The primary disease processes that give rise to swellings and tumors of the oral cavity include cysts, mucous extravasation and retention in the minor salivary glands, foci of granulation tissue and inflammation, abscesses and connective-tissue proliferations that are well defined or encapsulated, as well as infiltrative sarcomas. Figure 23–1 is a representation of the processes that cause soft-tissue tumefactions in the mouth. Both epithelial and connective-tissue disease processes can present as masses. Benign and malignant surface epithelial tumors are discussed in the Chapters 15 and 20, respectively. From a clinical perspective the three most important defining characteristics of any soft-tissue swelling are location, coloration, and palpable nature.

As for location, certain diseases tend to occur in specific sites to the exclusion of others. Table 23–1 lists the most common lesions according to site. This is not to say that these sites are exclusive, since many lesions can in fact occur anywhere in the mouth; rather, this tabulation catalogues the most likely lesions for that site in terms of overall prevalence. Coloration is dependent upon the tissues present in the mass and the depth of the lesion. Table 23–2 lists the most frequently encountered colorations observed with soft-tissue masses and indicates the lesions that most often present with a given coloration. In general, yellow-appearing lesions are comprised of lymphoid tissue or adipose tissue, red
Swellings and Tumors of the Oral Cavity and Face

Swellings are vascular, blue swellings are mucinous or venous, and brown swellings contain melanin or blood pigments. Lesions with normal mucosal pink coloration are generally composed of fibrous tissues or some other tissues lying deeper in the connective tissues.

Table 23–1 Orofacial Soft-Tissue Swellings according to Site

<table>
<thead>
<tr>
<th>Site</th>
<th>Type of Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraoral</td>
<td></td>
</tr>
<tr>
<td>Lips and buccal mucosa</td>
<td>Fibroma, mucocele, mesenchymal tumor, salivary tumor, squamous cell carcinoma</td>
</tr>
<tr>
<td>Gingiva</td>
<td>Parulis, pyogenic granuloma, peripheral fibroma, peripheral giant cell granuloma, peripheral ossifying fibroma, gingival cyst, peripheral odontogenic tumors, squamous cell carcinoma</td>
</tr>
<tr>
<td>Palate</td>
<td></td>
</tr>
<tr>
<td>Dorsolateral tongue</td>
<td>Fibroma, granular cell tumor, pyogenic granuloma, squamous cell carcinoma</td>
</tr>
<tr>
<td>Ventral tongue and oral floor</td>
<td>Mucocele, ranula, lymphoid aggregates, lymphoepithelial cyst, osteocartilagenous choristoma, squamous cell carcinoma</td>
</tr>
<tr>
<td>Face and neck swellings</td>
<td></td>
</tr>
<tr>
<td>Masseteric region</td>
<td>Cellulitis, space infection, jaw cysts and tumors, masseteric hypertrophy</td>
</tr>
<tr>
<td>Parotid region</td>
<td>Sialadenitis, sialolithiasis, salivary neoplasm</td>
</tr>
<tr>
<td>Submandibular region</td>
<td>Lymphadenopathy, sialolithiasis, salivary neoplasm</td>
</tr>
<tr>
<td>Lateral neck</td>
<td>Lymphadenopathy, mesenchymal neoplasm, branchial cleft cyst, metastatic carcinoma, lymphoma, carotid body tumor</td>
</tr>
<tr>
<td>Anterior neck</td>
<td>Goiter, thyroid neoplasm, thyroglossal cyst</td>
</tr>
<tr>
<td>Face</td>
<td>Seborrheic keratosis, basal cell carcinoma, adnexal skin tumors, squamous cell carcinoma, melanoma</td>
</tr>
</tbody>
</table>

Table 23–2 Masses with Coloration or Pigmentation

<table>
<thead>
<tr>
<th>Color</th>
<th>Soft-Tissue Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue-purple</td>
<td>Hemangioma, varix, hematoma, peripheral giant cell granuloma, mucocele, Kaposi sarcoma</td>
</tr>
<tr>
<td>Red</td>
<td>Hemangioma, pyogenic granuloma, Kaposi sarcoma</td>
</tr>
<tr>
<td>Brown</td>
<td>Nevus, hematoma, seborrheic keratosis, Kaposi sarcoma, melanoma</td>
</tr>
<tr>
<td>Black</td>
<td>Melanoma</td>
</tr>
<tr>
<td>Yellow-orange</td>
<td>Lymphoid aggregates, lymphoepithelial cyst, lipoma, granular cell tumor</td>
</tr>
</tbody>
</table>

In terms of frequency, the majority of oral mucosal masses are reactive proliferations, such as fibrous hyperplasias, pyogenic granulomas, and mucous extravasation reactions. Mesenchymal and salivary neoplasms are uncommon, and lymphomas and sarcomas are rare causes of oral swelling. Indeed, the probability that a mucosal mass is a reactive or hyperplastic process is probably 50-fold compared to a true neoplastic process. In most instances, biopsy is necessary to arrive at a definitive diagnosis. Aspiration or incision and drainage may be performed as a diagnostic procedure when the mass is consistant with an abscess.

