Understanding the pathology of the oral cavity and pharynx requires a relatively sophisticated knowledge of the anatomy of the region. This article begins with a review of the relevant anatomy. A discussion of pathologic entities follows and is divided into congenital, inflammatory, and neoplastic lesions.

ANATOMY

The oral cavity includes the oral tongue (the anterior two thirds of the tongue); hard palate; floor of the mouth; retromolar trigone; lips; buccal mucosa; alveolar ridges; and gingiva (Fig. 1). The mandible and maxilla are the defining bony margins of the oral cavity. These are the structures in the anterior aspect of the upper aerodigestive system as opposed to the pharynx, which is predominately posterior to the oral cavity and larynx.

The superior most portion of the pharynx is the nasopharynx, which has three subsites defined by the American Joint Committee on Cancer: (1) the posterosuperior wall, (2) the lateral wall, and (3) the anteroinferior pharyngeal wall. The posterosuperior wall extends to the skull base and the underlying tissue includes the retropharyngeal fat and the longus colli and capitis muscle complex. Along the lateral nasopharyngeal wall there are two indentations and one protrusion into the lumen. The indentations (recesses) are the eustachian tube orifice and the fossa of Rosenmüller (lateral pharyngeal recess), whereas the protrusion is the torus tubarius (the cartilaginous portion of the eustachian tube) (Fig. 2). The posterior pharyngeal wall extends from the skull base to the level of the plane of the hard palate–soft palate junction.

The oropharynx includes the base of the tongue; the vallecula; the soft palate and uvula; the lateral pharyngeal walls including the palatine tonsils and tonsillar pillars; and the posterior pharyngeal wall, extending from the plane of the soft palate–hard palate junction to the level of the pharyngoepiglottic folds at the hyoid bone (Fig. 3). The base of the tongue is the posterior one third of the tongue behind the plane of the circumvallate papillae.

The hypopharynx extends posteriorly from the lateral pharyngoepiglottic folds inferiorly to the upper esophageal segment and includes the postcricoid segment, the piriform sinuses, and the lateral pharyngeal walls (Fig. 4). Importantly, the anteromedial margins of the piriform sinuses are the lateral walls of the aryepiglottic folds (a portion of the supraglottic larynx).

The importance of knowing the distinctions between the nasopharynx, oral cavity, oropharynx, and hypopharynx lies in the T (tumor) of the TNM staging of squamous cell carcinoma of the aerodigestive system. In most sites, T4 lesions (the most advanced T stage) usually imply extension beyond the margins of the specific region being examined. Therefore, a lesion that arises in the base of the tongue that extends into the oral tongue becomes T4 as it leaves the oropharynx. Thus, knowledge of the anatomy is critical to the accurate T staging of head and neck cancers. The T staging helps define treatment options and allows for more objective data collection for tumor registries.

To review, the junctions between oral cavity and oropharynx are at the hard palate and soft palate, the oral tongue and base of the tongue, and the floor of the mouth and tonsil. Between the nasopharynx and oropharynx the demarcation posteriorly is at the plane of the soft palate and its superior and inferior surface. Between the oropharynx
Figure 1. Oral cavity anatomy. A, Axial T1-weighted MR scan demonstrates the masseter muscle (M), the pterygoid musculature (P), the orbicularis oris muscle (O), the buccinator muscle (black arrow), the intrinsic muscles of the tongue (i), the maxillary teeth (T), the retromolar trigone (white arrow), the pterygomandibular raphe (star), the palatine tonsil (asterisk), and the mandible (arrowhead). B, T1-weighted MR section goes through the genioglossus muscle (g), the styloglossus muscle (s), the hyoglossus muscle (hm), and the junction of the styloglossus and hyoglossus muscles intertwining (short arrow), medial to the sublingual glandular tissue. The mylohyoid muscle (m) is barely visible at its superior attachment to the mylohyoid line of the mandible. High signal intensity fat is seen in the midline lingual septum just medial to the genioglossus muscle (g). The mylohyoid muscle forms the floor of the mouth. C, The buccinator muscle (B), genioglossus muscle (g), geniohyoid muscle (H), anterior belly of the digastric muscle (d), mylohyoid muscle (solid black arrow), platysma (open black arrow), hard palate (P), and masseter muscle (M), can be identified.
ORAL CAVITY AND PHARYNX

Figure 2. Nasopharyngeal anatomy. A, At the superior most portion of the nasopharynx, seen on this T1-weighted axial scan, the relationship of the longus capitus muscle (asterisk), clivus (C), and nasal septum (S) can be appreciated. B, At a somewhat lower level, the classic anatomy of the nasopharynx with the lateral recess (fossa of Rosenmuller) (arrow), torus tubarius (T), and the eustachian tube orifice (curved arrow) can be identified. The separation between the tensor veli palatini muscle and the levator veli palatini muscles can be seen with the intervening fat (open black arrow). The pterygoid muscle (P), temporalis muscle (asterisk), and masseter muscle can be seen.

