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15.3 ORAL CAVITY AND OROPHARYNX

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15.3.1 Anatomy

The oral cavity is surrounded anteriorly by the lips. The lips are supplied with a rich sensory innervation and fine-motor control to provide a complex sphincter mechanism. The lips function to prevent the loss of saliva and food materials from the oral cavity. They close around food utensils to assist in the introduction of food materials into the mouth. The lips are also instrumental in the production of labiodental and plosive sounds for speech. The motor nerve supply is from the facial nerve. Damage to the marginal mandibular branch of the facial nerve denervates the depressor labialis, causing the lower lip to turn into the oral cavity, creating an asymmetric smile. Sensation to the lips is provided by branches of the maxillary and mandibular branches of the trigeminal nerve. Loss of sensation interferes with the protective function of the labial sphincter, often causing sialorrhea and mild feeding problems. The space between the lips and the alveolar ridge is the vestibule. It extends externally around the horseshoe-shaped dental arches. The detailed anatomy of the dental arches that support the teeth and gingival mucosa, which covers the alveolar ridges, is discussed in Sec. 16.1.1. The floor of the mouth is a horseshoe-shaped area juxtaposing the medial alveolar ridge and includes the ventral (under) surface of the tongue. The lingual frenulum divides the floor of the mouth in the midline. The geniohyoid, mylohyoid, genioglossus, and hyoglossus muscles provide support to the floor of the mouth.

THE TONGUE

The tongue is an important structure for communication and mastication. Fine-motor abilities of the tongue permit proper articulation for speech and also provide the necessary manipulation of food materials to create a finely prepared bolus for swallowing. The tongue is divided into an anterior two-thirds (which lies within the oral cavity) and a posterior one-third (which lies within the oropharynx). The division between these two areas is demarcated by the sulcus terminalis, which is the location of the circumvallate papillae that form a “V” with the apex posteriorly at the foramen cecum. The foramen cecum is the embryologic derivative of the thyroid gland. If the thyroid gland fails to descend, a lingual thyroid may be visible on the dorsum of the tongue in this area.

The mucosal covering of the tongue is composed of a mixture of papillae. The circumvallate papillae are the largest and are located posterior on the tongue. Smaller fungiform papillae are located on the lateral aspects and the tip of the tongue. The filiform papillae are the most abundant and cover the dorsum of the tongue. Taste buds are richly demonstrated on the circumvallate and fungiform papillae. The filiform

papillae contain no taste buds.

The musculature of the tongue is composed of intrinsic and extrinsic muscle groups. The extrinsic muscles are the genioglossus, the hyoglossus, and the styloglossus. These muscles provide support for the tongue and assist in positioning the tongue forward and backward as well as upward and downward. The genioglossus can protrude the tongue, and its superior fibers depress the tongue tip and assist in retraction. The hyoglossus muscle flattens the tongue. The styloglossus is the primary retractor of the tongue. The intrinsic muscles of the tongue, transverse, vertical, and longitudinal muscles, are responsible for changing the shape of the tongue and are integral to both speech and swallowing.

The blood supply to the tongue is supplied by a branch of the external carotid system, the lingual artery. Sensation is supplied to the anterior two-thirds of the tongue via the lingual nerve, which is a branch of the mandibular division of the trigeminal nerve. The posterior one-third of the tongue is innervated by the glossopharyngeal nerve. Special taste afferents from the anterior tongue course through the lingual nerve to the chorda tympani nerve within the middle ear. Motor function of the tongue is controlled by the hypoglossal nerve. Injury to the hypoglossal nerve will result in a tongue that deviates to the injured side on protrusion.

PALATE, VELOPHARYNGEAL SPHINCTER, AND PHARYNX

The roof of the mouth consists of the palate. The bony hard palate is in continuity with the soft palate posteriorly. The hard palate consists of three fused bones. The paired palatine processes of the maxilla have fused in the midline and comprise the majority of the hard palate. The premaxilla is the triangular piece of bone that consists of the alveolus containing the four upper incisors. The soft palate consists of muscles that originate from the posterior edge of the hard palate and the skull base. The blood supply to the palate is from the palatine branches of the maxillary artery. Sensory innervation to the palate is supplied by branches of the maxillary division of the trigeminal nerve, whereas motor innervation is supplied through the pharyngeal plexus of the vagus nerve. Only the tensor veli palatini muscle receives innervation from the motor division of the trigeminal nerve.

