy reveals a yellowish exudate in the shallow clefts of the red and edematous adenoid folds. Treatment is dependent on culture results and is identical to those listed for specific pharyngeal infections.

L. **Acquired immunodeficiency syndrome (AIDS)** is caused by infection with the human immunodeficiency virus (HIV). It manifests as the presence of one or more opportunistic diseases associated with immunodeficiency.

Disease transmission is usually through sexual contact, parenteral exposure to blood or blood products, and perinatally from mother to child.

Currently, high-risk groups are homosexual men, intravenous drug users, children born to HIV-infected mothers, and blood transfusion recipients of unscreened blood products. Sexual contact with HIV-infected persons has increased the rate of infection in the heterosexual population.

1. **Signs and symptoms.** Otolaryngologic manifestations of AIDS include Kaposi's sarcoma (skin, external ear canal, nose, nasopharynx, oropharynx, and larynx), candidiasis (oropharyngeal, oesophageal), parotid enlargement (sometimes cystic), cervical lymphadenopathy, herpetic ulcers (nasal vestibule and upper lip), and sensorineural hearing loss.

2. **Diagnosis** is made by serologic tests, which require patient consent.

3. **Therapy**

   a. **Azidothymidine (AZT)** prolongs patient survival but has a significant incidence of serious side effects.

   b. **Treatment** for specific infections (such as antifungal therapy for candidiasis) is employed whenever possible.
c. Preventative measures such as condom use for sexually active people, clean needles for intravenous drug users, screening of blood products, and universal precaution for health care workers are all essential.

IV. Noninfectious etiology

Pharyngeal trauma (eg, heat, foreign body), irritant inhalation or ingestion (ammonia, lye, acid), and dryness (eg, mouth breathing) are among some of the causes to be considered in a patient presenting with pharyngeal lesions, pain, or both. These causes are not detailed here.

A. Pemphigus is a progressive disease of unknown etiology. Skin and mucous membranes are characterized by scattered bullae, which can involve other organs and lead to death unless treated with immunosuppressive agents or steroids.

1. Signs and symptoms

   a. More than half of the patients first present with oropharyngeal lesions. Patients complain primarily of throat pain, odynophagia, or tongue pain. The vesicles and bullae are painless at first, but with rupture and secondary infection, the pain is severe.

   b. On physical examination, the fibrinous exudate covering tender, superficially eroded areas of the oral cavity and oropharynx is characteristic. Often the lesions remain limited to this area for several weeks before spreading to other skin and mucous membrane sites.

2. Diagnosis is made by a high index of suspicion prompting a subsequent biopsy, which reveals acantholysis of the suprabasal cell region and immunofluorescent antibodies to a specific intracellular antigen at the site of acantholysis.

3. Therapy. Management involves vigorous and early systemic corticosteroid therapy (prednisone, 120-240 mg), along with adjuvant immunosuppressive drugs (methotrexate, azathioprine). Hospitalization, isolation (if the skin is denuded, as in a burn patient), and topical and systemic antibiotics for secondary infection may be necessary. Viscous lidocaine, milk of magnesia, diphenhydramine (Benadryl), dyclonine (Dyclone 0.5%), and saline mouth washes may be helpful for local pain and oropharyngeal cleansing.

B. Pemphigoid includes pemphigoid and benign mucous membrane pemphigoid (BMMP).

1. Signs and symptoms. These diseases are usually much more benign than pemphigus. BMMP often presents with oral cavity lesions, most commonly affecting the buccal mucosa, gingiva, and palate. The lesion begins as a blister, which then ruptures, leaving a denuded area. The gingiva may be diffusely friable and hemorrhagic.

2. Diagnosis is made by immunofluorescence study of the biopsied lesion. IgG antibodies react with the basement membrane, as opposed to in pemphigus, where the antibodies react at the site of acantholysis.
3. **Therapy.** Systemic steroids are used in these diseases, which respond much better than does pemphigus.

C. **Erythema multiforme** is an acute, self-limited disease of the skin and mucous membranes in response to various etiologies, including drugs (eg, penicillin, sulfa) and infection (eg, herpes, mycoplasma). Over half the cases are idiopathic.

1. **Signs and symptoms.** Oral vesicles and bullae quickly rupture to become erosions that are friable and bleed. Ulcerations covered with a white pseudomembrane are characteristic. Cutaneous manifestations, often involving the palms and soles, consist of the sudden onset of erythematous patches or plaques that erupt symmetrically. They may then develop into characteristic "target lesions." These oral lesions are extremely painful. The lips are frequently covered with a dark, hemorrhagic crust. This symptom complex may be initiated by drug reactions, herpes infections, or by an internal malignancy.

2. **Diagnosis** is made clinically.

3. **Therapy.** Treatment is supportive with hydration and topical anesthetic mouth rinses (dyclonine, diphenhydramine, plus milk of magnesia). Systemic steroids are sometimes used.

D. **Recurrent aphthous stomatitis**

1. **Signs and symptoms.** This disorder is characterized by recurring buccal and labial painful mucosal ulcers in an otherwise healthy patient. These patients usually have between two and six lesions with each episode and may experience several exacerbations per year. The etiology is unclear, but factors such as heredity, hormones, nutrition, stress, and local trauma have been suggested to play a role. An altered immune response remains a postulated etiology. Patients should be queried as to inflammatory bowel disease and diabetes.

2. **Diagnosis.** Physical examination reveals round, symmetric, shallow painful lesions on the buccal and labial mucosa. The hard palate and attached gingiva are spared. Major aphthous ulcers can be as large as 2-3 cm. Healing without scarring within 2 weeks is the rule for smaller lesions. It may take months for larger lesions to heal and scarring is common.

3. **Therapy.** Treatment with topical corticosteroid preparations, eg, triamcinolone acetonide (Kenalog in Orabase, qid), may be palliative. In severe cases, tetracycline mouth rinses (250-mg capsule dissolved in 50 mL of water qid) may be used, but with the risk of oral candidiasis and allergic reactions. For large ulcers, prednisone may be necessary 40 mg daily until healing is complete.

E. **Agranulocytosis.** Agranulocytosis is believed to occur as a rare reaction to a wide variety of drugs (eg, phenytoin (Dilantin), phenylbutazone, chloramphenicol, chlorpromazine, and meprobamate).

1. **Signs and symptoms**

   a. These patients are acutely ill with fever, sore throat, malaise, and prostration.
b. Physical examination reveals an ulcerative, sometimes gangrenous mucositis of the oropharynx, demonstrated by lesions that range from small superficial ulcers to a membranous gingivitis and pharyngitis.

2. Diagnosis. The total leukocyte count is often 1000-2000 cells/microL, and neutrophils are absent from the bone marrow. The differential must include leukemia, Vincent's angina, mononucleosis, diphtheria, or acute tonsillitis on initial examination.

3. Therapy. Immediate discontinuance of the offending drug - supportive measures and antibiotics to control secondary infections sustain the patient until bone marrow recovery occurs.