Figure 3. Oropharyngeal anatomy. A, On this axial T1-weighted scan, the plan of the circumvallate papillae can be identified (arrowheads), delineating the anterior margin of the base of the tongue (B). The palatine tonsils (T) are also a component of the oropharynx. B, Further inferiorly, the vallecula is the air space anterior to the epiglottis (white arrow) and posterior to the base of the tongue (B). The vallecula usually is separated into two sections by the median glossoepiglottic fold.
and hypopharynx the plane of the hyoid bone and pharyngoepiglottic fold separates the two with valleculae above and medial to piriform sinuses.

CONGENITAL LESIONS

Congenital lesions of the oral cavity and pharynx most commonly occur within the tongue. Here, one can have lesions due to the arrest or incomplete migration of thyroid tissue from the foramen cecum at its origin from the apex of the circumvallate papillae through the tongue base and floor of the mouth to the lower neck. Therefore, lingual thyroid tissue and thyroglossal duct cysts may occur within the oropharynx (base of the tongue); oral cavity (floor of mouth); or below and anterior to these structures. Thyroglossal duct cysts account for over two thirds of congenital neck anomalies. Although most (65%) thyroglossal ducts occur in the infrahyoid neck, approximately 25% may arise in the tongue base or bulge into the floor of the mouth (Fig. 5). They are usually unilocular cystic masses that may have high signal intensity on T1-weighted scans, but more often have signal intensity characteristics of pure cysts. In the suprathyroid location they are usually in the midline. On the other hand, if one finds a hyperdense tongue base mass on noncontrast CT scanning, one should strongly consider a lingual thyroid gland. Because this may be the only functioning thyroid glandular tissue in the patient's body, it behooves the radiologist to recommend a thyroid scintigram to search for other thyroid tissue. Otherwise, resecting the lingual thyroid gland necessitates lifetime thyroid replacement hormone and, if not recognized as such, may lead to cretinism in childhood.

Other congenital masses that may occur in the tongue include hemangiomas and other vascular malformations. Seventy percent of cavernous hemangiomas resolve on their own by the time of adolescence and 50% have an associated skin hemangioma. Most cavernous hemangiomas of the head and neck have recently been renamed venous vascular malformations. CT shows peripheral or diffuse enhancement in venous vascular malformations with or without phleboliths (Fig. 6). The lesions are lobulated, well-defined masses. MR imaging intensities may be heterogeneous. The differential diagnosis of vascular masses in the nose includes pyogenic granulomas, juvenile angiofibroma, and hemangiopericytomas. The classification proposed by Mulliken and Glowacki suggests that the term hemangioma be reserved for lesions with cellular proliferation that present in infancy and usually involute by adolescence. What was previously called capillary hemangiomas are now called capillary malformations, which are slow-flow lesions often associated with Sturge-Weber syndrome. Capillary hemangiomas may occur superficially in the skin around the oral cavity and nose where they are seen as well-circumscribed, intensely enhancing masses often with bony remodeling. In addition there are arteriovenous, venous, capillary, or lymphatic vascular malformations. The major distinction between hemangiomas and the venous vascular malformations is that the latter do not involute and may actually grow with time, hormonal influences, infection, thromboses, or trauma. Phleboliths are more characteristic of cavernous hemangiomas. Both sets of patients may present with a discolored skin lesion that is compressible.

Lymphangiomas may occur as a purely lymphatic-derived lesion or as part of a combination
Figure 6. Venous vascular malformation of the tongue. This contrast-enhanced CT scan shows phleboliths (arrows) in a mass that has enhancing and nonenhancing portions to it within the oral and oropharyngeal tongue. The lesion extends into the subcutaneous tissues around the mandible and into the submandibular gland and parapharyngeal fat on the right side.

lesion in association with hemangiomas. Lymphangiomas are often multiloculated and may have high signal intensity on T1-weighted scans and hyperdensity on CT if they have been previously traumatized or infected. The lesions may not necessarily remain within the tongue but may extend out and broach other fascial planes (Fig. 7). Although they characteristically do not show contrast enhancement, when they are combined with hemangiomas, the lesion may have focal areas of contrast enhancement.

Dermoid and epidermoid cysts also occur within the tongue and may have typical signal intensity characteristics of these lesions elsewhere in the head and neck region or intracranial compartment. Fluid attenuation in epidermoid cysts and fat in dermoids or teratomas may differentiate the two. Dermoid cysts of the tongue often have a characteristic appearance with what appears to be a mosaic within the lesion with polygonal hypodense areas surrounded by diffuse hypodense areas within the lesion. Alternatively, one might find dermoid cysts that are entirely hypodense and nonenhancing.

Other congenital lesions of this region include cutaneous and osseous lesions, such as lateral and medium cleft lips due to incomplete closures of the nasomedial process and nasolateral processes with the maxillary processes. Although these may simply include the soft tissue of the lip, occasionally they also extend through the bony structures of the maxilla and palate (cleft palate).

