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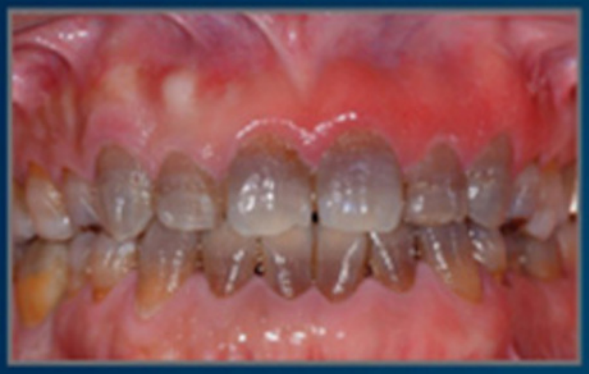
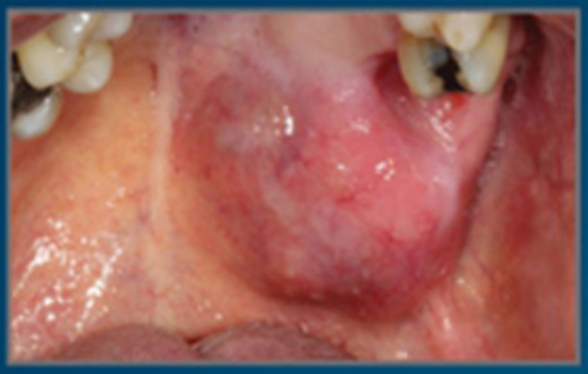
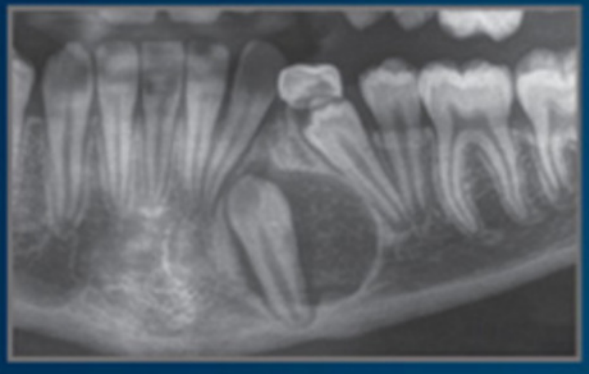
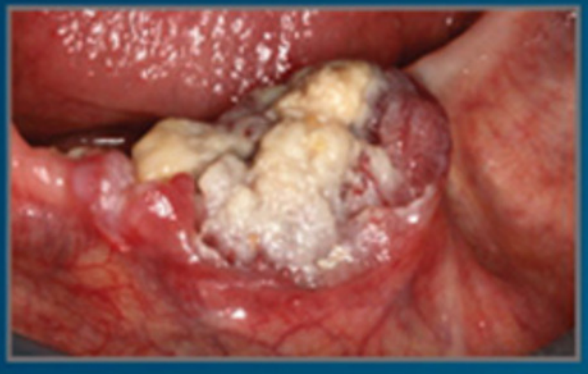
color atlas OF ORAL AND MAXILLOFACIAL DISEASES

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To our colleagues, many of whom have shared these cases with us.

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Preface

By its very nature, the discipline of pathology encompasses not only the microscopic diagnosis of disease, but also the recognition and diagnosis of lesions on a clinical and radiographic basis. As oral and maxillofacial pathologists, we naturally spend a significant amount of our time in the laboratory examining tissue specimens for diagnosis. However, we also have the great opportunity and privilege to see many patients in a clinical setting for both the diagnosis and management of various oral diseases. As a matter of fact, it is this fascination with disease on a clinical basis that first stimulated our interest to enter the specialty of oral and maxillofacial pathology.

With this in mind, we are pleased to offer this collection of photographs and radiographic images of oral, head, and neck diseases. These illustrations represent a compilation of what we consider to be among the best clinical teaching material that we have accrued over the course of our careers. The book has been designed primarily with the dental professional in mind, but it also should be useful to other health care providers who treat oral diseases, such as otolaryngologists and dermatologists.

In keeping with an atlas format, we have decided to include more pictures rather than more words. The chapters are organized by broad disease categories, which match the sequence of how we initially lecture about these topics in the classroom. A wide variety of lesions has been included, but we have tried to emphasize more commonly occurring and important disorders. No photomicrographs are included in this book. Although we obviously recognize the importance of histopathology in the diagnosis of disease, we think that the purpose of this book is better served by limiting it to clinical photographs and radiographs.

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Acknowledgments

We are deeply indebted to our friends and colleagues who have shared many of the images included in this atlas or who have referred patients for us to examine and photograph. We have attempted to be as thorough as possible in listing credit to these individuals in the figure legends. However, if anyone's name has been inadvertently omitted, please accept our apologies.

We would like to acknowledge some of the many teachers who have mentored us during our careers, particularly those individuals who stimulated and fostered our interest in clinical oral pathology. This list includes Drs. George Blozis, Jerry Bouquot, George Gallagher, Susan Müller, Charles Waldron, and Ronnie Weathers.

We also wish to thank Alexandra Mortimer, Jennifer Flynn-Briggs, Kate Mannix, Caroline Dorey-Stein, and Taylor Ball at Elsevier for their editorial expertise and patience as we worked on this project. Finally, our families deserve more personal thanks and praise for their love and support during the preparation of this book.

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Developmental Defects of the Oral and Maxillofacial Region

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Cleft Lip and Palate

Fig. **1.1**

Cleft lip (CL) is a common congenital anomaly that is caused by defective fusion of the medial nasal and maxillary processes during embryologic development. Approximately 80% of cases are unilateral and 20% are bilateral. **Cleft palate** (CP), which results from failure of the lateral palatal shelves to fuse, often occurs in conjunction with CL, although it also may develop as an isolated defect. CL alone and CL with CP are etiologically related conditions that can be grouped together as CL ± CP (CL with or without CP). CP only (CPO) represents a separate entity from CL ± CP. Orofacial clefting is seen with greater frequency in a variety of specific genetic syndromes, although more often it occurs in a sporadic fashion due to a combination of environmental and genetic factors. Factors known to increase the risk for clefts include maternal smoking, alcohol consumption, and phenytoin usage.

The frequency of CL ± CP varies considerably among different racial/ethnic groups. Among whites, the frequency is estimated at 1 per every 700 to 1000 births. The prevalence in blacks is much lower, with a rate of 0.4 cases per 1000 births. In contrast, the rate in Asian populations is about 1.5 times that seen in whites. The highest rate occurs in Native Americans, with a frequency of 3.6 per 1000 births. CL ± CP is more common in males, whereas CPO is more common in females.

Orofacial clefting can result in a variety of problems related to appearance, feeding, speech, hearing, and socialization skills. Management involves a dedicated craniofacial team, which may include specialists in genetics, oral and maxillofacial surgery, orthodontics, otolaryngology, pediatric dentistry, pediatric medicine, plastic surgery, prosthodontics, psychology, and speech pathology. Treatment may require multiple surgeries, with repair of CL usually accomplished around 2 to 3 months after birth and surgical correction of CP undertaken between 6 and 12 months of life.

Bifid Uvula (Cleft Uvula)

Fig. **1.2**

During the embryologic formation of the hard and soft palate, the lateral palatal shelves normally fuse in the midline. This fusion begins in the anterior region of the palate and progresses posteriorly to the uvula. If the fusion is not totally completed, then a **bifid uvula** may occur, which represents the most minimal manifestation of a cleft palate (CP). Sometimes a bifid uvula may be associated with a submucous palatal cleft in which the overlying mucosa is intact but there is a defect in the formation of the musculature of the soft palate. Submucous clefts also may be associated with a notched defect of the midline bone of the posterior hard palate. Bifid uvula is more common than complete CP, with an estimated overall prevalence of 1% to 2%. The frequency is much higher in Asian and Native American populations. In most instances, bifid uvula is an incidental finding that does not cause any problems. If an associated submucous CP is present, velopharyngeal insufficiency may be present, which can result in hypernasal speech. A bifid uvula can be associated with certain genetic conditions, such as van der Woude syndrome and Loeys-Dietz syndrome (hypertelorism, bifid uvula or CP, and aortic aneurysm with tortuosity).

Double Lip

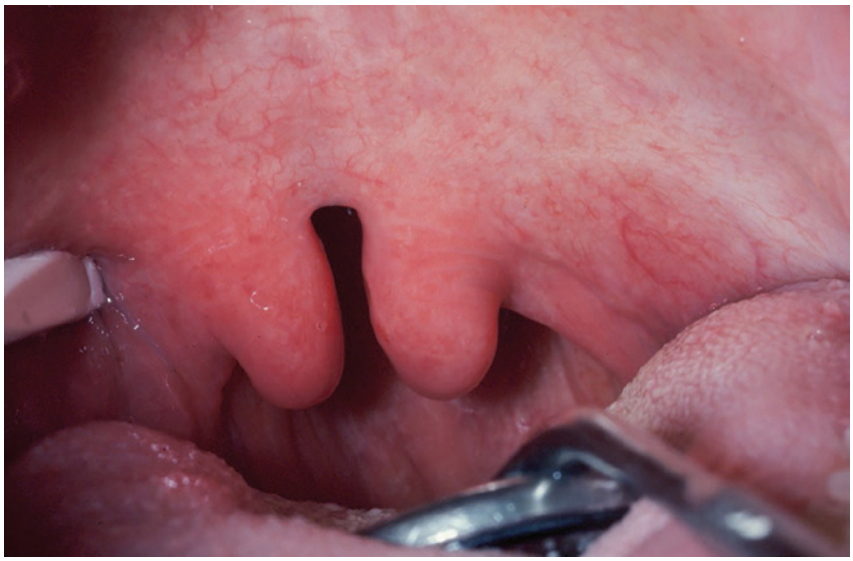
Fig. **1.3**

Double lip is an uncommon oral anomaly in which there is an excess fold of tissue along the mucosal surface of the lip. It either may be congenital or develop later in life. The upper lip is affected more frequently than the lower lip, although sometimes both lips are involved. The redundant tissue may be seen bilaterally in a symmetric fashion, or it may appear primarily on one side. When the lips are at rest, a double lip may not be noticeable; however, when the patient smiles, the excess tissue will become evident. Double lip occasionally may be a component of Ascher syndrome, which is characterized by the following triad: (1) double lip, (2) blepharochalasis (edema and sagging of the upper eyelid), and (3) nontoxic thyroid enlargement.

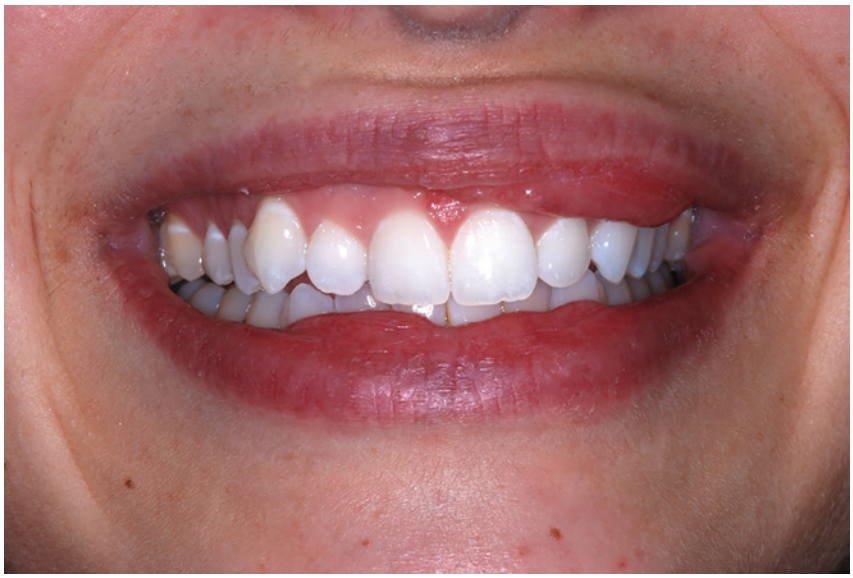
No treatment may be required for mild forms of double lip. However, more severe examples can be managed by surgical excision of the excess tissue for cosmetic purposes.

■ Figure **1.1**
Cleft Lip
Unilateral cleft of the left upper lip. (Courtesy Dr. Cathy Flaitz.)

■ Figure **1.2**
Bifid Uvula
A midline cleft divides the uvula into two lobes.



■ Figure **1.3**
Double Lip
An extra fold of tissue hangs down from the left upper lip.



Commissural Lip Pits

Fig. **1.4**

Commissural lip pits are tiny mucosal invaginations at the corners of the mouth near the vermilion border. Such depressions have been noted in 12% to 20% of the adult population, whereas among children the reported prevalence is only about 0.2% to 0.7%. Although such pits often are considered to be congenital defects, their increased frequency in adult patients suggests that they usually do not appear until later in life. Commissural lip pits occur more often in males than in females.

Commissural lip pits are typically asymptomatic unilateral or bilateral lesions that are discovered as incidental findings. They appear as small punctate depressions extending to a depth of 1 to 4 mm on the lip vermilion at the commissures. Because ducts from minor salivary glands may empty into the depth of the pit, a small amount of mucoid secretion sometimes can be expressed. Commissural lip pits have been associated with a higher prevalence of preauricular pits, but they are not associated with orofacial clefting. Because of their asymptomatic nature, treatment rarely is required. However, if excessive salivary secretions occur or secondary infection develops in a deep pit, then surgical excision may be considered.