F. Leukemia. As opposed to agranulocytosis, leukemia is characterized by neoplastic proliferation of one of the blood-forming cells, which then replaces the normal hematopoietic cells in the bone marrow.

Signs and symptoms. Symptoms are usually abrupt and consist of fever, prostration, infection, or bleeding, but may be insidious with progressive weakness, low-grade fever, bleeding tendencies, and recurrent infections. Often the patient presents to the otolaryngologist with fever, sore throat, and gingival bleeding. Findings can include swollen, purplish gingivae, tonsillar enlargement with exudate or necrotic ulcers, and pharyngeal mucous membrane ulcerations. These necrotic lesions must be differentiated from diphtheria, tuberculosis, Vincent's angina, pemphigus, and other bacterial or viral entities.

V. Deep neck space abscess

A. Peritonsillar abscess (quinsy) is a purulent collection between the tonsillar capsule and the fascia of the superior constrictor muscle (ie, the peritonsillar space). Organisms involved are streptococci (anaerobic species) and occasionally staphylococci. It is believed that infection deep in the tonsillar crypts penetrate the capsule (usually in the supratonsillar fossa) to spread into the peritonsillar space. The process may be localized to a collection of minor salivary glands (Weber's glands) in the superior pole of the tonsil. The abscess may occur early or late in the course of an acute tonsillitis and is often seen during or after appropriate antibiotic treatment of the initial tonsillitis. The abscess is usually unilateral and is rare in children.

1. Signs and symptoms

a. Symptoms. The patient often gives a history of a sore throat that initially improved but subsequently worsened, progressing to a unilateral painful throat. This can occur with or without antibiotic treatment. The temperature may be elevated and the patient demonstrates a thickened or "hot potato" voice. Odynophagia, drooling, and trismus (due to irritation of the internal pterygoid muscle) are common.

b. Physical examination reveals unilateral edema and erythema of the soft palate and anterior tonsillar pillar with medical displacement of the tonsil. Examination is often difficult due to trismus. Spontaneous drainage, if present, usually occurs in the superior portion of the anterior pillar area.
2. **Diagnosis.** Differentiation between cellulitis and abscess can be made with needle aspiration of the area lateral to the superior pole or at any point of obvious fluctuation. A large-bore (16- or 18-gauge) spinal needle, after topical or local anesthesia, should be used. The aspirate is sent for Gram stain and anaerobic and aerobic culture and sensitivity.

3. **Therapy**

   a. Needle aspiration is the treatment of choice. If there is a concern regarding potential reaccumulation, incision and drainage under local anesthesia is done immediately after aspiration.

   b. Aspiration is followed by outpatient treatment with oral antibiotics (penicillin 500 mg PO qid, modified by the results of the culture and sensitivity). Hourly irrigations with warm saline, liquid oral analgesics, and hydration are all important.

   c. Hospitalization for IV antibiotics and hydration is indicated for significant trismus, dehydration, inability to swallow, and significant obstruction.

   d. Immediate bilateral tonsillectomy is advocated for the pediatric patient in whom anesthesia would be required for incision and drainage.

   e. There is debate as to whether "interval tonsillectomy," performed 6-8 weeks after initial treatment for the peritonsillar abscess is indicated. In the absence of a prior history of recurrent tonsillitis, there is only a 10% chance of recurrent peritonsillar abscess.

**B. Retropharyngeal abscess** (see Chap. 1, IV.B.) is usually a disease of children less than 3 years of age. This relatively uncommon abscess is due to necrosis and suppuration of the lymph nodes in the retropharyngeal space, which lies between the buccopharyngeal fascia and the prevertebral fascia. The nodes, which drain the nasal passages, eustachian tube, sinuses, and pharynx, usually regress by age 3 or 4 years. This abscess may follow penetrating pharyngeal injuries, neglected middle ear infections, suppurative parotitis, or during active tuberculosis (Pott's disease).

1. **Signs and symptoms**

   a. **Symptoms.** The child lies with the head extended and tilted toward the uninvolved side, refuses food, and demonstrates nuchal rigidity. If old enough, the patient complains of odynophagia or dysphagia but does not have trismus. If the swelling becomes massive, obstructive symptoms of snoring, muffled cry, or dyspnea may be present. Later in the course, respiratory embarrassment occurs.

   b. **Physical examination.** There is cervical adenitis and a soft unilateral red bulging of the posterior pharyngeal wall, visible directly or by mirror examination.

2. **Diagnosis.** Fluctuance to palpation is often present, but caution must be exercised so as not to precipitate rupture with possible aspiration of pus, causing asphyxia or pulmonary infection. The temperature and white count are elevated. An inspiratory hyperextended lateral neck film shows an anterior bulging of the posterior pharyngeal wall. Fluoroscopy is
beneficial and reveals decreased mobility of the posterior pharynx. CT can help define the extent of an abscess. A chest film is indicated, because a retropharyngeal abscess is a likely source of mediastinitis.

3. Therapy

a. If seen early, before suppuration occurs, most cases of retropharyngeal adenitis are successfully treated with parenteral antibiotics that cover anaerobes, staphylococci, streptococci, H. influenzae, and other oral flora. A third-generation cephalosporin (ceftriaxone) should be considered initially.

b. Abscess formation necessitates incision and drainage in the operating room with the patient in the extreme Trendelenburg position (head down) to avoid aspiration of the pus from a ruptured abscess.

C. Pharyngomaxillary space (parapharyngeal) abscess. This space is pyramid shaped, with its base located at the base of the skull and its apex at the hyoid bone. Its lateral boundary is the fascia covering the internal pterygoid muscle, mandible, and deep surface of the parotid gland. Fascia covering the superior constrictor muscle serves as the medial boundary. The styloid process and its muscular attachments divide the space into anterior (the tonsillar fossa medially and internal pterygoid laterally) and posterior spaces (containing the carotid sheath and the ninth through twelfth cranial nerves). Pharyngeal infection is the most common source, but direct extension from infection in other cervical spaces (submaxillary, retropharyngeal, peritonsillar, masticator, parotid) is commonly seen. Bezold's abscess results from infection breaking through the mastoid tip cells medial to the digastric muscle and dissecting inferiorly into the pharyngomaxillary space.

1. Signs and symptoms

a. Fever, odynophagia, sore throat, ipsilateral otalgia and neck rigidity are common.

b. Marked trismus is due to internal pterygoid muscle inflammation. There is lateral neck swelling, especially posterior to the angle of the mandible. The posterior tonsillar pillar is deviated medially.

c. Cranial neuropathies, as well as septic thrombosis of the internal jugular vein or carotid artery rupture, can lead to disastrous complications.

2. Diagnosis. The x-ray findings of posterior pharyngeal swelling assist in the diagnosis; however, a computed tomography (CT) or MRI scan may give information as to the extent of the abscess.