Molecular and pathologic correlates of disease

The molecular aspects or oral soft-tissue swellings are poorly understood and have not received much attention in the experimental literature. Conversely, the underlying pathologic processes associated with the various lesions that produce tumefaction in the oral cavity are well defined in the oral and maxillofacial pathology literature.

As an overview of pathologic mechanisms, basic concepts are briefly presented here. The common masses that represent hyperplasias evolve as a consequence of irritation to the mucosal tissues by a dental appliance or by trauma, often the consequence of biting. The injured tissues respond to chronic and sometimes acute injury by proliferation of cells. The most commonly encountered hyperplasias are those involving fibroblasts. Injury to connective tissue results in fibroblastic proliferation of a benign nature, followed by collagen fibrillogenesis. Many fibrous hyperplasias are comprised of loose collagen and are soft to palpation, such as denture-induced fibrous hyperplasia and the common traumatic fibromas of the tongue and labial and buccal mucosae. In the gingiva, the periodontal tissues may be the targets of injury, particularly from irritants that may become entrapped in the gingival sulcus. Calculus, food particles, and foreign objects may be introduced into the sulcus, where they irritate the
fibrovascular connective tissues, periosteum, and periodontal ligament fibrous tissues. Proliferation of the fibrovascular connective tissue, along with inflammation, gives rise to pyogenic granulomas, whereas proliferation of the periosteal tissues, which contain osteoblasts and osteoclasts, gives rise to peripheral giant cell granulomas. When periodontal ligament fibroblasts proliferate, they retain the potential to elaborate bone and cementum, thereby giving rise to peripheral ossifying fibromas. Minor salivary glands are located everywhere in the oral cavity except on the anterior dorsal tongue and the attached gingiva. They are most easily damaged from accidental biting in the lower lip and sometimes in the buccal mucosa, whereas injuries to the palatal and upper lip glands are rare. Therefore, severence of the minor gland ducts after an acute biting episode frequently leads to mucous extravasation into the connective tissues of the lips and buccal mucosa. In these mucoceles, the extravasated mucus becomes encapsulated, or walled-off, by fibrous and granulation tissues, giving the appearance of a cyst. Less commonly, mucous plugs form in the ducts of minor glands and cause retention of mucus. The ducts undergo cystic dilation and are epithelial lined; such lesions are referred to as mucous retention cysts or sialoceles. Although rare, true salivary stones may arise in minor salivary ducts, and as they grow, they result in an enlargement within the submucosa that is movable and hard.

Table 23–3  Masses according to Palpation Characteristic

<table>
<thead>
<tr>
<th>Palpation Characteristic</th>
<th>Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soft, fluctuant</td>
<td>Mucocele, ranula</td>
</tr>
<tr>
<td></td>
<td>Developmental cysts</td>
</tr>
<tr>
<td></td>
<td>Sialoceles</td>
</tr>
<tr>
<td></td>
<td>Gingival cysts</td>
</tr>
<tr>
<td></td>
<td>Parulis</td>
</tr>
<tr>
<td></td>
<td>Space infections and abscesses</td>
</tr>
<tr>
<td>Soft, nonfluctuant</td>
<td>Lipoma</td>
</tr>
<tr>
<td></td>
<td>Fibroma</td>
</tr>
<tr>
<td></td>
<td>Organized mucocele</td>
</tr>
<tr>
<td>Firm, movable</td>
<td>Granulomas</td>
</tr>
<tr>
<td></td>
<td>Salivary adenomas</td>
</tr>
<tr>
<td></td>
<td>Adnexal skin tumors</td>
</tr>
<tr>
<td>Firm, fixed</td>
<td>Granular cell tumor</td>
</tr>
<tr>
<td></td>
<td>Seborrheic keratosis</td>
</tr>
<tr>
<td></td>
<td>Keratoacanthoma</td>
</tr>
<tr>
<td></td>
<td>Fibromatosis</td>
</tr>
<tr>
<td>Indurated, fixed</td>
<td>Basal cell carcinoma</td>
</tr>
<tr>
<td></td>
<td>Salivary adenocarcinomas</td>
</tr>
<tr>
<td></td>
<td>Squamous cell carcinoma</td>
</tr>
<tr>
<td></td>
<td>Melanoma</td>
</tr>
<tr>
<td></td>
<td>Sarcomas</td>
</tr>
<tr>
<td></td>
<td>Lymphomas</td>
</tr>
</tbody>
</table>

Acute infections of the soft tissues are uncommon; however, occasionally a foreign body, such as a small fish bone or material from a dental procedure may be implanted into the soft tissues and cause an acute reaction. These submucosal fluctuant abscesses are more often seen on the tongue. Of course the most common location for abscess in the oral cavity is the gingiva and vestibule. In such instances the abscess is a parulis from odontogenic infection or a periodontal abscess arising in a periodontal pocket. Pulp vitality testing of adjacent teeth, periapical radiographs, periodontal probing, and aspiration are all important procedures when attempting to establish a definitive diagnosis. For accurate cultures procured for identifying causative microorganisms, a pure suppurate is essential; this is to avoid contaminants.