The muscles of the soft palate act to create a dynamic sling. The coordinated movement of the six muscles of the soft palate allows the closure of the velopharyngeal sphincter separating the nasopharynx from the oropharynx during swallowing and speech. The levator veli palatini muscle is the primary elevator of the soft palate. The tensor veli palatini, in addition to assisting palate elevation, is the primary muscle responsible for opening the eustachian tube. Abnormalities in the orientation of this muscle or its insertion on the eustachian tube cartilage greatly predispose to chronic ear disease. The musculus uvulae is a paired muscle in the midline of the posterior soft palate. The bulk of the muscle is on the nasal surface of the soft palate, with small slips of muscle extending into the fleshy appendage hanging free from the posterior midline of the soft palate, which contracts to creat a bulge on the posterior nasal surface of the soft palate, contributing to closure of the velopharyngeal sphincter. The palatoglossus and the palatopharyngeus

muscles are incorporated into the anterior and posterior tonsillar pillars, respectively. The superior pharyngeal constrictor is a paired muscle that inserts onto the midline of

the posterior pharynx (median raphe) and extends around laterally to originate from the skull base. Contraction narrows the velopharyngeal sphincter.

The pharynx is a mucosally lined tube that extends from the skull base to the esophageal inlet. It is divided into three areas. The nasopharynx is located superiorly. It extends from the posterior choanae of the nasal cavity anteriorly to the level of the free edge of the soft palate inferiorly. It contains the eustachian tube and the adenoid. The oropharynx extends from the free margin of the soft palate to the vallecula. The vallecula is the space created by the junction of the tongue base with the epiglottis. The tip of the epiglottis and the lingual and fascial tonsils lie within the oral cavity. Anteriorly, the oropharynx extends to the anterior tonsillar pillars. The hypopharynx extends from the vallecula to the esophageal inlet. The larynx and pyriform sinuses are contained within the hypopharynx.

The pharyngeal constrictors are paired muscles lining the pharynx. They insert into the medial raphe on the midline of the posterior pharyngeal wall. The superior, middle, and inferior pharyngeal constrictors are responsible for the forces generated to clear materials from the pharynx during the act of swallowing. The salpingopharyngeus, stylopharyngeus, and palatopharyngeus extend from the skull base to insert into the lateral walls of the pharynx and elevate the larynx during swallowing. The innervation of all of the pharyngeal muscles is from the pharyngeal plexus of the vagus nerve except that the stylopharyngeus muscle is innervated by the glossopharyngeal nerve. Sensory innervation to the pharynx is via the glossopharyngeal nerve and pharyngeal plexus inferiorly and the maxillary division of the trigeminal nerve superiorly. The blood supply to the pharynx is from the ascending pharyngeal and superior thyroid arteries of the external carotid system.

SALIVARY GLANDS

The parotid duct (Stensen duct) enters the oral cavity through the buccal mucosa, adjacent to the second maxillary molar at the level of the gingival mucosa. The sublingual glands and the duct from the submandibular gland (Wharton duct) enter the floor of the mouth on either side of the lingual frenulum. The paired parotid glands produce the majority of the serous secretions in the mouth. The submandibular and sublingual glands produce a mucoid secretion. These glands produce secretions in response to gustatory stimuli. The majority of secretions in the mouth throughout the day are produced by minor salivary glands scattered throughout the mucosa lining the oral cavity and the pharynx.