3. Therapy. Treatment includes parenteral antibiotics cover anaerobes, streptococci, and staphylococci (especially if there is a history of trauma, iatrogenic or otherwise). The choice of antibiotics includes penicillin G, clindamycin, or cefoxitin, pending culture and sensitivity results. Surgical drainage may be indicated. High doses of penicillin or cephalosporin should be started and maintained until culture and sensitivity results are available. Nafcilin or oxacillin should be used if Staphylococcus is suspected.
D. **Submandibular space abscess Ludwig's angina** (see Chap. 1, IV.A.). The submandibular space consists of the sublingual space (between the floor of the mouth and mylohyoid muscle) and the submaxillary spaces (below the mylohyoid). Most abscesses (> 80%) are due to dental infections, but they can also be caused by trauma, lingual tonsillitis, or salivary gland disease. The submaxillary space is involved by abscesses of the first, second, or third molars breaking through the mandibular cortex below the mylohyoid line, while the sublingual space is infected with abscesses of the premolars or first molars, whose roots are above the mylohyoid insertion. Often a history of dental work within the past week is elicited.

Ludwig's original description in 1836 is still valid and describes the angina as (1) a rapidly spreading gangrenous cellulitis or phlegmon, which causes the brawny hardness of the tissues; (2) originating near the submandibular gland, but never involving only one space; (3) arising from direct extension rather than by lymphatics; and (4) producing gangrene and serosanguinous, putrid infiltration but little or no frank pus.

1. **Signs and symptoms**

   a. The patient experiences odynophagia, drooling, trismus, and cannot speak or swallow due to tongue involvement. Dyspnea, agitation, and aspiration occur as the swelling increases. Very high fevers are characteristic.

   b. Floor-of-mouth edema forces the tongue upward and posteriorly with sublingual infection, whereas submandibular induration, tenderness, and swelling are evident with submaxillary space involvement. Ludwig's angina is present when both spaces are affected. As with the other neck space infections, a leukocytosis with left shift is present.

2. **Therapy.** Treatment is with surgical decompression, high-dose parenteral antibiotics (see parapharyngeal abscess), control of the airway either by tracheotomy or by nasotracheal intubation, and removal of suspected teeth if panorex evaluation identifies a source. Early control of the airway is of utmost importance, whether or not surgery is performed.

VI. **Allergic edema**

A. **Quincke's edema,** presumed to have an allergic etiology, involves acute uvular and soft palatal edema.

1. **Signs and symptoms.** The patient presents with the sudden onset of a muffled voice and fullness of the throat. Clinical examination reveals a watery palatal and uvular edema, often with the uvula resting on the tongue. One should question the patient for a history of trauma, caustic substance ingestion, known allergies, and be alert for developing laryngeal edema.

2. **Therapy.** Treatment with IM or SQ epinephrine (1:1000 units, 0.3-0.5 mL) and steroids - hydrocortisone sodium-succinate (100-200 mg IV) or dexamethasone (Decadron, 10 mg IV) - is indicated if edema is massive. Antihistamines, such as diphenhydramine (25-50 mg IV), have also been used, although their effects are not as immediate as epinephrine. Steroids are used q6-8h for resistant cases.
B. Angioedema (see Chap. 1, VI). Angioedema is characterized by transient, localized, painless swelling of the subcutaneous tissue or submucosa of various parts of the body, including the skin and mucosa of the oral cavity, pharynx, larynx, and gastrointestinal tract. Episodes may be triggered by exposure to specific allergens, physical stimuli, or emotional stress. Two forms occur: a well-publicized but rare hereditary angioedema (occurring in fewer than 0.5% of cases of chronic urticaria or 2% of all cases of angioedema) and a more common nonhereditary allergic form. Pharyngeal and laryngeal involvement is more common in the hereditary form, where mortality ranges from 5-50% due to glottic edema. Hereditary angioedema is an autosomal dominant genetic disease in which there is a deficiency of the inhibitor of the first component of complement (C1 esterase).

1. Signs and symptoms involve nonpitting swelling without the sharply defined, raised border characteristic of urticaria of the face, extremities, genitalia, lips, tongue, or pharynx. Pain or itching is absent. The edema may cause respiratory obstruction.

2. Diagnosis. Angioedema is suspected in the patient with a history of previous episodes, a positive family history (in the hereditary form), the typical clinical presentation, and the absence of signs of infection. Laboratory confirmation of low or absent levels of C1 esterase inhibitor or low levels of C4 or C2 secures the diagnosis.

3. Therapy. Treatment is prevention, with avoidance of provocative factors. For an acute attack, epinephrine, 0.5 mL of 1:1000 solution IM, and steroids should be administered, with prompt airway control by intubation or tracheotomy if laryngeal involvement is progressing. Distinction between hereditary and nonhereditary forms is important, as long-term use of such drugs as epsilon aminocaproic acid has been used in the familiar disorder. Purified C1 esterase inhibitor, if available, reverses the acute episode. Danazol, a synthetic androgen, or stanazolol, an anabolic steroid, appear to be effective for long-term therapy, bringing the C1 esterase inhibitor into normal range.

VII. Tissue hypertrophy

A. Adenotonsillar hypertrophy. The adenoid is a mass of lymphoid tissue located on the posterosuperior wall of the nasopharynx, which forms part of Waldeyee's ring. This lymphoid tissue undergoes physiologic hypertrophy and hyperplasia in response to upper respiratory infections, usually greatest between 2 and 5 years of age. Infection of the adenoids rarely occurs alone and usually involves an adenotonsillar inflammation with hypertrophy and enlargement as a response. Repeated infections lead to hyperplasia with microscopic demonstration of active germinal centers, inflammatory infiltrates, and scanty fibrosis in surgical specimens.

1. Signs and symptoms

   a. Symptoms are usually obstructive and include mouth breathing, restless sleep, nasal obstruction with snoring, purulent rhinitis, and hyponasal speech.

   b. Nasal airway obstruction may cause altered midface development, with a narrow and arched palate, demonstrable in serial cephalometric radiographs.
c. Eustachian tube obstruction with subsequent serous otitis media, hearing loss, and perhaps recurrent acute otitis media, may occur. Adenoid hypertrophy alone rarely appears to be the sole factor causing otitis media.

2. Diagnosis. Direct examination of the oral cavity shows enlarged tonsils. Lateral radiographs of the nasopharynx reveal narrowing or obliteration of the airway by enlarged adenoid tissue. Obstructive apnea can be demonstrated on a sleep study (see next section).

3. Therapy. Adenotonsillectomy is required and may occasionally be done emergently to relieve airway obstruction. Non-infectious indications for adenotonsillectomy include cor pulmonale, sleep apnea, severe upper airway obstruction, and malocclusion secondary to chronic mouth breathing.