Chronic foci of inflammation may also account for submucosal masses. Lymph nodes are located in the buccal mucosa and in health are not palpable. Sometimes they become irritated or drain a local viral or bacterial infection and become enlarged. This enlargement is referred to as reactive lymphoid hyperplasia in which both T cells and germinal-center B cells undergo immune-mediated proliferation. Foreign bodies, in addition to acute infections, may induce granuloma formation. Recall that many foreign bodies can cause a giant cell reaction with accompanying chronically inflamed granulation tissue. Oral mucosal foreign body granulomas are seen with many dental materials, including amalgam and dental cements, handpiece oil (oil granulomas), and vegetable particles (legume or pulse granulomas).

A group of idiopathic diseases, collectively known as orofacial granulomatosis, is histologically characterized by multiple, often confluent foci of granulation tissue with giant cell formation in the absence of foreign material or a specific infectious agent. These are termed noncaseating granulomas, because unlike the granulomas of tuberculosis, there is no focus of caseous necrosis. Included in the orofacial granulomatosis group of lesions are Crohn disease, sarcoidosis, Melkerson-Rosenthal syndrome, and cheilitis granulomatosa (see Chapter 24). Submucosal masses comprised of granulomas also occur in response to infectious agents and are known as specific granulomatous inflammatory lesions. Included here are such specific infections as tuberculosis and deep fungal infections, the most common of which is histoplasmosis. The specific infectious granulomas are typically multinodular with an erythematous granular surface. Wegener granulomatosis is a systemic disease with multiple organ involvement that also manifests as a red granular swelling, usually confined to the fixed gingiva, so-called strawberry gums.

Swellings that diffusely involve the gingiva are the result of pathologic leukocytic infiltrates, such as might be encountered in leukemia, proliferation of granulation tissue in instances of nonspecific hyperplastic gin-
givitis, and overproduction of collagen in cases of drug-induced gingival hyperplasia (eg, Dilantin, nifedipine, cyclosporine), or the rare hereditary condition, familial fibromatosis gingivae.

As stated previously, true connective-tissue neoplasms are uncommonly seen in the oral cavity as compared with reactive and inflammatory masses. They may derive from any of the submucosal tissues, such as fibroblasts, lipocytes, nerve sheath, smooth muscle, skeletal muscle, vessels, osteoblasts, and chondroblasts. Sarcomas of these tissues are extremely rare. The proliferations contain cellular elements that are histogenically related to the normal tissues from which they arise and are named according to their histologic differentiation. The benign entities derived from mesenchymal or connective-tissues are variably encapsulated or are at least well circumscribed. A specific microscopic diagnosis is essential, since not all connective-tissue tumors behave in the same way. Some are aggressive, such as myofibromatoses, and have a tendency for local recurrence. Others are benign and have no tendency for recurrence after excision.

Minor salivary tumors also present as submucosal masses (see Chapter 26). They are most commonly found in the palate and buccal mucosa, but can arise in any location where minor glands are located. Clinically, benign salivary gland tumors are nonulcerated and show normal surface coloration, whereas the malignant types often exhibit surface telangiectasia, can be ulcerated, and are usually firm to palpation, owing to cellular proliferation. As with connective-tissue tumors, the salivary adenomas and adenocarcinomas are classified according to histologic patterns of differentiation. Recall that normal glands are comprised of ducts, acini, and myoepithelial cells. The various salivary-gland tumors are nonulcerated and show normal surface coloration, whereas the malignant types often exhibit surface telangiectasia, can be ulcerated, and are usually firm to palpation, owing to cellular proliferation. The common pleomorphic adenoma is a lesion comprised of benign proliferations of ducts and myoepithelial cells. In the malignant category, adenoid cystic carcinoma and polymorphous low-grade adenocarcinoma are composed of solid-tumor islands with additional foci of ductal formations. Mucoepidermoid carcinoma shows both acinar and ductal cells with squamous (epidermoid) and mucous acinar cell differentiation. Mucoepidermoid carcinomas show variations in cell patterns that allow for assignment into low- or high-grade subgroups that correlate with good and poor prognosis, respectively.

Masses of the facial skin can also, as in the mouth, be represented by inflammatory, infectious, developmental, and neoplastic processes. Diffuse swellings occur in edematous states and in inflammatory conditions, such as dental-space infections, cellulitis, and allergic reactions. Focal masses are often the consequence of either benign or malignant tumors that arise from the surface epithelium, adnexal structures (hair follicles, sweat glands, sebaceous glands), and the dermal connective tissues. The most common focal cancerous swelling of the face is basal cell carcinoma. It has been shown that these tumors, as well as those associated with the basal cell nevus syndrome, harbor mutations in the “patched” gene, a membrane tumor suppressor involved in the sonic hedgehog morphogen pathway. Jaw keratocysts harbor these same mutations.