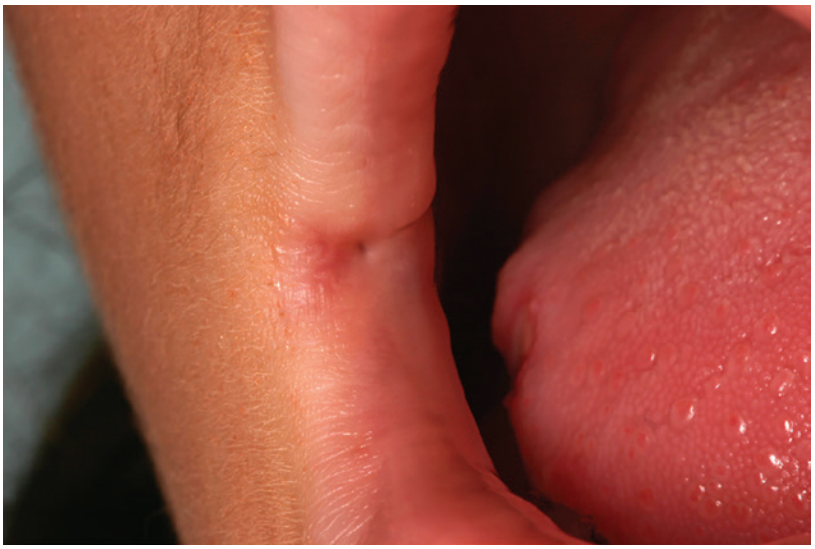
Paramedian Lip Pits and Van der Woude Syndrome

Figs. **1.5 and 1.6**

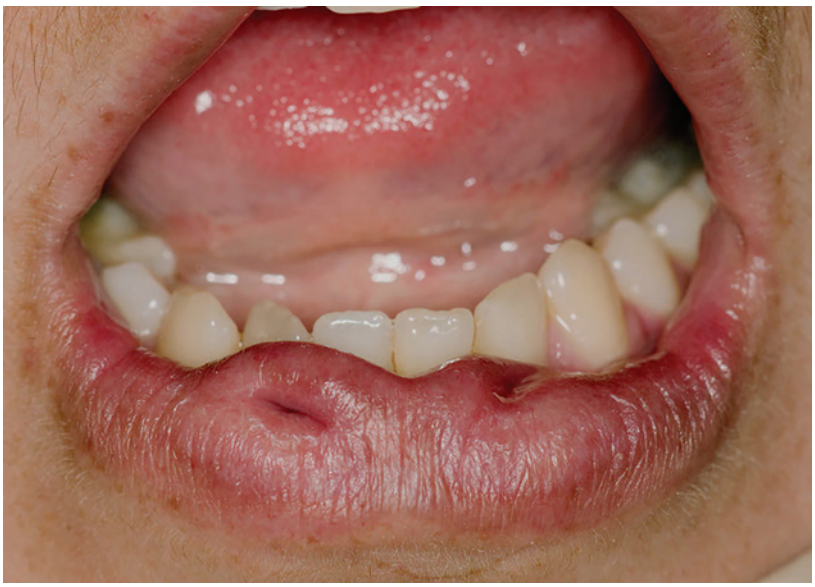
Paramedian lip pits are rare congenital invaginations that are seen on the vermilion border of the lower lip, lateral to the midline. Such lesions are usually bilateral, although in some instances a single pit may be found more centrally positioned or lateral to the midline. Paramedian lip pits are significant because they usually are associated with **van der Woude syndrome**, an autosomal dominant condition that also includes cleft lip (CL) and/or cleft palate (CP). Van der Woude syndrome is the most common form of syndromic orofacial clefting, occurring in 1 out of every 40,000 to 100,000 births. It is estimated that 2% of all CL and CP cases are part of van der Woude syndrome, which is caused by mutations of the gene that encodes interferon regulatory factor 6 (IRF6). Some people with paramedian lip pits and van der Woude syndrome may not demonstrate clefting or they may exhibit only a submucosal CP; however, such individuals can transmit the full syndrome to their offspring. Paramedian lip pits also can be a feature of two other syndromes that include orofacial clefting: popliteal pterygium syndrome and Kabuki syndrome.

Paramedian lip pits appear as blind sinuslike depressions that can extend to a depth of 1.5 cm. A humped swelling sometimes surrounds the central pore. Salivary secretions may be expressed because of minor salivary gland ducts that empty into the depth of the pit. If the pits are a cosmetic problem for the patient, then surgical excision can be performed.

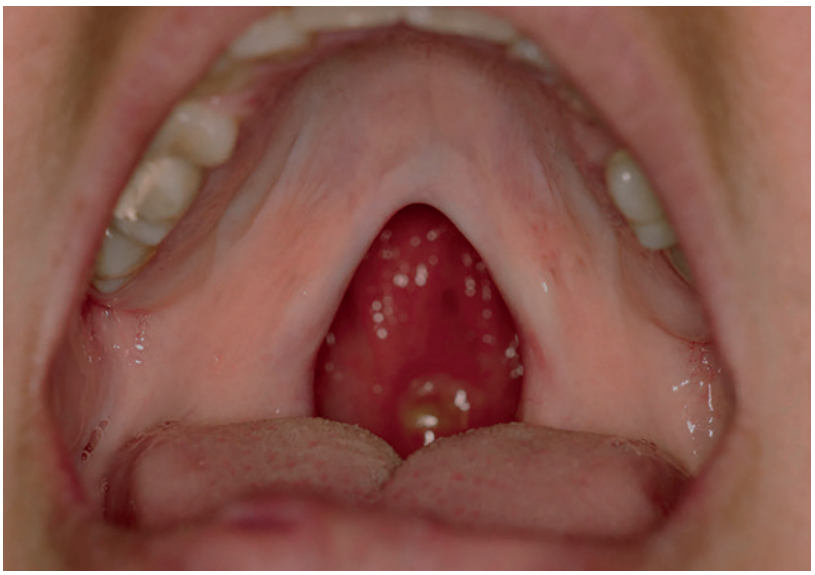
■ Figure **1.4**
Commissural Lip Pit
A punctate depression is present at the right labial commissure.



■ Figure **1.5**
Paramedian Lip Pits in Van der Woude Syndrome
Bilateral pits are seen adjacent to the midline of the lower lip vermilion. (Courtesy Dr. Nadarajah Vigneswaran.)



■ Figure **1.6**
Cleft Palate in Van der Woude Syndrome
Same patient as depicted in Fig. 1.5 with a cleft of the soft palate. (Courtesy Dr. Nadarajah Vigneswaran.)

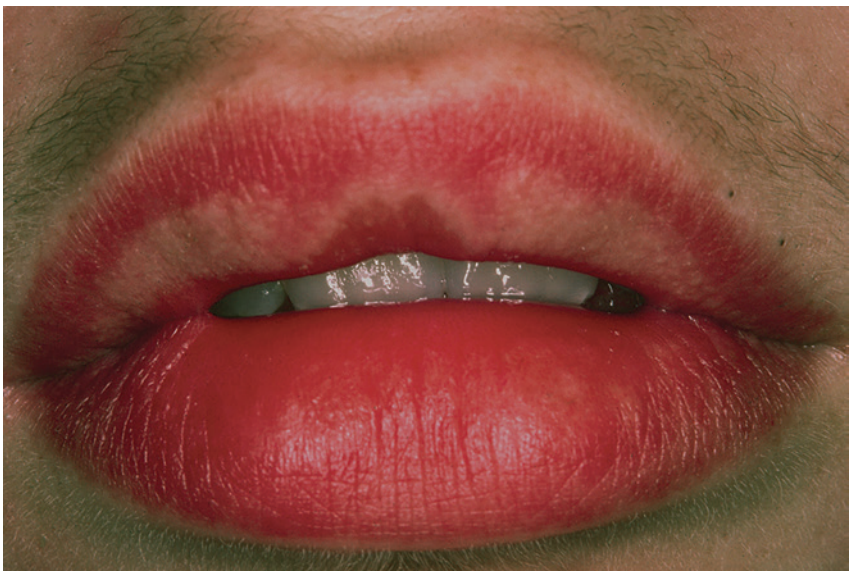


Fordyce Granules

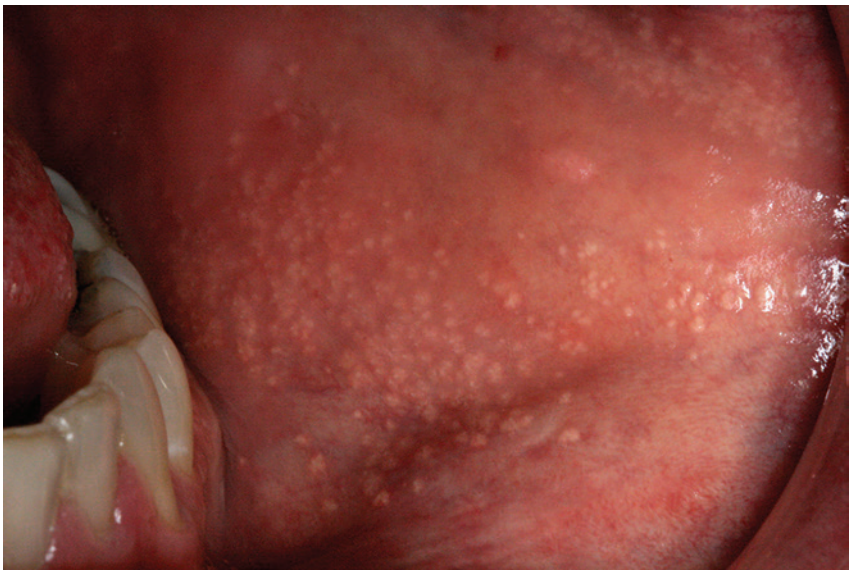
Figs. **1.7–1.9**

Sebaceous glands are common adnexal structures on the skin, where they typically are associated with hair follicles. However, sebaceous glands also can be found on mucosal surfaces, where they are known as **Fordyce granules**. In the oral cavity, Fordyce granules are seen most frequently on the upper lip vermilion and buccal mucosa, although they also may appear on the retromolar pad and tonsillar pillars. The term *Fordyce granules* also is used to describe sebaceous glands found on the genitalia. Although oral Fordyce granules often are regarded as ectopic structures, they are found in over 80% of adults, suggesting that these glands represent a normal anatomic variation. Their prevalence is higher in adults than in children, probably because of hormonal influences. Clinically, they appear as tiny yellowish or chamois-colored papules ranging from 1 to 3 mm in diameter. Some patients may exhibit only isolated lesions, whereas others may have well over 100 such papules that focally may appear tightly packed and almost confluent. Because Fordyce granules are asymptomatic normal anatomic structures, no treatment is necessary. Occasionally such glands can become hyperplastic or form keratin-filled pseudocysts, which might prompt biopsy to confirm the diagnosis. Extremely rare examples of sebaceous tumors in the oral cavity, which may have arisen from Fordyce granules, have been described.

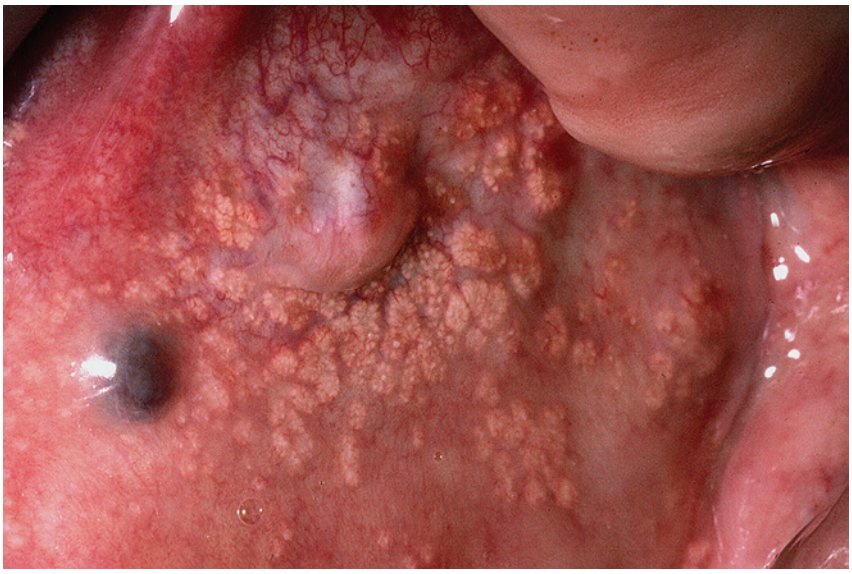
■ Figure **1.7**
Fordyce Granules
Confluent yellow papules on the upper lip vermilion.



■ Figure **1.8**
Fordyce Granules
Cluster of yellow papules on the left buccal mucosa.



■ Figure **1.9**
Fordyce Granules
Multiple prominent sebaceous glands on the right buccal mucosa. The parotid papilla is located near the center, and a varix can be seen toward the anterior buccal mucosa.