B. Obstructive sleep apnea. Sleep apnea is defined as cessation of airflow at the nares and mouth for at least 10 seconds. Central apnea is the absence of both airflow and respiratory effort; obstructive apnea is the absence of airflow in the presence of respiratory effort; and mixed apnea is a combination of both central and obstructive apneas. Sleep apnea is defined by 5 episodes of apneas and hypopneas per hour of sleep or 30 episodes over 7 hours.

Obstructive sleep apnea syndrome (OSAS) is caused by collapse and obstruction of the upper airway at the level of the oropharynx and/or hypopharynx; often the exact site(s) of obstruction is difficult to pinpoint. Contributing factors may include obesity, adenotonsillar hypertrophy, redundant oropharyngeal soft tissue, macroglossia, micrognathia, pharyngeal neoplasms, and certain medications.

1. Signs and symptoms. There is usually a history of loud snoring and restless sleeping; there may be enuresis and impotence. Daytime symptoms include hypersomnolence, early morning headaches, and difficulty concentrating.

Physical examination should include a Muller maneuver, where the flexible nasopharyngoscope is used to visualize the degree of collapse at first at the level of the soft palate, and then the base of tongue, while the patient is inspiring against a closed moth and nose.

2. Diagnosis

a. A polysomnogram is used to measure the frequency and type of apneic spells, oxygen saturation, stages of sleep, and cardiac arrhythmias.

b. Other tests include cephalometric x rays, CT scan, and/or fluoroscopy to measure the pharyngeal airway; pulmonary function tests, sleep latency tests, and continuous performance measures are also useful.

3. Therapy

a. Medication, such as protriptyline, may be helpful, but side effects limit its usefulness.
b. Weight-control measures are clearly beneficial for some patients.

c. Stimulants such as coffee and colas, as well as alcohol, should be avoided at bedtime.

d. Nasal CPAP (continuous positive airway pressure) is known to be effective for OSAS, but some patients will not tolerate the use of a mask.

e. Uvulopalatopharyngoplasty (UPPP) widens the oropharyngeal airway by excising portions of the soft palate, uvula, tonsils, and posterolateral pharyngeal walls. This alleviates snoring in most patients, but the number of patients where sleep apnea is cured depends in part on the experience of the operating surgeon, and whether the surgery is pre- or post-tonsillectomy. Fifty to ninety percent of patients experience symptomatic improvement. Negative prognostic factors include retro- or micrognathic mandible, a narrow mandibular arch, macroglossia, hypopharyngeal collapse, and weight greater than 125% of ideal body weight.

f. Nasal airway surgery, such as septal reconstruction, turbinate reduction, and polypectomy, can be done at the same time as the UPPP in patients with known nasal obstruction.

g. Mandibular advancement, geniotubercle and anterior hyoid advancement, and hyoid expansion are other surgical options that are not yet as well studied as UPPP.

h. Tracheotomy is indicated for patients with cor pulmonale, serious cardiac nocturnal dysrrhythmias, and chronic alveolar hypoventilation.

VIII. Congenital obstruction

A. Pierre-Robin syndrome. This congenital complex of U-shaped cleft palate, micrognathia, glossoptosis, and associated eye, ear, skeletal, and cardiac defects (esotropia, cataracts, deafness, spina bifida occulta) is present in about 1 of every 30,000 live births. An arrest in development, probably with multiple etiologies, is the basic defect. Neonatal airway obstruction and feeding problems due to the hypoplastic mandible and relative glossoptosis can be remedied by placing the infant in the prone position or by suturing the tongue to the mandibular labial sulcus until the mandible reaches more normal proportions. The use of the McGovern nipple (a baby nipple with a large open tip) to provide an oral airway is of value until more definitive repair is undertaken. The prognosis is good if the patient survives infancy.

B. Thornwaldt's bursa or nasopharyngeal cyst represents a midline persistence of an embryonic communication between the notochord and the roof of the pharynx.

1. Signs and symptoms

a. Occlusion of the ostia by inflammation causes cyst formation. Subsequent infection may cause headache, persistent postnasal discharge, unpleasant taste sensation, nasopharyngeal crusting, and nasal or eustachian tube obstruction.
b. Examination reveals a cystic, perhaps fluctuant, lesion of the posterior wall of the nasopharynx.

2. Therapy. Treatment requires use of antibiotics and drainage, if necessary, with definitive marsupialization and excision of epithelial lining at a later date.

C. Choanal atresia (see Chap. 1, VII.A.). Failure of the buccopharyngeal membrane to become patent during the seventh and eighth weeks of embryonic life results in choanal atresia. It may be unilateral (right side is affected more than the left) or bilateral, bony (90%) or membranous, complete or incomplete. A familial tendency is recognized.

1. Signs and symptoms

a. Symptoms of bilateral nasal obstruction should be noted immediately after birth. Cyclic dyspnea and cyanosis, which disappears with crying and mouth breathing, is classic. Oropharyngeal breathing is an acquired habit, not learned for weeks, and neonates may die unless an oral airway is provided. Affected infants tightly purse their lips, struggle for air, have sternal retractions, and become cyanotic. Unilateral atresia is not ordinarily life-threatening.

b. Less immediate signs are feeding difficulties (cyanosis while sucking), aspiration, viscid nasal discharge, or constant mouth breathing.

c. Unilateral obstruction obviously has less marked symptoms and may not be discovered for years. There is chronic unilateral rhinorrhea and a hyponasal voice. The suggestion of chronic unilateral maxillary sinusitis may lead to the proper diagnosis.

2. Diagnosis should be made in the delivery room by passing a soft catheter through each nares into the nasopharynx to assess choanal patency. Radiologic confirmation can be performed with Lipiodol or other radiopaque substance placed in the nares. A CT scan delineates this skull base anomaly and is the diagnostic procedure of choice.

3. Therapy. Immediate treatment is establishing an oropharyngeal airway. Intubation or tracheotomy is usually not necessary. The McGovern nipple technique (see A.) or variations of it have proved successful over the years. The infant may eventually learn to coordinate breathing and feeding, but modern anesthesia and surgical techniques are now available for definitive surgical repair in the neonatal period, so surgery is no longer delayed. A transnasal or transpalatal approach is used.

IX. Cysts and neoplasms

A. Nasopharyngeal. Nasal obstruction, rhinorrhea (especially unilateral), epistaxis, hyponasal voice, cranial nerve palsy, hearing loss secondary to serous otitis media, facial pain, or a neck mass should alert the physician to the possibility of a nasopharyngeal mass.
1. Benign masses of the nasopharynx

a. Nasopharyngeal angiofibroma (juvenile angiofibroma). This uncommon, highly vascular neoplasm predominantly affects adolescent males and arises from tissues in the vault of the nasopharynx. The etiology and pathogenesis are poorly understood, but an endocrine-related phenomenon is suspected. The tumor is locally destructive and commonly erodes the base of the skull. Intracranial extension can occur.

(1) Signs and symptoms are often not representative of the size of the lesion. Nasal obstruction (90%), epistaxis (80%), palatal or cheek fullness, and exophthalmus may be present.