**Clinical features of oral swellings**

The clinical features for the more common mucosal swellings vary according to each specific entity. As has already been emphasized, it is crucial that the clinician take note of the location, coloration, surface texture, and palpable nature of the mass before attempting to secure a definitive diagnosis. If the lesion shows the features of an abscess, then diagnostic testing for odontogenic or periodontal origin must be performed by obtaining radiographs, pocket probing, pulp vitality testing, and identifying pyogenic suppuration. If no apparent infectious source is uncovered, then biopsy may be the chief method for procurement of a definitive diagnosis. In the case of diffuse gingival enlargement, interrogation with regard to drug use is imperative. As in all cases of oral lesions, the clinician must obtain a thorough medical history, knowing that some swellings may be associated with systemic diseases. When biopsy is to be undertaken, a decision must be made as to whether the biopsy will be incisional or excisional, a consideration based on both the size of the lesion and the possibility of malignancy. If malignancy has a high priority in the differential diagnosis, then incisional biopsy is indicated (see Chapter 20). The lesional and diagnostic tissue lies deep in the submucosa, and therefore, an incisional biopsy must be taken to a significant depth within the tumefaction. A wedge or pie-shaped incisional biopsy is advisable in these situations, to get an adequate specimen.

**Traumatic fibroma**

Focal fibrous hyperplasia as a consequence of trauma underlies the pathogenesis of this common benign oral tumor. It is pink in color yet may have a white keratotic surface if it is repeatedly irritated. Most fibromas are round, dome shaped sessile, soft masses (Figure 23–2). They vary greatly in size and are usually asymptomatic. The most common sites are the lips, commissures, buccal mucosa, and tongue. When traumatic fibromas occur on the gingiva they are commonly referred to as peripheral fibromas. Some show unique histologic features and are designated gingival fibromas. Another variant that can occur anywhere in the mouth is the
giant cell fibroma, not to be confused with giant cell granuloma, an aggressive lesion of the gingiva. Fibromas are treated by simple excision as well as trying to identify and remove a possible causative irritant, which is not often apparent.

Inflammatory fibrous (denture) hyperplasia

The irritation from an overextended denture flange can irritate the submucosal connective tissue. This tends to occur under dentures when alveolar ridge resorption has caused the denture to overseat. The irritating flanges induce multinodular flabby masses along the maxillary or mandibular vestibule (Figure 23–3). This so-called epulis fissuratum, an older term for fibrous hyperplasia associated with denture irritation, was used in a descriptive sense because so many of these common lesions are lobulated, with intervening fissural depressions. The most common locations are the anterior maxillary and mandibular vestibules, but they can be located anywhere along the sites of denture compression and irritation. Recall that in the palatal vault denture hyperplasias are diffuse and papillary, a lesion termed inflammatory papillary hyperplasia.

Mucous extravasation phenomenon (mucocele)

Mucoceles are the result of minor salivary gland duct severage with resultant escape of mucus into the submucosal connective tissues. With no conduit for excretion, the mucus collects in the connective tissues, creating a pseudocyst (they lack an epithelial lining) which becomes walled-off with granulation tissue and if not removed, the wall becomes fibrotic. The duct severage is the consequence of biting, usually in the lower lip. Mucoceles rarely involve the upper lip yet may occur at any site where minor salivary glands are located.

The clinical presentation is that of a soft-tissue cyst that is soft and fluctuant (Figure 23–4). When superficial they are faintly blue; if walled-off or fibrosed they may feel more solid and have normal mucosal coloration. Occasionally, the patient pops the lesion (with a pin or by biting) and it resolves; however, it usually recurs. More often, is the tendency to remain or even enlarge. Surgical excision of the cystic tissue should be accompanied by removal of the underlying “feeder” minor glands. Large mucoceles in the floor of the mouth arise from severage of the sublingual or even the major submandibular duct and are referred to as ranulas. Some of these mucous extravasations extend deeply into the intrinsic muscles of the tongue and mylohyoid, so-called plunging ranulas.

Mucous retention cyst

True cysts of the minor salivary ducts are referred to as mucous retention cysts or sialocysts. These lesions mani-
fest the same clinical characteristics as the mucocele, being soft fluctuant bluish masses. Whereas some are probably true blind cysts lined by salivary ductal epithelium, others are ductal dilatations that develop as a consequence of ductal obstruction or occlusion by mucous plugs. These latter retentive cysts tend to occur in the buccal mucosa of older adults. Treatment is complete excision.

Reactive gingival tumefactions

Four pathologically related proliferations are encountered on the gingiva, usually arising in the interdental or gingival papilla region. All are reactions to irritation from calculus or particles that become wedged into the gingival sulcus (toothpick fragments, popcorn kernels, etc.). The host reacts to the irritant by hyperplasia of the endogenous tissues in the site, which include fibrovascular connective tissues, periodontal ligament fibroblasts, and periosteal tissues. Proliferation of granulation tissue gives rise to pyogenic granulomas, which may fibrose to peripheral fibroma; periodontal ligament cells give rise to cells capable of osteogenesis and cementogenesis, causing the ossifying fibroma; periosteal progenitor cells, including osteoblasts and osteoclasts, proliferate, producing a lesion termed peripheral giant cell granuloma (Figure 23–5). The term peripheral is employed to distinguish these common lesions from their less common counterparts that arise within the jaw bones. Pyogenic granulomas and peripheral ossifying fibromas are common in pregnancy.