TONSILS AND ADENOIDS

The separation between the oral cavity and the oropharynx is a line extending from the anterior tonsillar pillars (the palatoglossus muscle) across the foramen cecum of the tongue to the opposite side. The tonsils lie within the oropharynx. The faucial tonsils are paired collections of lymphoid tissue. The tonsils are bounded anteriorly by the palatoglossus muscle (anterior tonsillar pillar) and the palatopharyngeus muscle (posterior tonsillar pillar). The superior pharyngeal constrictor muscle is deep to the tonsil. The blood supply to the tonsil is from the external carotid system. The tonsillar branches of the ascending pharyngeal and lesser palatine arteries enter the superior pole of the tonsil. Tonsillar branches from the facial, lingual, and ascending palatine arteries enter the inferior pole. The nerve supply to the tonsil is via the

glossopharyngeal nerve inferiorly and branches of the lesser palatine nerves superiorly. The pharyngeal tonsil (adenoid) resides in the nasopharynx. The lingual tonsil resides in the base of the tongue. Superficial bands of lymphoid tissue connect these four masses of tonsil tissue. *Waldeyer ring* refers to this encircling mass of lymphoid (tonsillar) tissue.

The function of the tonsils (and adenoids) is to process antigens and present them to the germinal centers of the lymphoid follicles. This modulates both B- and T-cell populations within the tonsil in early childhood. With increasing age the tonsil and adenoid tissue atrophies.

EMBRYOLOGY

The oral cavity begins as a depression that invades into the developing embryo. It invaginates until the ectoderm of the stomodeum contacts the endoderm of the primitive foregut, creating the buccopharyngeal membrane. This membrane degenerates at 4 weeks of gestation, providing continuity between the ectodermally derived oral cavity and the endodermally derived oropharynx.

The five branchial arches are mesodermal condensations on the lateral cervical area of the embryo and are separated by branchial clefts externally and branchial pouches internally. The cleft is ectodermally lined, whereas the pouch is endodermally lined.

The first arch develops into the mandible, portions of the ossicles, and muscles associated with these structures. The second arch contributes to portions of the ossicles, the styloid process, portions of the hyoid bone, facial muscles, posterior belly of the digastric muscle, and the buccinator muscles. The third arch differentiates into portions of the hyoid bone and pharyngeal muscles. The fourth arch develops into the anterior/superior portions of the larynx, and the fifth arch contributes to the posterior, larynx, cricoid, and intrinsic muscles of the larynx and the inferior pharyngeal constrictor muscle.

The pharyngeal pouches give rise to a variety of structures. The first pouch becomes the middle ear cavity. The first branchial cleft gives rise to the external ear canal. The tonsils are formed from contributions from both the first and second pouches invading into the surrounding mesoderm. Lymphatic tissue then invades these primitive structures between the third and fifth months of gestation. The third pouch gives rise to the thymus gland and the inferior parathyroid glands. The fourth pouch develops into the thyroid gland and the superior parathyroids.

The hard palate is divided into a primary and secondary palate. The primary palate (containing the anterior alveolus and the four upper incisors) is derived from the medial nasal swelling. The secondary palate (the area posterior to the incisive canals) is formed by the medial growth of the lateral palatine processes of the maxilla. The primary palate has completed development by the seventh week of gestation, the secondary palate completes its fusion between weeks 10 and 12 of gestation. Clefts of the soft palate are generally associated with clefts of the secondary hard palate. Complete clefts involve the primary, secondary, and soft palate structures.

The anterior two-thirds of the tongue is derived from ectoderm, whereas the posterior one-third is derived from endoderm of the primitive foregut. Swellings begin to condense during the fourth week of gestation and are complete by the seventh week. The fungiform and filiform papillae develop by the 11th week, and the circumvallate

papillae develop between weeks 8 and 20.

The floor of the mouth is a first arch derivative. The salivary glands are of ectodermal origin and are derived from the first pouch, developing between weeks 5 and 8.

