Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 4: The Pharynx

Chapter 21: Non-neoplastic Diseases of the Tonsils and Adenoids

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Embryology

Within the first 3 weeks of embryonic life, the developing tissues that become the embryonic disc thicken. The proximal head fold is produced by increased growth of these tissues, whereas surface invagination of ectoderm creates the primitive oral pit or stomodeum. Subsequently, a cephalic portion of the yolk sac becomes enclosed within the developing embryo to form a portion of the foregut. This later produces the pharynx as a tubelike diverticulum of entoderm. This site is separated from the developing stomodeum by the buccopharyngeal membrane, which later ruptures to produce continuity between the primitive oral cavity and the pharynx (Patton, 1969; Hamilton et al, 1972; Arey, 1974; Corliss, 1976).

The primitive face and neck can be seen by the fourth week of development and occurs with the establishment of five ectodermal grooves that lie caudal to the stomodeum and lateral to the pharynx. These grooves are separated by surface elevations that become the branchial or visceral arches. As development proceeds, the arches extend ventrally to merge with the arches of the opposite side. Simultaneously, entodermal pouches at the level of each groove evaginate from the developing pharynx and later contribute tissue to the forming eustachian tube, the parathyroid and thymus glands, and portions of the thyroid gland (Patton, 1969; Arey, 1974; Langman, 1981).

Through growth and lateral expansion of the pharynx, the second pharyngeal pouch is largely absorbed into the pharyngeal wall. However, dorsal remnants of the pouch persist to become the epithelium of the palatine tonsils. The tonsillar pillars are formed from the second and third branchial arches through dorsal extension of mesenchyme into the developing soft palate (Parkinson, 1951).

The actual development of the palatine tonsils can be seen in fetal studies during the 14th gestational week when mesenchyme underlying the mucosa of the forming tonsillar fossa is invaded by mononuclear cells (von Gaudecker and Müller-Hermelink, 1982). These latter cells are formed by lymphoblastic stem cells or pre-bursa-derived cells. Thereafter, mesenchymal condensations differentiate into the tonsillar lymphoid tissue. By the 16th gestational week, the tonsillar primordium can be seen to have a distinct organization that later shows nodular structures (or primary follicles) after the sixth month of development. Invagination of surface epithelium into adjacent connective tissue gives rise to the tonsillar crypts that subsequently

become infiltrated by thymus-derived lymphocytes. As development proceeds, the tonsils enlarge and the epithelial crypts branch, degenerate, and re-form. This process occurs even after birth (Schaeffer, 1953; Arey, 1974). The tonsillar capsule, as well as the internal connective tissue of the tonsil, is apparent by the 20th gestational week and develops from mesenchyme contiguous to the tonsillar tissues. Functional germinal centers may be lacking until birth and then are prevalent thereafter (Sakai, 1983).

The development of the lingual tonsil represents lymphoid infiltration into the base of the tongue and occurs at the same approximate times as does development of the palatine tonsils. This formation may also involve epithelial ingrowth into connective tissue that has already condensed at this site (Arey, 1947, 1974). Peritonsillar mucous glands also form and include excretory ducts around which lymphocytes can be found. These ducts commonly open into saccular crypts, or they may even enter the developing tonsillar lymphatics.

During the fourth to sixth weeks of development, lymphatic tissue also forms within the posterior wall of the nasopharynx to become the pharyngeal tonsil or adenoid (Arey, 1947, 1974). This tissue develops modified crypts that come to represent epithelial folds and may also cover dilated ducts of subjacent mucous glands. Enlargement of this tissue has been suggested to represent tissue responsiveness to the rich blood supply of the nasopharynx as well as possibly being a local manifestation of postnatal infection (Snook, 1934). In any event, the adenoids grow rapidly in infancy and early childhood and then develop more slowly until puberty, after which a gradual decline in size occurs (Handelman and Osborne, 1976).

Anatomy

The lingual, pharyngeal, and palatine tonsils compose the bulk of the so-called Waldeyer's ring of lymphatic tissue that lines the walls of the nasopharynx and oropharynx. This ring is completed by lymphoid tissue located on the lateral pharyngeal wall described as the lateral pharyngeal bands.

The Lingual Tonsil

Nonencapsulated nodular masses of lymphoid tissue located within the base of the tongue form the lingual tonsil. The sulcus terminalis, anterior to the circumvallate papillae, separates the lingual tonsil from the mucosa of the anterior portion of the tongue. Between the lymphoid follicles of the tonsil are tubular pockets or crypts of stratified squamous epithelium into which lymphocytes may penetrate (Blood and Fawcett, 1975). These lymphocytic cells may subsequently degenerate to produce plugs of detritus with desquamated epithelial cells and bacteria. The lingual tonsils are connected with adjacent mucous glands whose ducts open either within the tonsillar crypts or onto their free epithelial surfaces.

The lymphoid follicles of the lingual tonsil vary in number from 30 to 100 and are somewhat irregular in size and form (Schaeffer, 1953). These follicles may be arranged in longitudinal rows, with corresponding folds of mucous membrane. Smaller nodules of lymphoid

tissue may also be present between the primary follicles of the lingual tonsil, but they can be poorly defined.

The arterial blood supply of the lingual tonsils is provided by the lingual branches of the external carotid arteries (Schaeffer, 1953). The venous drainage occurs primarily through the lingual veins to the internal jugular vein. Lymphatic plexuses can be found around the tonsilar follicles, and they drain primarily to the superior deep cervical or jugular nodes. The sensory nerve supply to the lingual tonsils and the base of the tongue is provided by the lingual branch of the glossopharyngeal nerve, although some small contribution may come from the superior laryngeal branch of the vagus nerve.

Pharyngeal Tonsil

The pharyngeal tonsil (or adenoid) is formed by numerous variable folds of lymphoid tissue within the mucous membrane of the roof and posterior wall of the nasopharynx. These folds can also extend into the lateral recesses of Rosenmüller behind each eustachian tube orifice. In addition, *Gerlach's tonsil* is formed by small inclusions of lymphoid tissue within the lip of each eustachian tube orifice (Schaeffer, 1953; Boies, 1954; Hollinshead, 1982; Adams et al, 1989).

The lymphoid accumulations in the nasopharynx can be diffuse or nodular and are covered by stratified squamous epithelium (Bloom and Fawcett, 1975). The cleft-like invaginations of this epithelium resemble crypts but are not homologous to the true crypts of the palatine tonsils. Mucous glands are present beneath the lymphatic tissue of the pharyngeal tonsil and can drain through variously shaped ducts to surface mucosa.

The arteries of the pharyngeal tonsils originate from the pharyngeal branches of the external carotid artery (Schaeffer, 1953). These vessels include the ascending pharyngeal artery and the minor palatine branches of the maxillary artery, as well as palatine branches of the facial artery. Venous drainage passes through the pharyngeal plexuses to the internal jugular vein. Lymphatic drainage occurs first to the retropharyngeal lymph nodes before entering the deep jugular nodes. Sensory innervation is derived from nasopharyngeal branches of the glossopharyngeal and vagus nerves (Schaeffer, 1953; Hollinshead, 1982).

Palatine Tonsil

The palatine (faucial) tonsils can be either flattened or pedunculated ovoid bodies of lymphoid tissue. They are located in the posterolateral oropharynx in fossae that are described separately as the sinus tonsillaris. Although each tonsil may be variable in size, the length of the adult tonsil measures 20 to 25 mm, its width ranges from 15 to 20 mm, and its thickness is about 12 mm. The average weight of the adult tonsil has been found to be 1.5 gm (Schaeffer, 1953).

