INITIATION

The primary teeth form in dental crypts that arise from a band of epithelial cells incorporated into each developing jaw. By 12 wk of fetal life, each of these epithelial bands (dental laminae) has five areas of rapid growth on each side of the maxilla and the mandible, seen as rounded, budlike enlargements. Organization of adjacent mesenchyme takes place in each area of epithelial growth, and the two elements together are the beginning of a tooth.

After the formation of these crypts for the 20 primary teeth, another generation of tooth buds forms lingually (toward the tongue), which will develop into the succeeding permanent incisors, canines, and premolars that eventually replace the primary teeth. This process takes place from ≈3 mo of gestation for the central incisors to about 10 mo of age for the 2nd premolars. The permanent 1st, 2nd, and 3rd molars, on the other hand, arise from extension of the dental laminae distal to the 2nd primary molars; buds for these teeth develop at ≈4 mo of gestation, 1 yr of age, and 4–5 yr of age, respectively.

HISTODIFFERENTIATION-MORPHODIFFERENTIATION. As the epithelial bud proliferates, the deeper surface invaginates and a mass of mesenchyme becomes partially enclosed. The epithelial cells differentiate into the ameloblasts that lay down an organic matrix that forms enamel; the mesenchyme forms the dentin and dental pulp.

CALCIFICATION. After the organic matrix has been laid down, the deposition of the inorganic mineral crystals takes place from several sites of calcification that later coalesce. The characteristics of the inorganic portions of a tooth can be altered by (1) disturbances in formation of the matrix, (2) decreased availability of minerals, or (3) the incorporation of foreign materials. Such disturbances may affect the color, texture, or thickness of the tooth surface. Calcification of primary teeth begins at 3–4 mo in utero and concludes postnatally at ≈12 mo with mineralization of the 2nd primary molars (Table 304-1).

ERUPTION. At the time of tooth bud formation, each tooth begins a continuous movement toward the oral cavity. The times of eruption of the primary and permanent teeth are listed in Table 304-1.

## ANOMALIES ASSOCIATED WITH TOOTH DEVELOPMENT

Both failures and excesses of tooth initiation are observed. Developmentally missing teeth can result from environmental insult, a genetic defect involving only teeth, or the manifestation of a syndrome. Anodontia, or absence of teeth, occurs when no tooth buds form (ectodermal dysplasia, or familial missing teeth) or when there is a disturbance of a normal site of initiation (the area of a palatal cleft). The teeth that are most commonly absent

### TABLE 304-1. Calcification, Crown Completion, and Eruption

<table>
<thead>
<tr>
<th>TOOTH</th>
<th>FIRST EVIDENCE OF CALCIFICATION</th>
<th>CROWN COMPLETED</th>
<th>ERUPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PRIMARY DENTITION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maxillary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central incisor</td>
<td>3–4 mo in utero</td>
<td>4 mo</td>
<td>7½ mo</td>
</tr>
<tr>
<td>Lateral incisor</td>
<td>4½ mo in utero</td>
<td>5 mo</td>
<td>8 mo</td>
</tr>
<tr>
<td>Canine</td>
<td>5½ mo in utero</td>
<td>9 mo</td>
<td>16–20 mo</td>
</tr>
<tr>
<td>First molar</td>
<td>5 mo in utero</td>
<td>6 mo</td>
<td>12–16 mo</td>
</tr>
<tr>
<td>Second molar</td>
<td>6 mo in utero</td>
<td>10–12 mo</td>
<td>20–30 mo</td>
</tr>
<tr>
<td>Mandibular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central incisor</td>
<td>4½ mo in utero</td>
<td>4 mo</td>
<td>6½ mo</td>
</tr>
<tr>
<td>Lateral incisor</td>
<td>4½ mo in utero</td>
<td>6½ mo</td>
<td>7½ mo</td>
</tr>
<tr>
<td>Canine</td>
<td>5½ mo in utero</td>
<td>9½ mo</td>
<td>16–20 mo</td>
</tr>
<tr>
<td>First molar</td>
<td>5½ mo in utero</td>
<td>6 mo</td>
<td>12–16 mo</td>
</tr>
<tr>
<td>Second molar</td>
<td>6½ mo in utero</td>
<td>10–12 mo</td>
<td>20–30 mo</td>
</tr>
<tr>
<td><strong>PERMANENT DENTITION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maxillary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central incisor</td>
<td>3–4 mo</td>
<td>4–5 yr</td>
<td>7–8 yr</td>
</tr>
<tr>
<td>Lateral incisor</td>
<td>10 mo</td>
<td>4–5 yr</td>
<td>8–9 yr</td>
</tr>
<tr>
<td>Canine</td>
<td>4–5 mo</td>
<td>6–7 yr</td>
<td>11–12 yr</td>
</tr>
<tr>
<td>First premolar</td>
<td>1½–1½ yr</td>
<td>5–6 yr</td>
<td>10–11 yr</td>
</tr>
<tr>
<td>Second premolar</td>
<td>2–2½ yr</td>
<td>6–7 yr</td>
<td>10–12 yr</td>
</tr>
<tr>
<td>First molar</td>
<td>4½ yr</td>
<td>6–7 yr</td>
<td>12–13 yr</td>
</tr>
<tr>
<td>Second molar</td>
<td>2½–3 yr</td>
<td>7–8 yr</td>
<td>12–13 yr</td>
</tr>
<tr>
<td>Third molar</td>
<td>7–9 yr</td>
<td>12½–15 yr</td>
<td>17–21 yr</td>
</tr>
<tr>
<td>Mandibular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central incisor</td>
<td>3–4 mo</td>
<td>4–5 yr</td>
<td>6–7 yr</td>
</tr>
<tr>
<td>Lateral incisor</td>
<td>3–4 mo</td>
<td>4–5 yr</td>
<td>7–8 yr</td>
</tr>
<tr>
<td>Canine</td>
<td>4½–5 mo</td>
<td>6–7 yr</td>
<td>9–10 yr</td>
</tr>
<tr>
<td>First premolar</td>
<td>1½–2 yr</td>
<td>5–6 yr</td>
<td>10–12 yr</td>
</tr>
<tr>
<td>Second premolar</td>
<td>2½–3½ yr</td>
<td>6–7 yr</td>
<td>11–12 yr</td>
</tr>
<tr>
<td>First molar</td>
<td>4½ yr</td>
<td>6–7 yr</td>
<td>11–12 yr</td>
</tr>
<tr>
<td>Second molar</td>
<td>2½–3 yr</td>
<td>7–8 yr</td>
<td>11½–13 yr</td>
</tr>
<tr>
<td>Third molar</td>
<td>8–10 yr</td>
<td>12–16 yr</td>
<td>17–21 yr</td>
</tr>
</tbody>
</table>