Leukoedema

Figs. **1.10 and 1.11**

Leukoedema is a bilateral, white, opalescent appearance of the buccal mucosa that may represent a normal variation in anatomy. The white appearance is created by an increase in thickness of the surface epithelium, which includes numerous cells with prominent intracellular edema. Leukoedema exhibits a predilection for blacks, among whom it has been described in 70% to 90% of adults and 50% of children. In whites, the condition often has a milder presentation and may be hardly noticeable. Leukoedema in blacks may appear more obvious because of the contrast between the edematous mucosa and background pigmentation. Although leukoedema is considered to be a developmental lesion, the white color can be more prominent in smokers and may become less severe after smoking cessation.

Leukoedema appears as a diffuse, milky, gray-white change in the color of the buccal mucosa, which should be bilateral and symmetric. Often, the mucosal surface appears somewhat folded, resulting in white streaks or wrinkles. The diagnosis can be confirmed easily by stretching and everting the cheek, which will result in disappearance of the opalescent white change. No treatment or biopsy is necessary.

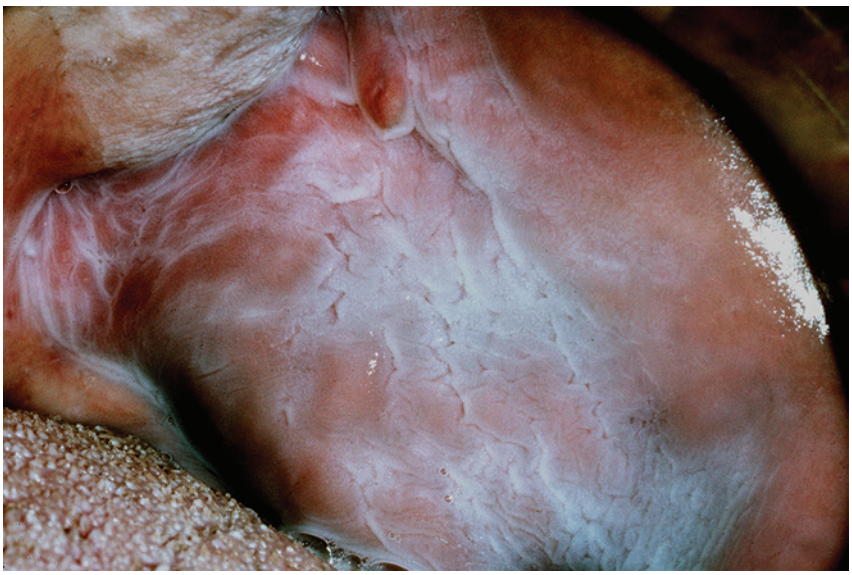
Ankyloglossia

Fig. **1.12**

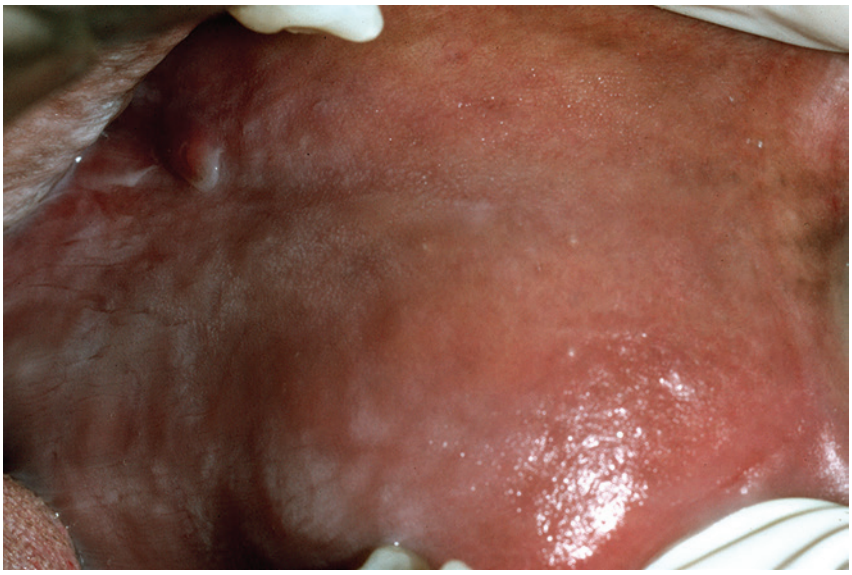
Ankyloglossia ("tongue-tie") refers to a short or tight attachment of the lingual frenum to the ventral tongue, which results in limited tongue mobility. Ankyloglossia has been reported in 2% to 16% of neonates, with a male predilection. However, because the tongue normally is short at birth and then grows longer at the tip, the prevalence is much lower in adults. The term *anterior ankyloglossia* is used for examples in which the attachment of the frenum extends toward the tip of the tongue. *Posterior ankyloglossia* is often more difficult to appreciate, being caused by short submucosal collagen fibers in the posterior midline floor of the mouth that prevent full tongue motion. Although tongue-tie has been thought to contribute to speech difficulties, most patients have only minor difficulties and can compensate for any limitation in tongue movement. It also has been theorized that ankyloglossia might contribute to gingival recession if the frenum attaches high on the lingual alveolar mucosa. With the increased prevalence of breast-feeding over the past several decades, lactation experts believe that tongue-tie can contribute to feeding difficulties, such as inability of the baby to attach to the nipple and nipple pain.

Because most cases of ankyloglossia do not result in significant clinical problems, treatment is often unnecessary. For infants with breast-feeding difficulty, frenotomy (clipping and freeing the frenum) may improve the ability to nurse. However, there is insufficient evidence to support prophylactic surgical correction of tongue-tie in an effort to improve speech development.

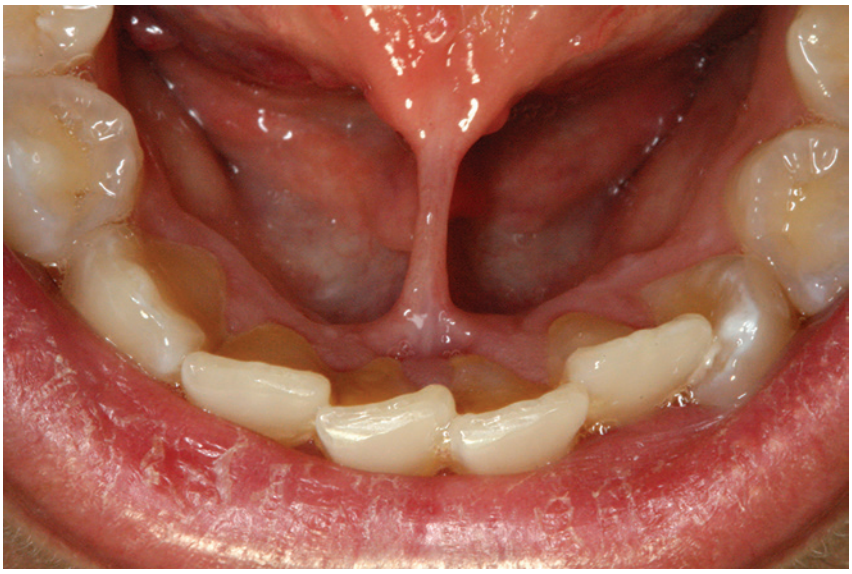
■ Figure **1.10**
Leukoedema
Milky white appearance affecting almost the entire buccal mucosa.



■ Figure **1.11**
Leukoedema
Same patient as seen in Fig. 1.10, showing disappearance of the white change when the cheek is stretched.



■ Figure **1.12**
Ankyloglossia
The lingual frenum attaches from the tip of the tongue to the lingual alveolar mucosa.



Lingual Thyroid

Figs. **1.13 and 1.14**

The thyroid gland originally develops at the base of the tongue in the foramen cecum area and then migrates to its normal pretracheal location during early embryologic life. However, if the embryonic gland does not undergo normal migration, then a **lingual thyroid** may develop at the midline of the dorsal tongue at the junction of the anterior two-thirds and posterior third. Other potential sites for ectopic thyroid tissue include the floor of the mouth and anterior neck. The prevalence of symptomatic or clinically evident lingual thyroid has been estimated at 1 in every 100,000 persons, with a female-to-male ratio of 5:1. However, autopsy studies have revealed incidental remnants of thyroid tissue on the posterior dorsal tongue in up to 10% of both men and women. Hypothyroidism will be present in 33% to 62% of patients with lingual thyroids, and some examples are discovered in newborns as part of screening for congenital hypothyroidism. Other cases may not be diagnosed until later in childhood or during adult life. In most instances, a lingual thyroid represents the only thyroid tissue that is present.

Many examples of lingual thyroid are asymptomatic and will be discovered incidentally upon routine oral examination or during evaluation for tonsillectomy or upper respiratory infections. Symptomatic patients may report the sensation of a mass or foreign body, hoarseness, cough, dysphagia, or snoring. The diagnosis can best be supported by a radioactive iodine scan. Other imaging tests may include magnetic resonance imaging (MRI), ultrasonography, or computed tomography (CT).

Asymptomatic lingual thyroids may not require treatment, although when associated hypothyroidism is present, thyroid hormone replacement is needed. Hormone replacement therapy sometimes results in shrinkage of a lingual thyroid. For symptomatic cases, radioactive iodine therapy or surgical resection may become necessary if hormone replacement therapy does not produce shrinkage. The risk of carcinoma development within a lingual thyroid is low, occurring in approximately 1% of cases.

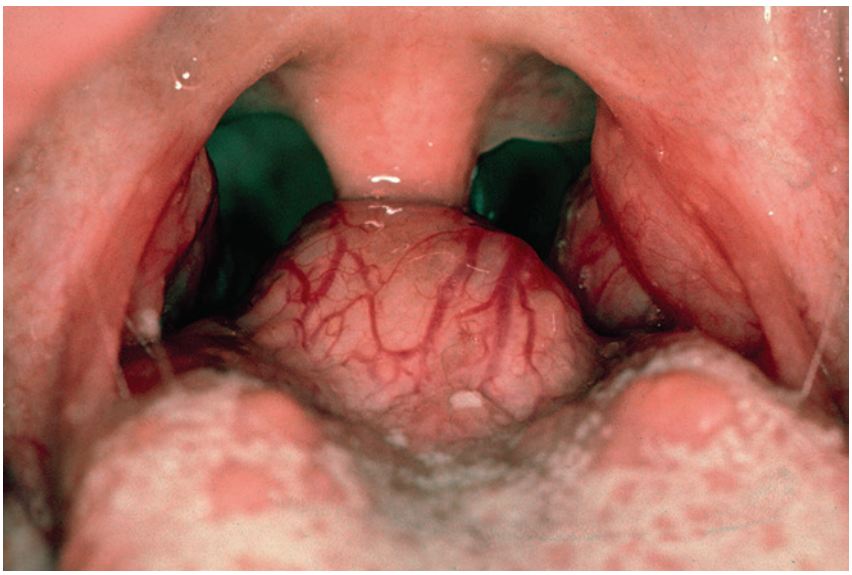
Fissured Tongue

Fig. **1.15**

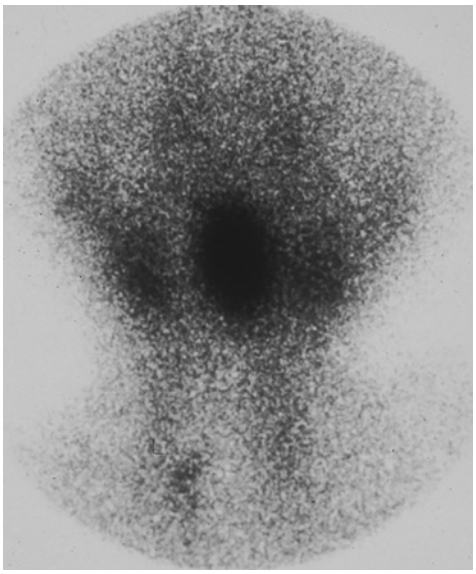
Fissured tongue is a benign condition characterized by the presence of multiple grooves or fissures on the dorsal tongue. The cause is unknown, although genetic factors may play a role. Fissured tongue has a strong association with erythema migrans (geographic tongue), with many patients demonstrating both conditions simultaneously. It is possible that longstanding geographic tongue may contribute to the development of a fissured tongue. A variety of other factors also have been associated with a greater prevalence of fissured tongue, including psoriasis and tobacco usage. Fissured tongue also may be a component of Melkersson-Rosenthal syndrome (in association with orofacial granulomatosis and facial nerve paralysis). The reported prevalence of fissured tongue varies widely, probably related to the stringency of the criteria used to make the diagnosis. The frequency ranges from 2% to 5% of the overall population in some studies, whereas other studies indicate a prevalence in the range of 10% to 20%. Fissured tongue is uncommon in children, but it increases in frequency with age, reportedly reaching as high as 30% or more in older adults.

Patients with fissured tongue exhibit multiple grooves on the dorsal tongue, which may range from 2 to 6 mm in depth. Some patients may have a central midline fissure with smaller fissures branching off at 90-degree angles. Other patients may have numerous grooves crisscrossing the tongue, separating the surface into small islands. Sometimes one or more of these islands may develop into nodular fibroma-like growths. Fissured tongue is usually asymptomatic, although some patients may complain of mild burning or soreness. No treatment is necessary, although daily tongue brushing can help to remove any entrapped food or debris that might act as a source of irritation.