INFLAMMATORY LESIONS

The most common inflammatory lesions to affect the oral cavity and pharynx are of odontogenic or tonsillar origin. Odontogenic infections are covered elsewhere in this issue; however, a few comments are appropriate with respect to oral cavity pathology. Odontogenic infections of the maxillary or mandibular molars can produce abscesses or infection of the buccal, masseteric, sublingual (fossa), and submandibular spaces. Palatal, mucosal, and facial abscesses can be seen. Extension of odontogenic infection can lead to abscesses of the secondary spaces, specifically the lateral (para) pharyngeal, retropharyngeal (danger zone), and prevertebral spaces. From the masticator space, odontogenic infections can spread into the retropharyngeal trigone or floor of the mouth and present with abscesses that might seem remote from the teeth. Usually there is superficial cellulitis—true osteomyelitis is rare. The whole oral cavity may be inflamed on the side of the infection; extraction of the infected teeth may actually exacerbate symptoms (Fig. 8).

Although pharyngitis is a universally endemic infection particularly common in children, imaging is reserved for potential complications of pharyngitis and or tonsillitis. Peritonsillar abscesses can occur and may present with symptomatology of sore throat and neck pain as well as high fever and trismus. In a review of head and neck space infections in children, the most common site was the peritonsillar location (49%), followed by the retropharyngeal space (22%), and the submandibular region (14%). Infections in the first two sites are likely the sequelae of tonsillitis, pharyngitis, or upper respiratory tract viral infections. From a peritonsillar location, infection may spread into the parapharyngeal space and cause septic thrombophlebitis of the internal jugular vein or septic aneurysms of the internal carotid artery. Plain films may demonstrate widening of the prevertebral soft tissue, but CT or MR imaging are most
Figure 8. Odontogenic infection. This CT scan shows diffuse infiltration of the subcutaneous tissues and masticator space caused by an odontogenic infection. Within the masseter muscle there are areas of fluid density representing abscess formation (asterisk). The parapharyngeal fat (arrow) is displaced posteromedially by the masticator space process.

Helpful to delineate the peripherally enhancing abscess collection that must be drained.

Occasionally, a second branchial cleft cyst drains into the tonsillar fossa and this should be considered when a fistula there is identified. Alternatively, actinomycosis or dermal sinus tracts could cause peritonsillar or tonsillar fistulae. After a tonsillitis, one might identify calcifications (tonsilloliths), which are a marker of chronic or previous tonsillitis. This is less commonly seen in the adenoidal tissue.

Mucous retention cysts due to inflamed mucous secreting glands may occur anywhere along the oral cavity and pharynx. In the nasopharynx Tornwaldt's cyst may be unique in that it is hyperintense on T1-weighted scans and is a midline lesion occurring due to retraction of the nasopharyngeal mucosa during the ascent of the notochordal tissue (Fig. 9).24

Inflammation in the buccal mucosa opposite Stensen's duct or in the floor of the mouth along Wharton's duct may coexist with salivary calculi and sialadenitis. Microabscesses may occur within the glands and lead to ductal inflammation (sialodochitis) and subsequent purulent drainage into the oral cavity.

With adjacent infections, the retropharyngeal space may demonstrate hypodensity and enlargement. Often, this represents edema rather than true inflammation (or abscess); however, a spectrum of disease from edema to retropharyngitis (a cellulitis of the retropharyngeal space) to phlegmon (a diffuse infected locular collection in the retropharyngeal space) to a retropharyngeal abscess may occur. Spread of infection down the retropharyngeal or danger space may lead to mediastinitis because the space extends to the upper and middle thoracic levels.

**BENIGN NEOPLASMS**

Benign neoplasms of the oral cavity and oropharynx include those derived from minor salivary gland tissue. Pleomorphic adenomas (benign mixed tumors) are the most common benign tumors of the minor salivary gland and are most commonly located in the hard or soft palate (Fig. 11).4 They may appear bright on T2-weighted scanning and typically show contrast enhancement. Canalicular adenomas are next most common in the palate and are radiologically indistinguishable from pleomorphic adenomas. Occasionally, pleomorphic adenomas may be heterogeneous lesions that may have fat or calcification within them.

A pseudomass of the oral cavity and pharynx is not (simple ranula) perforate into the submandibular space is that the former is treated surgically from an incision below the mandible, whereas the latter might be treated with intraoral or transoral excision.

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lymphoid hyperplasia. This may be seen in the nasopharyngeal adenoidal tissue, the oropharyngeal palatine tonsillar tissue, and the oropharyngeal lingual tonsillar tissue. Although lymphoid hyperplasia or lymphoid tissue enlargement is common in young age (less than 30 years old), it may also be seen in adults over 30 years old who have had chronic infections, HIV infection, other viral illnesses, or as part of the posttransplant lymphoproliferative disorder. Patients who chronically irritate their oral and oropharyngeal mucosa (smokers, alcohol abusers, chewing tobacco enthusiasts, or betel nut chewers), or who have recurrent chronic rhinitis or sinusitis have a propensity for lymphoid hyperplasia.