include the 3rd molars, the maxillary lateral incisors, and the mandibular 2nd premolars.

If the dental lamina produces more than the normal number of buds, supernumerary teeth occur, most often in the area between the maxillary central incisors. Because they tend to disrupt the position and eruption of the adjacent normal teeth, their identification by radiographic examination is important. Supernumerary teeth also occur with cleidocranial dysplasia (see Chapter 308) and in the area of cleft palates.

Twinning, in which two teeth are joined together, is most often observed in the mandibular incisors of the primary dentition. It can result from gemination, fusion, or concrescence. Gemination is the result of the division of one tooth germ to form a bifid crown on a single root with a common pulp canal; an extra tooth appears to be present in the dental arch. Fusion is the joining of incompletely developed teeth that, owing to pressure, trauma, or crowding, continue to develop as one tooth. Fused teeth are sometimes joined along their entire length; in other cases, a single wide crown is supported on two roots. Concrescence is the attachment of the roots of closely approximated adjacent teeth by an excessive deposit of cementum. This type of twinning, unlike the others, is found most often in the maxillary molar region.

Disturbances during differentiation can result in alterations in dental morphology, such as macrodontia (large teeth) or microdontia (small teeth). The maxillary lateral incisors may assume a slender, tapering shape (peg-shaped laterals).

Amelogenesis imperfecta represents a group of hereditary conditions that manifest in enamel defects of the primary and permanent teeth without evidence of systemic disorders (Fig. 304-1). The teeth are covered by only a thin layer of abnormally formed enamel through which the yellow underlying dentin is seen. The primary teeth are generally affected more than the permanent teeth. Susceptibility to caries is low, but the enamel is subject to destruction from abrasion. Complete coverage of the crown may be indicated for dentin protection, to reduce tooth sensitivity, and for improved appearance.

Dentinogenesis imperfecta, or hereditary opalescent dentin, is an analogous condition to amelogenesis imperfecta in which the odontoblasts fail to differentiate normally, resulting in poorly calcified dentin (Fig. 304-2). This autosomal dominant disorder may also occur in patients with osteogenesis imperfecta. The enamel-dentin junction is altered, causing enamel to break away.

The exposed dentin is then susceptible to abrasion, in some cases worn to the gingiva. The teeth are opaque and pearly, and the pulp chambers are generally obliterated by calcification. Both primary and permanent teeth are usually involved.

Localized disturbances of calcification that correlate with periods of illness, malnutrition, premature birth, or birth trauma are common. Hypocalcification appears as opaque white patches or horizontal lines on the tooth; hypoplasia is more severe and manifests as pitting or areas devoid of enamel. Systemic conditions, such as renal failure and cystic fibrosis, are associated with enamel defects. Local trauma to the primary incisors can also affect calcification of permanent incisors.

Fluorosis (mottled enamel) can result from systemic fluoride consumption >0.05 mg/kg/day during enamel formation. This high fluoride consumption can be caused by residing in an area of high fluoride content of the drinking water (>2.0 ppm), swallowing excessive fluoridated toothpaste, or inappropriate fluoride prescriptions. Excessive fluoride during enamel formation affects ameloblastic function, resulting in inconspicuous white, lacy patches on the enamel to severe brownish discoloration and hypoplasia. The latter changes are usually seen with fluoride concentrations in the drinking water >5.0 ppm.