The lateral or attached surface of the palatine tonsil is covered by a firm fibrous capsule that is loosely continuous with the pharyngobasilar fascia that overlies the superior constrictor

muscle of the pharynx (Schaeffer, 1953; Hollinshead, 1982). This capsule is in intimate contact with the deep surface of the tonsil and extends into the parenchyma of the tonsil to contribute to connective tissue septae. These septae conduct the tonsillary nerves and vessels. The mucosa of the medial or free surface of the tonsil is composed of stratified squamous epithelium that is variably pitted with the openings of the tonsillar crypts or fossulae.

The palatine tonsil is bounded posteromedially by the palatopharyngeus muscle, or posterior pillar, which originates from the caudal plexus and inserts on the aponeurosis of the soft palate. The palatoglossus muscle, or anterior pillar, lies anterolaterally and originates from the inferior surface of the tongue to also insert on the palatal aponeurosis, forming an arch with the palatopharyngeus muscle over the tonsil.

Variable folds or plicae are formed by the mucosa overlying the tonsil between each tonsillar pillar. The plica triangularis is a variable fold that lies dorsal to the glossopalatine arch between the tongue and the inferior palatoglossus muscle. This position means that the anterior tonsillar fossa is a potential recess between the plica and the tonsil and is best defined when the overlying mucosa is not adherent to the medial surface of the tonsil. A similar mucosal fold at the posterior inferior border of the tonsil may overlie the variable posterior tonsillar fossa. The plica semilunaris is a third fold of mucosa and drapes the apex of the tonsil between the soft palate and the anterior and posterior tonsillar pillars. Beneath this folds lies the supratonsillar fossa, which is also variable in size and shape.

Each palatine tonsil is composed of connective tissue as well as lymphoid tissue and contains lymphoid cells in various stages of development (Bloom and Fawcett, 1975). The tonsillar crypts are unique and represent tubular invaginations of surface epithelium within the substance of each tonsil. Although the crypts may be irregular in shape, they usually extend through the depth of the tonsillar parenchyma to be surrounded by lymphoid nodules. Exfoliated epithelium as well as keratin debris and foreign particles are frequently present within these crypts. Mucous glands are located peripheral to the tonsil but also drain within the tonsil and are similar to those glands associated with the lingual and pharyngeal tonsils.

The palatine tonsils have a rich blood supply that originates from branches of the external carotid artery. The main trunk of this latter vessel is usually lateral to the tonsil, whereas the internal carotid artery lies approximately 2 cm posterolateral to it (Schaeffer, 1953). The major vessels to the tonsil include the ascending pharyngeal and ascending palatine arteries, which lie deep to the tonsillar fossae. Other vessels include the anterior tonsillar branches of the dorsal lingual artery, the inferior tonsillar branches of the facial artery, and the superior tonsillar branches of the descending palatine artery. Venous drainage occurs through a pericapsular plexus of veins into the lingual or pharyngeal veins and then passes into the internal jugular vein (Hollinshead, 1982).

There are no afferent lymphatics to the tonsil (Parkinson, 1951). Efferent lymphatics are formed from peritonsillar plexuses and are filled by the peritonsillar musculature, which forces lymphoid cells from the tonsillar crypts into the lymphatic ducts. These ducts then pass to the

superior deep cervical nodes as well as to the deep jugular nodes. Alternate lymphatic pathways to the submandibular and superficial cervical nodes also exist (Schaeffer, 1953). More recently, Nowara and Pabst (1986) have shown experimentally that peripherally labeled lymphocytes can pass from one lymphoid organ to another, with a small number of these cells passing to the tonsil. However, these findings may represent only hematogenous dissemination of the cells rather than true passage through an afferent lymphatic system to the tonsil.

The palatine tonsil receives sensory innervation from the lesser palatine nerves, which transmit afferent fibers to the sphenopalatine ganglion. Additional sensory fibers are formed by tonsillar branches of the glossopharyngeal nerve (Schaeffer, 1953). A nervous plexus around the tonsil has been described as the circulus tonsillaris, but it may in actuality represent glossopharyngeal nerve fibers immediately posterior to the tonsillar fossa.

Immunology

The role of the pharyngeal lymphoid tissue in host immune defenses has yet to be defined, although lymphocyte production is an established function. The most predominant of these lymphocytes is the B-lymphocyte (or bursa-equivalent lymphocyte), which can subsequently produce immune globulins for participation in antibody-mediated host reactions. The T-lymphocytes (or thymus-derived cells) are also present, and these cells are responsible for cell-mediated cytotoxicity (Lawlor and Fischer, 1981).

Empiric evidence suggests that the tonsils play an important role during early life in providing host resistance to infection as a result of the inspired air stream eddying around the pharyngeal tissues before passing to the lower airway (Ogden, 1961). Host resistance may also be reflected by the temporary enlargement of the tonsils and adenoids during adolescence. Pathogenic bacteria have been found within the tonsils without having caused disease, implying that these organisms may somehow be made less virulent and can then act as possible antigens to provoke antibody formation (Bloom and Fawcett, 1975). It has also been suggested that regional lymph nodes may play a similar role, with infection developing only after there has been an overaccumulation of debris and microorganisms (Boies, 1954; Hill, 1960; Bloom and Fawcett, 1975; Adams et al, 1989).

The actual part played by the tonsils and adenoids in immunology remains controversial (Ogden, 1961; Mawson et al, 1968; Feinstein and Levitt, 1970). Certain immune-deficient states, such as the Bruton agammaglobulinemia, seem to show that these tissues have some role in immune balance, since the tonsils and other lymphoid tissues are poorly developed in affected persons in contrast to the same tissues in normal individuals (Alexander and Good, 1970). Conversely, enlargements of the tonsils and pharyngeal lymphoid tissues have been found in some patients with pre- or acquired immune deficiency syndrome (Gomperts et al, 1985). Such persons have defective T4/Leu-3-positive T-lymphocytes that are centrally responsive for regulating the immune response to viral, fungal, and possibly tumor-related antigens (Lotze, 1985).

Most clinical studies have failed to demonstrate significant alterations of immune globulin in patients with recurrent tonsillar infections or have not shown any objective changes in the incidence of systemic infections in post-tonsillectomy patients (Chamovitz et al, 1960; Stewart and Lumsden, 1961; Ritter, 1967; Bläker, 1975; Gastpar, 1984). Ogra (1971)) did find altered gamma-A poliovirus antibodies in children following adenotonsillectomy, and Veltri and colleagues (1972) reported the lowering of serum IgG from abnormally high levels to relatively normal ones following tonsillar surgery. Lal and associates (1984) have also found high levels of immunoglobulins to be lowered by surgery. However, the significance of these findings still remains to be defined.

Tissue studies have shown that the tonsillar lymphocytes can express surface or secretory immunoglobulins (such as IgA), which can provide local tissue resistance to pathogenic organisms (Ogra, 1969; Tourville et al, 1969). These lymphocytes can be stimulated in vivo by antigen recognition and T-cell help so that they will further differentiate into immunoglobulin-producing plasma cells (Mudde et al, 1984). Additionally, natural killer cells have been found in tonsillar tissues and are specialized subpopulations of lymphoid cells that may play an important role in host resistance (von Gaudecker et al, 1984).