15.3.2 Evaluation of the Oral Cavity and Pharynx

PHYSICAL EXAMINATION

The oral examination begins anteriorly with a systematic evaluation of structures from anterior to posterior, from left to right. The floor of the mouth is evaluated by having the patient elevate the tongue. In small children the tongue will often need to be elevated mechanically. The retromolar trigone (the area among the inferior aspect of the anterior tonsillar pillar, medial aspect of the mandible, and lateral aspect of the tongue) needs to be evaluated by pushing the lateral tongue medially to expose this region. The faucial arches need to be closely evaluated for signs of abnormality. The tonsils should be evaluated for signs of inflammatory changes as well as debris collecting within the crypts of the tonsil. Tonsillar size should be graded on a 1 to 4 scale: 4+ tonsils touch in the midline. Tonsils that are 1+ in size are contained within the tonsillar fossa; 2+ tonsils extend to the medial extent of the tonsillar pillars; 3+ tonsils extend beyond the tonsillar pillars. The oropharyngeal inlet should also be evaluated for adequacy. The tonsils may be of relatively small size but, when combined with a small oropharyngeal inlet, may be obstructing. The posterior pharyngeal wall should be evaluated for symmetry. Granular tissue may often be seen on the posterior pharyngeal wall and represent small areas of lymphoid tissue. Lateral pharyngeal bands are frequently present and represent mild inflammatory changes on the posterior pharyngeal wall secondary to nasopharyngeal drainage or other irritation of this lymphoid tissue.

The soft palate should be evaluated both at rest and in motion. The uvula deserves close attention. A bifid uvula may be a sign of a submucosal cleft of the soft palate. On phonation the soft palate should elevate. Motion of the soft palate should be symmetric. Intraoral palpation is also warranted. The floor of the mouth should be palpated for any sign of stone development within salivary ducts or a mass developing within the floor of the mouth. The hard palate should always be palpated, paying particular attention to the posterior aspect of the hard palate. A posterior projection should be apparent, signifying a normal condition. A notching of the posterior aspect of the hard palate may represent a submucous cleft palate. The buccal area should also be palpated, feeling for stones and also to express saliva from the parotid glands. The tongue should be palpated looking for abnormalities within the substance of the tongue.

ENDOSCOPIC AND RADIOLOGIC EVALUATION OF THE PHARYNX

Flexible endoscopy can be performed easily and safely in an office setting without sedation using a topical anesthetic on the nasopharynx. The nasopharynx is examined for adequacy of velopharyngeal closure, the soft palate for the presence of a submucosal cleft, the tongue base and the lingual tonsils are assessed for size and inflammatory changes, and the hypopharynx and larynx are visualized for signs of cysts, masses, or inflammation.

Radiologic evaluation often begins with a lateral neck x-ray, which provides good

visualization of the nasopharynx and assesses the overall size of the adenoid pad. Retropharyngeal and inflammatory processes on the posterior pharyngeal wall may also be identified. Computed tomography and magnetic resonance imaging provide fine detail of specific abnormalities in the oral cavity and oropharynx and the relationship to surrounding structures. Sialograms are rarely performed. Sialograms require the cannulation of either the parotid duct or the submandibular duct and the installation of contrast material to visualize the ductal system within these glands. Abnormalities within the ducts (sialectasia or stones) can be identified. Infectious complications of this procedure increase in the face of active sialadenitis.

15.3.3 Congenital Malformations of the Tongue and Pharynx

CLEFT LIP AND PALATE

A cleft lip results from incomplete fusion of embryonic structures surrounding the primitive oral cavity. The clefts may be unilateral or bilateral. They are often associated with clefts of the palate (Fig. 15-15). Clefts of the palate vary greatly in their extent. Cleft palates may involve only the soft palate or may extend into the hard palate. The cleft may extend through the hard palate and the alveolar ridge and may be in continuity with a cleft of the lip. A combination of cleft lip and palate leads to significant cosmetic deformities of the nose. The structural support of the nose is not present, leading to abnormal lower lateral cartilage development and nasal septum development. Dental abnormalities are also common, as discussed in Sec. 16.8. For further discussion of cleft palate management see Sec. 10.3.4.

FIGURE 15-15 Clefts of the lip and alveolus create significant nasal deficiencies because of the lack of support of the nasal base.

A submucous cleft palate may not be recognized until the child is several years of age. The muscular development of the soft palate is similar to that seen in a child with a cleft palate. The levator muscles do not attach in the midline of the soft palate but rather to the posterior edge of the hard palate. There is a mucosal covering

such that there is no obvious defect of the soft palate. Palpation of the hard palate will frequently demonstrate a notch in the posterior aspect of the hard palate. Close inspection of the uvula will often demonstrate a bifid uvular structure. A blue line (zona pellucida) may be seen in the midline of the soft palate because of the lack of musculature in the midline. A notch of the posterior hard palate may also be palpable. A submucous cleft palate does not necessarily require repair, but velopharyngeal insufficiency with hypernasal speech and an increased incidence of otitis media often result.

