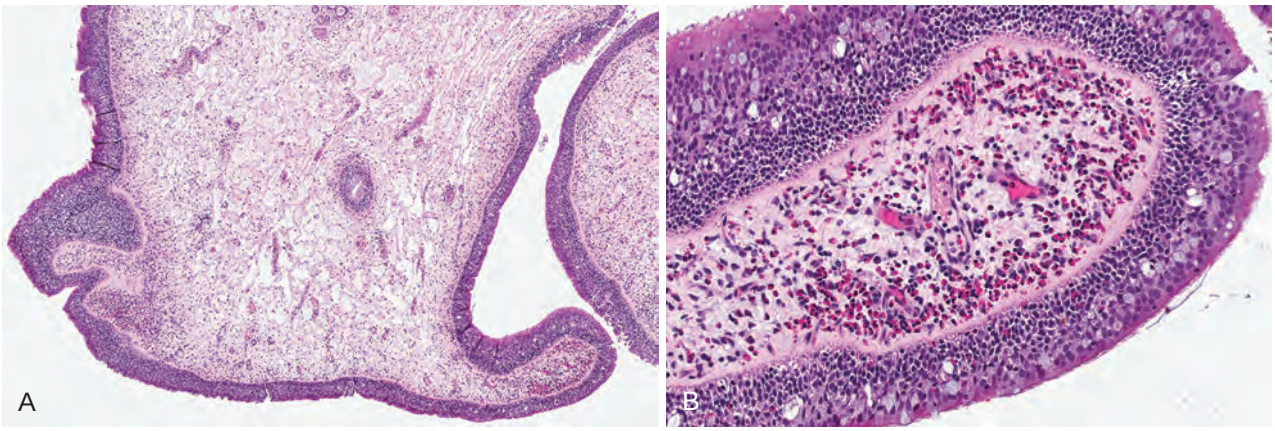

Figure 16-8 **A,** Nasal polyps. Low-power magnification showing edematous masses lined by epithelium. **B,** High-power view showing edema and eosinophil-rich inflammatory infiltrate.

inhabitants of the oral cavity, and the inflammatory reaction is entirely nonspecific. Impairment of drainage of the sinus by inflammatory edema of the mucosa is an important contributor to the process and, when complete, may impound the suppurative exudate, producing *empyema* of the sinus. Obstruction of the outflow, most often from the frontal and less commonly from the anterior ethmoid sinuses, occasionally leads to an accumulation of mucous secretions, producing a so-called *mucocele*.

Acute sinusitis may, in time, give rise to *chronic sinusitis*, particularly when there is interference with drainage. There is usually a mixed microbial flora, largely of normal inhabitants of the oral cavity. Particularly severe forms of chronic sinusitis are caused by fungi (e.g., mucormycosis), especially in patients with diabetes. Uncommonly, sinusitis is a component of *Kartagener syndrome*, which also includes bronchiectasis and situs inversus (Chapter 15). All these features are secondary to defective ciliary action. Although most instances of chronic sinusitis are more uncomfortable than disabling or serious, the infections have the potential of spreading into the orbit or of penetrating into the surrounding bone to give rise to osteomyelitis or spreading into the cranial vault, causing septic thrombophlebitis of a dural venous sinus.

Necrotizing Lesions of the Nose and Upper Airways

Necrotizing ulcerating lesions of the nose and upper respiratory tract may be produced by the following:

- Acute fungal infections (including mucormycosis; Chapter 8), particularly in patients with diabetes and immunosuppressed patients
- Granulomatosis with polyangiitis, previously called Wegener granulomatosis (Chapter 11)
- Extranodal NK/T-cell lymphoma, nasal type, is a lymphoma in which the tumor cells harbor EBV (Chapter 13). These neoplasms are typically seen in males who are in the fifth or sixth decade of life and most commonly occur in individuals of Asian and Latin American descent. Ulceration and superimposed bacterial infection frequently complicate the process. At one time these lesions were almost always rapidly fatal as a result of uncontrolled spread of the lymphoma and penetration into the cranial vault, or secondary bacterial infection and blood-borne dissemination of the infection. Currently, localized cases can often be controlled with radiotherapy, but relapse and recurrences remain common and are associated with poor outcomes.

Nasopharynx

Although the nasopharyngeal mucosa, related lymphoid structures, and glands may be involved in a wide variety of specific infections (e.g., diphtheria, infectious mononucleosis) and by neoplasms, the only disorders we describe here are nonspecific inflammations; tumors are discussed separately.

Inflammations

Pharyngitis and *tonsillitis* are frequent features of viral upper respiratory infections. Most commonly implicated are the rhinoviruses, echoviruses, and adenoviruses, and, less frequently, respiratory syncytial viruses and the various strains of influenza virus. In the usual case, there is reddening and edema of the nasopharyngeal mucosa, with reactive enlargement of nearby tonsils and lymph nodes. Bacterial infections may be superimposed on these viral infections, or may be primary invaders. The most common offenders are the β-hemolytic streptococci, but sometimes *Staphylococcus aureus* or other pathogens may be implicated. The inflamed nasopharyngeal mucosa may be covered by an exudative membrane (pseudomembrane), and the nasopalatine and palatine tonsils may be enlarged and covered by exudate. A typical appearance is of enlarged, reddened tonsils (due to reactive lymphoid hyperplasia) dotted by pinpoints of exudate emanating from the tonsillar crypts, so-called *follicular tonsillitis*.

The major importance of streptococcal "sore throats" lies in the possible development of late sequelae, such as rheumatic fever (Chapter 12) and glomerulonephritis (Chapter 20). Whether recurrent episodes of acute tonsillitis favor the development of chronic tonsillar enlargement is open to debate, but regardless of cause the enlargement of the lymphoid tissue invites the tender mercies of the ENT surgeon.