Both tonsillar tissues and adenoids have been found to produce immunoglobulin E, which is believed to mediate reaginic hypersensitivity reactions (Ishizaka et al, 1969). Other substances isolated from human tonsillar lymphocytes have included interferon, lymphotoxin, and prostaglandins (Klimpel et al, 1975; Marchenko et al, 1976; Morimoto et al, 1985). Lymphokines have also been found in tonsillar tissues, and they may inhibit nucleotide incorporation in lymphocytes to contribute to their regulation (Szabó et al, 1984). It has also been suggested that these latter substances may be activated by constant exposure to bacterial antigens or mitogens (Ennas et al, 1984). Such findings may explain in part the immunologic role of the pharyngeal lymphatic tissues during the early years of growth and development. However, as has been stressed in the past, the true relationship of the tonsils and adenoids to immune factors is *still* not clearly understood.

Microbiology

Bacteriologic cultures of the throat made within 12 hours after birth are usually sterile, with organisms beginning to appear following the beginning of oral feedings (Boies, 1954; Adams et al, 1989). The earliest organisms to be isolated from the throat tend to correspond to those recovered from the mother's skin and may include staphylococci. Nonhemolytic streptococci can be found in the throat thereafter, as can gram-negative cocci and diphtheroids. Hemolytic streptococci are not commonly found in the throats of healthy infants but may be cultured as part of the normal oral flora of healthy adults.

The normal adult flora contains both gram-positive and gram-negative organisms, as well as anaerobic bacteria. Gram-positive bacteria usually predominate and include lactobacilli, filamentous forms such as *Actinomyces* and *Leptothrix,* micrococci, *Neisseria,* diphtheroids, *Bacteroides,* and nonsporulating bacteria (LeJeune and Lewis, 1950; Burrows, 1973; Jokinen et

al, 1976). These latter organisms may be beta-lactamase producing and function synergistically in disease states (Tunér and Nord, 1983; Brook, 1983). Pathogenic bacteria have been found in the normal oral cavities of children up to 5 years of age without causing disease, with their frequency decreasing as age increases (Ingvarsson et al, 1982). The reason for this is unclear.

Other organisms found in the oral cavity include pleuropneumonialike forms and saprophytic fungi (Burrows, 1973). Spirochetes can also occur but not without the presence of teeth, and they require the presence of gingival crevices for their survival (Socransky and Maniganiello, 1971). As previously noted a viral flora is present in the oral cavity and can include adenoviruses, myxoviruses, picornaviruses, and coronaviruses (Enders, 1949; Gwaltney, 1971). Not all of these organisms can be readily cultured so that a culture performed by any specific method may yield a distorted analysis of the actual flora present. In addition, the oral flora can be altered by diet or its deficiencies as well as by other factors. Antibiotics usually produce a coliform flora, whereas disease states may result in a predominating flora that contributes to the existing disease (Miller and Bohnhoff, 1949).

Some evidence exists in the literature for a definite association between viral respiratory tract infections and subsequent bacterial colonizations (Selinger et al, 1981; Ramirez-Ronda et al, 1981). This occurrence may represent the consequence of local defects in cellular immunity, macrophage dysfunction, or altered mucociliary transport to effect clearance of microorganisms from the pharynx. Actual viral infection of affected epithelial cells may then render these cells more susceptible to bacterial adherence and allow secondary infections to occur.

Non-Neoplastic Diseases

Palatine Tonsils

Acute Follicular (Lacunar) Tonsillitis

Acute tonsillitis is usually due to a self-limiting infection of either or both tonsils. Its severity can vary, depending on the virulence of the infecting organisms and the resistance of the patient. The most prevalent bacterial organisms to be cultured from affected patients are beta-hemolytic streptococci, although staphylococci, pneumococci, and *Hemophilus* organisms can also be found, as can various anaerobic bacteria (Lotter and Allen, 1972; Zelenka et al, 1974; Sugita and Ichikawa, 1975; Brook, 1983; Ruuskanen et al, 1984). In addition, viral pathogens are quite common and can include influenza, parainfluenza, herpes simplex, coxsackievirus, echovirus, rhinovirus, and respiratory syncytial virus. Douglas and co-workers (1984) have found that in preschool children, viral causes of tonsillitis are more likely, whereas in older children, various bacterial causes of tonsil infection tend to be more common.

Acute tonsillitis usually occurs clinically in adolescents and young adults and is less common in other age groups. Certain conditions predispose patients to tonsillar infections and include fatigue, exposure to extremes of temperature, pre-existing upper respiratory infections, as well as known metabolic or immune disorders.

The onset of acute tonsillitis can be quite sudden and may be associated with chills and fever. Sore throat soon follows, and difficulty in swallowing occurs when inflammation involves the pharyngeal muscles. Patients may also complain of systemic problems such as headache, malaise, and joint pains. These symptoms commonly persist for 4 to 6 days and then gradually resolve unless complications occur.

Examination of the throat during the acute phase of tonsillitis usually shows enlarged injected tonsils that may be covered to varying degrees with patches of a whitish exudate. This exudate is usually limited to the tonsillar fossae, particularly over the openings of the tonsillar crypts, and is soft and friable. Although parenchymal ulcerations can occur, this tonsillar exudate usually is not adherent to underlying tissues. Such exudates are described as being *follicular* when multiple small patches are found and *membranous* or *pseudomembranous* when coalescence occurs.

In addition to tonsillar inflammation or infection, pharyngitis may also occur, involving all lymphatic tissues in the throat. The tongue can be coated, and thick tenacious mucus may be present within the oral cavity. Tender cervical adenopathy is also common.

Laboratory evaluations usually indicate the occurrence of an infection, so that leukocytosis is invariably found in peripheral blood smears. In the more acute processes, target cells may appear. Gram stains are useful for the diagnosis of bacterial tonsillitis. When viral tonsillitis is suspected, specific agglutination studies can be done, although such studies usually are not warranted and are not cost-effective. Bacterial cultures are most helpful for the diagnosis of infections remaining refractory to early empiric therapy.

Although acute tonsillitis is usually self-limited, persistent infection may be complicated by peritonsillar abscesses, infections deep in the neck, and septicemia. Inflammation with peritonsillar edema may cause airway obstruction. Moreover, the tonsils may also seed infection to the brain, heart, and lungs, as well as to other distant sites.

Treatment of acute tonsillitis involves the elimination of infection and the provision of adequate supportive care. Sufficient fluid intake and rest, as well as warmth and proper hygiene, are important. Lavages with diluted (3 per cent) hydrogen peroxide or warm saline solutions are useful in maintaining adequate oral hygiene. Analgesics should be given as needed. In most persons, aspirin or acetaminophen preparations are sufficient, whereas stronger medications may be required in some patients. Certain topical analgesics contain cetylpyridinium or sodium phenolate and may themselves be irritating to the pharyngeal mucosa. One exception is a dilute solution of diphenhydramine (12.5 mg/5 mL), which can be gargled and swallowed.