Clinically either facial-buccal or lingual gingiva may be involved and there may be a history of rapid growth. The pyogenic granuloma is usually red; fibromas and ossifying fibromas are pale pink; and giant cell granulomas are bluish; and all can become ulcerated with a white pseudomembranous surface (Figure 23–6, 23–7, and 23–8). The peripheral giant cell granuloma is the more aggressive of these lesions, often eroding underlying alveolar bone and even causing root resorption (Figure 23–9).

**Figure 23–4** A, Mucocele of the lower lip; B, photomicrograph showing pooling of mucin under the surface epithelium with minor gland lobules in the deeper connective tissue; C, ranula in the floor of the mouth.

**Figure 23–5** Diagram depicting the various reactive lesions of the gingiva.
Wide surgical excision is the treatment. Recurrence occurs in over 20% of the cases, which can be minimized by adequate surgical excision coupled with root planing.

Peripheral odontogenic cysts and tumors

Although odontogenic tumors are encountered central in the jaws, recall that the dental lamina arises from the alveolar mucosa, and odontogenic rests, which can give rise to neoplasms and cysts, are located in the gingiva. The most common entity is the gingival cyst of the adult, a lesion that appears as a nodule on the attached gingiva and may erode the underlying cortex (Figure 23–10). Benign odontogenic tumors also occur here, the peripheral odontogenic fibroma being the most common (Figure 23–11). Rare peripheral odontogenic tumors that appear as gingival masses include ameloblastoma, dentinogenic ghost-cell tumor, and calcifying epithelial odontogenic tumor.

Diffuse gingival enlargements

Gingival hyperplasias can be nonspecific, drug-induced, hormonally-related, granulomatous, or even neoplastic. Nonspecific gingival hyperplasias often have the appearance of multifocal pyogenic granulomas, being soft, hemorrhagic, and fiery red. There is a systemic underlying hormonal influence in the pathogenesis of hyperplastic gingivitis when the patient is a female entering puberty or gravid (Figure 23–12). Such instances are often referred to as puberty and pregnancy gingivitis, respectively. The gingival lesions are typically associated with formation of pseudopockets.

Drug-induced gingival hyperplasias are diffuse, and the lesions may be of normal coral pink coloration or red and inflamed. Phenytoin, calcium channel blockers, and cyclosporine used in the treatment of seizure disorders, hypertension and cardiovascular disease, and immunosuppression for organ transplantation, respectively, are all responsible (Figures 23–13 and 23–14). The fibrosis of cyclosporine enlargement is not limited to the gingival tissues; indeed, renal, pulmonary, and retroperitoneal fibrosis are complications of this drug. These lesions do not resolve with drug withdrawal.

Familial fibromatosis gingivae is a rare disorder that is inherited as an autosomal dominant trait. The enlarged gingivae are firmly fibrotic and devoid of significant inflammatory erythema as a rule (Figure 23–15). Periodic gingivectomies are often requested by the patient for esthetic as well as functional reasons.

Wegener granulomatosis is a multisystem immunopathologic disease that affects the lungs, kidneys, skin, and middle ear. The enlarged gingiva is red and granular, often termed strawberry gums (Figure 23–16). A histopathologic diagnosis of Wegener granulomatosis warrants a systemic workup to examine for other sites of involvement. The serologic marker antineutrophil cytoplasmic antibody (ANCA) is of diagnostic importance, being found in over 85% of cases.

Varix

Focal varices are most common in the lower lip and are probably the consequence of trauma, such as lip biting, to the submucosal vessels. Venous channels proliferate and become dilated. These lesions may be flat or, more often, raised blue or purple masses (Figure 23–17). Some will blanch on diascopy (exerting direct pressure on the
lesion to detect blanching of vascular channels). Those that fail to blanch are often thrombosed. Treatment is elective. Injection with a sclerosing agent, such as Sotradecol™, may be effective. Surgical excision and laser ablation are common treatment options.

**Pulsatile labial artery**

The labial branch artery may develop a small aneurysmal dilatation that appears as a nodule or linear elongated mass of the lower lip. The lesion is usually of normal color because the vessel is deep and surrounded by the usual arterial muscularis coat. Sometimes pulsations can be observed visibly; in others palpation is required to detect the pulsatile nature of this vascular anomaly. If the patient elects to have the lesion removed, the

---

**Figure 23–7**  
*Figure 23–7*  
*A*, Peripheral ossifying fibroma is coral pink in color; *B*, photomicrograph of peripheral ossifying fibroma.

**Figure 23–8**  
*Figure 23–8*  
*A*, Peripheral giant cell granuloma; *B*, photomicrograph depicted the multifocal nature of giant cell granuloma extending to the base of the cut margin.

**Figure 23–9**  
*Figure 23–9*  
Radiograph showing saucerized zone of alveolar ridge resorption from an overlying peripheral giant cell granuloma.
mucosa should be incised and the vessel blunt dissected, followed by ligation and excision.

Parulis

Odontogenic infections that evolve into periapical inflammatory lesions may perforate the cortex, with drainage into the oral soft tissues. Focal drainage of an acute inflammatory process creates a tract that delivers suppurative material into the gingival submucosa. These drainage tracts may occur anywhere from the free gingival margin down to the vestibule (Figure 23–18). This submucosal abscess, or parulis, is associated with an endodontically involved necrotic tooth. Radiographs and pulp vitalometry testing disclose the incriminating tooth. If all teeth in the region of the parulis are vital, then the lesion may represent a focal periodontal abscess, and in such cases, a deep pocket is identifiable and probing causes exudation.