Juvenile angiofibromas probably arise near the sphenopalatine foramen and grow into the nasopharynx early in their course. They are characterized by their hypervascularity and their growth along the foramina of the pterygopalatine fossa (Fig. 12). Clinically, they occur almost exclusively in adolescent boys and they present with epistaxis. A higher incidence in the Far East than in America has been noted. Grading of the lesions is by extent of spread: intracranially or into the orbit (grade 3); into the pterygopalatine fossa or masticator space (grade 2); or confined to nasopharynx (grade 1). Imaging is very helpful in grading and treating these lesions. The vascular supply to these tumors is usually through branches of the internal maxillary, ascending pharyngeal, and ascending palatine arteries. Classically, the lesions displace the posterior wall of the maxillary antrum forward (Holman-Miller sign).

Schwannomas and amyloidomas might present as benign masses in the oral cavity or oropharynx. The latter might be hyperdense on noncontrast CT scan, raising the possibility of a lingual thyroid gland in the differential diagnosis. Benign bony masses include osteomas, fibrous dysplasia, and other fibro-osseous lesions of the mandible and maxilla. Odontogenic bony masses include cementomas, Pindborg tumors, odontomas, and ameloblastomas.

**MALIGNANT NEOPLASMS**

Most of the imaging that is performed in the pharynx and oral cavity is done to stage squamous cell carcinomas of the head and neck. Because each site has unique issues associated with it, this article separates the discussion of cancers into the regions of the nasopharynx, oropharynx, hypopharynx, and oral cavity.

**Nasopharyngeal Carcinoma**

Most nasopharyngeal carcinomas are associated etiologically with exposure to the Epstein-Barr vi-
Figure 12. Juvenile angiofibroma. A, This CT scan shows the classic appearance of a juvenile angiofibroma displacing the posterior wall of the maxilla anteriorly and extending from the nasal cavity across an enlarged pterygopalatine fossa into the masticator space on the left side. B, In a different patient, the MR scan shows the classic appearance of signal voids (small arrows) within the angiofibroma indicating its hypervascular nature.

ruses and there is also a high rate of occurrence in individuals of Southeast Asian descent, particularly in the Cantonese dialect group of Chinese. Undifferentiated carcinomas and nonkeratinizing carcinomas are very highly associated with Epstein-Barr virus exposure and account for 63% and 12%, respectively, of nasopharyngeal carcinomas. Squamous cell carcinoma represents the remaining 25% of cancers in this region and has the least avid association with Epstein-Barr virus exposure.

Nasopharyngeal carcinomas are staged according to subsite (posterosuperior wall, lateral wall, and anteroinferior wall) and presence of spread outside of the nasopharynx as follows:

- **T1**: Tumor confined to the nasopharynx
- **T2**: Tumor extends to soft tissues of oropharynx or nasal fossa
- **T3**: Tumor invades bony structures or paranasal sinuses
- **T4**: Tumor with intracranial extension or involvement of cranial nerves, infratemporal fossa, hypopharynx, or orbit

When evaluating a patient for nasopharyngeal carcinoma it is important to attempt to determine whether or not more than one subsite of the nasopharynx is involved because this distinction increases the T staging from T1 to T2 (Fig. 13). Note the presence or absence of invasion into the nasal cavity or the oropharynx (T3 stage) or invasion of the skull base or cranial nerves (T4 stage). Because the higher the stage the worse the prognosis, accurate staging is important for patient consultation and treatment management.

MR imaging is the study of choice to stage nasopharyngeal carcinoma because it more accurately depicts the tumors and demonstrates the skull base or the cranial nerve invasion of T4 cancers. Most nasopharyngeal carcinomas occur along the lateral wall of the nasopharynx and, because this may be a confusing area where adenoidal tissue may be asymmetric in normal individuals, it is important to be able to distinguish normal variation from early cancer. Mere asymmetry in the degree of aeration of the fossa of Rosenmüller is not a reliable sign for nasopharyngeal carcinoma because retained secretions and asymmetric lymphoid hyperplasia can produce this finding. Obliteration of the hyperintense fat stripe between the tensor and levator veli palatini muscles on a T1-weighted scan may be one of the earliest suggestions that a nasopharyngeal carcinoma may be present. A second reliable imaging feature is the infiltration of the parapharyngeal fat, which occurs quite early in nasopharyngeal carcinoma. At the time of presentation 60% of patients with nasopharyngeal carcinoma have parapharyngeal fat infiltration. Obstruction of the eustachian tube with a serous middle ear effusion or otomastoiditis may also be a harbinger of nasopharyngeal cancer. The anterosuperior width of the adenoidal tissue should be under 1 cm in adults; widening should raise the possibility of cancer versus lymphoid hyperplasia. Sagittal MR imaging may be the best way to gauge the adenoidal width.