Systemic antibiotics are necessary for the treatment of bacterial tonsillitis, although care must be taken not to use medications to which the patient may be allergic. Usually, penicillin, erythromycin, or tetracycline is sufficient as an antibiotic, although tetracycline should *not* be used in children because of the dental staining produced in teeth that have not yet erupted. The actual dosage of the antibiotic used is dependent on the age and weight of the patient being

treated. However, specific bacterial cultures should be analyzed for antibiotic sensitivities in those patients refractory to therapy *before* any medications are adjusted. Moreover, if antibiotics are used, a full course of therapy should be given for periods of 7 to 10 days, since lesser treatment may produce antibiotic-resistant bacteria (Hill, 1960; Simpson et al, 1967; Burrows, 1973). Hospital management usually is not necessary unless the patient becomes dehydrated and proper home care is not available.

Differential Diagnosis. Other disease states may mimic acute tonsillitis, making a careful differential diagnosis of other diseases extremely important for successful therapy.

In the past, *diphtheria* has been mistaken for uncomplicated bacterial tonsillitis, with lethal sequelae. However, the onset of diphtheritic infections may be more gradual, with less pronounced systemic infections. Although cervical adenopathy may not be prominent, hoarseness, stridor, and croupy coughing frequently occur. The diphtheritic membrane is a firm, leathery gray tissue that is firmly adherent to the tonsils and pharyngeal mucosa and is pathognomonic for infection. Attempts at removal of this membrane will produce bleeding from the underlying tissues. Airway obstruction by the membrane is possible, and the organism can produce an exotoxin that is both neurotoxic and cardiotoxic. The actual diagnosis of infection is made by the demonstration of the Klebs-Löffler bacillus by gram stain or culture.

Scarlet fever may result in a thick membranous tonsillitis as well as marked erythema of the oropharyngeal mucosa. A characteristic "strawberry" tongue with prominent lingual papillae may occur, as may a diffuse erythematous papular skin rash. Diagnosis is made by the demonstration of beta-hemolytic streptococci in throat cultures and by immune testing, which may include either Dick's test or the Schültz-Charlton blanching phenomenon, in which convalescent serum causes the skin rash to fade (Burrows, 1973; Vaughan and McKay, 1975; Behrman and Vaughan, 1987).

Vincent's angina, or trench mouth, results in an ulcerative gingivitis and stomatitis with pharyngitis; it is caused by simultaneous infection of the oral cavity with a spirochete and a *Bacteroides* organism *(Fusobacterium fusiforme)* and is invariably found in the presence of poor dental and oral hygiene. A gray necrotic pseudomembrane can form to cover either tonsil or the pharyngeal mucosa. This pseudomembrane results from the necrosis of surface mucosa and contains the infecting organisms. Slough of the membrane from underlying tissues will produce bleeding. Diagnosis is based on clinical examination, which will reveal the characteristic oral ulcerations, and identification of the causative organisms through methylene blue smears or appropriate cultures.

Oral manifestations of *infectious mononucleosis* are relatively common and may vary from mild inflammations to frank ulcerations. An irregular white membrane can cover either tonsil to resemble a membranous tonsillitis. However, tonsillar inflammation may also result from coincidental infections with anaerobic bacteria. Diagnosis is usually based on other clinical manifestations of the disease. Posterior cervical adenopathy is common, as is generalized lymphadenopathy. Splenomegaly is also common. Peripheral blood smears are characteristic and

will show a lymphocytosis with large immature mononuclear cells. Agglutination test results, such as the Paul-Bunnel or Wampole test, are positive.

Other disease states that may produce membranous or pseudomembranous tonsillitis include agranulocytosis, leukoplakia, and pemphigus, as well as leukemias and epithelial malignancies. Lesions may be characteristic, or the precipitating disease process can be defined by other clinical findings as well as by microscopic evaluations of tissue biopsy specimens. Fungi, syphilitic gummata, and tuberculosis can also produce tonsillar lesions, and these entities will be considered further in relation to chronic infections of the tonsils.

Chronic Tonsillitis

Chronic tonsillitis represents persistent inflammation of the tonsils as a result of recurrent acute or subclinical infections. Tonsillar enlargement may occur through parenchymal hyperplasias or by fibrinoid degeneration with obstruction of the tonsillar crypts, although chronic scarification may also produce relatively small tonsils. Chronic tonsillitis is commonly a disease of adults, but it can occur at any age.

The pathogenic organisms causing chronic tonsillitis are similar to those involved in acute infections of the tonsils and are most commonly caused by gram-positive bacteria. However, other bacterial or viral pathogens can be isolated from infected tissues and are important for the administration of appropriate therapy (Lotter and Allen, 1972; Caers and Hensgens, 1974; Zelenka et al, 1974; Sugita and Ichikawa, 1975).

Patients with recurrent tonsillitis usually complain about recurrent sore throats that are only partially relieved by supportive measures. There may be associated febrile episodes or systemic complaints such as malaise and joint pain. Cervical adenopathy is relatively common and can be more pronounced during active periods of infection. Halitosis results from debris obstructing the tonsillar crypts, which then become secondarily infected. In addition, there may be an increased incidence of upper respiratory infections (with involvement of the ears or sinuses) as well as recurrent systemic infections.

Examination of the throat will show tonsils of various sizes, in which caseous debris or purulent material may obstruct the tonsillar crypts. The tonsillar pillars may show signs of chronic inflammation or scarring. However, if unilateral inflammation or enlargement of either tonsil is present, possible malignant change should be suspected, and an adequate biopsy should be done.

Treatment of chronic tonsillitis is generally symptomatic but does include those measures used in the management of acute inflammations, such as rest, fluids, analgesics, and antibiotics when indicated. Definitive therapy involves tonsillectomy, particularly since affected tonsils may become a chronic nidus for infection or their blood flow may be so reduced that any medical therapy given may be ineffective (Özdemir et al, 1985).

Differential Diagnosis. Subacute or chronic forms of those diseases that may be mistaken for acute tonsillitis must also be considered in the differential diagnosis of chronic tonsillitis. These entities include diseases that can produce a pseudomembrane over the tonsils, such as Vincent's angina, infectious mononucleosis, and the like. However, granulomatous diseases of the pharynx are more likely to be confused with chronic bacterial tonsillitis and include tuberculosis, syphilis, pathogenic mycoses, and collagen diseases (Shambaugh, 1945; Parkinson, 1951; Simpson et al, 1967; Beeson and McDermott, 1975).

At the present time, pharyngeal *tuberculosis* is invariably secondary to active pulmonary disease, although infected milk can also cause primary pharyngeal infection (and scrofula). In this disease, the pharyngeal mucosa will be found to have irregular, shallow ulcerations. They contain pale granulations from which the acid-fast mycobacterium can be smeared or cultured. Involvement of the pharynx by *lupus erythematosus* is usually associated with facial or systemic disease. Superficial ulcerations that heal by scar formation occur in the throat, although recurrences are possible. Pharyngeal *gummas* tend to form as painless swellings, which later ulcerate before healing takes place by scarification. Diagnostic sequelae are persistent perforations of the soft palate or tonsillar pillars. *Leprosy* will produce pharyngeal nodules or ulcerations that subsequently heal with extensive tissue loss and scarring. *Actinomycosis* results in the formation of firm, painless mucosal swellings that can later ulcerate and suppurate. The organism can then be found in the discharge as characteristic sulfur granules. Among the mycoses causing possible pharyngeal infections, *blastomycosis* can produce irregular superficial ulcerations with soft granulating bases, whereas coccidioidal granulomas result in diffuse granular thickening of the soft palate and tonsillar pillars with superficial epithelial erosions.