Specific granulomas

Granulomatous inflammation is characterized by granulomas with multinucleated giant cells. This type of histologic reaction is seen in foreign body reactions, orofacial granulomatosis, and such specific infections as tuberculosis and deep fungal infections. Specific granulomas occur most often in the tongue, vestibule, and buccal mucosa, where they appear as submucosal nodules, some being multinodular. Foreign body reactions are commonly found to contain fruit or vegetable material (puls granulomas), dental materials, or oil from handpieces, thereby representing iatrogenic lesions. Granulomas that represent specific microbial infections are often red with a granular “strawberry” appearance. They are generally firm to palpation. Biopsy with specific microbial stains usually allows the pathologist to identify the genus of the microorganism. Orofacial granulomatosis includes sarcoid, sarcoid-like diseases, and Crohn disease (see Chapter 24).
Ectopic lymphoid tissue and benign lymphoepithelial cysts

Ectopic lymphoid tissue is commonly seen in the oral cavity where it appears as a yellow nodular or multinodular mass (Figure 23–19). The common sites are the floor of the mouth and the soft palate. Many of these lymphoid aggregates emulate tonsilar tissue in that epithelial lined crypts extend into the lymphoid tissue and some become impacted with keratin, exhibiting a cystic appearance.

Amyloidosis

Amyloid is a pathologic fibrillar protein that accumulates within the connective tissues and is associated with certain neoplasms. Chemically, there are over 15 separate varieties, yet only three are of clinical significance: amyloid light chain (AL) protein, derived from plasma-cell-generated immunoglobulin light chains; amyloid-associated (AA) protein, which is made in the liver, and beta-2-microglobulin. Amyloid light chain protein is associated with primary amyloidosis and becomes deposited in tissues in patients with B lymphocyte proliferations, multiple myeloma being the most prevalent. Amyloid-associated protein is deposited in secondary amyloidosis, such as inflammatory lesions and tuberculosis, and beta-2 microglobulin is associated with long-term renal dialysis. In the oral cavity, these deposits are usually encountered on the tongue as multiple or, less
often, single nodules (Figure 23–20). The presence of amyloid in oral biopsies is determined by Congo red staining with subsequent demonstration of green birefringence under polarized light. The fluorochrome thioflavin T also stains amyloid, yet is not specific.

**Mesenchymal neoplasms**

A variety of neoplasms arise from the submucosal connective tissues, and such tumors appear as nodular swellings. They may show distinct clinical features, such as hemangioma and lymphangioma, or they may be nondescript, simply presenting as pink, smooth-surfaced tumefactions. Hemangiomas and lymphangiomas are considered to be developmental lesions, since these lesions often proliferate in infancy or childhood, and may spontaneously resolve during teenage years (see Chapter 25). Most mesenchymal tumors are found in the tongue or buccal mucosa, but they can occur anywhere in the mouth. The more common are nerve sheath tumors, including neurilemoma (schwannoma) and neurofibroma (Figure 23–21). The granular cell tumor is generally considered to be a nerve sheath tumor as well (Schwann cell origin) and is most commonly found in the tongue, where it appears as a yellow, smooth-surfaced, firm plaque or nodule (Figure 23–22).

Lipomas are typically located in the buccal mucosa, appearing as soft, yellow, single or multinodular masses (Figure 22–23). Choristomas are benign growths that aberrantly arise in locations that do not harbor the progenitor cells from which they arise. In the oral cav-
ity, chondroid (cartilaginous) choristomas are typically encountered as hard nodules in the submucosa of the tongue. Other benign mesenchymal neoplasms that are occasionally encountered in the oral cavity are rhabdomyoma, leiomyoma, nodular fasciitis, solitary fibrous tumor, and fibrous histiocytoma, to mention but a few (Figures 23–24 and 23–25). These specific entities are all treated by surgical excision, and have variable tendencies for recurrence.

Figure 23–22  A, Granular cell tumor of the tongue; B, photomicrograph of granular cell tumor with overlying pseudoepitheliomatous hyperplasia.

Figure 23–23  Lipoma with yellow coloration of the buccal mucosa.

Figure 23–24  Submucosal tumor of the lip.

Figure 23–25  Photomicrograph of a well-defined mesenchymal neoplasms, in this case a leiomyoma.
**Aggressive proliferations**

Certain mesenchymal proliferations are characterized by rapid growth and can reach large proportions; some actually invade adjacent soft and hard tissues despite their inability to metastasize. All are defined by their microscopic appearance. These aggressive mesenchymal tumors often lie deep within the facial tissues, floor of the mouth, tongue, or neck, and are firm to palpation. If there is any invasion of contiguous tissues, they are partially fixed, as assessed by palpation. Included in this group are fibrous histiocytoma, aggressive juvenile fibromatosis, hemangiopericytoma (some of which show malignant behavior), and hemangioendothelioma. Open biopsy or needle aspiration cytology are acceptable diagnostic procedures. Wide excision is generally required.