(2) Physical examination reveals a grayish or red mass in the nasopharynx.

(3) Diagnosis can consistently be made with radiologic techniques; thus, biopsy is not recommended, as significant hemorrhage can result. Plain films demonstrate "bowing" of the posterior wall of the maxillary sinus, and the carotid arteriographic appearance is diagnostic of a vascular tumor. CT scan with enhancement demonstrates a vascular mass lesion. Erosion of the orbital wall, zygomatic arch, and base of skull may also be noted.

(4) Treatment is surgical removal, preceded by embolization of arterial feeders. Irradiation as a primary treatment is rarely indicated.

c. Choanal polyps. These large, soft, gelatinous masses originate from the mucosa of the maxillary sinus and extend through the sinus ostia into the nasal cavity; they then prolapse through the choana into the nasopharynx. Traditional treatment is by excision through a Caldwell-Luc approach to the maxillary antrum and polyp base. Endoscopic removal is sometimes possible by a combined intranasal and anterior wall antrotomy approach.

c. Other benign neoplasms and pseudotumors of the nasopharynx are teratomas (usually seen shortly after birth as obstructive upper airway masses), mixed tumors, adenomas, chondromas, and cysts.

2. Malignant neoplasms of the nasopharynx

Nasopharyngeal malignancies represent approximately 2% of all malignancies in Caucasians and up to 15% in the Southern Chinese population. The male-female ratio is approximately 3:1. No familial tendency has been established, and most patients in the United States are 50-70 years old. High titers of antibody to EBV appear in about 70% of patients.

Eighty-five percent of these malignant neoplasms are epidermoid carcinomas (this includes lymphoepitheliomas or nonkeratinizing epidermoid cancers). Of the remainder, half are lymphomas, with adenocarcinomas, plasmacytomas, melanomas, or sarcomas occasionally seen. Fifty percent of cases present with metastatic cervical nodes as the chief complaint.

a. Signs and symptoms. Nasal symptoms include purulent or bloody rhinorrhea, posterior epistaxis, or nasal obstruction. Obstruction of the eustachian tube by edema or tumor results in a conductive hearing loss secondary to serous otitis media. An adult with unilateral
serous otitis media must be suspected of having a nasopharyngeal cancer. Ophthalmic symptoms may be related to neurologic compromise, with tumor involvement of the fifth and sixth cranial nerves being the most frequent. Ophthalmoplegia, pain, diplopia, or a decreased corneal reflex may occur. Metastasis to the jugular foramen nodes (jugular foramen syndrome) may involve the ninth through twelfth cranial nerves and the sympathetic trunk (causing unilateral Horner syndrome). Due to the rich lymphatic supply and late symptomatology, 60-80% of patients will already have cervical metastasis on admission to the hospital.

b. Examination with direct or indirect nasopharyngoscopy, careful neurologic evaluation, radiologic evaluation of the skull base, with eventual transnasal or transoral biopsy, establishes the diagnosis.

c. The primary treatment is radiation therapy directed to the nasopharynx and both sides of the neck. Repeated radiation may be used for the 30-60% of patients with recurrent disease. Interval neck dissection should be considered in cases with persistent cervical nodes after the completion of radiation. Carefully selected recurrences may be removed via an infratemporal skull base approach. The prognosis is guarded with a 5-year overall survival rate of about 30-35% for all stages of disease.

B. Oro- and hypopharyngeal. Depending on the anatomic location, these masses present with sore throat, a sensation of a lump in the throat, dysphagia, voice changes,odynophagia, otalgia, and possible trismus.

1. Benign lesions

a. Mucous retention cysts arising from minor salivary glands occur in the valleculae and aryepiglottic folds, and may cause respiratory distress in the newborn; however, they are usually seen as incidental findings on indirect laryngeal examination and most often require no therapy.

b. Squamous papillomas are small, pink, mullberrylike masses on the soft palate, uvula, or tonsillar fossae. They are viral in origin and do not portend malignancy. Surgical excision of persistent papillomas is advised. These may be associated with respiratory papillomatosis.

c. Parapharyngeal tumors present as lateral oropharyngeal or palatal masses with few symptoms. Most are benign (pleomorphic adenomas, neurilemmomas, lymphangiomas, teratomas, or leiomyomas) and are approached surgically through an upper lateral neck incision. Large tumors in the region may require an intraoral as well as external approach with a mandibular osteotomy, or parotidectomy.

d. Aberrant thyroid tissue located in the base of the tongue in the region of the foramen cecum or between the epiglottis and circumvallate papillae may result from failure of the thyroid anlage to descend into its normal position in the neck. Females are predominantly affected and may complain of a foreign body sensation, dysarthria, or dysphagia, especially during periods of increased endocrine activity.
A biopsy will differentiate thyroid tissue from other tumors such as amyloid, angiomas, fibromas, papillomas, gummas, lingual tonsil carcinomas, lymphomas, or sarcomas. A thyroid scan must be performed, as this aberrant tissue may be the patient's only thyroid tissue, and in many cases it is hypofunctional. Rarely is therapy necessary although thyroid suppression may be necessary in symptomatic patients.

2. Malignant neoplasms of the oropharynx. Smaller tumors of the tonsil, pharyngeal walls, or base of the tongue are often asymptomatic, and growth is usually advanced by the time the patient seeks medical care.

a. Signs and symptoms. Vague sore throat, foreign body sensation, local irritation with rough or hot food, odynophagia, referred otalgia, and a neck lump are common presenting complaints. Most patients are males, 50-80 years old, and have a history of chronic alcohol or tobacco use. More than 50% will have clinically positive cervical nodes when first examined. About 90% of tonsil cancers are epidermoid carcinomas, with about 10% being lymphomas. Ninety-five percent of base of tongue cancers are epidermoid in type.

b. Physical examination reveals a firm exophytic or ulcerated lesion. Direct visualization and palpation of both the primary tumor and the neck at the time of biopsy are important for proper staging and therapy.

c. Combined therapy with radiation, surgery, and/or chemotherapy can be planned. Endoscopic evaluation of the entire upper aerodigestive tract should be performed because of the high (up to 15%) incidence of multiple separate tumors.

3. Malignant tumors of the hypopharynx include cancers of the pyriform fossa, posterior hypopharyngeal wall, and postcricoid region. Ninety-five percent of pyriform sinus and posterior pharyngeal wall cancers are epidermoid carcinomas. Most are poorly differentiated infiltrating tumors. These neoplasms spread by direct as well as lymphatic extension. More than half of the patients present with palpable cervical nodes, and local disease is often extensive by the time symptoms of dysphagia, voice alteration, otalgia, aspiration, stridor, throat pain, or weight loss prompts the patient to seek medical assistance.

Indirect laryngoscopy demonstrates a large sessile or exophytic superficially ulcerative mass. MRI and CT scans and barium swallows are helpful in delineating the extent of disease, but tumor mapping at the time of direct laryngoscopy and biopsy are of utmost importance in treatment planning. Combined treatment with surgery, irradiation, and chemotherapy is then coordinated.

