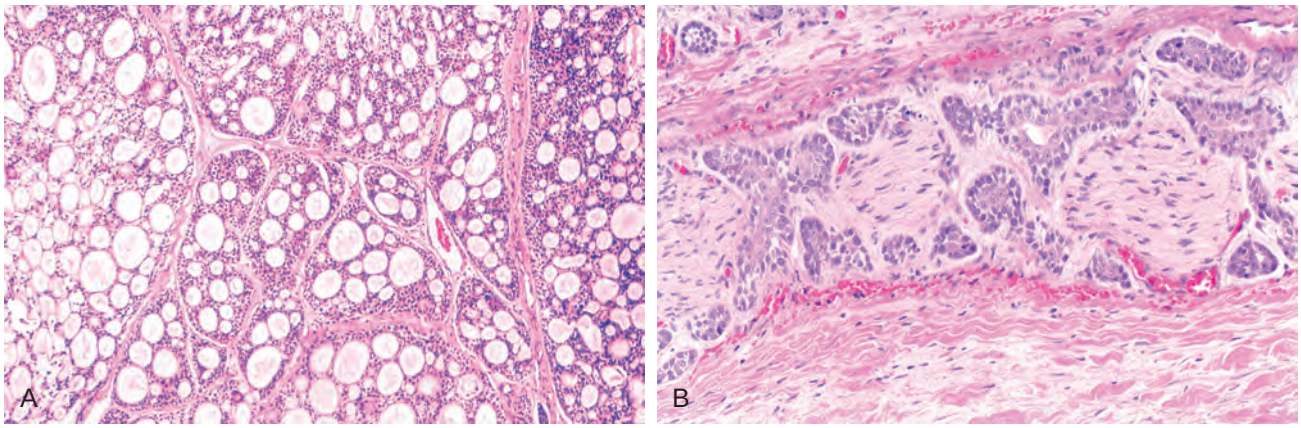

Figure 16-19 Adenoid cystic carcinoma in a salivary gland. **A,** Low-power view. The tumor cells have created a cribriform pattern enclosing secretions. **B,** Perineural invasion by tumor cells.

have been reported in the nose, sinuses, upper airways, breast, and elsewhere.

MORPHOLOGY

In gross appearance, they are generally small, poorly encapsulated, infiltrative, gray-pink lesions. On histologic evaluation, they are composed of small cells having dark, compact nuclei and scant cytoplasm. These cells tend to be disposed in tubular, solid, or cribriform patterns reminiscent of cylindromas arising in the adnexa of the skin (Chapter 25). The spaces between the tumor cells are often filled with a hyaline material thought to represent excess basement membrane (Fig. 16-19*A*). Other less common histologic patterns have been designated as tubular and solid variants.

Although slow growing, adenoid cystic carcinomas are unpredictable tumors with a tendency to invade perineural spaces (Fig. 16-19*B*), and they are stubbornly recurrent. Eventually, 50% or more disseminate widely to distant sites such as bone, liver, and brain, sometimes decades after attempted removal. Thus, although the 5-year survival rate is about 60% to 70%, it drops to about 30% at 10 years and 15% at 15 years. Neoplasms arising in the minor salivary glands have, on average, a poorer prognosis than those that arise in the parotid glands.

Acinic cell carcinoma is composed of cells resembling the normal serous acinar cells of salivary glands. It is relatively uncommon, representing only 2% to 3% of salivary gland tumors. Most arise in the parotids; the remainder arise in the submandibular glands. It rarely involves the minor glands, which normally have only a scant number of serous cells. Like Warthin tumor, it is sometimes bilateral or multicentric. The tumors are generally small, discrete lesions that may appear encapsulated. On histologic examination, they reveal a variable architecture and cell morphology. Most characteristically, the cells have clear cytoplasm but the cells are sometimes solid and at other times vacuolated. The tumor cells are disposed in sheets or microcystic, glandular, follicular, or papillary patterns. There is usually little anaplasia and few mitoses, but some tumors are occasionally slightly more pleomorphic.

The clinical course of these neoplasms is somewhat dependent on the level of pleomorphism. Overall, recurrence after resection is uncommon, but about 10% to 15% of these neoplasms metastasize to lymph nodes. The survival rate is in the range of 90% at 5 years and 60% at 20 years.

KEY CONCEPTS

Diseases of Salivary Glands

- **Sialadenitis** (inflammation of the salivary glands) can be caused by trauma, infection, or an autoimmune reaction.
- **Mucoceles** are caused by trauma to or blockage of a salivary gland duct, with consequent leakage of saliva into the surrounding connective tissue stroma.
- **Pleomorphic adenoma** is a benign, slow-growing neoplasm composed of a heterogenous mixture of epithelial and mesenchymal cells.
- **Mucoepidermoid carcinoma** is a malignant neoplasm of variable biological aggressiveness that is composed of a mixture of squamous and mucous cells.

SUGGESTED READINGS

Barnes L, Eveson JW, Reichart P, et al: *Pathology and genetics of head and neck tumors*, 2005, WHO, IARC, Chapter 6, Odontogenic Tumors, pp 284–327. [This chapter represents the most recent consensus regarding epidemiology, etiology, clinical features, and classification of odontogenic tumors.]

Dardick I: *Color Atlas/Text of Salivary Gland Pathology*, New York, 1996, Igaku-Shoin. [*Color atlas that discusses both normal salivary gland morphology and pathology. It also proposes hypotheses regarding the role of differentiation in the development as well as the histopathology of salivary gland neoplasia.*]

Ellis GL, Auclair PL: *Tumors of the salivary glands. AFIP atlas of tumor pathology*, Third series, Fascicle 9. Silver Spring MD, 2008, ARP Press. [*Comprehensive textbook on salivary gland pathology that is considered to be the gold standard.*]

Gillison ML, Broutian T, Pickard RK, et al: Human papilloma virus and rising oropharyngeal cancer incidence in the United States. *J Clin Oncol* 29:4294, 2011. [*A study using the Surveillance, Epidemiology, and End Results (SEER) Residual Tissue Repositories Program which demonstrated Increases in the population-level incidence and survival of oropharyngeal cancers in the United States since 1984 are caused by HPV infection.*]