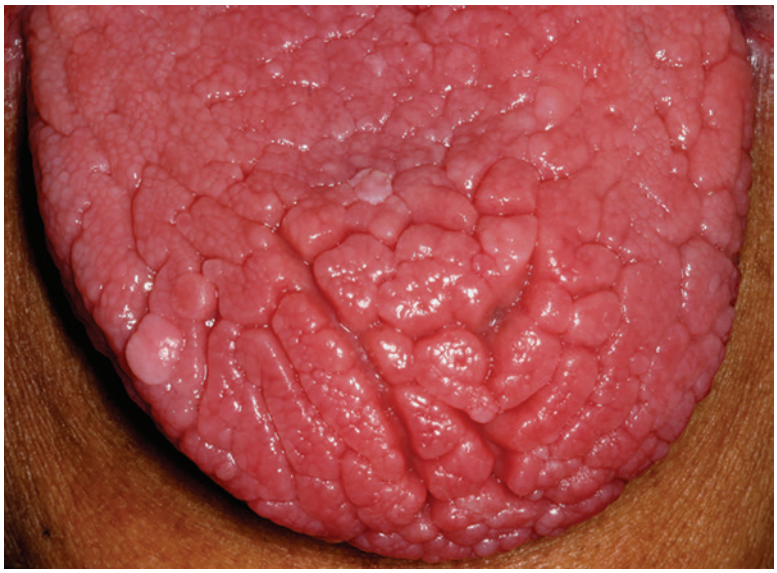
■ Figure **1.13**
Lingual Thyroid
Four-year-old girl with a mass of the posterior midline dorsal tongue.



■ Figure **1.14**
Lingual Thyroid
Radioactive iodine scan of the patient seen in Fig. 1.13, showing strong uptake in the tongue mass *(center)* with minimal uptake in the lower neck.



■ Figure **1.15**
Fissured Tongue
The tongue exhibits multiple cracks and grooves on the dorsal surface.



Hairy Tongue

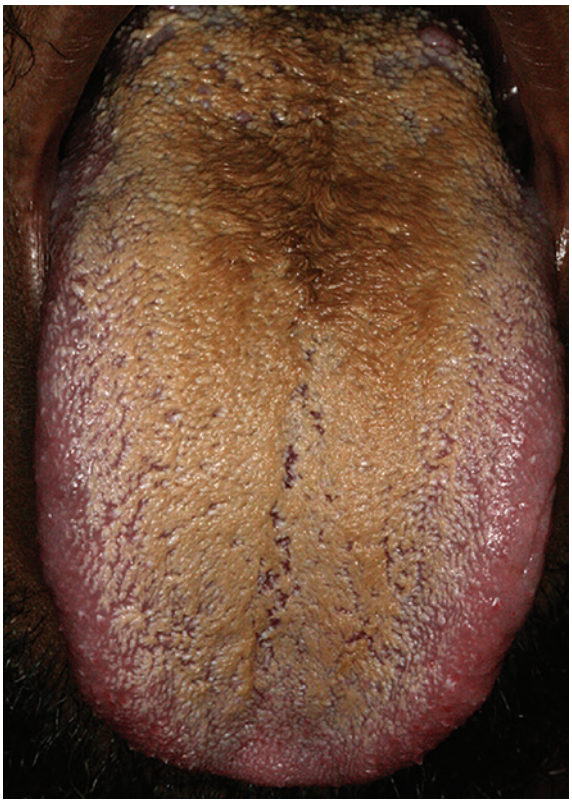
Figs. **1.16–1.19**

Hairy tongue is a common, benign condition of the dorsal tongue that is characterized by elongation and hyperkeratosis of the filiform papillae, mimicking the appearance of small hairs. Depending on the population studied, the prevalence ranges from 0.5% to 11.3%. Because the elongated papillae usually are pigmented secondary to smoking, coffee/tea consumption, or chromogenic bacteria, the condition often is referred to as *black hairy tongue*. However, many examples actually exhibit a brown or yellow color. The most common factor associated with the development of hairy tongue is smoking, although other causes include general debilitation, poor oral hygiene, xerostomia, and a history of radiation to the head and neck. Mild cases of hairy tongue will involve only the posterior midline region of the dorsal tongue. More severe examples can result in a generalized thick, matted appearance that involves most of the dorsal tongue surface. Hairy tongue is usually asymptomatic, although extreme elongation of the papillae has been known to cause gagging in some patients. Because of the accumulation of bacteria on the rough surface, halitosis is a possible sequela.

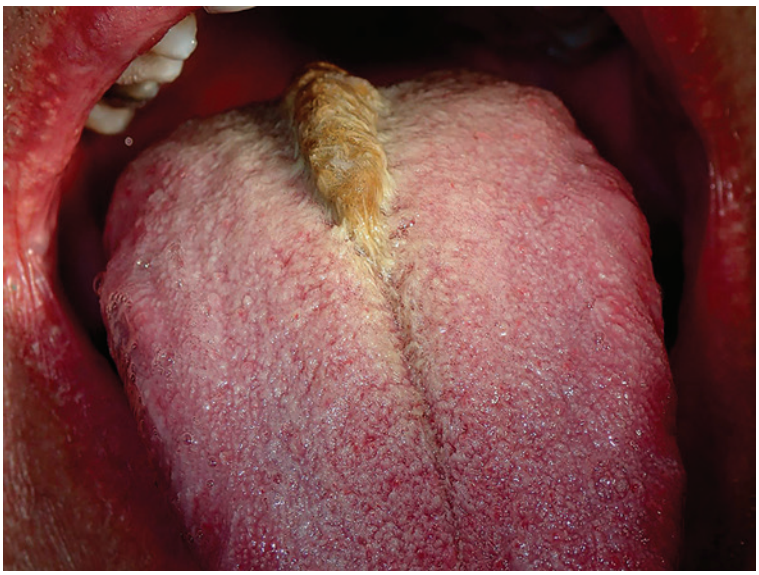
Some patients exhibit accumulation of bacteria and dead epithelial cells on the dorsal tongue surface without the development of hair-like elongation of the filiform papillae—a condition sometimes known as a *coated tongue*. Temporary "pseudo–black hairy tongue" can occur in patients who have used bismuth subsalicylate to treat upset stomach because the bismuth in such preparations can react with traces of sulfur in the saliva to produce bismuth sulfide.

Although hairy tongue is a benign condition, it is unsightly and can contribute to bad breath. Treatment includes elimination of causative factors (if possible) and improved oral hygiene. Periodic brushing/scraping of the tongue with a toothbrush or tongue scraper can promote desquamation of the excessive keratin layer and bacterial debris. Because hairy tongue is not caused by a yeast infection, clinicians should avoid unnecessary treatment with antifungal medications.

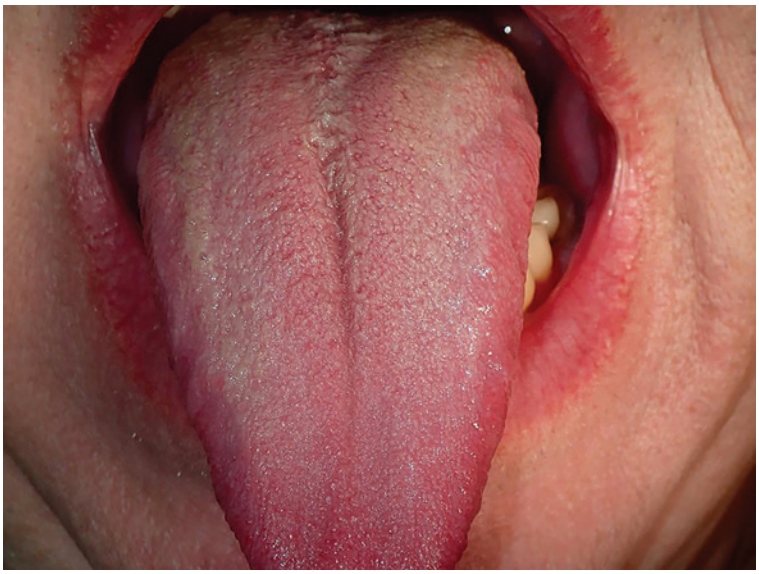
■ Figure **1.16**
Hairy Tongue
The tongue exhibits multiple elongated filiform papillae with brown staining.



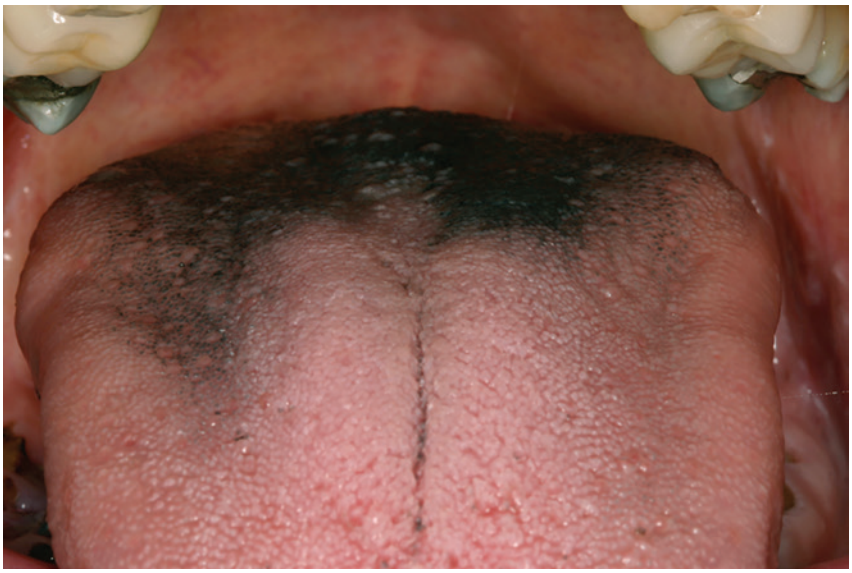
■ Figure **1.17**
Hairy Tongue
The filiform papillae show prominent elongation in the midline region of the posterior dorsal tongue. (Courtesy Dr. Scott Wietecha.)



■ Figure **1.18**
Hairy Tongue
Same patient as seen in Fig. 1.17, showing resolution of the lesion after regular brushing of her tongue. (Courtesy Dr. Scott Wietecha.)



■ Figure **1.19**
Bismuth Staining
The dorsal tongue exhibits black staining, which developed after the patient used bismuth subsalicylate for an upset stomach.



Varicosities (Varices)

Figs. **1.20 and 1.21**

A **varicosity**, or **varix**, is an abnormally dilated, tortuous vein. In the oral cavity, varicosities develop most frequently on the ventrolateral tongue, although such lesions can occur in other locations, especially the labial and buccal mucosa. They are rare in children but common in older adults, which suggests that age is an important factor in their development. Sublingual varicosities have been described in two-thirds of people over 60 years of age. Age-related weakening of blood vessel walls and loss of tone in the surrounding connective tissue may contribute to such vascular dilation. Also, oral varices have been reported to occur more often in patients with varicose veins of the legs and in those with a history of smoking, cardiovascular disease, and/or hypertension.

Clinically, oral varicosities appear as blue to purple blebs or soft nodules, which should blanch on compression. Blanching may be demonstrated clinically by pressing against the lesion with a glass slide, a technique known as *diascopy*. Sublingual varicosities usually are multiple and bilateral in distribution, although varices in other locations may occur as isolated lesions. Because the flow of blood will slow down within a dilated vessel, varicosities are prone to thrombosis. Such a lesion typically will not blanch under pressure because the thrombus cannot be pressed into the adjacent vasculature. A thrombosed varix will feel firmer, similar to a BB beneath the mucosal surface. However, unlike deep vein thromboses in the leg, a thrombosed oral varix poses minimal risk of embolism.

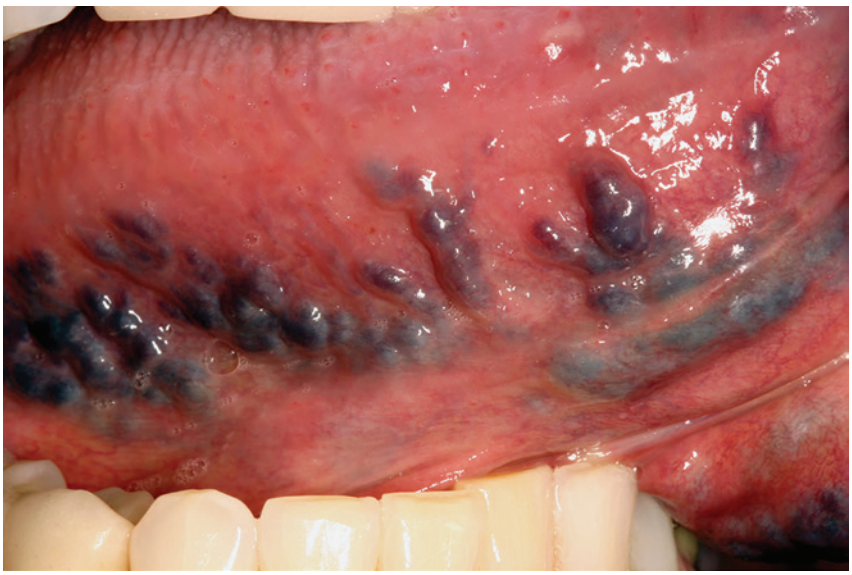
Oral varicosities are usually innocuous lesions that can be diagnosed clinically and, therefore, do not require treatment. However, surgical excision can be performed for thrombosed varices, for aesthetically displeasing varicosities on the lips, or in situations where the diagnosis must be confirmed.