MR imaging also has the advantage in demonstrating infiltration of the retropharyngeal fat (obliteration of the hyperintense fat stripe behind the nasopharyngeal mucosa but anterior to the longus capitis–collis complex). Abnormal signal intensity and enhancement may be seen in infiltrated prevertebral muscles; preservation of normal signal intensity and contour best predicts lack of invasion. It is not uncommon to see nasopharyngeal carcinoma grow through these prevertebral muscles and into the clivus or skull base marrow without a tremendous amount of distortion of the normal architecture of the tissue there (Fig. 14). Once the lesion has violated the retropharyngeal space, extension along the cranial nerves via the numerous foramina at the skull base (most commonly the
fifth cranial nerve branches) can occur readily.\(^6\)\(^7\) Growth along the eustachian tube into the middle ear cavity or along the skull into the jugular foramen such that the 9th, 10th, and 11th cranial nerves may be affected are two other routes of spread of nasopharyngeal carcinoma.\(^5\)

Skull base erosion is seen in up to one third of cases of nasopharyngeal carcinoma.\(^6\) Chong et al's paper showed that MR imaging was superior to CT in detecting tumor that involves the clivus (23%), petrous apex (30%), foramen ovale (25%), and sphenoid wing (14%).\(^6\)

Because the TNM staging of head and neck cancers also emphasizes the nodal chains (N staging) it is important to observe carefully the retropharyngeal lymph nodes of Rouvière as well as the high jugular chains (levels 2 and 3) for the presence of pathologic lymphadenopathy.

Nasopharyngeal carcinoma is not a surgical disease. Combined modality chemoradiation and radiotherapy is the primary treatment regimen. Therefore, the main role of the diagnostic radiologist is to determine accurate staging; to define the margins of the tumor for the therapeutic radiologist (radiation oncologist); and to assess the efficacy of radiation or chemotherapy. Once radiation dosage limits have been reached, brachytherapy (implants) may be used and occasionally surgical debulking of anteroinferior disease may be contemplated. Usually chemotherapeutic manipulations are the last option for patients with residual or recurrent disease.

Lymphoma, minor salivary gland carcinomas, and rhabdomyosarcomas may also occur in the nasopharynx. The absence of necrosis in a large lesion might imply the diagnosis of lymphoma. The presence of perineural spread of a tumor suggests adenoid cystic carcinoma, the most common malignancy of the minor salivary glands; however, squamous cell carcinoma and lymphoma could track along nerves too. A child with a nasopharyngeal malignancy might suggest a rhabdomyosarcoma. Nonetheless, squamous cell carcinoma must always be considered at the top of the list.

**Oropharyngeal Squamous Cell Carcinoma**

The tonsil and tongue base are the most common sites of origin of oropharyngeal squamous cell carcinoma. Staging of oropharyngeal carcinoma consists of size criteria for T1 through T3 cancers as follows:

- **T1:** Tumor is 2 cm or less in its greatest dimension
- **T2:** Tumor is more than 2 cm but not more than 4 cm in its greatest dimension
- **T3:** Tumor is more than 4 cm in its greatest dimension
- **T4:** Tumor invades adjacent structures, including bone (mandible or maxilla); soft tissues of the neck; or deep (extrinsic) muscles of the tongue, pterygoid, hard palate

Extension outside of the oropharynx constitutes T4 cancer. Therefore, it is important to remember the boundaries of the oropharynx (hard palate–soft palate junction along the posterior pharyngeal wall, oral tongue–base of tongue, vallecula–epiglottis) in order to determine whether spread has occurred beyond the confines of the oropharynx.

In addition, there are a handful of critical areas that determine the extent of surgery for oropharyngeal carcinoma. These include the (1) pre-epiglottic fat, (2) mandible, (3) maxilla, (4) prevertebral musculature, (5) pterygopalatine fossa, (6) pterygomandibular raphe, and (7) midline of or deep invasion into the base of the tongue. Identifying whether there is tumor infiltration of each of

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**Figure 13. Nasopharyngeal carcinoma.** A. The primary tumor (arrow) is less easy to identify specifically than the lymph node metastasis (+). The fat stripe between the levator and tensor veli palatini muscles has been interrupted (curved arrow). B. This is the more classic appearance of nasopharyngeal carcinoma with extensive infiltration at the time of diagnosis, with obliteration of the retropharyngeal fat anterior to the longus colli (C) muscles. Growth into the nasal cavity is present as well as parapharyngeal spread.
Figure 14. Skull base invasion by nasopharyngeal carcinoma. A. The sagittal T1-weighted MR scan is helpful in identifying the infiltration of nasopharyngeal carcinoma into the skull base with obliteration of what was the clivus and the anterior arch of C1. The tumor (T) extends below the plane of the hard palate and soft palate into the oropharynx. B. The gross bony destruction is often better identified on a CT scan.

Pre-epiglottic Fat Invasion. It is important to assess for invasion of the pre-epiglottic fat. A tumor of the base of the tongue that does not involve the pre-epiglottic fat can be resected without requiring a portion of the supraglottic larynx to be included in the operative specimen. On the other hand, if the pre-epiglottic fat is invaded, the patient often requires a partial supraglottic laryngectomy or, at worse, a total laryngectomy. The combination of base of tongue surgery with laryngeal surgery often leads to a poor quality of life in which both swallowing and speaking are compromised. The pre-epiglottic fat is best assessed with sagittal T1-weighted scans and axial T1-weighted scans in which soft tissue signal intensity within the fat suggests cancerous involvement (Fig. 15). Occasionally, adjacent inflammation or peritumoral edema or partial volume effects might stimulate pre-epiglottic fat invasion.