Postcricoid carcinoma usually presents with dysphagia, intermittent cough secondary to aspiration, a lump-in-the-throat sensation, local pain, excess salivation, and weight loss. Indirect laryngoscopy may reveal only interarytenoid edema and pooling of secretions. Radiologic studies confirm the presence of a mass, and direct endoscopy determines the geography of the disease. The inferior boundary must be defined for the proper application of surgery and/or radiation therapy.

4. Dysphagia (oropharyngeal). Dysphagia is defined as difficulty swallowing. It is distinguished from odynophagia, which is pain on swallowing.
The swallow is divided into the oral, pharyngeal, and oesophageal phases. The oral phase starts when the tongue begins its anterior to posterior motion, which strips the bolus along the hard palate. The oral phase ends when the bolus passes the anterior tonsillar pillars, which begins the pharyngeal phase. The pharyngeal phase ends when the bolus passes through the pharyngoesophageal segment (PES). The esophageal phase then begins; it ends when the bolus passes through the lower esophageal sphincter (LES).

**a. Signs and symptoms.** The patient who has difficulty propelling the bolus through the oropharynx may describe the bolus getting stuck in the throat, often requiring regurgitation to dislodge the bolus. Nasal regurgitation implies palatal dysfunction. Coughing, sometimes associated with aspiration, may lead to pneumonia. Other associated symptoms include a change in voice, weight loss, or diet change. Odynophagia may indicate the presence of either neoplasia or infection.

**b. Physical examination includes assessment of palatal elevation, tongue strength and mobility, vocal cord mobility, pooling of secretions, presence of masses in the neck, naso-, oro-, or hypopharynx.**

**c. Diagnosis**

(1) Oropharyngeal swallowing videofluoroscopy studies the oropharyngeal phases of the swallow by giving the patient small amounts of liquid, paste, and solid barium. Various parameters are studied, including the presence and mechanisms of aspiration. Therapeutic maneuvers such as variations in bolus texture and head position are attempted to facilitate a normal swallow. A swallowing videofluoroscopy should be done for any patients with dysphagia, especially if aspiration is suspected.

(2) A standard barium swallow uses a large bolus of liquid barium. It is helpful for visualizing any structural lesions, esophageal motility, or gastroesophageal reflux. Patients with solid food dysphagia, odynophagia, history of reflux, or persistent dysphagia with a normal swallowing videofluoroscopy should have a standard barium swallow.

(3) Other diagnostic procedures include manometry, swallowing ultrasound, and EMGs.

**d. There are numerous diseases that are associated with dysphagia. These include neurologic (CVA, cerebral palsy, multiple sclerosis, and Parkinson's), musculoskeletal (polymyositis, oculopharyngeal muscular dystrophy), idiopathic (cricopharyngeal achalasia), and structural (neoplasms, oropharyngeal or laryngeal resections). Two diseases will be discussed:**

(1) Hypopharyngeal diverticulum (Zenker's diverticulum). Patients with Zenker's diverticulum are usually males over 40 years of age and complain of longstanding dysphagia, food sticking in the throat, and noisy deglutition. They often must swallow water with their food and give a history of regurgitation of undigested food when supine. Aspiration pneumonia may result. The diverticula is thought to be caused by incoordination of the upper esophageal sphincter, with a congenital or acquired weakness in the muscular hypopharyngeal wall between the inferior constrictor muscle and the cricopharyngeus muscle. A sack forms
from the posterior hypopharyngeal mucosa, entering the prevertebral space and protruding to the left as it enlarges. Physical examination may reveal a soft, compressible, nontender mass deep to or behind the left sternocleidomastoid muscle. Barium swallow with cineradiography should be diagnostic.

Treatment is a diverticulectomy or diverticulopexy combined with a cricopharyngeal myotomy through an external approach. Endoscopic cautery or laser division of the common wall between the diverticulum and the pharyngeal lumen is reserved for poor surgical candidates.

(2) Cricopharyngeal achalasia. The PES, sometimes called the upper esophageal sphincter (UES), is contracted at rest and relaxes during a swallow period. PES dysfunction may result in a failure, incomplete, or delayed relaxation of the sphincter. This causes dysphagia, mostly for solids, which have difficulty passing through the PES. Pure cricopharyngeal achalasia is rare; it is most often seen in conjunction with diffuse pharyngeal phase dysfunction.

e. Treatment

(1) The underlying disease should be treated if possible.

(2) A cricopharyngeal myotomy (cutting the muscles of the PES) is indicated in patients with predominantly PES dysfunction. Oculopharyngeal dystrophy patients may also benefit from a myotomy. Any patient who undergoes a Zenker's diverticulectomy should have a concomitant myotomy.

(3) Swallowing therapy, performed by a trained swallowing therapist, may be quite helpful. This includes teaching the patient various maneuvers which, on video, have demonstrably improved the patient's swallowing dysfunction.

(4) If oral intake results in significant aspiration or inadequate caloric intake, non-oral feeding such as a gastrostomy or jejunostomy tube may be necessary.

Esophagus

I. Congenital

A. Cysts. Esophageal cysts are embryological remnants of uncertain origin, lined by the types of epithelium from which they arise (stratified squamous and ciliated columnar epithelia), with or without a muscle coat. Duplication of the esophagus is less common but may have a similar embryological derivation. Both anomalies rarely connect with the esophagus and may be associated with vertebral anomalies such as spina bifida and Klippel-Feil deformity, as well as intraspinal malformations.

1. Signs and symptoms. These anomalies are usually asymptomatic in adults and are discovered as incidental findings on chest or spine roentgenograms. If symptoms occur, they can include dysphagia, pain, bleeding, choking, and retrosternal discomfort.
2. Diagnosis. Plain films of the esophagus may demonstrate a posterior mediastinal mass. Contrast films of a cyst demonstrate a smoothly rounded extramucosal mass, usually in the middle or lower third of the esophagus. CT scans delineate a cystic mass.

3. Treatment of the symptomatic lesions is transthoracic enucleation, without opening the esophageal lumen.

B. Atresia. Esophageal atresia and tracheoesophageal fistula are believed to result from incomplete formation of the tracheoesophageal septum during the fourth week of gestation. The anomalies are commonly associated with defects of the gastrointestinal, genitourinary, and neurologic systems. Five categories of malformation have been described:

Type C accounts for 85-90% of these anomalies. The distal end of the upper esophagus ends blindly, and the superior end of the lower esophagus attaches into the posterior tracheal wall.

Type A. There is no communication between the upper and lower esophageal segments and no communication with the trachea.

Type B. The distal end of the upper esophageal segment communicates with the trachea, while the proximal end of the lower esophageal segment is blind.

Type D. Both the upper and the lower esophageal segments communicate with the trachea.

Type E, otherwise known as the H-fistula. The esophagus is uninterrupted but communicates with the trachea.