Discolored teeth can result from incorporation of foreign substances into developing enamel. Neonatal hyperbilirubinemia can produce blue to black discoloration of the primary teeth. Porphyria produces a red-brown discoloration. Tetracyclines are extensively incorporated into bones and teeth and, if administered during the period of formation of enamel, can result in brown-yellow discoloration and hypoplasia of the enamel. Such teeth fluoresce under ultraviolet light. The period at risk extends from ≈4 mo of gestation to 7 yr of life. Repeated or prolonged therapy with tetracycline carries the highest risk.

Delayed eruption of the 20 primary teeth can be familial or indicate systemic or nutritional disturbances such as hypothyroidism, cleidocranial dysplasia, trisomy 21, progeria, Albright osteodystrophy, incontinentia pigmenti, rickets, and multiple syndromes. Failure of eruption of single or small groups of teeth can arise from local causes such as malpositioned teeth, supernumerary teeth, cysts, or retained primary teeth. Premature loss of primary teeth is most commonly caused by premature eruption of the permanent teeth. If the entire dentition is advanced for age and sex, precocious puberty or hyperthyroidism should be considered.
Natal teeth are observed in ~1/2,000 newborn infants; usually, there are two in the position of the mandibular central incisors. Natal teeth are present at birth, whereas neonatal teeth erupt in the 1st mo of life. Attachment of natal/neonatal teeth is generally limited to the gingival margin, with little root formation or bony support. They may be a supernumerary or a prematurely erupted primary tooth. A radiograph can easily differentiate between the two conditions. Natal teeth are associated with cleft palate, Pierre Robin syndrome, Ellis–van Creveld syndrome, Hallermann-Streiff syndrome, pachyonychia congenita, and other anomalies. A family history of natal teeth or premature eruption is present in 15–20% of affected children.

Natal/neonatal teeth can occasionally result in pain and refusal to feed and, at times, can produce maternal discomfort because of abrasion or biting of the nipple during nursing. There is a remote danger of detachment, with aspiration of the tooth. Because the tongue lies between the alveolar processes during birth, it can become lacerated, and, occasionally, the tip is amputated (Riga-Fede disease). Decisions regarding extraction of prematurely erupted primary teeth must be made on an individual basis.

Exfoliation failure occurs when a primary tooth is not shed before the eruption of its permanent successor. Most often the primary tooth exfoliates eventually, but in some cases, the primary tooth may need to be extracted. This occurs most commonly in the mandibular incisor region.

TABLE 305-1.

<table>
<thead>
<tr>
<th>MEDICAL CONDITION</th>
<th>COMMON ASSOCIATED DENTAL OR ORAL FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleft lip and palate</td>
<td>Missing teeth, extra (supernumerary) teeth, shifting of arch segments, feeding difficulties, speech problems</td>
</tr>
<tr>
<td>Kidney failure</td>
<td>Mottled enamel (permanent teeth), facial dysmorphology</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>Stained teeth with extensive medication, mottled enamel</td>
</tr>
<tr>
<td>Immunosuppression</td>
<td>Oral candidiasis with potential for systemic candidiasis, cyclosporine-induced gingival hyperplasia</td>
</tr>
<tr>
<td>Low birthweight with prolonged oral intubation</td>
<td>Palatal groove, narrow arch</td>
</tr>
<tr>
<td>Heart defects with susceptibility for bacterial endocarditis</td>
<td>Bacteremia from dental procedures or trauma</td>
</tr>
<tr>
<td>Neurophil chromatic deficiency</td>
<td>Juvenile periodontitis (loss of supporting bone around teeth)</td>
</tr>
<tr>
<td>Neuroendocrine dysfunction</td>
<td>Oral trauma from failing malocclusion (open bite); gingivitis from lack of hygiene</td>
</tr>
<tr>
<td>Prolonged illness (generalized) during tooth formation</td>
<td>Enamel hypoplasia of crown portions forming during illness</td>
</tr>
<tr>
<td>Prolonged illness (generalized) seizures</td>
<td>Enamel hypoplasia</td>
</tr>
<tr>
<td>Maternal infections</td>
<td>Gingival enlargement if pharynx is used</td>
</tr>
<tr>
<td>Vitamin D-dependent rickets</td>
<td>Siderophylia—abnormally shaped teeth</td>
</tr>
</tbody>
</table>

Disorders of the teeth and surrounding structures may occur in isolation or in combination with other systemic conditions (Table 305-1). Most commonly, medical conditions that occur during tooth development may affect tooth formation or appearance. Damage to teeth during their development is permanent.