Treatment of a child with a cleft palate requires a team approach. Craniofacial teams are generally available at major pediatric centers throughout the country. Surgical repair of the lip is generally performed at approximately 10 weeks of age, with repair of the palate being performed between 9 and 12 months of age. Early intervention with caregivers familiar with children with clefts of the palate is essential to assist the family in learning how to feed the child. Because of a cleft of the palate, a negative intraoral pressure cannot be generated for sucking. Breast-feeding is frequently unsuccessful, and bottle feeding with the conventional nipple may be difficult. Formula is essentially poured into the oral cavity at a paced rate by the feeder. Special bottle-feeding systems are commercially available to improve the oral nutrition and ease of feeding these children, but often a standard nipple can be utilized. Consultation with either an occupational therapist or speech pathologist with experience in feeding problems of children with clefts of the lip and palate is often useful.

OTHER CONGENITAL ABNORMALITIES OF THE ORAL CAVITY AND PHARYNX

Variations of the dentition and oropharyngeal mucosal lining are discussed in Sec. 16.5.

LINGUAL ANKYLOGLOSSIA

This abnormality is a common disorder, also known as “tongue-tie,” in which the lingual frenulum limits the movement of the anterior tongue tip. On protrusion of the tongue there is frequently a heart-shaped deformity that is created as a very short lingual frenulum tethers the midline of the tongue. The tongue frequently has difficulty protruding beyond the alveolar ridge. Infants may have difficulty attaching to the breast during breast-feeding or to the nipple of a bottle. Surgical correction of ankyloglossia is rarely necessary. The initial feeding difficulties experienced by a newborn often relate to neurologic immaturity and lack of experience of the child and mother. With time and instruction the ability to breast-feed will increase. In some cases, however, a frenulectomy is beneficial and can be performed in the newborn in an office setting. The

tongue is elevated with a special retractor, and the thin lingual frenulum is divided with scissors. No anesthetic is required. Negligible bleeding is encountered. In the older child, general anesthesia is frequently required because of a lack of cooperation.

Speech difficulties secondary to ankyloglossia are quite rare. In the English language the tongue needs only to touch the upper teeth. It is more frequently the case that oromotor dysfunction is leading to the articulation errors that are being attributed to the short lingual frenulum. There are some social activities that may lead a patient to a frenulectomy, such as the inability to lick an ice cream cone. If the patient is unable to protrude the tongue, this activity can be awkward.

LINGUAL THYROID

Failure of the thyroid tissue to decend into the neck from its site of origin in the tongue base may result in a raised violaceous mass that is often visible in the base of the tongue. A lingual thyroid is more common in girls, and in general, this thyroid tissue does not function normally. The size of the mass tends to increase over time. Frequently, signs of airway obstruction will lead the patient to a physician for evaluation. Thyroid hormone replacement will generally cause a reduction in size of the abnormal thyroid remnant.

THYROGLOSSAL DUCT CYSTS

A thyroglossal duct cyst is a cystic mass in the midline of the neck. Occasionally, this cystic mass may present above the hyoid, within the substance of the tongue. Generally, there are no symptoms unless the cyst becomes infected, which leads to a rapid increase in size of the mass. Respiratory difficulties can ensue. Lingual thyroglossal duct cysts need to be differentiated from lingual thyroids. An ultrasound should demonstrate a thyroid gland in its normal location in the base of the neck with essentially normal structure. A nuclear medicine scan can also be utilized to identify functioning thyroid tissue. Thyroglossal duct cysts need to be surgically removed. They are generally excised through a transcervical approach, with the central portion of the hyoid bone removed when a cyst presents within the neck. Thyroglossal duct cysts remaining within the substance of the tongue can be removed endoscopically.

CLEFT TONGUE

Oral-facial-digital syndrome I is an inherited X-linked dominant trait and usually includes a cleft of the tongue with multiple hyperplastic frenula, hypoplasia of the nasal alar cartilages, a medial cleft of the upper lip, an asymmetric cleft of the palate, digital malformations, and mild mental retardation. Fifty percent of these patients have hamartomas between the lobes of a divided tongue. Mohr syndrome (oral-facial-digital syndrome II) is an autosomal recessive condition associated with a conductive hearing loss, cleft lip, a high arched palate, a lobulated nodular tongue, hypoplasia of the body of the mandible, polydactyly, and syndactyly.