1. Signs and symptoms. Infants in whom the upper esophageal segment ends blindly are unable to swallow even their own secretions, with resultant regurgitation, choking, and aspiration. Infants in whom the upper esophageal segment fistulizes to the trachea frequently experience life-threatening pulmonary complications, particularly if the anomalies are not noted immediately after birth and the infants are fed. H-fistulae may be relatively asymptomatic if the fistula tract is small and may go undetected for many years. Symptoms include recurrent pneumonitis, failure to thrive, and coughing, especially when swallowing liquids.

2. Diagnosis. In all but the H-fistula, a catheter passed through the nose will not enter the stomach. Anteroposterior (AP) and lateral chest and abdominal films with a catheter in position are often sufficient to make the diagnosis. In the type A and B anomalies, no abdominal gas is visible, whereas gas is present in types C, D, and E. If the diagnosis is still in doubt, instillation of a small amount of sodium diatrizoate (Hypaque) under fluoroscopic control may be helpful, though at the risk of additional soiling of the tracheobronchial tree.

3. Treatment is surgical repair of the fistula and atresia as soon as the infant is medically stabilized. Repair usually consists of a one-stage esophageal anastomosis and closure of the fistula. If a primary anastomosis is not feasible, the procedure may be performed in two or more stages.
C. Stenosis and webs. Congenital stenoses and congenital webs are discussed together, since the terms webs, bands, strictures, stenoses, and rings have all been used to refer to similar anomalies. These entities are rare, usually identified in the midesophagus, and may be associated with tracheoesophageal fistula.

1. Signs and symptoms. The degree of dysphagia is a function of the amount of esophageal obstruction. Infants with significant narrowing may be unable to handle their own secretions. With a minimum of narrowing, however, they may have no difficulty swallowing until solid food is added to their diet, at which time intermittent regurgitation of undigested food may develop with or without aspiration.

2. Diagnosis. Contrast studies of the esophagus constitute the most reliable means of demonstrating such anomalies. As webs frequently project from the anterior wall of the esophagus, it is important to obtain oblique or lateral views as well as AP views.

3. Treatment. Symptomatic webs and stenoses usually respond to dilatation. If the narrowing is secondary to the presence of a cartilaginous ring (a tracheobronchial tree remnant), surgical resection with esophageal reanastomosis may be necessary.

II. Motility disorders

A. Achalasia denotes the combination of impaired relaxation of the gastroesophageal sphincter with abnormal esophageal motility. Its etiology is unknown, but it has been associated with a decreased number of ganglion cells in Auerbach's plexus and with a decreased number of cells in the dorsal motor nucleus of the vagus nerve. Chagas's disease, caused by Trypanosoma cruzi, produces changes similar to those seen in achalasia but is also associated with dilatation of the colon, ureters, and other viscera.

1. Signs and symptoms. Patients experience the gradual onset of dysphagia, with the sensation of food sticking in the lower esophagus, or discomfort referred to the area of the suprasternal notch. These symptoms may be precipitated by the intake of cold food or liquid. The patient may find that by making repetitive swallowing efforts or by washing the food down with fluids the discomfort is temporarily relieved. Regurgitation of undigested food, either delayed or immediate, may occur. In advanced achalasia, repeated aspiration of esophageal contents may lead to pneumonitis, lung abscess, and bronchiectasis. Bleeding and pain are uncommon.

2. Diagnosis. Early in the course of achalasia, the distal esophagus may appear tapered on barium swallow, with disorganized contractions. Later, the esophagus dilates and may assume a sigmoid shape. An air-fluid level may be identified, and passage of contrast material into the stomach is slow. Manometry demonstrates a lack of esophageal peristalsis and impaired relaxation of the gastroesophageal sphincter on deglutition, with normal or elevated resting pressures. Esophagoscopy is not necessary to make the diagnosis of achalasia but should be performed in patients whose symptoms are of short duration to rule out a distal esophageal malignancy.
3. Treatment

a. Dilatation. Mechanical dilatation of the gastroesophageal sphincter with bougies provides only transient improvement. However, balloon dilators positioned within the gastroesophageal sphincter and expanded with air or fluid offer 60-70% of patients long-term improvement after only one or two dilatations.

b. Surgery. Surgical therapy consists primarily of modifications of the Heller procedure, in which a transthoracic esophagomyotomy is performed, incising the circular fibers of the gastroesophageal sphincter and leaving the esophageal mucosa intact. These procedures provide long-term improvement in approximately 80% of patients. They may be considered as alternatives to balloon dilatation or employed if dilatation fails.

B. Diffuse esophageal spasm (DES) is a disorder in which the smooth muscle contractions are simultaneous and repetitive.

1. Signs and symptoms. Pain referred to the neck, jaw, arms, or shoulders is the usual symptom. Dysphagia and regurgitation may also be seen.

2. Diagnosis. Contrast cineradiography may reveal diffuse irregular esophageal spasm, diffuse constant narrowing, or pseudodiverticulosis. Hiatal hernias are often seen. Manometry of the lower esophageal sphincter may demonstrate increased tone but complete relaxation on swallowing.

3. Treatment

a. Medical. Medications including sedatives, tranquilizers, anticholinergics, antispasmodics, and H₂ blockers each have a modicum of success. Oral or sublingual nifedipine (10-20 mg before meals) may provide relief of associated chest pain and has efficacy for the subgroup with less pressure elevation.

b. Dilatation. A few patients will benefit from bouginage.

c. Surgery. Surgical therapy consisting of an extended esophagomyotomy and repair of hiatal hernia, if present, may be helpful for severely symptomatic patients.

C. Gastroesophageal reflux (GER) is the result of an incompetent gastroesophageal sphincter. It is commonly associated with a sliding (but not paraesophageal) hiatal hernia; however, not all patients with sliding hiatal hernias have reflux and not all patients with reflux have sliding hiatal hernias. Esophageal damage, the result of the erosive activity of regurgitated gastric contents, may range from diffuse erythema to deep ulceration and transmural fibrosis. Complications include bleeding, stricture formation, shortening of the esophagus, perforation, replacement of the lower esophageal squamous epithelium by columnar epithelium (Barrett's esophagus), and motility disturbances. Patients with Barrett's esophagus have a 10-15% incidence of esophageal carcinoma.

1. Signs and symptoms. The most common symptom is "heart-burn," burning substernal or epigastric distress. Dysphagia is common and is manifested as food "sticking"
during the first few swallows of a meal, perhaps secondary to spastic contraction of the lower esophagus or to spasm of the upper esophageal sphincter. Coughing secondary to regurgitation and aspiration noted with recumbency may also occur.

2. **Diagnosis.** The most sensitive studies for reflux are the acid perfusion, pH monitoring, and acid-clearing tests. Contrast cineradiography is not as accurate for this entity, although reflux of contrast material into the lower esophagus may be observed. Esophagoscopy is used to examine the esophageal mucosa for the pathologic sequelae of reflux.