Mandibular Invasion. Imaging is utilized to attempt to identify tumor adjacent to the mandible, cortical erosions, infiltration of marrow fat, or tumor on both sides of the mandible. Particularly when a lesion arises on the alveolar surface of the mandible, single-plane imaging may be insufficient to determine mandibular invasion. For this reason, when the issue of mandibular involvement is raised, axial and coronal images are recommended either with CT or MR imaging (Fig. 16). Depending on the extent of involvement, the oral cavity and oropharyngeal cancer adjacent to the mandible are treated differently. If tumor abuts the mandible but is not fixed to the periosteum
mandible, the periosteum is resected for margin. For tumor fixed to or superficially invading the periosteum or cortex, inner or outer cortex resection can be performed for margin control. Marginal resection can be performed for superficial alveolar (oral cavity) cancers. For invasion into the marrow, limited or more extended segmental resections of the mandible may be undertaken. Once the cortex has been violated or marrow has been infiltrated, a more extended mandibular resection is required for cure because primary radiation incurs the risk of osteoradionecrosis at doses high enough to sterilize the bone disease. In most cases, microvascular free flaps are used to replace the bone and to achieve a cosmetic result in which facial deformity is not evident. Flaps are usually separated into several categories: site (local, regional, distant); tissue (cutaneous, fasciocutaneous, musculocutaneous, osteomusculocutaneous); and blood supply (random, axial, pedicled, free). Modern techniques of inserting osteointegrated implants into bone grafts (often distant osteomusculocutaneous free flaps of the fibula) afford the patient an opportunity to have a dental surface capable of mastication. After radiation therapy or in individuals who have carious teeth, marrow changes may occur that might simulate tumor infiltration but may actually represent radiation fibrosis, osteoradionecrosis, osteomyelitis, or periodontal disease. These conditions are important to identify to facilitate treatment planning.

Maxillary Invasion. The maxilla is more commonly involved with oral cavity and specifically retromolar trigone cancers than with oropharyngeal cancers. Nonetheless, a soft palate cancer may affect the maxilla and tonsillar cancers may spread to the retromolar trigone and from there infiltrate the maxilla. Partial maxillectomies are relatively well tolerated by patients as long as appropriately tailored obturators are constructed that separate the nasal cavity from the oral cavity and oropharynx. Otherwise, regurgitation of food products into the nasal cavity or phonation difficulties, such as velopharyngeal insufficiency, may arise from this common cavity. After the maxilla the tumor may grow into the maxillary sinus or the pterygopalatine fossa (vide infra).

Prevertebral Muscle Invasion. If a cancer is fixed to the prevertebral musculature (longus capitis–longus colli complex), the patient is deemed unresectable. Although the imaging findings of high signal intensity on T2-weighted scans in the muscles, contrast enhancement of the muscle, or nodular infiltration of the muscles suggest neoplastic infiltration, in fact these findings have not been very reliable (Fig. 17). The surgical evaluation at the time of panendoscopy or open exploration remains the gold standard, despite the fact that in rare instances a plane can be found between tumor and the prevertebral musculature. Because the pre-
vertebral musculature is so close to the spinal canal and spinal cord, there are some issues with regard to curative radiotherapy in individuals who have infiltration in this location. At the very least the radiologist should suggest the possibility of prevertebral musculature when the aforementioned findings are present or there is obliteration of the retropharyngeal fat stripe by cancer.

**Pterygopalatine Fossa Invasion.** Extension to the pterygopalatine fossa or to other avenues of the fifth cranial nerve raises the possibility of perineural spread of the cancer to the skull base. Losing the tumor at the skull base due to spread along the cranial nerves happens infrequently with squamous cell carcinoma when the radiologist cautions the surgeon about this possibility; on the other hand, this is typical for adenoid cystic carcinoma where remote perineural spread is almost normal. Once again, MR imaging appears to have some advantages in evaluating the nerves over CT, showing abnormal enlargement and enhancement of the nerve. Foraminal enlargement is a reliable finding on CT. If one sees infiltration of the fat of the pterygopalatine fossa, atrophy of the muscles innervated by the trigeminal nerve, or abnormal enhancement in Meckel’s cave on CT, perineural invasion is implied. Perineural spread of tumor may be antegrade or retrograde and may show skip lesions radiographically.

**Pterygomandibular Raphe Invasion.** The pterygomandibular raphe stretches from the medial pterygoid muscle’s insertion on the medial pterygoid plate to the mandible and separates the anterior tonsillar pillar and the retromolar trigone. Tumor can spread along this plane superiorly to the temporalis muscle, medially into the pterygomandibular space where the lingual and inferior alveolar nerves run, or inferiorly into the floor of the mouth. If tumors spread anteriorly from the medial pterygoid plate they enter the pterygopalatine fossa.