VARIATIONS IN GROWTH PATTERNS. Growth patterns are classified into three main types of occlusion, determined when the jaws are closed and the teeth are held together (Fig. 306-1). According to the Angle Classification of Malocclusion, in class I occlusion (normal), the cusps of the posterior mandibular teeth interdigitate ahead of and inside the corresponding cusps of the opposing maxillary teeth. This relationship provides a normal facial profile. In class II malocclusion, “buck teeth,” the cusps of the posterior mandibular teeth are behind and inside the corresponding cusps of the maxillary teeth. This common occlusal disharmony is found in ~45% of the population. The facial profile may give the appearance of a “receding chin” (retrogнатia) or protruding front teeth. The resultant increased space between upper and lower anterior teeth encourages finger sucking and tongue-thrust habits. Additionally, children with pronounced class II malocclusions are at greater risk of damage to the incisors due to trauma. In class III malocclusion, “underbite,” the cusps of the posterior mandibular teeth interdigitate a tooth or more ahead of their opposing maxillary counterparts. The anterior teeth appear in cross bite with the mandibular incisors protruding beyond the maxillary incisors. The facial profile gives the appearance of a “protruding chin” (prognathia).

CROSS BITE. Normally, the mandibular teeth are in a position just inside the maxillary teeth, so that the outside mandibular cusps or incisal edges meet the central portion of the opposing maxillary teeth. A reversal of this relation is referred to as a cross bite. Cross bites can be anterior, involving the incisors; can be posterior, involving the molars; or can involve single or multiple teeth.

OPEN AND CLOSED BITES. If the posterior mandibular and maxillary teeth make contact with each other, but the anterior teeth are still apart, the condition is called an open bite. Open bites are due to trauma. Additionally, children with pronounced class II malocclusions are at greater risk of damage to the incisors due to trauma. In class III malocclusion, “underbite,” the cusps of the posterior mandibular teeth interdigitate a tooth or more ahead of their opposing maxillary counterparts. The anterior teeth appear in cross bite with the mandibular incisors protruding beyond the maxillary incisors. The facial profile gives the appearance of a “receding chin” (retrogнатia).

DENTAL CROWDING. Overlap of incisors can result when the jaws are too small or the teeth are too large for adequate alignment of the teeth. Growth of the jaws is mostly in the posterior aspects of the mandible and maxilla, and, therefore, inadequate space for the teeth at 7 or 8 yr of age will not resolve with growth of the jaws. Spacing in the primary dentition is normal and favorable for adequate alignment of successor teeth.

DIGIT SUCKING. Various and conflicting etiologic theories and recommendations for correction have been proposed for digit sucking in children. Prolonged digit sucking can cause flaring of
the maxillary incisor teeth, an open bite, as well as a posterior cross bite. The prevalence of digit sucking decreases steadily from the age of 2 yr to ~10% by the age of 5. The earlier the habit is discontinued after the eruption of the permanent maxillary incisors (age 7–8 yr), the greater the likelihood that there will be lessening effects on the dentition. A variety of treatments have been suggested, from behavioral modification to insertion of an appliance with extensions that serves as a reminder when the child attempts to insert the digit. The greatest likelihood of success occurs in cases in which the child desires to stop. Stopping the habit, however, will not rectify a malocclusion caused by a prior deviant growth pattern.

**INCIDENCE AND EPIDEMIOLOGY.** The incidence of cleft lip with or without cleft palate is ~1/750 white births; the incidence of cleft palate alone is ~1/1,500 white births. Clefts of the lip are more common in males. Possible causes include maternal drug exposure, a syndrome-malformation complex, or genetic factors. Although both may appear to occur sporadically, the presence of susceptibility genes appears important. There are families in which a cleft lip or palate, or both, is inherited in a dominant fashion (van der Woude syndrome), and careful examination of parents is important to distinguish this type from others, because the recurrence risk is 50%. Ethnic factors also affect the incidence of cleft lip and palate; the incidence is highest among Asians and Native Americans, and lowest among blacks. The incidence of associated congenital malformations (chromosomal aneuploidy, holoprosencephaly) and of impairment in development is increased in children with cleft defects, especially in those with cleft palate alone. The risks of recurrence of cleft defects within families are discussed in Chapters 80 and 83.

**CLINICAL MANIFESTATIONS.** Cleft lip may vary from a small notch in the vermilion border to a complete separation involving skin, muscle, mucosa, tooth, and bone. Clefts may be unilateral (more often on the left side) or bilateral and may involve the alveolar ridge. Deformed, supernumerary, or absent teeth are associated findings.

A secondary cleft palate occurs in the midline and may involve only the uvula or may extend into or through the soft and hard palates to the incisive foramen. When associated with cleft lip, the defect may involve the midline of the soft palate and extend into the hard palate on one or both sides, exposing one or both of the nasal cavities as a unilateral or bilateral cleft palate. The palate may also present with a submucosal cleft indicated by a bifid uvula, partial separation of muscle with intact mucosa, or a palpable notch at the posterior of the palate.

**TREATMENT.** A complete program of habilitation for the child with a cleft lip or palate may require years of special treatment by a team consisting of a pediatrician, plastic surgeon, otolaryngologist, oral and maxillofacial surgeon, pediatric dentist, prosthodontist, orthodontist, speech therapist, geneticist, medical social worker, psychologist, and public health nurse. The child’s physician should be responsible for seeking the coordinated use of specialists and for parental counseling and guidance.