An unusual cause of chronic tonsillitis is *leishmaniasis,* with the protozoan found in the cytoplasm of infected histiocytes (Laudadio, 1984). *Crohn's disease* has also been found clinically as tonsillitis, with tonsils showing aphthouslike ulcerations and granulomas (Mones and Merrell, 1984). However, such cases almost invariably occur in patients with systemic manifestations of disease.

The common complaints occurring in patients with granulomatous pharyngitis or tonsillitis are chronic sore throats and dysphagias that vary in severity. Diagnosis is based on serologic studies, tissue smears or cultures, radiographs, and biopsies. However, malignant disease of the tonsil cannot be overemphasized in the differential diagnosis of tonsillar inflammations. Diagnosis is based on tissue biopsy, which is repeated as needed for a definitive diagnosis.

Peritonsillar Abscess (Quinsy)

The peritonsillar abscess is a localized accumulation of pus within the peritonsillar tissues, which usually results from suppurative infection of the tonsils. This infection penetrates the tonsillar capsule (usually at a superior location) and extends into the connective tissue space between the capsule and the posterior wall of the tonsillar fossa. It can then remain localized or can dissect through the constrictor muscle into the adjacent retropharyngeal space (Hill, 1960; Simpson et al, 1967; Ryan et al, 1970).

Any of the microorganisms causing acute or chronic tonsillitis may be the causative organism of a peritonsillar abscess, although some evidence has now accumulated to indicate that anaerobic bacteria are a frequent cause of these infections (Brook, 1983).

Clinically, patients with peritonsillar abscesses commonly have had persistent sore throats that progressively worsen in spite of seemingly adequate treatment. Localization of pain then occurs, although bilateral abscesses may infrequently develop. The patient is invariably quite sick. Fever is present and ranges from 102 to 105°F. Chills, malaise, and nausea occur. Dysphagia can become a prominent symptom, and the patient may not be able to eat or even swallow his or her own secretions. Thus, salivation or drooling occurs, and the breath becomes rancid. Trismus results from inflammation of the pterygoid muscles. Speech becomes difficult, and the voice assumes a characteristic nasal or thickened "hot potato" quality when patients attempt to minimize pain on opening the mouth. Dehydration is a common clinical occurrence, as is tender cervical adenopathy.

Adequate examination of the throat can be difficult in the presence of trismus and may require the judicious use of an oral topical anesthetic solution (such as lidocaine or benzocaine). When a true abscess is present, there is usually marked injection and edema of the involved peritonsillar tissues, with bulging of the affected tonsillar pillars or soft palate. The tonsil itself may be obscured by adjacent tissue edema or may be covered with mucopus. Swelling of the uvula results in its displacement to the unaffected side. Although most abscesses have a superior location behind the tonsil, inferior extension of pus can produce supraglottic edema and airway obstruction.

Initial treatment of a peritonsillar abscess requires attentive nursing care, adequate hydration, and parenteral antibiotics. Penicillin in high doses is usually the drug of choice unless contraindicated by allergy or the possible occurrence of coagulase-positive organisms. Tetracycline is an excellent alternative antibiotics for adults, although clindamycin is now considered to be the antibiotic of choice in managing beta-lactamase-producing bacteria (Tunér and Nord, 1983; Brook, 1983). However, cultures and sensitivity analyses should be obtained as soon as feasible so that refractory infections can be treated appropriately. Supportive measures include attention to oral hygiene (using peroxide or saline lavages), rest warmth, analgesics, and antipyretics.

Medical measures alone may abort an early peritonsillar abscess or peritonsillar cellulitis. Herzon (1984) has indicated that a number of localized abscesses can be adequately treated by needle aspirations in conjunction with appropriate antibiotic therapy. However, some type of surgical evacuation is usually required once loculated pus forms, although spontaneous rupture of abscesses has occasionally occurred into the oral cavity through the tonsil or the tonsillar pillars (Boies, 1954; Stewart and Lumsden, 1961).

Technique of Incision and Drainage. Topical anesthesia for drainage of a peritonsillar abscess may be preferred, since injectable solutions can result in additional contamination of uninvolved soft tissues. Topical 4 to 5 per cent cocaine or 2 per cent tetracaine can be placed

against the involved tonsillar tissues, using cotton pledges affixed to Jackson-type forceps. Topical anesthetic solution can also be placed intranasally to the sphenopalatine ganglion for supplemental anesthesia. Increment dosages of an intravenous analgesic (diazepam, meperidine, morphine, and so on) can also be needed. However, in the case of small children or uncooperative patients, general endotracheal anesthesia is best.

The preferred placement of the awake patient is in a sitting or partially reclining position, with the head supported against a secure headrest. When general anesthesia is required, the head-down or Trendelenburg position should be employed so that any pus drained can be readily removed by suction. In addition, the airway should be protected with an endotracheal tube. Adequate illumination is imperative and can be provided by either a head mirror or a Lempert lamp.

The primary objective of the procedure is the rapid localization and adequate drainage of the abscess. Its location can usually be defied by bulging of the tonsillar pillars or by palpation of the site of fluctuation. A Luer-Lok type of syringe is useful for the careful administration of local anesthesia (when indicated), but it can also be used for the aspiration of pus from the presumed abscess bed. A No. 11 scalpel blade with adhesive tape guarding all but the tip is best used to carefully incise the mucosa overlying the abscess. An angulated closed forceps or hemostat can then be insinuated into the abscess cavity and opened, which usually provides a sufficient tract for drainage. A supratonsillar approach, if feasible, has the advantage of sparing the tonsillar pillars from later scarring and should provide an adequate approach to posterior lesions. Under most circumstances, drainage can be quickly established through relatively small incisions so that recovery should take place within 4 to 5 days if adjunctive medical therapy is appropriate.

On occasion, peritonsillar cellulitis has been mistaken for a peritonsillar abscess. In such cases, attempts at tissue drainage will be nonproductive. Similar results may also occur when attempts are made to drain early peritonsillar abscesses. Under these circumstances, drainage procedures should be terminated and medical management alone employed. With proper therapy, favorable resolution should occur within several days.

There is now a sizable literature available documenting the value of abscess tonsillectomy, or tonsillectomy *à chaud,* particularly since antibiotic levels may be insufficient to control infection in such tonsils (Maisel, 1982). These tonsillectomies can be quite useful in draining an abscess or in those patients with recurrent tonsillitis or recurrent abscesses (Bonding, 1973; Joshi et al, 1974; Christensen et al, 1983; Holt and Tinsley, 1981). However, patients *must* be carefully selected, since severe toxicity may be present when they are seen initially, and they may do poorly if a general anesthetic is required and no attempt is made to begin parenteral fluids and antibiotics before surgery.

Although there are no extensive prospective studies available to define the true incidence of recurrent tonsillitis or peritonsillar abscesses after an initial abscess has occurred, many clinicians throughout the world *still* believe that definitive tonsillectomy is justified, particularly

in patients with histories of recurrent tonsillitis. In these cases, surgery should be done as soon as all local inflammatory changes have resolved. In the literature, such surgery done 3 to 4 days after drainage of an abscess is known as tonsillectomy *à tiede* and is *à froid* when done 4 to 6 weeks after the incision and drainage (Harma et al, 1979).