3. **Treatment**

   a. **Reflux precautions** include elevation of the head of the bed, weight reduction, bland diet, the wearing of loose-fitting abdominal garments, and refraining from heavy lifting, alcohol, coffee, and tobacco. H₂ blockers have made symptom relief possible for most patients.

   b. **Medications** include antacids, cimetidine, ranitidine, and omeprazole.

   c. **Surgery.** Symptoms that are refractory to medical therapy often respond to a fundoplication, which restores the competence of the gastroesophageal sphincter. Strictures usually respond to dilatation with subsequent fundoplication, although severe strictures may require resection with interposition of small bowel or colon.

III. Acquired

A. **Foreign bodies.** Dysphagia secondary to pharyngeal or esophageal obstruction from foreign bodies is usually observed in children less than 10 years old, in the mentally retarded, and in adults over 50 years of age who obstruct with meat or bones. The narrowest part of the gastrointestinal tract is the upper esophageal sphincter and one-half to two-thirds of foreign bodies are found at this level, primarily in children. Mid- or lower esophageal obstruction is more common in adults.

   1. **Signs and symptoms.** Foreign body obstruction produces dysphagia and occasionally pain of sudden onset. Associated coughing or choking is seen if the foreign body is impacted at or just below the laryngeal inlet. The degree of dysphagia correlates with the amount of obstruction.

   2. **Diagnosis.** Plain AP and lateral roentgenograms of the chest and neck may demonstrate the foreign body if it is radiopaque. The mediastinum and prevertebral space should be examined for the presence of air, which would indicate an esophageal perforation. Only in questionable cases, with normal plain films and without significant obstruction, is the use of contrast material indicated. Contrast studies may be performed with barium or with a small piece of cotton soaked with barium.
3. Treatment

a. Emergency. Difficult respiration, aspiration, or the suspicion of a perforation warrant emergency esophagoscopy.

b. Medical. Sedation with narcotics, barbiturates, or diazepam may produce sufficient esophageal relaxation in milder cases to permit passage of the foreign body. The use of papain to digest foreign bodies consisting of meat is not recommended, as necrotic esophageal epithelium may also be digested.

c. If the foreign body does not pass or is unlikely to do so because of its size or shape, removal using a rigid esophagoscope is recommended.

B. Diverticula. Nearly all esophageal diverticula are acquired and seen in adults. Pulsion diverticula are the result of prolonged or excessive intraesophageal pressure that produces mucosal herniation through weak points in the esophageal musculature. The pharyngoesophageal and gastroesophageal sphincter areas are common sites. These diverticula are usually not covered by muscle. Traction diverticula are most often the result of inflammatory mediastinal lymph nodes and differ also from pulsion diverticula by having a muscular coat.

1. Parabronchial (midesophageal) diverticula are rarely symptomatic. Affected individuals infrequently experience dysphagia, odynophagia, or suffer the complication of fistulization to the respiratory tract.

2. Epiphrenic diverticula of the lower esophagus are less common than pharyngoesophageal diverticula and are usually secondary to functional or mechanical obstruction of the gastroesophageal sphincter.

   a. Signs and symptoms. Although epiphrenic diverticula are usually asymptomatic, symptoms may include dysphagia, food retention, and regurgitation. Since these diverticula are often associated with other lower esophageal disorders such as achalasia, diaphragmatic hernia, esophagitis, and stricture, symptoms of these other disorders may predominate.

   b. Diagnosis. Epiphrenic diverticula are most accurately diagnosed with contrast radiography. Other diagnostic tests may be performed if other lower esophageal disorders are found or suspected.

   c. Treatment. The only available therapy is surgical and is reserved for severely symptomatic patients. Transthoracic diverticulectomy is performed with a gastroesophageal myotomy.

IV. Tumors

A. Benign

1. Intramural tumors. The most common benign esophageal tumor is the leiomyoma, which is usually seen in the middle or lower third of the esophagus.
a. **Signs and symptoms.** Leiomyomas are usually asymptomatic until they reach a diameter of approximately 5 cm. Dysphagia, fullness, and retrosternal pressure are then noted.

b. **Diagnosis.** A barium swallow demonstrates a smoothly rounded filling defect that appears to be half in and half out of the esophagus. Less often, the leiomyomas may be large and circumferential. On esophagoscopy, they are generally seen to be covered with normal esophageal epithelium, although ulceration may also be seen.

c. **Treatment.** A thoracotomy is performed and the tumor enucleated, leaving the esophageal epithelium intact.

2. **Pedunculated intraluminal tumors.** Pathologically, such tumors are fibromas, myxomas, and lipomas, and they are usually found in the cervical esophagus.

a. **Signs and symptoms.** Symptoms include dysphagia, aspiration, and regurgitation. Because these tumors are pedunculated, they may prolapse into the hypopharynx and even the larynx, producing dyspnea and choking.

b. **Diagnosis.** Carefully performed contrast studies and esophagoscopy are necessary to detect these tumors. They may easily be overlooked because they are often not bulky and may be covered with normal esophageal epithelium.

c. **Treatment.** Therapy consists of surgical removal, either endoscopically or through a lateral neck incision.

3. **Other benign tumors** include sessile mucosal and submucosal lesions, such as papillomas. Hemangiomas usually present with bleeding, which may be life-threatening, and therefore should be removed.

B. **Malignant.** Dysphagia and odynophagia may result from malignant tumors located in the base of the tongue, hypopharynx (including the pyriform sinuses and postcricoid area), and esophagus. Ninety percent are squamous cell carcinomas, and the remainder, located near the gastroesophageal sphincter, are largely adenocarcinomas. Leiomyosarcomas, melanomas, and verrucous carcinomas have also been reported. Factors implicated in the etiology of these tumors include tobacco, alcohol, nitrosamines, lye burns, achalasia, reflux esophagitis, and Plummer-Vinson syndrome. They are usually seen in the sixth and seventh decades of life.

1. **Signs and symptoms.** The most common symptom is progressive dysphagia. Other symptoms include weight loss, vague retrosternal pressure, and hematemeses.

2. **Diagnosis** is usually made by a combination of contrast radiography and esophagoscopy with biopsy of the mass or cytologic washings if no mass is apparent.

3. **Treatment.** Surgery, and to date, chemotherapy, have not favorably altered survival. Radiotherapy is used either alone or in combination. The overall 5-year survival is a dismal 15-25%, except for squamous cell carcinoma of the lower esophagus, which carries a somewhat more favorable prognosis. Carcinoma of the body of the esophagus may be radiated or resected, and a gastric pull-up or colonic interposition procedure performed. Lower tumors
may require esophagogastricectomy. Similar procedures may be performed for the purpose of palliation. Other palliative procedures include dilatation, gastrostomy, jejunostomy, and insertion of a bypass tube. The Nd-YAG laser has been used endoscopically to establish a serviceable lumen.