Caliber-Persistent Artery

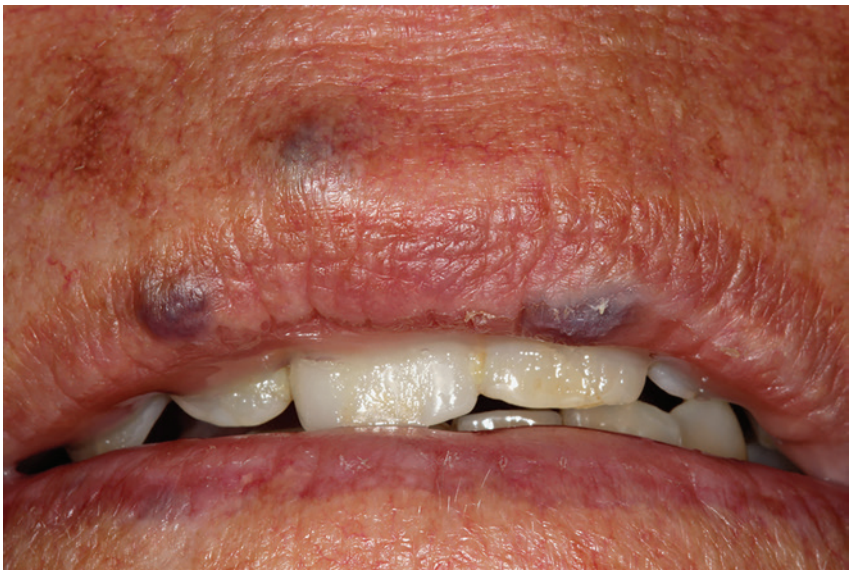
Fig. **1.22**

Larger arterial vessels normally are found within the deeper connective tissues. However, occasionally a large branch of an artery will extend close to the mucosal surface without a reduction in its diameter—a vascular anomaly known as a **caliber-persistent artery**. This lesion is seen most frequently in older adults, suggesting that it may represent an age-related phenomenon related to loss of tone within the surrounding connective tissues. Caliber-persistent arteries almost always occur on the lower or upper labial mucosa; some patients may develop lesions on both lips or bilaterally. The lesion appears as a curvilinear or papular elevation that can appear bluish in color. The artery may become less obvious when the lip is stretched. Pulsation may be noted within the vessel, although it may be difficult to feel this pulse through gloved fingers. The lesion is usually asymptomatic, although overlying mucosal ulceration has been reported in a few examples. Because of its benign nature, no treatment is necessary. However, sometimes a biopsy will be performed because the lesion is mistaken for a mucocele or hemangioma. In such instances, significant bleeding may be encountered.

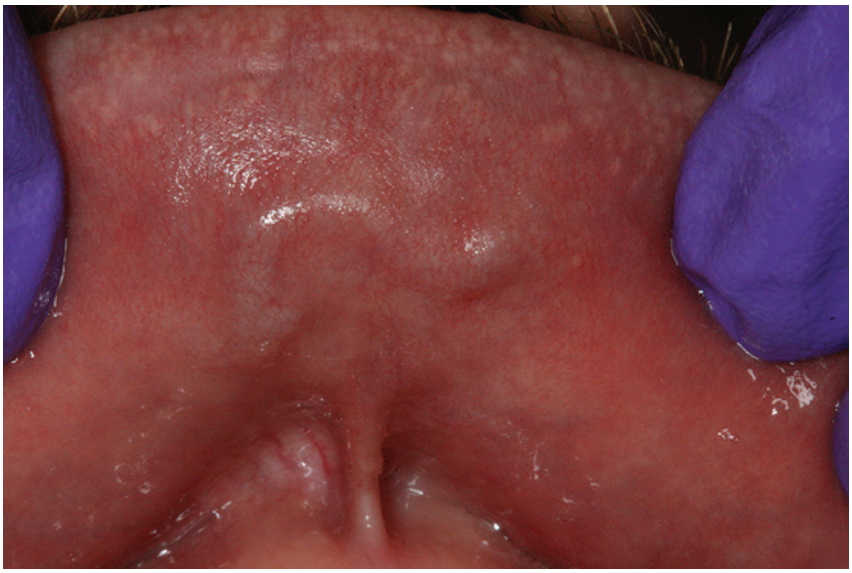
■ Figure **1.20**
Varicosities
Multiple dilated purple veins found bilaterally on the ventrolateral tongue.



■ Figure **1.21**
Varicosities
Three separate varices can be seen on the skin and vermilion border of the upper lip.



■ Figure **1.22**
Caliber-Persistent Artery
A slightly blue, arcuate vessel can be seen on the upper lip mucosa.



Coronoid Hyperplasia

Fig. **1.23**

Hyperplasia of the coronoid process of the mandible is an uncommon bony anomaly that limits the ability to open the mouth. As the jaw translates forward when the patient tries to open the mouth, the elongated coronoid process impinges on the body or arch of the zygomatic bone. **Coronoid hyperplasia** is diagnosed most frequently in teenagers, although some examples have been documented in newborns and older adults. The mean age at diagnosis is 23 to 25 years of age. Bilateral cases are four to five times more common than unilateral examples. Coronoid hyperplasia also is three to five times more common in males than in females, which suggests that there could be an endocrine influence. Heredity also may play a role because cases have been noted in siblings.

Bilateral coronoid hyperplasia presents with a progressively worsening ability to open the mouth, which typically develops over a period of several years. In unilateral examples, the mandible may deviate toward the affected side. Radiographic examination will reveal elongation of the coronoid process(es), which is often best demonstrated on CT imaging. Treatment consists of coronoidectomy or coronoidotomy, which usually is accomplished via an intraoral approach. Postoperative physiotherapy plays an important role in trying to preserve the increased oral opening.

Condylar Hyperplasia

Fig. **1.24**

Condylar hyperplasia is an uncommon bony malformation characterized by excessive growth of one or both of the mandibular condyles. The classification system developed by Wolford and associates describes four major categories: type 1—an accelerated and prolonged aberration of the "normal" condylar growth mechanism that can be either bilateral (type 1A) or unilateral (type 1B); type 2—unilateral enlargement caused by an osteochondroma; type 3—unilateral enlargement caused by other benign tumors of the condyle; and type 4—unilateral enlargement caused by malignant tumors of the condyle. Types 1 and 2 are the most common forms of condylar hyperplasia, with types 3 and 4 being much rarer. Approximately 60% of cases of type 1 condylar hyperplasia occur in females; this female predilection is even higher for type 2 cases (76%). The condition usually is discovered in teenagers and young adults.

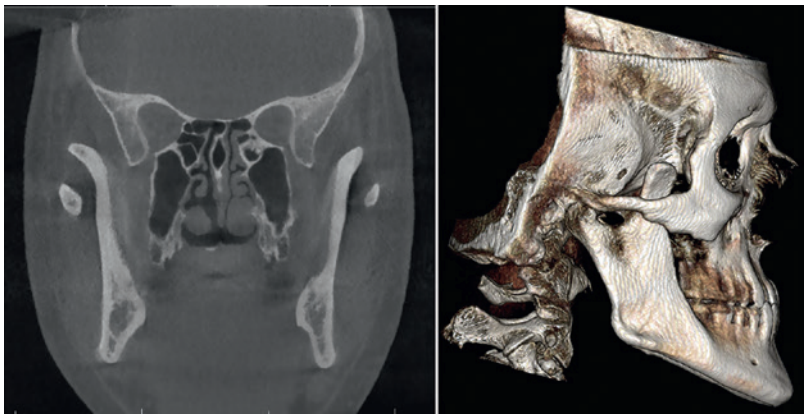
Condylar hyperplasia classically presents as a progressively worsening asymmetry of the face, which may be associated with prognathism, crossbite, and open bite. In some cases, compensatory maxillary growth occurs with tilting of the occlusal plane. Radiographs will show elongation or enlargement of the affected condyle. In addition, some cases may exhibit some degree of expansion or asymmetry of the entire ramus. Treatment usually requires condylectomy, which may be combined with orthodontic therapy and osteotomies of the mandible and maxilla.

Bifid Condyle

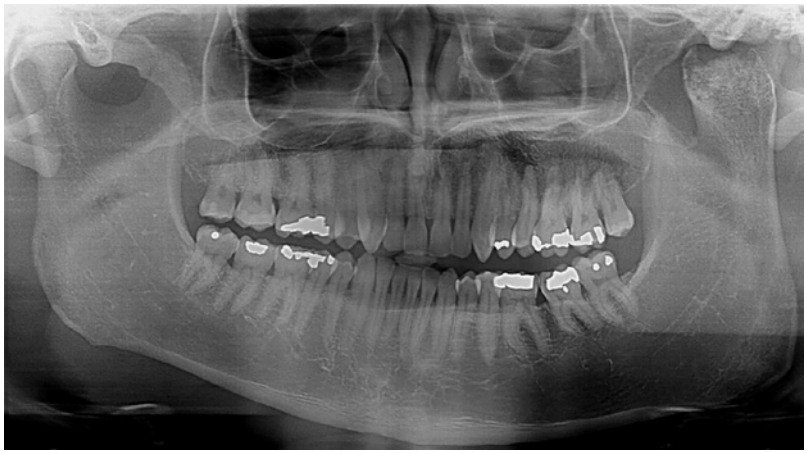
Fig. **1.25**

A **bifid condyle** is an uncommon bony anomaly in which the head of the mandibular condyle is divided into two lobes by a central groove. The reported prevalence of bifid condyle varies from 0.02% to 4.5%, probably related to the criteria used and whether the study was performed on plain radiographs, cone beam CT, or dry mandibles. Regardless, the lesion usually is discovered as an asymptomatic, incidental radiographic finding. In some instances, the patient may report a popping or clicking noise in the temporomandibular joint.

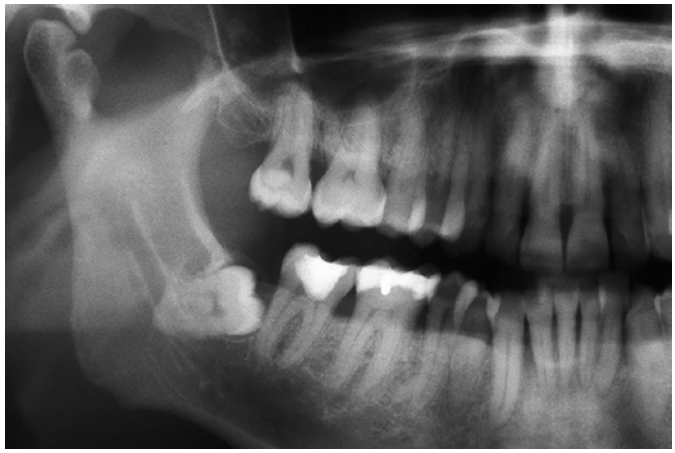
Most bifid condyles have medial and lateral lobes that are divided by an anteroposterior groove. Less commonly, anterior and posterior heads are noted, which are separated by a transverse groove. Bifid condyles usually are unilateral, although occasionally both condyles may be affected. The etiology is uncertain, although various theories have been suggested. Some examples with anterior and posterior lobes may be due to trauma, such as a fracture during childhood. Because most bifid condyles are asymptomatic, no treatment is necessary. However, if the patient complains of joint problems, appropriate temporomandibular therapy may be considered.



■ Figure **1.23**
Coronoid Hyperplasia
Coronal cone beam computed tomography and three-dimensional reconstruction showing elongation of the coronoid processes. (Courtesy Dr. Peter Green.)



■ Figure **1.24**
Condylar Hyperplasia
The left mandibular condyle shows prominent enlargement.



■ Figure **1.25**
Bifid Condyle
The right condylar head shows two lobes divided by a central groove.

Exostoses

Figs. **1.26 and 1.27**

An **exostosis** is a nodular protuberance of dense cortical bone. The most common and best-known exostoses of the jaws are the torus mandibularis and torus palatinus, which are discussed later in this chapter. However, exostoses can arise from the cortical surface in other areas of the jaws, especially along the buccal aspect of the alveolar processes or palatal to the maxillary molars. The specific cause of exostoses is uncertain, although they likely are related to both genetic factors and local stresses placed on the bone through occlusal function.

Buccal exostoses ("buttressing bone") appear as a bilateral row of smooth bony nodules along the facial alveolar process of the mandible and/or maxilla. The prevalence of buccal exostoses in different studies varies from 0.09% to nearly 19%, probably depending on the diagnostic criteria used and the population studied. Palatal exostoses occur along the lingual aspect of the maxillary molars. Such lesions are more common in males and may be unilateral or bilateral. The reported prevalence of palatal exostoses also varies widely, ranging from 8% to 69% in various studies. Many patients with buccal or palatal exostoses also have palatal and/or mandibular tori. If an exostosis is large enough, a relatively increased density of the bone might be noticed on radiographs.