The immediate problem in an infant born with a cleft lip or palate is feeding. Although some advocate the construction of a plastic obturator to assist in feedings, most believe that with the use of soft artificial nipples with large openings, a squeezable bottle, and proper instruction, feeding of infants with clefts can be achieved with relative ease and effectiveness.

Surgical closure of a cleft lip is usually performed by 3 mo of age, when the infant has shown satisfactory weight gain and is free of any oral, respiratory, or systemic infection. Modification of the Millard rotation-advancement technique is the most commonly used technique; a staggered suture line minimizes notching of the lip from retraction of scar tissue. The initial repair may be revised at 4 or 5 yr of age. Corrective surgery on the nose may be delayed until adolescence. Nasal surgery can also be performed at the time of the lip repair. Cosmetic results depend on the extent of the original deformity, healing potential of the individual, absence of infection, and the skill of the surgeon.

Because clefts of the palate vary considerably in size, shape, and degree of deformity, the timing of surgical correction should be individualized. Criteria such as width of the cleft, adequacy of the existing palatal segments, morphology of the surrounding areas (width of the oropharynx), and neuromuscular function of the soft palate and pharyngeal wall affect the decision. The goals of surgery are the union of the cleft segments, intelligible and pleasant speech, reduction of nasal regurgitation, and avoidance of injury to the growing maxilla.

In an otherwise healthy child, closure of the palate is usually done before 1 yr of age to enhance normal speech development. When surgical correction is delayed beyond the 3rd yr, a con-
Syndromes with Oral incompetence (VPI) can also occur in children with an inherent condition, such as a submucous cleft palate or similar anomaly. This may explain why a speech defect does not become apparent in some children. One mechanism is that neuromuscular function is adequate, compensation in palatopharyngeal movement may take place and the speech defect may improve, although speech therapy is necessary. In other cases, the uvula, soft palate or pharynx may not be in the correct position and the speech defect may not resolve. Nasal resonance (nasality) is a characteristic of the child with a cleft palate that can also be produced by neuromuscular abnormalities such as frontal bossing, mandibular prognathism, and a broad nasal base. Tooth eruption can be delayed. The primary teeth can be abnormally retained while the permanent teeth remain unerupted. Supernumerary teeth are common, especially in the premolar area. Although the erupted teeth are usually free of hypoplasia, variations in the size and shape of the teeth are common. Restoration of the erupted primary and permanent teeth should be performed when carious lesions are present. Extensive dental rehabilitation therapy may be needed to maintain effective mastication.

Osteogenesis imperfecta is often accompanied by effects on the teeth, termed dentinogenesis imperfecta (see Chapter 304, Fig. 304.2). Depending on the severity of presentation, treatment of the dentition varies from routine preventive and restorative monitoring to covering affected posterior teeth with stainless steel crowns, to reduce abrasion from chewing. Dentinogenesis imperfecta may also occur in isolation without the bony effects.

Another syndrome, cleidocranial dysplasia, has orofacial variations such as frontal bossing, mandibular prognathism, and a broad nasal base. Tooth eruption is often delayed. The primary teeth can be abnormally retained while the permanent teeth remain unerupted. Supernumerary teeth are common, especially in the premolar area. Although the erupted teeth are usually free of hypoplasia, variations in the size and shape of the teeth are common. Restoration of the erupted primary and permanent teeth should be performed when carious lesions are present. Extensive dental rehabilitation therapy may be needed to maintain effective mastication.

Clayton's syndrome (see Chapter 308) is a heterogeneous group of conditions in which oral manifestations range from little or no involvement (the dentition is completely normal) to cases in which the teeth can be totally or partially absent or malformed. Because alveolar bone does not develop in the absence of teeth, the alveolar processes can be either totally or partially absent, and the resultant overclosure of the mandible causes the lips to protrude. Facial development is otherwise normal. Teeth, when present, can range from normal to small and conical. If aplasia of the buccal and labial mucous glands is present, dryness and irritation of the oral mucosa can occur. People with ectodermal dysplasia may need either partial or full dentures, even at a young age. The clinical signs vary, and symptoms of VPI are similar to those of a cleft palate. There may be hypernasal speech (especially noted in the articulation of pressure consonants such as p, b, d, t, h, v, f, and s); conspicuous stricture of the nares during speech; inability to whistle, gargle, blow out a candle, or inflate a balloon; loss of liquid through the nose when drinking with the head down; oral fluid; and hearing loss. Oral inspection may reveal a cleft palate or a relatively short palate with a large oropharynx; absent, grossly asymmetric, or minimal muscular activity of the soft palate and pharynx during phonation or gagging; or a submucous cleft. The latter is suggested by a bifid uvula, by a translucency membrane in the midline of the soft palate (revealing lack of continuity of muscles), by palpable notching in the posterior border of the hard palate instead of a posterior nasal spine process, or by forward or V-shaped displacement or groove in the soft palate during phonation or gagging.