Complications. If not adequately treated, peritonsillar abscesses can seed infections to both regional and distant sites (Hill, 1960; Steward and Lumsden, 1961; Ballantyne and Groves, 1979). Local venous thrombosis or phlebitis may occur, and endocarditis, nephritis, peritonitis, and brain abscess have been found. Supraglottic edema may occur from extension of infection, necessitating an emergency tracheotomy. Involvement of the pharyngomaxillary space may require external drainage through the submandibular triangle. Necrotizing fascitis has occurred as a fatal complication (Wenig et al, 1984). In surviving patients, further extension of infection may produce perichondritis of the thyroid cartilage. Aspiration of blood or pus has produced pneumonitis or pulmonary abscess. Equally dramatic has been spontaneous hemorrhage from any of the carotid or jugular vessels through vessel erosions. Temporizing measures require immediate pressure packs and fluid replacement. Surgical exploration is necessary and major vessel ligations may be required.

Intratonsillar Abscess (Phlegmonous Tonsillitis)

A tonsillar abscess is a relatively uncommon occurrence and represents a localized accumulation of pus within the tonsil per se (Simpson et al, 1967; Ballenger, 1987). Such abscesses may result from acute follicular tonsillitis with obstruction of crypts or from spontaneous rupture of a peritonsillar abscess into the tonsil.

The signs and symptoms of a tonsillar abscess are similar to those of peritonsillar lesions. Treatment should be medically directed following drainage of the abscess. If warranted, a definitive tonsillectomy can be performed at a later date when adjacent inflammation has resolved.

Tonsillar Hypertrophy

As previously discussed, tonsillar hypertrophy begins in early childhood and continues until puberty, after which atrophic changes may occur (Hill, 1960; Stewart and Lumsden, 1961). The exact cause of this change is unknown, but it may represent a composite of varied factors involving diet, genetics, and humoral changes. The tonsils can also enlarge following repeated episodes of local or systemic infections, especially those associated with bacterial or viral causes.

In tonsillar hyperplasias, enlargement is due to an overall increase in cell numbers in the tonsillar parenchyma. Thus, prominent cellular activity can be found within the germinal centers of the tonsillar follicles. When tonsillar hypertrophy follows inflammatory changes, the size increase occurs predominantly in the tonsil's connective tissue stroma or as a result of cellular debris or salt deposits obstructing the tonsillar crypts. In *Tangier disease*, a yellow-orange hypertrophy of the tonsil results from massive cholesterol storage within the tonsil and is a

consequence of alpha-lipoprotein deficiency (Huth et al, 1970).

The size of the tonsil is of little clinical significance unless the tonsil is large enough to produce mechanical obstruction and impair the patient's ability to eat or breathe. Enlarged tonsils may also result from malignant disease, and suspicion of such disease justifies biopsy (or tonsillectomy) for tissue diagnosis. It must be emphasized, however, that enlarged tonsils alone are insufficient reason for their removal unless other clinical considerations exist (as discussed in Chapter 22). Anxious patients or their parents must often be reassured that multiple minor pharyngeal complaints are not necessarily related to tonsillar infections, particularly since many such infections can begin as a result of postnasal discharge causing secondary pharyngitis or tonsillitis. When these infections do occur, supportive measures are indicated and follow-up care should be provided as required.

Hyperkeratosis of the Tonsils

(Keratosis Pharyngeus)

Hyperkeratosis pharyngeus is characterized by the development of white horny projections on the lymphoid tissues of the pharynx as well as on the tonsils and adenoids. Hyperkeratosis usually occurs in young adults but may also be found in older individuals. Debilitated patients may be predisposed to this condition, but it may also occur as an incidental finding in asymptomatic patients. The problem occurs clinically when hyperkeratosis is confused with chronic follicular tonsillitis (Hill, 1960; Anderson, 1980; Ballenger, 1987).

The fungus *Leptothrix buccalis* was formerly considered to be the causative organism, but this theory is now in doubt, since the fungus can be a common saprophyte in the oral cavity. Lymphoid keratosis may be related to some metabolic disturbance and is produced by excessive cornification within the tonsillar crypts.

Clinical symptoms in this condition can be lacking or vague. Patients can complain of nondescript pharyngeal irritations or foreign body sensations. The keratotic excrescences may also produce a recurrent cough or cause the need for frequent swallowing. However, there should be no associated systemic symptoms.

Examination of the throat reveals multiple discrete, white, minute projections from the tonsillar crypts or other pharyngeal lymphoid tissues. There is usually no evidence of local inflammation unless the cryptic debris present becomes secondarily infected. Because the keratotic projections are frequently adherent to the underlying tissues, their blunt removal can leave a bleeding base.

Treatment of symptomatic hyperkeratosis consists primarily of supportive measures and involves efforts to improve general health or correct obvious metabolic disorders. The keratoses can be removed from sites of local irritation with a small sharp curette. Unfortunately, the task may be tedious and the results disappointing. Other measures include the electrocoagulation of

involved tissues and the applications of topical astringents. Symptomatic measures alone are perhaps best, combined with reassurance to the patient that the problem is noninjurious and is usually self-limited (unless secondary infections occur).

Tonsilloliths (Calculi of the Tonsils)

Spiculated concretions of varying sizes and consistencies can form around or within the substance of the tonsils (Hill, 1960; Harding, 1962; Samant and Gupta, 1975). Repeated episodes of inflammation may produce fibrosis at the openings of the tonsillar crypts. Bacterial and epithelial debris then accumulate within these crypts and contribute to the formation of a retention cyst. Calcification occurs with the deposition of inorganic salts to form calcium phosphate or carbonates, following which gradual enlargement of the concretion takes place. Certain calculi may form from the accumulation of keratohyaline masses in the crypts, with secondary concentric deposits of salts occurring around these masses (Hadi and Samara, 1985). These processes may be stimulated by the oral flora, which may include fungi or actinomycetes-like organisms. The resultant calculus can be quite large and may even ulcerate through the tonsillar surface.

Tonsilloliths occur more frequently in adults than in children. Symptoms are comparable to those found in patients with tonsillar hyperkeratoses and include non-specific chronic sore throats and (referred) otalgias. A foreign body sensation may also exist in the back of the throat, and patients may complain of chronic or recurrent halitosis.

Inspection of the throat will show the tonsillolith embedded within the affected tonsil. Local inflammatory changes will be present if there is secondary infection, and the tonsil may be enlarged as a result. Gentle palpation will indicate local induration.

The differential diagnosis of tonsilloliths includes a foreign body, a calcified granuloma, malignancy, and an elongated styloid process (which is palpable deep to the tonsillar fossa). Isolated cartilage or bone is usually derived from embryonic rests originating from the second or third branchial arches and can also be mistaken for a tonsillolith.

Treatment should be based on supportive care in addition to removal of concretions from the tonsillar crypts. When possible, curettage can be used, although larger lesions may require local excisions under topical or regional infiltration anesthesias. When clinically indicated, tonsillectomy is definitive therapy.

Elongated Styloid Process

The normal styloid process is a slender bony projection from the inferior aspect of the temporal bone medial to the stylomastoid foramen. Its usual length varies from 1.5 to 5 cm (Moffat et al, 1977). On occasion, an elongated styloid process or an ossified stylohyoid ligament can project into the tonsillar fossa or impinge on vital structures in the neck and pharynx to cause varied neurologic or vascular symptoms. Most patients are asymptomatic, but some may complain

about throat discomfort following tonsillectomy. This occurrence possibly results from scar formation at the tip of the styloid process behind the tonsillar fossa with irritation of the pharyngeal mucosa (Loeser and Caldwell, 1942; Eagle, 1948; Hill, 1960).