15.3.4 Inflammatory Disorders of the Tonsils and Pharynx

Inflammatory disorders of the oral cavity and tongue are discussed in Chap. 16. Pharyngitis presents with symptoms of sore throat, pain on swallowing, mild fever, and malaise. The pharynx and tonsils are usually erythematous on examination, and an exudate may be present over enlarged tonsils. Shotty cervical lymphadenopathy is often

present. Common bacterial pathogens causing tonsillitis and pharyngitis are β-hemolytic *Streptococcus, Streptococcus pneumoniae, Hemophilus influenzae, Peptostreptococcus*, and *Diphtheroids.* The symptoms of bacterial pharyngitis are identical to infections caused by viruses. There are no reliable clinical findings that permit the differentiation of viral from bacterial pharyngitis. Throat cultures are necessary to direct treatment. Infectious mononucleosis must always be considered in cases of exudative tonsillitis with marked lymph node involvement in the posterior cervical chain. The diagnosis of mononucleosis is made by serology.

Patients with a history of recurrent adenotonsillitis (four to six bouts per year), or relapsing infection within 2 weeks of antibiotic treatment, may benefit from prophylactic antibiotic therapy for 6 to 8 weeks. Trials of an antistaphylococcal antibiotic or rifampin may be helpful in irradiating *Staph. aureus* or β-hemolytic *Streptococcus* carriage. Chronic or breakthrough infections despite adequate antibiotic therapy, infections requiring hospitalization, or

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complications developing from a bout of acute adenotonsillitis are indications for adenotonsillectomy. Adenotonsillectomy should not be reserved solely for recurrent streptococcal infections because repeated episodes of any bacterial infection are significant. Surgery is best performed when the acute inflammatory response has subsided, generally 6 weeks after the last infection. In some circumstances, however, a tonsillectomy is necessary during an acute infection if there are life-threatening complications such as airway compromise or spread of infection to the parapharyngeal space as a result of a peritonsillar abscess.

Infections of the tonsils invade the substance of the tonsillar tissue. These infections may extend into the surrounding tissues as well. A peritonsillar cellulitis is an infection that has extended deep to the tonsil and causes local tissue inflammation. At this stage, intravenous antibiotics usually resolve the infection. At times, the cellulitis progresses, and a coalescence of the infection leads to development of a peritonsillar abscess. The abscess collection and surrounding soft tissue edema cause medial displacement of the tonsil and asymmetry of the posterior soft palate with the uvula generally deviating away from the site of infection, and the soft palate bulges downward. Often trismus and ipsilateral otalgia are associated with a peritonsillar abscess. A characteristic muffled (hot potato) voice is heard. Treatment of a peritonsillar abscess requires drainage of the abscess. This may be accomplished in the emergency room with needle aspiration, with incision and drainage, or by tonsillectomy. A delayed tonsillectomy is generally recommended in patients with a peritonsillar cellulitis/abscess if there is a preceding history of recurring throat or tonsil infections.

Lymph nodes in the retropharynx and parapharyngeal space hypertrophy in response to infection. At times, the node becomes overwhelmed with the infection and becomes necrotic. Cellulitis may progress to abscess. Surgical drainage of the abscess in the retropharyngeal or parapharyngeal space is required. Cellulitis will generally respond to high doses of intravenous antibiotics. Peritonsillar abscess, parapharyngeal space abscess, and retropharyngeal abscess all have the potential to cause life-threatening complications if diagnosis and treatment are delayed. A mass effect can cause airway obstruction. Infection may also spread along natural tissue planes upward to the skull base, causing meningitis, or inferiorly into the mediastinum. In cases in which surgical drainage is required, intubation must be performed carefully because the act of

intubation can rupture the abscess cavity with purulent material soiling an unprotected airway.