Veloopharyngeal incompetence may also be demonstrated radiographically. The head should be carefully positioned to obtain a true lateral view; one film is obtained with the patient at rest and another during continuous phonation of the vowel a as in “boom.” The soft palate contacts the posterior pharyngeal wall in normal function, whereas in velopharyngeal incompetence the contact is absent. Most accurate evaluations of VPI are accomplished by the use of nasoendoscopy. In selected cases, the palate may be retropositioned or pharyngoplasty performed using a flap of tissue from the posterior pharyngeal wall. Dental speech appliances have also been used successfully. The type of surgery used is best tailored to the findings on nasoendoscopy.

Many syndromes have distinct or accompanying facial, oral, and dental manifestations (Apert syndrome, Chapter 592; Crouzon disease, Chapter 592; Down syndrome, Chapter 81).

Dental appliances may be needed. The primary dentition may be retained for an extended period of time. Extensive dental rehabilitation therapy may be needed to maintain effective mastication.

Ectodermal dysplasias (see Chapter 648) are a heterogeneous group of conditions in which oral manifestations range from little or no involvement (the dentition is completely normal) to cases in which the teeth can be totally or partially absent or malformed. Because alveolar bone does not develop in the absence of teeth, the alveolar processes can be either totally or partially absent, and the resultant overclosure of the mandible causes the lips to protrude. Facial development is otherwise normal. Teeth, when present, can range from normal to small and conical. If aplasia of the buccal and labial mucous glands is present, dryness and irritation of the oral mucosa can occur. People with ectodermal dysplasia may need either partial or full dentures, even at a young age. The clinical signs vary, and symptoms of VPI are similar to those of a cleft palate. There may be hypernasal speech (especially noted in the articulation of pressure consonants such as p, b, d, t, h, v, f, and s); conspicuous stricture of the nares during speech; inability to whistle, gargle, blow out a candle, or inflate a balloon; loss of liquid through the nose when drinking with the head down; oral fluid; and hearing loss. Oral inspection may reveal a cleft palate or a relatively short palate with a large oropharynx; absent, grossly asymmetric, or minimal muscular activity of the soft palate and pharynx during phonation or gagging; or a submucous cleft. The latter is suggested by a bifid uvula, by a translucency membrane in the midline of the soft palate (revealing lack of continuity of muscles), by palpable notching in the posterior border of the hard palate instead of a posterior nasal spine process, or by forward or V-shaped displacement or groove in the soft palate during phonation or gagging.

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Ectodermal dysplasias (see Chapter 648) are a heterogeneous group of conditions in which oral manifestations range from little or no involvement (the dentition is completely normal) to cases in which the teeth can be totally or partially absent or malformed. Because alveolar bone does not develop in the absence of teeth, the alveolar processes can be either totally or partially absent, and the resultant overclosure of the mandible causes the lips to protrude. Facial development is otherwise normal. Teeth, when present, can range from normal to small and conical. If aplasia of the buccal and labial mucous glands is present, dryness and irritation of the oral mucosa can occur. People with ectodermal dysplasia may need either partial or full dentures, even at a young age. The clinical signs vary, and symptoms of VPI are similar to those of a cleft palate. There may be hypernasal speech (especially noted in the articulation of pressure consonants such as p, b, d, t, h, v, f, and s); conspicuous stricture of the nares during speech; inability to whistle, gargle, blow out a candle, or inflate a balloon; loss of liquid through the nose when drinking with the head down; oral fluid; and hearing loss. Oral inspection may reveal a cleft palate or a relatively short palate with a large oropharynx; absent, grossly asymmetric, or minimal muscular activity of the soft palate and pharynx during phonation or gagging; or a submucous cleft. The latter is suggested by a bifid uvula, by a translucency membrane in the midline of the soft palate (revealing lack of continuity of muscles), by palpable notching in the posterior border of the hard palate instead of a posterior nasal spine process, or by forward or V-shaped displacement or groove in the soft palate during phonation or gagging.

Veloopharyngeal incompetence may also be demonstrated radiographically. The head should be carefully positioned to obtain a true lateral view; one film is obtained with the patient at rest and another during continuous phonation of the vowel a as in “boom.” The soft palate contacts the posterior pharyngeal wall in normal function, whereas in velopharyngeal incompetence the contact is absent. Most accurate evaluations of VPI are accomplished by the use of nasoendoscopy. In selected cases, the palate may be retropositioned or pharyngoplasty performed using a flap of tissue from the posterior pharyngeal wall. Dental speech appliances have also been used successfully. The type of surgery used is best tailored to the findings on nasoendoscopy.
Dental caries of the primary dentition usually begins in the pits and fissures. Small lesions may be angles of the mouth and the ears, deformed pinnae, atypical hair growth extending toward the cheeks, receding chin, and large mouth. Facial clefts, abnormalities of the ears, and deafness are common. The disorder is autosomal dominant, often with incomplete penetrance. The mandible is usually hypoplastic; the ramus may be deficient, and the coronoid and condylar processes are flat or even aplastic. The palatal vault may be either high or cleft. Infrequently, unilateral or bilateral macrostomia, or failure of embryonic fusion of the maxillary and mandibular processes, may occur. Dental malocclusions are frequent. The teeth may be widely separated, hypoplastic, or displaced or have an open bite. Orthodontic and routine dental treatments are indicated.