**Bilateral or Deep Invasion of the Tongue Base.** Nowhere else in the head and neck is bilaterality of disease more important with respect to patient quality of life. With base of tongue cancers, the difference between a hemiglossectomy and total glossectomy is critical to a patient’s quality of life. Most individuals are able to function quite well both from a swallowing standpoint as well as a speaking standpoint with half of their tongue present. It is important to realize that the vascular (lingual artery) and nervous (lingual and hypoglossal nerve) supply to the tongue enters from the base of tongue and runs along the sublingual space to supply the tongue from posterior to anterior. Once the base of the tongue has been resected, neurovascular grafting cannot be achieved to preserve function in the anterior aspect of the tongue so the whole side of the tongue may need to be resected. If the midline of the base of the tongue has been violated to any significant degree by cancer, the possibility of having a complete resection with adequate margins while maintaining a functioning tongue is remote. Therefore, a total glossectomy is often recommended, leaving the patient with no means to form a bolus of food. If a small mobile portion of the base of the tongue is preserved, functional recovery with flap reconstruction is much improved. With large pectoralis or bulky microvascular flaps, one can usually get the patient swallowing again after total glossectomy so that lifelong feeding by gastrostomy tubes is not required. The risk of aspiration into the larynx is also reduced when bulky flaps are used. Clear enunciation is also nearly impossible without the tongue, although intelligible speech may be achieved. In some institutions total glossectomies are never performed, leaving radiotherapy with or without implants or chemotherapy the only options for cancer cure.

T2-weighted MR images and postgadolinium enhanced scans may be very useful to determine the true extent of tongue cancers. Proximity of tumor to the lingual artery, nerve, and hypoglossal nerve may be evident by observing the margin of tumor versus the enhancing vessels on a contrast-enhanced CT scan. Remembering that 2-cm margins are typically necessary; however, this issue is less useful in practical experience. There is a greater association of lymph node metastases when tumor infiltration of the vessels, nerves, and sublingual space occurs. By the same token, perineural invasion implies a worse prognosis.

Nodal drainage of the oropharynx is to the submandibular chain (level 1) and the high jugular (levels 2 and 3) chains. One might also see retropharyngeal lymph nodes with more superior oropharyngeal carcinomas or advanced disease.

Lymphomas may occur in the lymphoid tissue of the base of the tongue or tonsil and may simulate proliferative lymphoid hyperplasia. The coexistence of large lymphadenopathy and systemic symptoms suggests lymphoma over benign lymphoid hyperplasia; however, often the imaging characteristics are identical. Rhabdomyosarcomas
may also occur within the tongue. In the sarcomatous category, hemangiopericytomas and synovial sarcomas may occur in the oropharynx and hypopharynx, respectively. Soft palate minor salivary gland neoplasms tend to be more benign in their histology and growth than hard palate ones.

**Hypopharyngeal Carcinoma**

Staging of hypopharyngeal cancers is as follows:

T1: Tumor limited to one subsite of the hypopharynx and 2 cm or less in greatest dimension

T2: Tumor involves more than one subsite of hypopharynx or an adjacent site or measures more than 2 cm but not more than 4 cm in greatest diameter without fixation of hemilarynx

T3: Tumor measures more than 4 cm in greatest dimension or with fixation of hemilarynx

T4: Tumor invades structures (e.g., thyroid/cricoid cartilage, carotid artery, soft tissues of neck, prevertebral fascial muscles, thyroid or esophagus)

Cancers of the hypopharynx are often described in association with laryngeal carcinoma and this issue of the Radiologic Clinics of North America is also organized along these lines. Nonetheless, a few important issues with regard to hypopharyngeal carcinoma must be made. The first is that it is rare to identify a hypopharyngeal carcinoma early in its course because these lesions are usually clinically silent. Therefore, involvement of the supraglottic larynx is not uncommon and often one is contemplating both laryngeal as well as pharyngeal surgery when dealing with a hypopharyngeal carcinoma. The second point to make is that the lesions often extend through the thyrohyoid membrane into the soft tissues of the neck. In fact, this tumor is the critical component to the swallowing mechanism. One can eat nearly normally with most oral tongue resections depending upon the amount of tongue tissue remaining after resection. Phonatory issues also are different between the sites of cancer. The tip and anterior portion of the tongue are more important with creating certain consonant sounds, such as T, D, G, J, and Z.

Nodal disease is less frequent with superficial oral cavity primary cancers than oropharyngeal ones. The exact numbers from different series vary widely, but roughly 30% of patients with oral cavity cancers have nodes at presentation, whereas the percentage for oropharyngeal cancers runs approximately 65%..

Nodal spread impacts significantly on patient outcome (reducing 5-year survival by 50%), emphasizing the importance of identifying pathologic nodes in all patients with cancer.

With oral cavity cancers, the issues of depth of skin invasion, pterygomandibular raphe invasion, maxilla invasion, and pterygopalatine fossa invasion (the latter secondary to retromolar trigone cancer) remain important (Fig. 18). If the disease is limited or superficial, transoral resection with reconstruction by skin grafting, local flaps, or healing by secondary intention can be utilized. More extensive skin grafting may be required with oral cavity cancers that invade superficially than the oropharyngeal cancers, which tend to occur in deeper tissues of the head and neck.