15.3.5 Adenotonsillar Hypertrophy with Airway Obstruction

Adenotonsillar hypertrophy can cause obstruction of the upper airway. Children often present with chronic snoring, interruption of airflow during inspiration, and frequently restless sleep behavior. The work of breathing increases as airway obstruction increases. Respiratory rate irregularity also increases as the work of breathing increases, but frank apnea secondary to adenotonsillar hypertrophy is uncommon in children except with severe obstruction. The evaluation and causes of sleep apnea are also discussed in Sec. 23.5.3.

Snoring is the hallmark of upper airway obstruction and may be caused by enlarged adenoid tissue or tonsils or both. When a questionable history of nighttime breathing patterns is obtained, it is often useful to have the parents record sound alone or videotape the child while sleeping to more adequately allow the physician to evaluate reports of noisy breathing, apneic episodes, and irregular breathing patterns.

Obesity is not a common feature of children with obstructive apnea. Many children present with poor weight gain because of poor eating habits. Daytime behavioral habits ranging from agitation to somnolence are often reported by parents or teachers. In severely affected children, pulmonary hypertension and cor pulmonale may develop, but these are uncommon.

If large tonsils are evident on physical examination, adenotonsillectomy is usually curative. If the tonsils are small on examination, a lateral neck radiograph may demonstrate adenoid hypertrophy. Lateral neck x-rays need to be interpreted in light of the dynamics of the upper airway. The relative size of the nasopharyngeal airway will be related to patient posture, general muscle tone, rate of air exchange, and inspiration pressures. Because static films cannot adequately capture the changing relationships of the upper airway, fluoroscopy (sometime with sedation) may provide a better evaluation in questionable cases.

Formal polysomnography is required only if neuromuscular disorders are thought to have an impact on the upper airway or in patients who exhibit central or mixed apneic episodes. Polysomnography is too expensive and time intensive for the routine diagnosis of adenotonsillar hypertrophy with airway obstruction. In those children with a convincing history of airway difficulties during sleep and enlarged tonsils or adenoids, adenotonsillectomy is usually curative. Adenoidectomy alone may resolve symptoms of obstruction in patients with small tonsils. In the presence of apnea, however, it is generally thought that both tonsillectomy and adenoidectomy should be performed.

15.3.6 Tumors of the Oropharynx

The most common benign salivary gland tumor is a mixed or pleomorphic adenoma, which generally develops within the parotid gland but can present as a mass extending from any salivary gland. Mucoepidermoid carcinoma is the most common salivary gland malignancy during childhood, most often originating in the sublingual glands. These lesions are treated by surgical excision.

Other malignant neoplasms that may involve the head and neck include

rhabdomyosarcomas, Langerhans histiocytosis, and lymphoma. Extranodal tissue involvement is common in non-Hodgkin lymphomas, with approximately 25% originating in extranodal sites and one-third of these involving structures in the head and neck. Therefore, asymmetric progressive enlargement of the tonsils or adenoids should raise suspicion of a non-Hodgkin lymphoma, and tonsillectomy should be performed as a biopsy procedure. Similarly, asymmetric enlargement of the tonsils or adenoids may be caused by lymphoproliferative disease in immunosuppressed patients following solid organ transplantation.

15.3.7 Pharyngeal Trauma

The approach to oral soft tissue and tooth injuries is discussed in Sec. 16.9. Pharyngeal trauma most commonly results from a child falling while an object is in his or her mouth. The object may impale the roof of the mouth and the posterior pharyngeal wall. The internal carotid artery is adjacent to the tonsillar bed, and therefore a puncture injury at this site has the potential for devastating vascular and neurologic sequelae. If a Horner syndrome is identified

at the time of initial evaluation, carotid injury should be suspected. In rare cases, the carotid intima may be injured, and delayed embolic events (up to 3 days following the injury) with stroke may occur. These objects will often impale the roof of the mouth and the posterior pharyngeal wall. Families need to be instructed to immediately seek medical attention should any alterations in mental status develop following this type of injury. Angiograms or magnetic resonance angiography are not warranted unless there is a high suspicion of carotid artery damage.

Lacerations of the soft palate rarely require repair. Large lacerations that will tend to trap food particles should be closed. Antibiotic coverage is generally not required.

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