**Hemifacial microsomia** is usually characterized by unilateral hypoplasia of the mandible and can be associated with partial paralysis of the facial nerve, macrostomia, blind fistulas between the angles of the mouth and the ears, and deformed external ears. Severe facial asymmetry and malocclusion can develop because of the absence or hypoplasia of the mandibular condyle on the affected side. Congenital condylar deformity tends to increase with age. Early craniofacial surgery may be indicated to minimize the deformity. This disorder can be associated with ocular and vertebral anomalies (oculo-auriculo-vertebral spectrum, including Goldenhar syndrome); therefore, radiographs of the vertebrae and ribs should be considered to determine the extent of skeletal involvement.

**Etiology.** The development of dental caries depends on interrelationships between the tooth surface, dietary carbohydrates, and specific oral bacteria. Organic acids produced by bacterial fermentation of dietary carbohydrates reduce the pH of dental plaque adjacent to the tooth to a point at which demineralization occurs. The initial carious lesion appears as an opaque white spot on the enamel; and with progressive loss of tooth mineral, cavitation occurs.

A group of microorganisms, *mutans streptococci*, are associated with the development of dental caries. These bacteria have the ability to adhere to enamel, produce abundant acid, and survive at low pH. Once the enamel surface cavitates, other oral bacteria (lactobacilli) colonize the tooth, produce acid, and foster further tooth demineralization. Demineralization from bacterial acid production is determined by the frequency of carbohydrate consumption and by the type of carbohydrate. Sucrose is the most cariogenic sugar because one of its by-products during bacterial metabolism is glucan, a polymer that enables bacteria to adhere more readily to tooth structures. The cariogenic potential of a nursing bottle of a sweetened beverage that is continuously consumed throughout the night or at nap times is much greater than that of the same volume of drink consumed at a single meal. Similarly, sticky candies retained orally for long periods (sucrose in sticky candies) is more cariogenic than the sugar in food products retained for short times.

**Epidemiology.** The incidence of dental caries has decreased in developed countries in the past 30 yr but remains highly prevalent among low-income children and children from developing countries. The decrease is due to advances in prevention, particularly in the use of fluorides. More than half of the children in the United States have dental caries, with most of those having caries primarily in the pits and fissures of the occlusal (biting) surfaces of the molar teeth.

**Clinical Manifestations.** Dental caries of the primary dentition usually begins in the pits and fissures. Small lesions may be
difficult to diagnose by visual inspection, but larger lesions present as cavitations of the occlusal surface. The 2nd most frequent location of caries is approximal sites (contact surfaces between the teeth), which in many cases can only be detected by intraoral radiographs. Caries lesions of the exposed smooth (buccal and lingual) surfaces are generally found only in children with rampant caries (Fig. 309-1).

Rampant caries in infants and toddlers, referred to as early childhood caries (ECC), nursing bottle caries, or baby bottle tooth decay, is ascribed to inappropriate bottle-feeding. Although the combination of a child infected with cariogenic bacteria and the frequent ingestion of sugar, either in the bottle or in solid foods, is critical, other factors such as enamel hypoplasia of primary teeth because of nutritional deficiencies during pregnancy or because of premature birth may have a role. Reports have also associated “at will” breast-feeding in older infants with early childhood caries. Interestingly, cow’s milk is less associated with caries, possibly due to the higher calcium and phosphorus contents of cow’s milk relative to breast milk.

Early childhood caries are common, with a reported prevalence of 30–50% in children from low socioeconomic backgrounds and as high as 70% in some Native American groups. It may occur as early as 12 mo of age, long before children visit a dentist. Pediatricians have the responsibility to both examine the child’s teeth for caries and to establish a “dental home” (refer to a dentist) before a child at risk for ECC is 1 yr of age. Risk factors include high-frequency sugar consumption (prolonged and frequent drinking from bottle or sippy cup, frequent eating of sugar-containing snacks), children of low socioeconomic status, immigrant children, parents or siblings with high caries rates, and evidence of defects on the teeth.

Children who develop caries at a young age are known to be at high risk for developing further caries as they get older. Therefore, the appropriate prevention of early childhood caries can result in the elimination of major dental problems in toddlers and less decay in later childhood.

COMPLICATIONS. If left untreated, dental caries usually destroy most of the tooth and invade the dental pulp (Fig. 309-2), leading to an inflammation of the pulp (pulpitis) and significant pain. Pulpitis can progress to necrosis, with bacterial invasion of the alveolar bone causing a dental abscess (Fig. 309-3). Infection of a primary tooth may disrupt normal development of the successor permanent tooth. In a small percentage of cases, this process may lead to sepsis and facial space infection.
The periodontium includes the gingiva, alveolar bone, cementum, and periodontal ligament (see Fig. 309-2).