Impingement of the styloid process or scar can affect the trigeminal, facial, glossopharyngeal, vagal, and cervical sympathetic nerves. This produces recurrent non-specific throat discomfort, dysphagia, increased salivation, and gagging. A foreign body sensation in the throat may also be experienced. The styloid process can also impinge on the carotid arteries to affect circulation or produce regional tenderness or headaches. In addition, otalgia or tinnitus may be present.

The diagnosis of the styloid process syndrome can be made by palpation of the process deep to the tonsillar fossa or by lateral and anteroposterior radiographic views of the skull. Xeroradiography or even contrast angiography may be helpful.

Management of the styloid process syndrome should consist of either supportive measures or surgical removal of the process. However, patients must be carefully selected, since nonspecific pharyngeal complaints can easily be attributed *incorrectly* to involvement of the styloid process. Eagle's (1948) technique for correction involves shortening of the styloid process through the tonsillar fossa. The muscles at the tip of the process are removed, following which the tip is excised with a surgical rongeur. The method described by Loeser and Caldwell (1942) employs an external approach to the styloid process and has the advantage of protecting vital structures under direct vision. A transverse incision is made anterior to the sternocleidomastoid muscle to provide free access to the deep structures of the neck medial to the angle of the jaw. The parotid fascia is reflected superiorly, and the contents of the carotid sheath are retracted posteriorly. Adequate exposure can be achieved by this method, but care must be taken to identify and protect the facial nerve at the stylomastoid foramen. The styloid process can then be removed by rongeur after the bone is stripped of its fascial and muscular attachments.

The Pharyngeal Tonsils (Adenoids)

Adenoid Hypertrophy

Enlargement of the nasopharyngeal lymphoid tissue or pharyngeal tonsil is relatively common in preadolescent or adolescent children, as previously discussed. The cause of this enlargement is unknown, but it may represent a response to repeated infections or may only reflect generalized lymphoid hypertrophy (Stewart and Lumsden, 1961; Simpson et al, 1967; Handleman and Osborne, 1976).

Hypertrophied adenoids can obstruct the eustachian tube orifices to cause middle ear infections or impede ventilation of the posterior paranasal sinus ostia. Resultant sinus infections may involve multiple factors, since persistent postnasal discharge associated with chronic rhinitis or sinusitis can itself cause secondary adenoid infection and subsequent adenoid hypertrophy. In addition, an allergic diathesis may be reflected by the size of the adenoids or the nasopharyngeal

lymphatic tissues.

The clinical symptoms of adenoid enlargement vary with the age of the patient and can be associated with the patient's general condition as well as with the anatomic size of the nasopharynx. Thus, in a small nasopharynx, adenoid tissues may prove to be clinically significant, whereas a larger adenoid mass may be relatively innocuous in an anatomically larger nasopharynx.

The most common symptom attributed to adenoid enlargement is nasal obstruction, with resultant mouth breathing or snoring during sleeping. Nasal discharge of mucus is common. Children with chronic adenoid hypertrophy may have characteristic facial features that reflect the need for chronic breathing through the mouth; these features are described as the "adenoid facies". Because the nasal arch is broad and flattened, the nasolabial furrow may be lost and a pinched nostril appearance is produced (Boies, 1954; Adams et al, 1989). The sucking of infants is usually impaired. The mouth is invariably open, and the upper lip is elevated to improve the oral intake of air. This occurrence may also give some prominence to the maxillary incisor teeth. The voice is characteristically "flat" as a result of the chronic nasal congestion. Affected children may also have a dull general appearance, with lack of chest development and a suggestion of chronic illness. These children may also have frequent headaches and upper respiratory tract infections. In addition, there may be vague systemic complaints such as gastrointestinal upsets or personality disturbances. Epistaxis can occur as a result of adenoid enlargement altering nasal ventilation and air flow patterns to dry the nasal mucosa. cracking of the mucosa during sleep will produce blood oral or nasal secretions.

The diagnosis of adenoid enlargement can readily be made by the careful use of a small pharyngeal mirror or a gently placed nasopharyngoscope. These findings can be confirmed by direct inspection with palpation under some form of anesthesia, especially in young or uncooperative patients. When indicated, lateral x-ray views of the nasopharynx may prove useful for diagnosis (Goldman and Bachman, 1958).

As discussed in Chapter 22, the management of symptomatic adenoid hypertrophy tends to be surgically directed if supportive measures fail to relieve complaints. Such therapies include the judicious use of antihistamines, decongestants, nasal saline douches, and the like. Allergic desensitization may also be required in some patients.

Acute Adenoiditis (Pharyngeal Tonsillitis)

The adenoids are commonly involved in throat inflammation affecting the tonsils or lymphoid tissues along the lateral pharyngeal walls. The organisms present are comparable to those identified in tonsillar infections (Boies, 1954; Hill, 1960; Simpson et al, 1967; Adams et al, 1989).

In the patient with adenoiditis only, the throat is invariably sore, and varying degrees of dysphagia occur. Fever, malaise, headache, and the like can be found. Obstruction of the posterior

nasal choanae may produce sinusitis, whereas eustachian tube obstruction can result in middle ear effusions (with complaints of otalgia or decreased hearing).

Examination of the throat reveals erythema and edema of the pharyngeal lymphoid tissues, with localized pustules or mucopus if bacterial infection is present. Cervical adenopathy is common.

Treatment is similar to that for acute tonsillitis. This includes adequate hydration, rest, oral hygiene, analgesics, and antipyretics (such as aspirin or acetaminophen preparations). Decongestants and antihistamines may be useful. Antibiotics are needed to treat bacterial infections and will have to be adjusted accordingly if organism resistance is encountered. Uncomplicated adenoiditis resolves within a short time, allowing the patient to return to normal activity.

Chronic Adenoiditis

Chronic infection of the adenoids is commonly the end result of acute inflammations that have failed to completely resolve. This condition may follow recurrent purulent rhinosinusitis or may be associated with chronic tonsillitis. Adenoid hypertrophy may be the consequence, with obstruction of the posterior nasal choanae or eustachian tube orifices.

Chronic adenoid inflammation can be bacterial or viral in origin, although consideration should also be given to other disease states that can mimic adenoiditis. This differential diagnosis is similar to that for inflammations of the tonsil, as discussed previously, and can include granulomas (tuberculosis, syphilis, and so on), collagen disease, and neoplasms (Boies, 1954; Hill, 1960; Stewart and Lumsden, 1961; Adams et al, 1989).

Complaints related to chronic adenoiditis are comparable to those associated with acute inflammations. Rhinorrhea, sinusitis, middle ear problems, and the like may occur.

Examination of the nasopharynx reveals the presence of enlarged lymphoid tissues that are chronically inflamed. Purulent discharge or mucopus is frequently present.

Management of chronic adenoiditis tends to be surgically directed. Allergy is a possible factor and should be evaluated for possible treatment, although in actuality, allergic causes are less common than might be anticipated (Tos et al, 1979; Loesel, 1984).