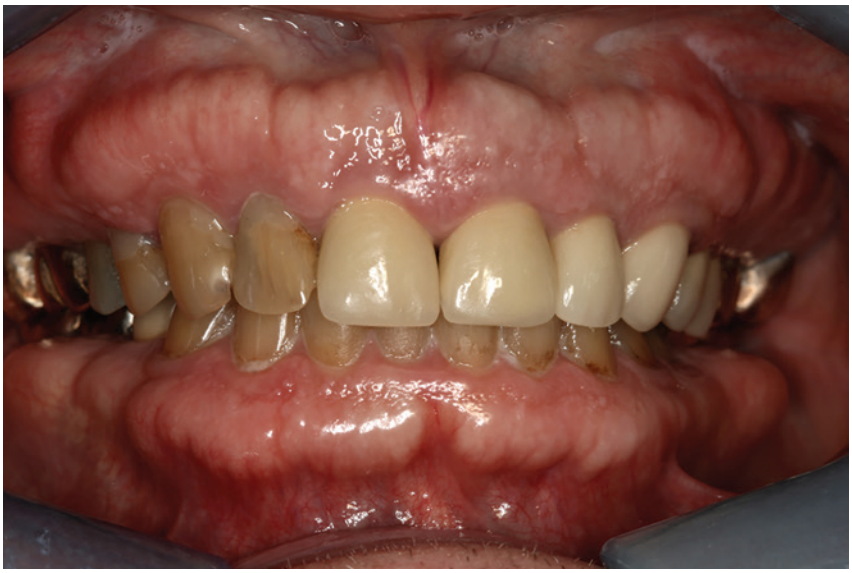
Exostoses are usually asymptomatic, although trauma to the thin overlying mucosa sometimes can result in superficial ulceration. No treatment is required for most exostoses. However, surgical removal can be performed if repeated ulceration and pain occur or if the location of the lesion interferes with the fabrication of a dental prosthesis.

Reactive Subpontine Exostosis (Subpontic Osseous Hyperplasia)

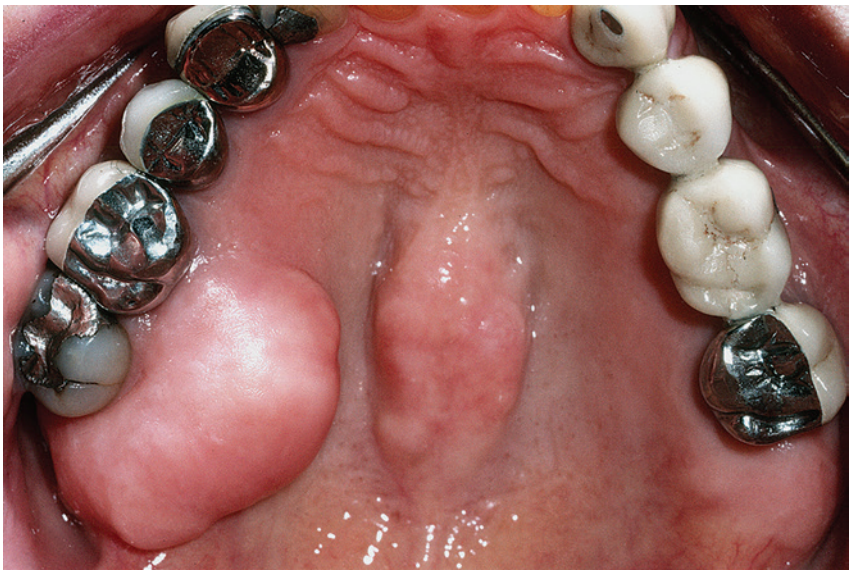
Fig. **1.28**

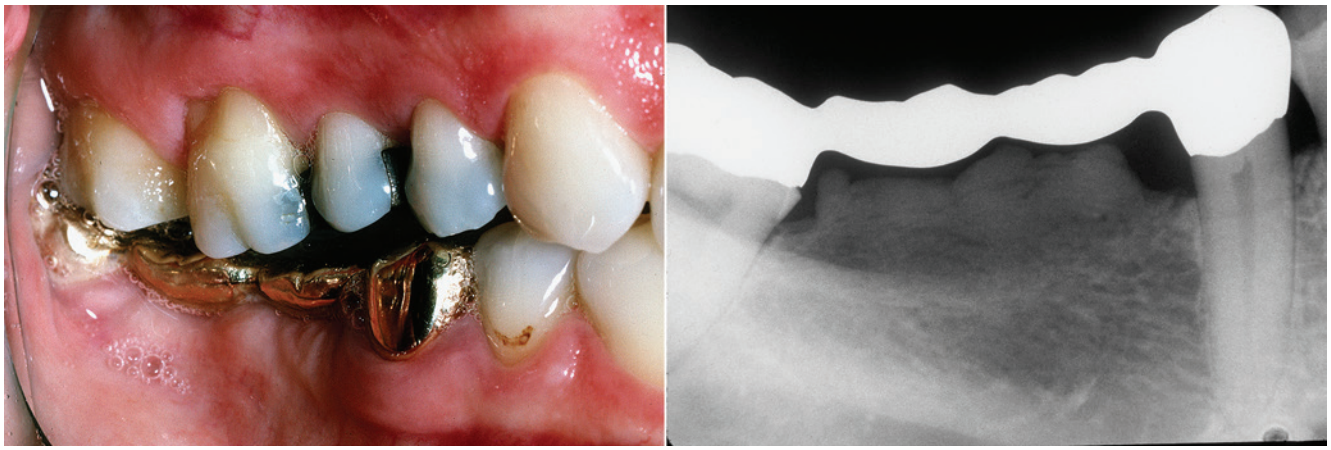
The **reactive subpontine exostosis** is a rare type of osseous hyperplasia that develops beneath the pontic of a fixed bridge. In almost all instances, such lesions occur in association with a posterior mandibular bridge. It is theorized that occlusal stresses carried through the abutment teeth of the bridge may stimulate the formation of new cortical bone under the central pontic. Such exostoses usually are discovered incidentally and do not require any treatment. However, if continued growth of the exostosis places pressure against the pontic or if it interferes with oral hygiene, then surgical removal can be performed.

■ Figure **1.26**
Buccal Exostoses
Confluent bony nodules affect the maxillary and mandibular facial alveolar processes.



■ Figure **1.27**
Palatal Exostosis
A prominent nodular mass of dense bone is present lingual to the maxillary molars. The patient also has a midline torus palatinus.





■ Figure **1.28**
Reactive Subpontine Exostosis
A nodular mass of bone has grown up beneath the pontics of this mandibular fixed bridge.

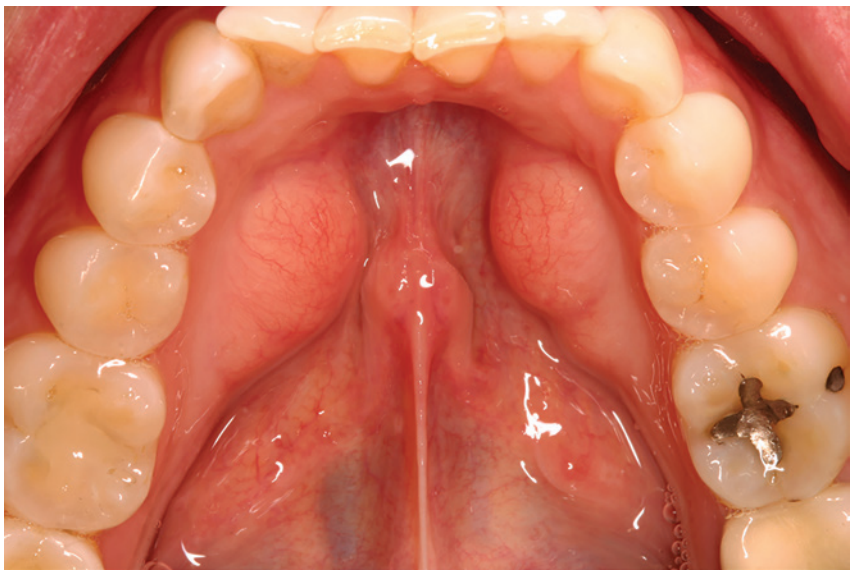
Torus Mandibularis

Figs. **1.29–1.32**

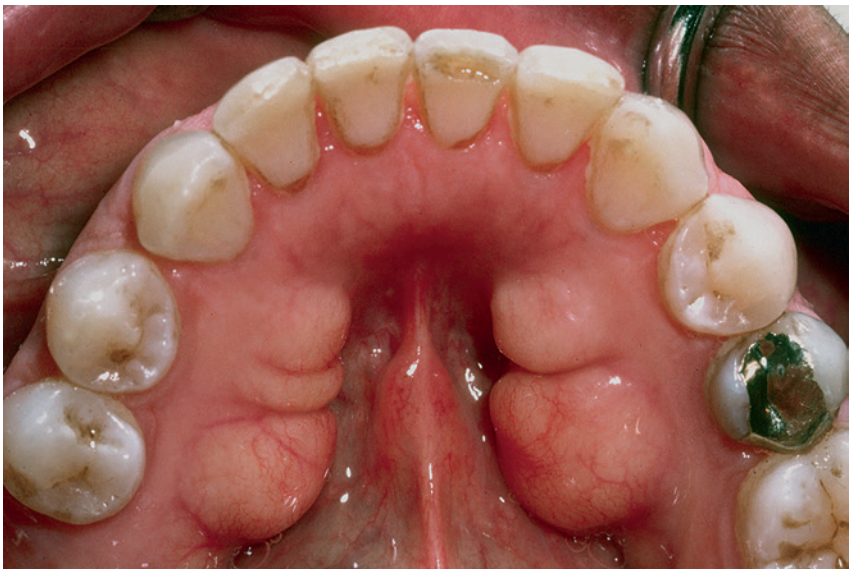
The **torus mandibularis** is a common form of exostosis that develops along the lingual aspect of the mandible above the mylohyoid line. As with other jaw exostoses, the etiology is likely multifactorial, being related to genetic susceptibility and environmental factors (such as occlusal stresses). The reported prevalence of mandibular tori varies widely, which may be related to the population studied and the diagnostic criteria used. In various studies from around the world, the frequency has ranged from as low as 3% in Malaysia to as high as 58% in Japan.

Mandibular tori typically occur in the premolar region, but larger examples also can involve the canine and first molar areas. In most instances, they are bilateral and symmetric, although unilateral examples sometimes may be noted. Most lesions occur as single bony nodules; larger examples can appear as a row of variably sized lobules that may result in a radiopacity superimposed on the roots of the mandibular teeth. In rare instances, tori may grow so large that they actually meet in the midline ("kissing tori"). Mandibular tori usually are noted as incidental findings, although trauma may result in transient superficial ulceration or abrasion. Asymptomatic tori do not require any treatment, but surgical removal may be required to accommodate a mandibular prosthesis. On occasion, mandibular tori may recur if teeth are still present in the region.

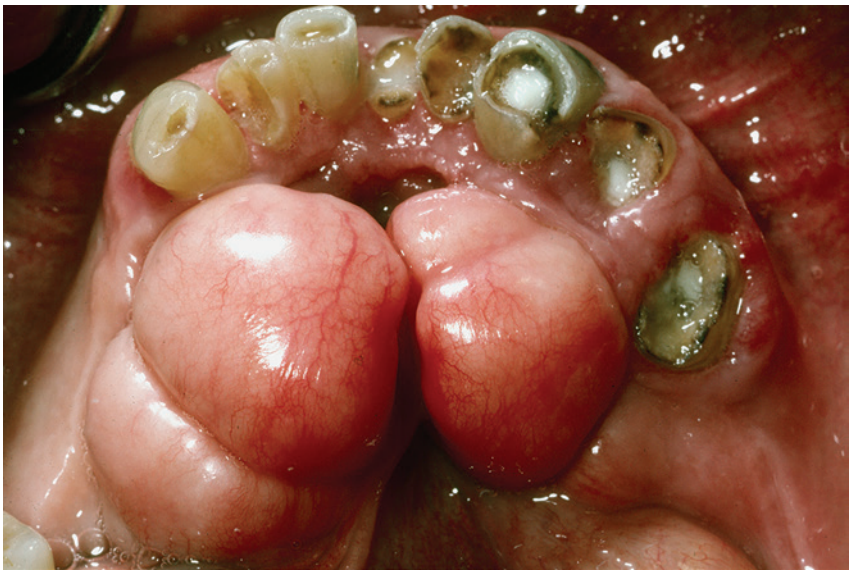
■ Figure **1.29**
Torus Mandibularis
Bilateral bony nodules are present on the lingual mandible in the premolar region.