Another issue that should be raised with oral cavity cancers is the high rate of metachronous and synchronous lesions. Field cancerization accounting for synchronous cancers and cancer occurrence remote from a primary tumor both in time and space are not uncommon phenomena identified with oral cavity cancers. This is probably because the entire oral cavity mucosa is exposed

**Oral Cavity Squamous Cell Carcinoma**

The floor of the mouth is the most common site of oral cavity cancer after the lips. Most occur close to the midline frenulum of the tongue. The lateral and undersurface of the oral tongue are the most common sites of involvement of the tongue. The staging of oral cavity cancer is similar to that of the oropharynx in that it is largely based on size criteria as follows:

T1: Tumor is 2 cm or less in its greatest dimension

T2: Tumor more than 2 cm but not more than 4 cm in its greatest dimension

T3: Tumor more than 4 cm in its greatest dimension

T4: Tumor invades adjacent structures, including mandible, maxilla, skin, extrinsic tongue muscles of the tongue root, and soft tissues of the neck, maxillary sinus

T4 infiltration outside of the oral cavity, however, requires the radiologist to do more than just measure the size of the lesion. The issues with regard to oral cavity cancer are similar to that of the oropharynx with respect to mandible, maxilla, and bilateral involvement. Nonetheless, a few distinctions with oropharyngeal squamous cell carcinoma must be made.

Although bilaterality is important with all tongue lesions, it is the base of the tongue that is the critical component to the swallowing mechanism. One can eat nearly normally with most oral tongue resections depending upon the amount of tongue tissue remaining after resection. Phonatory issues also are different between the sites of cancer. The tip and anterior portion of the tongue are more important with creating certain consonant sounds, such as T, D, G, J, and Z.

Oral cavity cancer is the most common aerodigestive system carcinoma to involve primarily the carotid sheath and encase the carotid artery. Finally, it is important to note that hypopharyngeal carcinomas are one of the only extralaryngeal primary tumors to erode and infiltrate the thyroid, cricoid, and arytenoid cartilages. The piriform sinus, from which many of these tumors arise, extends to a level just below the true vocal cords. Lateral growth of a piriform sinus cancer may destroy the thyroid and cricoid cartilages.

Because of their silent nature, most patients with hypopharyngeal carcinoma end up with a laryngectomy and a neopharynx, often without a functioning laryngeal voice. Metastatic disease to the cervical and retropharyngeal lymph nodes also occurs commonly with hypopharyngeal carcinoma as do retropharyngeal nodes.
to the various carcinogens of smoking, chewing tobacco, betel nuts, and alcohol use. That the dependent portions of the oral cavity are at higher risk than the nondependent superior portions of the oral cavity suggests that pooling of toxins in the dependent portions of the oral cavity is the carcinogenic effect.

For buccal mucosa, alveolar, and gingival cancers, the presence or absence of dental amalgam strongly influences the type of imaging study one should recommend as well as the quality of images produced. In our experience, CT scans tend to be degraded from metallic dental amalgam to a greater extent than MR images. In either case, multiplanar imaging is recommended in order to assess the maxilla or mandible for invasion.

One must also be cognizant of the role of the nasopalatine nerves, greater and lesser palatine canals, inferior alveolar canal, and pterygopalatine fossa as avenues for the possible spread of cancers along nerves. Ultimately, the foramen rotundum and foramen ovale should be assessed with imaging to ensure that intracranial extension of tumor along the cranial nerves has not occurred.13

As with squamous cell cancers elsewhere, the presence of lymphadenopathy in association with an oral cavity carcinoma portends a very poor prognosis. In the oral cavity, the primary drainage sites are the level 1 submental and submandibular nodes and the level 2 high jugular chain. In general, oral cavity cancers spread to lymph nodes later than those from the nasopharynx, oropharynx, and hypopharynx, possibly due to the ectodermal derivation of the oral tongue as opposed to the endodermal derivation of the other structures listed.

One final point with regard to the oral cavity should be made. Remember that the lips are the most common site of oral cavity cancer. Imaging for lip cancer is reserved for large cancers or ones with deep spread to the bone. The neck should be imaged to evaluate for adenopathy in large or recurrent tumors. The mental and inferior alveolar canals and nerves should be assessed for disease in transit.

LYMPHOMAS OF THE ORAL CAVITY AND MINOR SALIVARY GLANDS

Lymphomas of the oral cavity are relatively uncommon, usually affecting the base of the tongue or tonsil (portions of the oropharynx) (Fig. 19). Minor salivary gland cancers seem to predominate in the hard palate (where there is the highest concentration of minor salivary glands) and are of variable signal intensity on all pulse sequences (Fig. 20). In the M.D. Anderson series, the most
common minor salivary gland tumor of the palate was the adenoid cystic carcinoma, followed by terminal duct adenocarcinoma and mucoepidermoid carcinoma. Bone invasion (31%), perineural spread (35%), and sinonasal involvement (31%) are common with palate minor salivary gland malignancies. Large size, higher grade, and positive surgical margins are poor prognostic factors for palatal malignancies. Nodal metastases are rare.

References