Tornwaldt's Bursa (Pharyngeal Bursitis, Nasopharyngeal Cyst)

Some mention should be made of Tornwaldt's bursa, since it can be found clinically on rare occasions and may mimic chronic inflammations of the nasopharynx (Godwin, 1944; Hill, 1960; Simpson et al, 1967). The bursa is presumed to represent persistence of an embryonic communication between the anterior tip of the notochord and the roof of the pharynx. It is located in the middle of the posterior wall of the nasopharynx, extending toward the tubercle of

the occipital bone (Hollinshead, 1982). This communication usually persists in the fetus until the second month of development, after which resolution occurs. However, adhesions may form to result in its persistent patency.

Symptoms can be varied. A dull occipital headache may occur or there may be persistent postnasal discharge with crusting in the nasopharynx. Varying degrees of nasal or eustachian tube dysfunction can occur associated with rhinorrhea or middle ear effusions. Sore throat is common, as is persistent low-grade fever.

It is important to differentiate a retention cyst in the median cleft of the posterior nasopharynx, which results from chronic inflammation of the lymphoid tissues, from Tornwaldt's bursa. This differentiation may prove to be academic in that signs and symptoms of the bursa and cyst are the same. Microscopic examination of the pharyngeal bursa, however, usually shows pharyngeal mucosa on one surface and an epithelial cyst lining on the other, whereas the retention cyst demonstrates only lymphoid tissue, which possibly contains infected necrotic or cystic areas (Godwin, 1944).

In the presence of a bursa, examination of the nasopharynx will reveal a cystic or fluctuant lesion in the posterior wall of the nasopharynx. A variably sized opening may be present in the midline of the lesion although peripheral crusting or scab formation can be present. The asymptomatic bursa can be found during routine mirror pharyngoscopy and may show inflammatory changes that have narrowed or closed its pharyngeal communication.

Treatment of the infected bursa requires the use of appropriate antibiotics and surgical drainage as indicated. Definitive care involves marsupialization of the bursa or adequate removal of its lining membrane by curette or scissors dissection.

The Lingual Tonsils

The lingual tonsils may be involved in disease processes that are similar to those affecting the palatine or pharyngeal tonsils (Shambaugh, 1945; Stewart and Lumsden, 1961; Simpson et al, 1967; Ballenger, 1987). This includes acute and chronic inflammations, granulomas, and the like. However, it cannot be overly stressed that frank malignancy can be masked by any benign disease process involving the pharyngeal tissues. Consequently, any lesion involving the base of the tongue or lingual tonsil that fails to respond to symptomatic therapies within a limited time should be considered malignant until demonstrated otherwise by tissue biopsy.

Hypertrophy of the Lingual Tonsil

Enlargement of the lingual tonsil is relatively common following puberty, but it may also result from lymphoid hyperplasia subsequent to repeated infections or may represent compensatory hypertrophy following palatine tonsillectomies. There may be no associated clinical symptoms, or some throat complaints may occur, which include local irritations, discomfort on swallowing, and the sensation of a lump in the throat. There may also be a recurrent

nonproductive cough as well as a thick voice with poor articulation.

Examination of the base of tongue, including finger palpation and indirect mirror evaluation, should readily demonstrate enlargement of the lingual tonsil. On occasion, this may be associated with prominent lingual varices. Lateral soft tissue radiographs can prove useful in some patients to objectively document the tissue changes present (Willet and Youngs, 1984).

Differential diagnosis includes those disease processes previously mentioned, as well as inclusion cysts, thyroglossal cysts, and a lingual thyroid gland.

Treatment of the enlarged lingual tonsil should essentially consist of symptomatic measures and may include analgesics or topical cautery of varices. When indicated, lingual tonsillectomy can be done under either local or general anesthesia. The procedure is essentially a limited posterior glossectomy and may require morcellation of the tissues to be removed. Bleeding can be profuse but should be controllable with pressure hemostasis, electrocautery, or the placement of fixation ligatures. Any tissues removed should be submitted for microscopic study to allow appropriate follow-up therapy to be given. Alternate methods include use of cryosurgery and the CO_2 laser.

Acute Lingual Tonsillitis

Inflammation of the lingual tonsil is similar to that involving the tonsils and adenoids. Predominant clinical symptoms include the sensation of a lump in the throat or painful swallowing. Fever, nonspecific headache, and the like are common; salivation can be excessive and speech may be impaired. Examination of the throat reveals injected lingual tonsils that may contain pustules. Cervical adenopathy is common. Management is similar to that for faucial tonsillitis.

Abscess of the Lingual Tonsils

An abscess involving the lingual tonsils is relatively uncommon and results from a localized infection that becomes refractory to medical care. This invariably follows lingual tonsillitis, surgery, or oral trauma, but may also result from central necrosis of a malignancy of the posterior portion of the tongue.

Predominant symptoms include localizing pain or dysphagia, excessive salivation, and trismus. Impaired speech occurs with progression of the lesion. Dyspnea results from infection involving supraglottic tissues to produce laryngeal edema.

Examination reveals an inflamed and swollen tongue base that is fluctuant or indurated. It is also extremely tender to palpation.

Treatment is comparable to that for peritonsillar or retropharyngeal abscesses and includes antibiotics, hydration, and analgesics. Incision and drainage is indicated as soon as definite

localization of pus is found and is preferably done with topical anesthesia alone. The abscess site or its loculations should be opened with a guarded angulated knife. If airway compromise is suspected, tracheotomy is indicated, since edema may rapidly cause obstruction of the airway.

Resolution of a lingual abscess is usually similar to that associated with peritonsillar infections. Failure to respond to antibiotics requires the careful adjustment of medications through culture evaluation and appropriate drug sensitivity analysis. Follow-up, including possible tissue biopsies, is then performed as required.

Airway Obstruction and Waldeyer's Ring

In the past, an uncommonly occurring but definite complication of tonsillar or adenoid hypertrophy has been cor pulmonale with pulmonary edema. A less profound but clinically significant consequence of the hypoxia produced by obstruction of the airway from any of the lymphoid tissue of Waldeyer's ring is the sleep apnea syndrome (Kravath et al, 1977; Lind and Lundell, 1982; Messer et al, 1984; Feinberg and Shabino, 1985). Fernbach and co-workers (1983) used fluoroscopy to examine children who were suspected of having sleep apnea syndrome and found that there was partial or complete collapse of the pharyngeal airway during inspiration. Abnormal movements involve the prevertebral soft tissues, tonsils, soft palate, tongue, and jaw. Fernbach's group further found that the resistance to air flow was inversely proportional to the pharyngeal radius (using Hagen-Poiseuille's law). In affected patients, hypertrophied tonsils and adenoids result in marked changes in resistance to air flow, and large negative inspiratory pressures are needed to draw air through the patent but impeded airway. These changes worsen as negative pressure causes soft tissues to further collapse into the narrowed airway. Chronic airway obstruction then produces symptoms carbon dioxide retention and results in bronchoconstriction and pulmonary vasoconstriction with increased pulmonary artery resistance.

Patients with the sleep apnea syndrome may experience varying symptoms that depend on the severity of the disorder (Ravindran, 1983). Daytime complaints have been early morning headaches, easy fatigability, as well as mood and behavior changes. Symptoms occurring during sleep have included loud snoring, restlessness or insomnia, enuresis, and nightmares.

Tests for sleep apnea have evolved through referral centers but have included careful physical examinations for anatomic abnormalities, lateral radiographs of the airway and fluoroscopy, serial blood gas determinations, pulmonary function tests, and sleep electroencephalographs.

In patients with proven sleep apnea syndrome or cor pulmonale, therapy is surgically directed and is discussed elsewhere in this text.