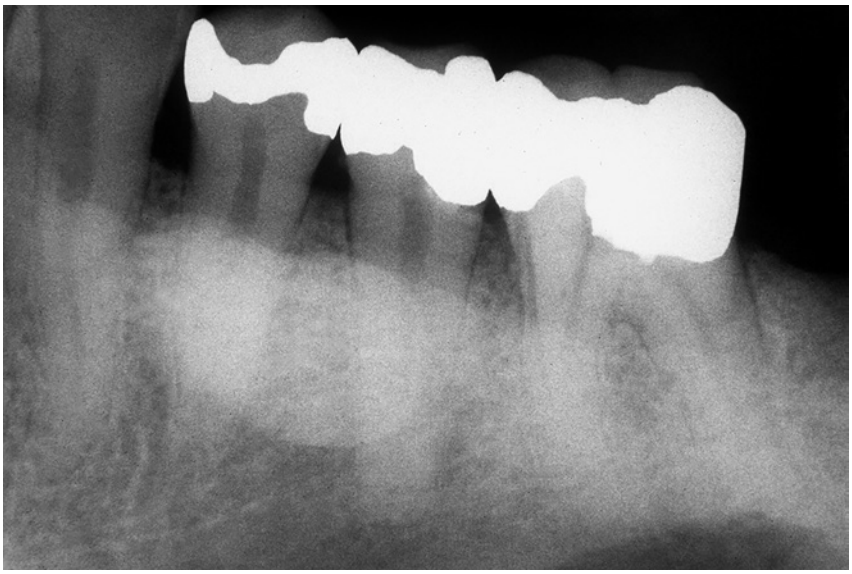
■ Figure **1.30**
Torus Mandibularis
Moderately large mandibular tori with a multilobulated appearance.



■ Figure **1.31**
Torus Mandibularis
Giant "kissing" mandibular tori.



■ Figure **1.32**
Torus Mandibularis
Periapical radiograph showing a radiopaque shadow superimposed across the roots of the mandibular teeth.



Torus Palatinus

Fig. **1.33**

The **torus palatinus** is a common exostosis that develops in the midline region of the hard palate. The reported prevalence of palatal tori, like that of mandibular tori, varies considerably, ranging from as low as 4% to more than 60%. This variation may reflect genetic differences among populations, clinical criteria used to make the diagnosis, and whether the determination was made on live patients or dried skulls. There appears to be a higher prevalence in Asian and Inuit populations. Almost all studies show that the torus palatinus occurs more often in women (female-to-male ratio equals 2:1).

The torus palatinus has a spectrum of clinical appearances, ranging from slight midline elevation of the palatal bone to large, multilobular masses. Sometimes they have been categorized on the basis of morphology into *flat*, *spindle*, *nodular*, and *lobular* subtypes. Most palatal tori are asymptomatic, and some patients may be unaware of their presence. Larger tori are more susceptible to trauma from eating, which occasionally results in superficial abrasion or ulceration. Most palatal tori do not require any treatment. However, surgical removal may be required prior to fabrication of a maxillary denture or if repeated trauma is bothersome to the patient.

Eagle Syndrome (Stylohyoid Syndrome; Carotid Artery Syndrome; Stylocarotid Syndrome)

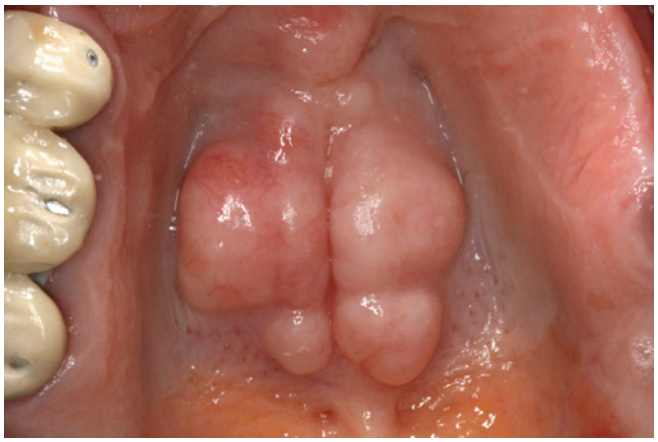
Figs. **1.34 and 1.35**

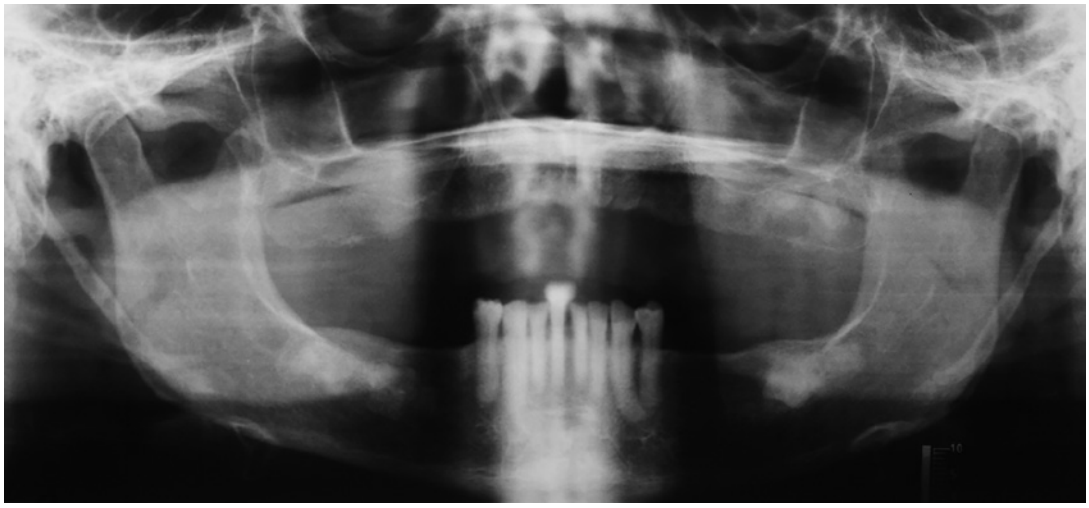
Eagle syndrome is an uncommon pain condition in which elongation of the styloid process or mineralization of the stylohyoid ligament results in a variety of clinical symptoms. The styloid process, a slender projection of bone arising from the inferior portion of the temporal bone, is connected to the hyoid bone in the neck by the stylohyoid ligament. The internal and external branches of the carotid artery are located on either side. Some degree of elongation of the styloid process or mineralization of the stylohyoid ligament is not unusual, although the reported prevalence varies widely, from 4% to greater than 40%. Regardless, only about 4% of individuals with radiographic evidence of such mineralization develop Eagle syndrome.

Eagle syndrome is characterized by unilateral pain in the anterior lateral neck, which may be precipitated by swallowing, turning the head, or yawning. This pain may radiate to the ear or temporomandibular joint. Other symptoms can include dysphagia and the sensation of a foreign body in the throat. In addition, compression of the adjacent carotid arteries can result in syncope, transient ischemic attacks, and even carotid artery dissection. Some authors distinguish between "classic" Eagle syndrome and stylohyoid syndrome. In classic Eagle syndrome, the symptoms develop after tonsillectomy, presumably due to development of scar tissue around the mineralized stylohyoid complex. **Stylohyoid syndrome** (**stylocarotid syndrome**) is not associated with a prior tonsillectomy but is thought to be due to direct impingement of the calcified stylohyoid complex upon the carotid arteries and adjacent sympathetic nerve fibers.

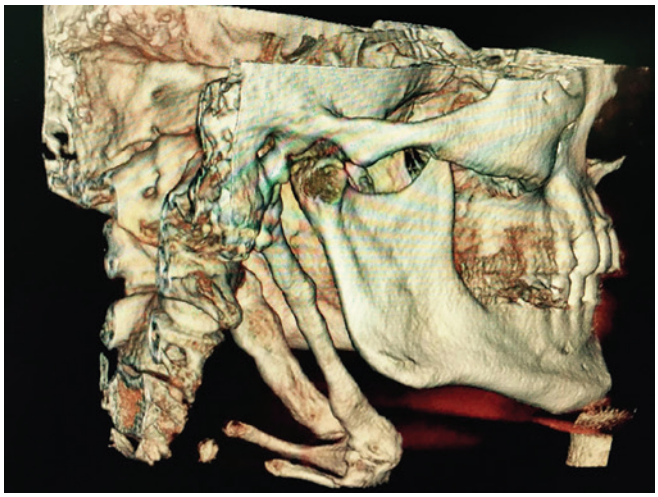
Treatment depends on the severity of the symptoms. Mild examples of Eagle syndrome may be managed conservatively using nonsteroidal antiinflammatory drugs and local injection of corticosteroids and anesthetics. More severe cases require partial surgical removal of the elongated styloid process, which can be accomplished by either a transoral tonsillar approach or an extraoral cervical approach. The prognosis after surgery is good.

■ Figure **1.33**
Torus Palatinus
Multilobulated bony growth of the hard palate.





■ Figure **1.34**
Eagle Syndrome
Panoramic radiograph showing bilateral mineralization of the stylohyoid ligament.



■ Figure **1.35**
Eagle Syndrome
Three-dimensional radiographic reconstruction showing complete ossification of the stylohyoid ligament, which attaches to the hyoid bone. (Courtesy Dr. Vicki Tatum.)

Stafne Defect (Lingual Mandibular Salivary Gland Depression; Latent Bone Cyst; Static Bone Cyst)

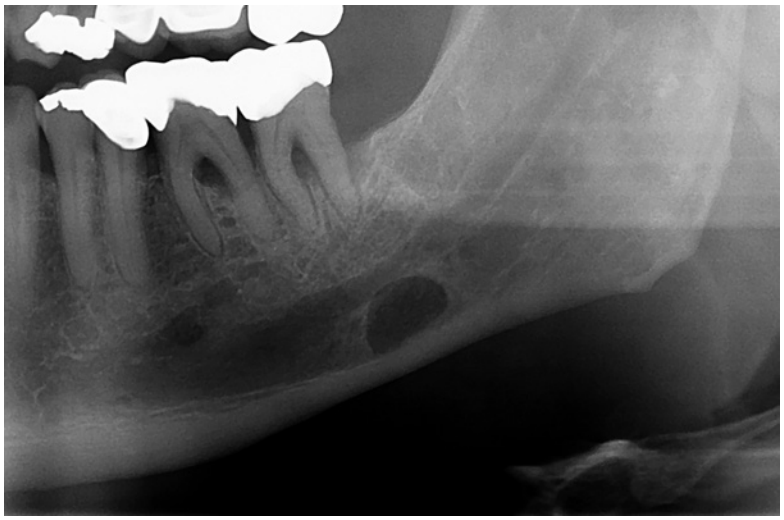
Figs. **1.36–1.39**

A **Stafne defect** is an uncommon radiographic anomaly of the mandible characterized by a cupped-out depression of the bony cortex adjacent to a major salivary gland. The lesion usually is related to the submandibular gland, although rare examples associated with the sublingual and parotid glands also have been described. Although Stafne defects generally are thought to be "developmental" in nature, they rarely are encountered in children, which indicates that these bony concavities gradually develop over time in adult patients. The posterior lingual submandibular type has been discovered on 0.08% to 0.48% of panoramic radiographs. There is a marked male predilection, with 80% to 90% of cases seen in men.

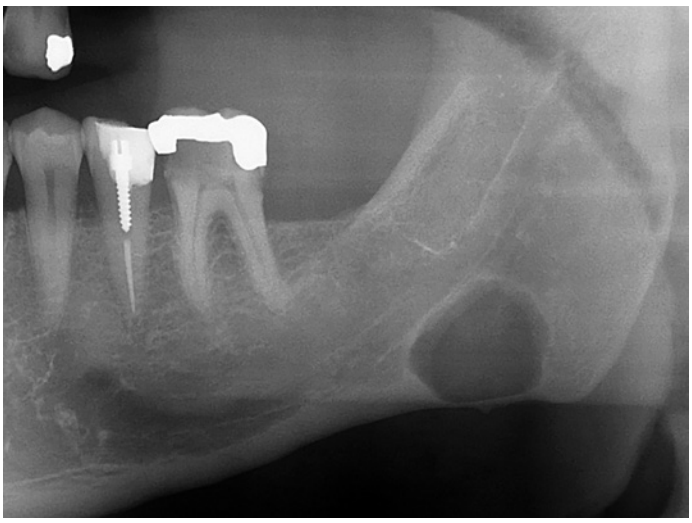
Stafne defects are asymptomatic lesions that typically are discovered as incidental findings on conventional dental radiographs. The classic posterior submandibular type appears as a well-circumscribed, corticated radiolucency near the angle of the mandible below the mandibular canal. On occasion, the lesion may involve the inferior border of the mandible, resulting in a palpable notch in this area. Anterior sublingual gland defects present as well-circumscribed radiolucencies located apical to the premolar or anterior teeth. Such a lesion may be mistaken for periapical pathosis. Exceedingly rare parotid examples may produce a radiolucency higher in the mandibular ramus. Computed tomography (CT), such as cone-beam CT, can be helpful to confirm that a suspected Stafne defect represents a cortical concavity rather than some other intrabony lesion.

Once discovered, a Stafne defect usually remains stable in size—hence the term *static bone cyst.* However, if discovered early enough in its formation, it is possible to see radiographic evidence of enlargement over time before the lesion becomes stable. No treatment is warranted for Stafne defects, and the prognosis is excellent.