**Squamous cell carcinoma**

Chapter 20 discusses oral malignant epithelial neoplasms in detail. Late-stage tumors often present as indurated ulcerated masses, although some are non-ulcerated. Most squamous cell carcinomas are located in the tongue and lips and in the floor of the mouth (Figure 23–26). For oral cancers in general, the more anterior the carcinoma is located in the mouth, the better the prognosis; the more posterior, the worse the prognosis. This may be attributable to delayed diagnosis (advanced stages), intrinsic cell proliferation differences, or greater lymphatic drainage in those areas. Incisional biopsy is recommended for diagnosing these tumefactive indurated lesions. Treatment planning is complex, with the more advanced-stage lesions requiring more aggressive treatment. Overall prognosis is poor, with only about 50% surviving.

**Salivary gland tumors**

Chapter 26 discusses each of the histologic types of salivary gland tumors. Some types that are commonly found in the major glands are rare or may never be found in the minor glands of the mouth, conversely, there are minor salivary gland tumors that rarely arise in the major glands. Intraoral minor gland tumors present as submucosal masses and are as apt to be malignant as they are to be benign. The most common site is the palate, where the mass is off the midline, arising in the posterior aspect of the hard palate or at the hard–soft palate junction (Figure 23–27). The buccal mucosa, upper lip, and ventral tongue are also common sites for these neoplasms. The benign tumors that occur in minor glands include the pleomorphic adenoma, monomorphic adenoma, and canalicular adenoma, the latter arising almost exclusively in the upper lip, where it may be multifocal. Polymorphous low-grade adenocarcinoma is a common malignant minor salivary gland tumor that almost never arises in the major glands. Other adenocarcinomas arising in the oral mucosa are mucoepidermoid carcinoma, adenoid cystic carcinoma, and adenocarcinoma not otherwise specified. There are many other rare histologic types that can arise in either major or minor glands. Clinically, the benign adenomas are typically smooth surfaced, nonul-
cerated nodules that are movable, unless located in the palate, where the tumor is trapped between the palatal bone and mucosa. Malignant salivary gland tumors are indurated (low-grade mucoepidermoid carcinoma being the exception) and may show surface ulceration and telangiectasia. Incisional biopsy is the diagnostic procedure of choice; fine-needle aspiration is also useful for diagnosis. Primary treatment is surgical removal. Based on the histologic type and surgical margins, postsurgical radiation therapy can be used. Adenomatous hyperplasia is seen in the soft palate and floor of the mouth and represents a benign nonneoplastic growth of normal salivary tissue (Figure 23–28).

**Sarcomas and lymphomas**

Malignant mesenchymal neoplasms are rarely encountered in the oral soft tissues, being much more prevalent in the neck. Sarcomas can arise anywhere, and cases have been reported in the tongue, buccal mucosa, and oral floor, being extremely rare in other locations (Figure 23–29). Rhabdomyosarcoma, fibrosarcoma, and malignant fibrous histiocytoma have been the more frequent sarcomas reported to arise in the oral cavity (Figure 23–30). These malignancies account for less than 5% of all oral cancers.

Whereas lymphomas are usually seen in the cervical lymph node chain, extranodal non-Hodgkin lymphomas are encountered in the oral cavity. They are far more common in the human immunodeficiency virus (HIV)-infected patient, where they tend to occur on the buccal and palatal gingiva. The masses are firm, rapidly growing, often multinodular, and may show surface ulceration. Microscopically they are usually high-grade lesions populated by monoclonal B lymphoblasts with a diffuse medium or large cell morphology. Patients with acquired immunodeficiency syndrome (AIDS) presenting with lymphoma generally succumb within 6 months of diagnosis. In the United States, HIV-associated lymphomas account for approximately 25% of all lymphomas reported each year (see Chapters 8 and 14).

Atypical lymphoproliferative lesions represent a lymphoid infiltrative disease of the palate that invades minor salivary tissue while leaving the extralobular ducts relatively well preserved. These lesions appear as unilateral, soft, boggy, diffuse swellings at the hard–soft palate junction. Some are polyclonal B-cell lesions that are probably reactive; others are monoclonal and likely represent low-grade mucosa-associated lymphoid tissue (MALT) lymphomas. These lesions are responsive to low-dose radiation therapy.

**Clinical features of facial tumors and swellings**

The swellings that are seen on the face can be clinically divided into two major types: those that are diffuse
and those that are focal nodules. As alluded to previously, diffuse swellings are usually inflammatory lesions, such as edema, emphysema, space infection, or cellulites. Facial asymmetry also may be seen when there is an underlying central lesion of the maxillary or mandibular bone and, of course, such lesions are bony hard. Radiographs are necessary diagnostic and evaluative tools.

Focal nodules of the face may be covered by normal skin or they may be verrucous, ulcerated, or pigmented. Most small facial nodules are sebaceous cysts, basal cell carcinomas, nevi, and seborrheic keratoses, the latter two being pigmented. Less common are squamous cancers, melanomas, and mesenchymal neoplasms.