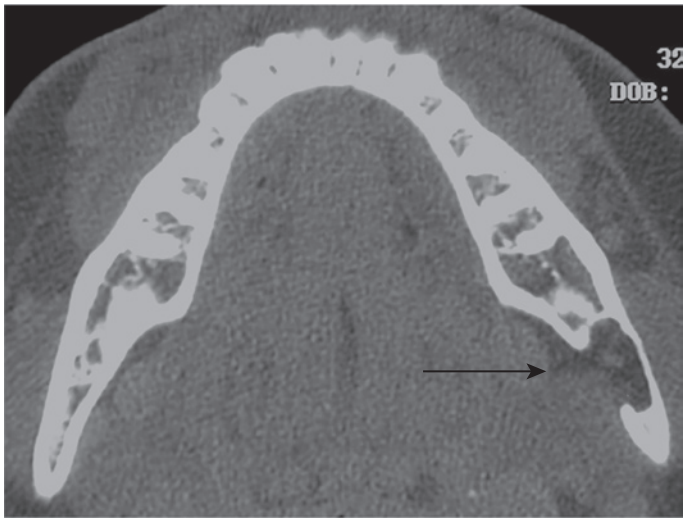
■ Figure **1.36**
Stafne Defect
Circumscribed radiolucency located below the mandibular canal near the inferior border of the mandible. (Courtesy Dr. Caleb Poston.)

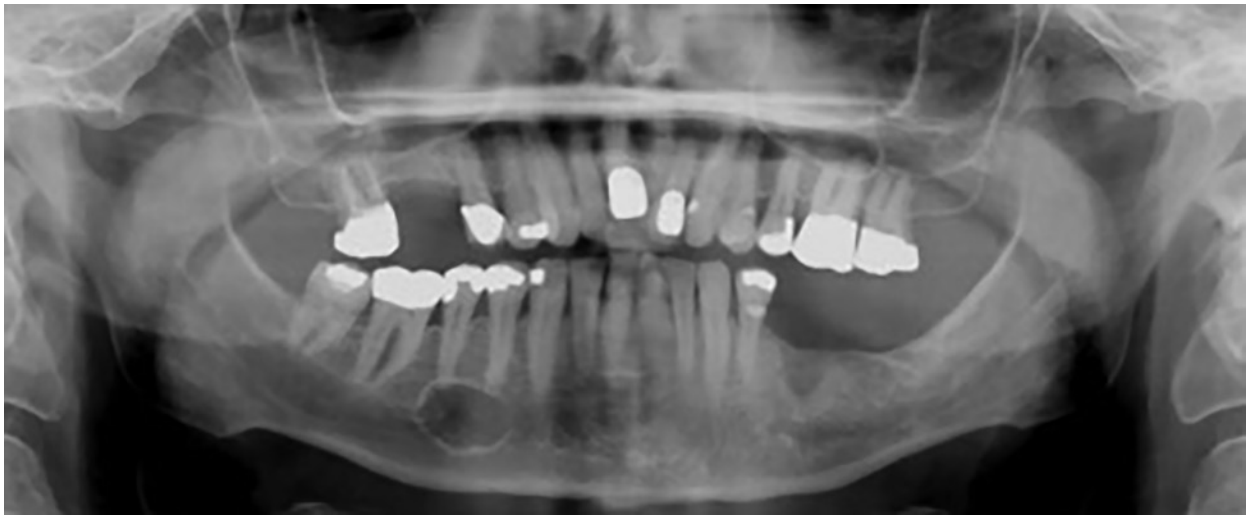


■ Figure **1.37**
Stafne Defect
Large radiolucency near the angle of the posterior mandible. (Courtesy Dr. Terry Day.)



■ Figure **1.38**
Stafne Defect
Axial computed tomography image showing a cupped-out defect on the lingual surface of the mandible *(arrow)*. (Courtesy Dr. Kim Tambini.)





■ Figure **1.39**
Stafne Defect
Anterior Stafne defect associated with the sublingual gland. The lesion appears as a well-circumscribed corticated radiolucency apical to the right mandibular premolars.

Epstein Pearls

Fig. **1.40**

Epstein pearls are tiny, congenital, keratin-filled cysts found at the junction of the hard and soft palate near the midline. Such lesions are quite common, having been reported in 55% to 89% of newborns. The terminology associated with various congenital oral cysts is confusing. Theoretically, Epstein pearls develop from epithelium entrapped along the line of fusion of the lateral palatal shelves. The term *Bohn nodules* has been used to describe similar keratin-filled cysts scattered across the posterior hard/soft palate junction, presumably arising from epithelial remnants of the developing minor salivary glands. However, over the years these two terms have been used interchangeably, sometimes also in reference to gingival cysts of the newborn.

Epstein pearls appear as one to several white papules on the posterior midline region of the hard palate at the junction with the soft palate. No treatment is necessary because the lesions are asymptomatic and typically will disappear within a few weeks.

Nasopalatine Duct Cyst (Incisive Canal Cyst)

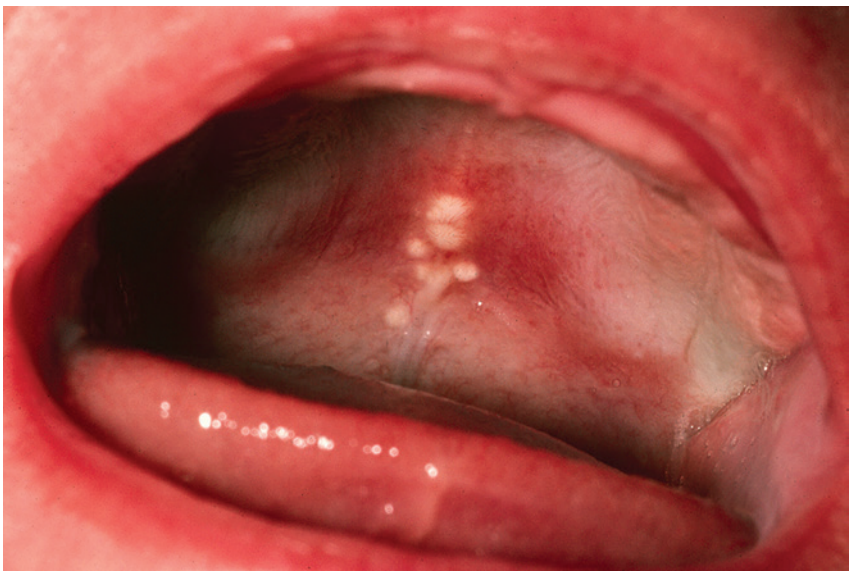
Figs. **1.41 and 1.42**

The most common nonodontogenic cyst of the oral cavity is the **nasopalatine duct cyst**, which is estimated to occur in 1% of the population. This developmental cyst arises from remnants of the nasopalatine ducts, which are paired embryonic passageways running through the incisive canal between the oral cavity and nasal cavity. Normally these ducts degenerate and disappear before birth, although epithelial remnants may remain in the incisive canal region and later give rise to a cyst. Nasopalatine duct cysts can develop at any age, but they are most commonly diagnosed in young to middle-aged adults. They occur twice as often in men as in women. Several examples have been reported to develop following placement of a dental implant in the area.

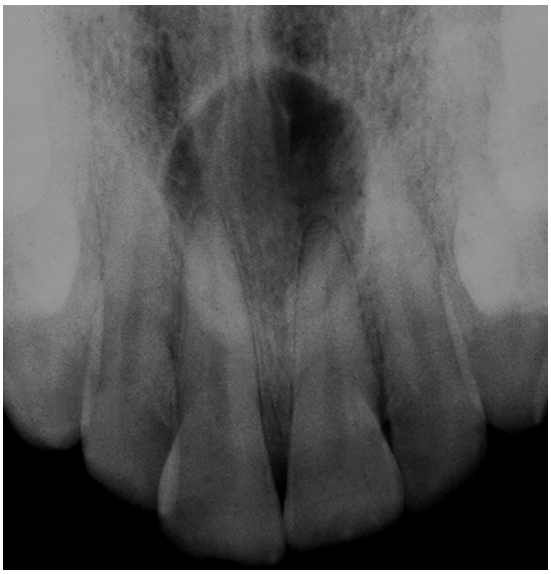
Small nasopalatine duct cysts may be discovered incidentally on dental radiographs, although larger lesions can produce symptoms such as swelling, pain, pressure, or drainage. Radiographically, the lesion appears as a well-circumscribed unilocular radiolucency that can range from less than 6 mm to more than 6 cm in diameter. Most examples are in the range of 1.0 to 2.5 cm. It is sometimes difficult to distinguish a small nasopalatine duct cyst from a large incisive canal. Generally a radiolucency smaller than 6 mm in diameter is considered to be a normal canal unless other signs or symptoms are present. Typically the radiolucency is found in the midline region of the maxilla superior to the apices of the central incisors, although some examples will extend laterally in an asymmetric fashion. In most cases, the radiolucency appears round to oval with a corticated rim. However, some examples may have the shape of an inverted pear or a heart because of resistance from the roots of the adjacent teeth or from superimposition of the nasal spine. Occasionally a nasopalatine duct cyst will occur solely in the soft tissue of the anterior palate ("**cyst of the incisive papilla**"). Such lesions may exhibit a bluish color due to the presence of fluid within the cyst lumen.

Nasopalatine duct cysts are treated by surgical enucleation, usually from a palatal approach. The lesion rarely recurs. Extremely rare examples of malignant transformation of the cystic lining have been reported.

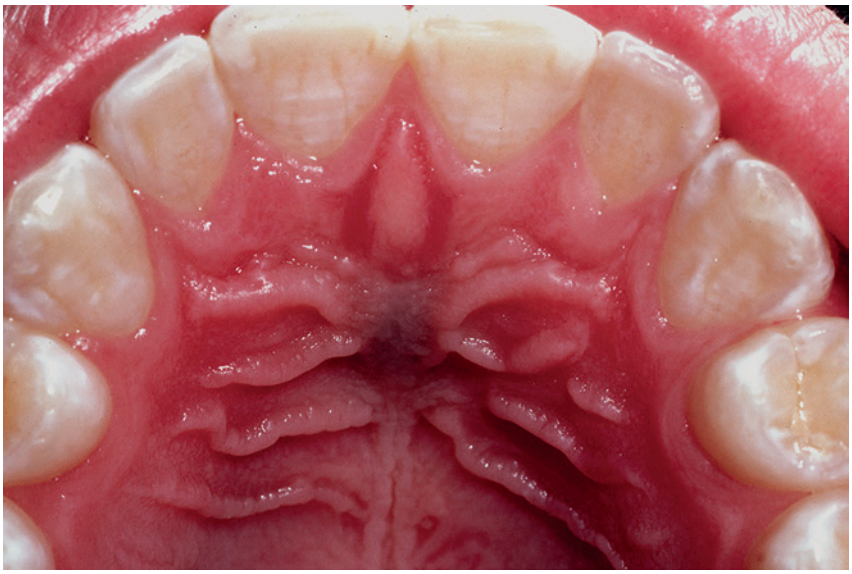
■ Figure **1.40**
Epstein Pearls
Newborn infant with multiple white papules found in the midline region at the junction of the hard and soft palate.



■ Figure **1.41**
Nasopalatine Duct Cyst
Well-circumscribed ovoid radiolucency of the anterior maxilla apical to the central incisors.



■ Figure **1.42**
Nasopalatine Duct Cyst ("Cyst of the Incisive Papilla")
Slight bluish soft tissue swelling located just behind the incisive papilla.



Epidermoid Cyst (Infundibular Cyst)

Figs. **1.43 and 1.44**

Keratin-filled cysts of the skin are common lesions that arise from hair follicles. The most common example is the **epidermoid cyst**, which is derived from the follicular infundibulum. Such lesions may develop secondary to inflammation of the hair follicle, and they are found most frequently in sites prone to acne, such as the head, neck, and back. Although follicular cysts of the skin often are referred to as *sebaceous cysts*, this term is a misnomer because neither the epidermoid cyst nor the pilar cyst (see next topic) arises from sebaceous glands. Another type of keratin-filled cyst of the skin is unrelated to hair follicles but instead arises secondary to traumatic implantation of the surface epithelium. Such lesions also can develop on oral mucosa and are designated as **epithelial** (or **epidermal**) **inclusion cysts**.

Epidermoid cysts on the skin occur more frequently in males than in females. They present as nodular subcutaneous growths that are often fluctuant to palpation. If the cyst becomes inflamed or infected, then the epidermal surface will appear red. Rupture of the cyst may release keratinaceous material, which typically will elicit a pronounced foreign-body response, resulting in pain and swelling. Epidermoid cysts rarely develop during childhood unless the patient has Gardner syndrome. Younger adults are more likely to develop these cysts on the face, whereas older adults are more likely to have them on the back. Epithelial inclusion cysts related to implanted epithelium usually appear as small, yellowish-white papules.

Epidermoid and epithelial inclusion cysts usually are treated by conservative surgical excision, and recurrence is uncommon. Extremely rare examples of malignant transformation of epidermoid cysts have been reported.

Pilar Cyst

Fig. **1.45**

In addition to the epidermoid cyst, the **pilar cyst** is the second type of follicular cyst that arises from the outer sheath of the hair follicle. Also known as a trichilemmal cyst or isthmus-catagen cyst, it comprises approximately 10% to 15% of skin cysts. Unlike the epidermoid cyst, the pilar cyst occurs most frequently on the scalp and exhibits a female predilection. A tendency to develop such cysts may run in families, and some patients will develop multiple lesions. Pilar cysts appear as movable nodules that typically shell out easily when surgically removed.