**Odontogenic infections**

Buccal drainage from a periapical abscess can result in significant facial swelling, which may localize over the mandible or, less frequently, below the zygoma. As the infection progresses through the buccal plate, bacteria and the host response to it result in purulent exudates. If this suppurative process is confined to spaces bordered by muscle and fascia, the diffuse swelling is soft or fluctuant and tender to palpation. Alternatively, if the infectious process infiltrates into muscle and leukocytic infiltrates are interposed within the substance of the muscle itself, then the lesion becomes indurated, a process termed cellulitis. The clinical distinction between cellulitis and space infection is germane to treatment, since the former cannot be incised and drained, whereas the latter is amenable to such intervention. Of course, the incriminated tooth must be treated as well. Depending upon the status of the tooth, endodontic therapy or extraction must be performed, and antibiotic therapy is indicated in these examples of more extensive spread of infection. In some instances, the periosteum reacts to underlying odontogenic infection, giving rise to proliferative periostitis (Figure 23–31).

**Soft-tissue emphysema**

On rare occasion, air may be forcefully introduced between tissue planes. This may occur during maxillary endodontic, periodontal, and oral surgery procedures in which compressed air is applied and separates the tissue planes. The entrapped air causes a diffuse facial swelling that is crepitant to palpation. A potentially lethal complication is vascular air embolism, an event that usually occurs shortly after the introduction of compressed air into the tissues. Aspiration of the swollen area may be attempted with caution, making sure not to puncture a major vessel in the process. Eventually, the trapped air is absorbed by the tissues.

**Seborrheic keratosis**

Sun exposure to the facial skin damages DNA and may induce proliferative responses in the surface epithelium. This exposure may result in the formation of seborrheic keratosis, a benign entity. This common lesion is usually seen on the forehead, temples, or malar regions of the face. The lesions are slightly tumefactive, brown in color, and have an oily texture. They vary considerably in size and are typically symmetrical, with smooth well-delineated borders. The differential diagnosis includes nevus and basal cell carcinoma. Most “seb Ks” can be removed by shave biopsy or laser ablation.

**Melanocytic lesions**

The common nevi are discussed in more detail in Chapter 22 on pigmentation; not all nevi, however, are pig-
mented. They can occur anywhere on the facial skin and are present from early childhood, with no history of any change or increase in size. In adults, they all represent intradermal nevi. The pigmented nevi are symmetrical, round and nodular. The nonpigmented nevi simply appear as nonproliferative nodules of normal skin coloration. Treatment is deferred unless the patient wants them removed for esthetic reasons, or if they are near the hairline and become easily irritated. Should any long-standing nevus begin to increase in size, ulcerate, or deepen in color, biopsy is recommended to rule out dysplastic change in a preexisting nevus. Melanomas are also discussed in Chapter 22.

Mesenchymal neoplasms

A variety of mesenchymal tumors can arise from the subcutaneous tissues of the facial skin. Hemangiomas appear red or purple when superficial (Figure 23–32), or they may not be discolored when they are deep and intramuscular (Figure 23–33). Neurilemomas, neurofibromas, and lipomas are relatively common connective-tissue tumors that appear as subcutaneous masses (Figure 23–34).

Basal cell carcinoma

Basal cell carcinomas (BCC) are the most frequently occurring malignancy in the United States, accounting for more than one million new cases each year. Sun exposure is also a factor in the etiology of BCC, which explains why those with fair skin and less melanocytic protection are more prone to the development of these skin cancers. They may appear as pearly nodules with surface telangiectasia, or they may present as nonhealing ulcers (Figure 23–35). The ulcerated basal cell carcinomas usually develop rolled borders. It is common for these skin cancers to be multifocal. They are more often seen on the upper face, helix of the ear, and scalp than on the lower face and lips. Wide local excision is required for cure, and despite adequate surgery, some recur, presumably owing to a field effect of dysplastic change that many occur in many areas of the facial skin. Radiation therapy is also effective in controlling BCC.

Prognosis is good, since few BCCs metastasize. However, they spread locally and insidiously, so if not diagnosed and treated early, the treatment defect can be disfiguring. Basal cell carcinoma does not arise in the mucosal tissues of the mouth. Some interesting cases have been reported of BCC occurring in skin grafts that have been placed in the mouth for repair or closure purposes.

Variant benign tumors of skin appendages are known as adnexal skin tumors. These lesions present as
smooth-surfaced nodules, because the cells of origin lie within the dermis. Most are benign and are treated by simple excision.

**Squamous cell carcinoma**

Arising de novo or from preexisting actinic keratoses, squamous cancers of the facial skin have potential for both regional node and distant metastases. The actinic keratoses are reddish-brown macules with a superficial scaley keratotic crust. They are typically found on the forehead, nose, cheeks, and lower lip. When carcinomatous change arises from these precancerous dysplasias, the lesions become tumefactive, indurated, and ulcerated (Figure 23–36). Ulcerated growths on the eyelids, particular the lower lid, tend to arise from malignant transformation of the dermally situated sebaceous glands (sebaceous carcinoma). Regional nodes become palpably enlarged when metastases evolve. Treatment consists of wide local excision and/or radiation therapy; management of the neck is contingent upon clinical or imaging findings of nodal disease. On the lips, squamous cell carcinoma is common, and BCC is rare (see Chapter 20).

**Suggested reading**