**GINGIVITIS.** Poor oral hygiene results in the accumulation of dental plaque at the tooth-gingival interface that activates an inflammatory response, expressed as localized or generalized redness and swelling of the gingiva. More than half of American schoolchildren experience gingivitis. In severe cases, the gingiva spontaneously bleeds and there is oral malodor. Treatment is proper oral hygiene (careful toothbrushing and flossing); complete resolution can be expected. Fluctuations in hormonal levels during the onset of puberty can increase inflammatory response to plaque. Gingivitis in healthy children is unlikely to progress to periodontitis (inflammation of the periodontal ligament resulting in loss of alveolar bone).

**AGGRESSIVE PERIODONTITIS IN CHILDREN (PREPUBERTAL PERIODONTITIS).** Periodontitis in children before puberty is a rare disease that often begins between the time of eruption of the primary teeth and the age of 4 or 5 yr. The disease occurs in localized and generalized forms. There is rapid bone loss, often leading to premature loss of primary teeth. It is often associated with systemic problems, including neutropenia, leukocyte adhesion or migration defects, hypophosphatasia, Papillon-Lefèvre syndrome, leukemia, and histiocytosis X. In many cases, however, there is no apparent underlying medical problem. Nonetheless, diagnostic work-ups are necessary to rule out underlying systemic disease.

Treatment includes aggressive professional teeth cleaning, strategic extraction of affected teeth, and antibiotic therapy. There are few reports of long-term successful treatment to reverse bone loss surrounding primary teeth.

**AGGRESSIVE PERIODONTITIS IN ADOLESCENTS (LOCALIZED JUVENILE PERIODONTITIS).** Aggressive periodontitis in adolescents is characterized by rapid alveolar bone loss, especially around the permanent incisors and 1st molars. Overall prevalence in the United States is <1%, but the prevalence among African Americans is reportedly 2.5%. This form of periodontitis is associated with a strain of Actinobacillus bacteria. In addition, the neutrophils of patients with aggressive periodontitis may have chemotactic or phagocytic defects. If left untreated, affected teeth lose their attachment and may exfoliate. Treatment varies with the degree of involvement. Patients diagnosed at the onset of the disease are usually managed by surgical or nonsurgical debridement in conjunction with antibiotic therapy. Prognosis depends on the degree of initial involvement and compliance with therapy.

**TEETHING.** Teething can lead to intermittent localized discomfort in the area of erupting primary teeth, irritability, low-grade fevers, and excessive salivation; however, many children have no apparent difficulties. Treatment of symptoms includes oral analgesics and ice rings for the child to “gum.” Similar manifestations can also arise when the 1st permanent molars erupt at about age 6 yr.

**CYCLOSPORINE- OR PHENYTOIN-INDUCED GINGIVAL OVERGROWTH.** The use of cyclosporine to suppress organ rejection or phenytoin for anticonvulsant therapy, and in some cases, calcium channel blockers, is associated with generalized enlargement of the gingiva. Phenytoin and its metabolites have a direct stimulatory action on gingival fibroblasts, resulting in accelerated synthesis of collagen. Phenytoin induces less gingival hyperplasia in patients who maintain meticulous oral hygiene.
Gingival hyperplasia occurs in 10–30% of patients treated with phenytoin. Severe manifestations may include (1) gross enlargement of the gingiva, sometimes covering the teeth; (2) edema and erythema of the gingiva; (3) secondary infection, resulting in abscess formation; (4) migration of teeth; and (5) inhibition of exfoliation of primary teeth and subsequent impaction of permanent teeth. Treatment should be directed toward prevention and, if possible, discontinuation of cyclosporine or phenytoin. Patients undergoing long-term treatment with these drugs should receive frequent dental examinations and oral hygiene care. Severe forms of gingival overgrowth are treated by gingivectomy, but the lesion recurs if drug use is continued.

**ACUTE PERICORONITIS.** Acute inflammation of the flap of gingiva that partially covers the crown of an incompletely erupted tooth is common in mandibular permanent molars. Accumulation of debris and bacteria between the gingival flap and tooth precipitates the inflammatory response. A variant of this condition is a gingival abscess due to entrapment of bacteria because of orthodontic bands or crowns. Trismus and severe pain may be associated with the inflammation. Untreated cases may result in facial space infections and facial cellulitis.

Treatment includes local debridement and irrigation, warm saline rinses, and antibiotic therapy. When the acute phase has subsided, extraction of the tooth or resection of the gingival flap is advocated. Early recognition of the partial impaction of mandibular 3rd molars and their subsequent extraction prevents recurrence. Early recognition of the partial impaction of mandibular 3rd molars and their subsequent extraction prevents recurrence. Early recognition of the partial impaction of mandibular 3rd molars and their subsequent extraction prevents recurrence. Early recognition of the partial impaction of mandibular 3rd molars and their subsequent extraction prevents recurrence.

**NECROTIZING PERIODONTAL DISEASE (ACUTE NECROTIZING ULCERATIVE GINGIVITIS).** Necrotizing periodontal disease, in the past sometimes referred to as “trench mouth,” is a distinct periodontal disease associated with oral spirochetes and fusobacteria. It is not clear, however, whether bacteria initiate the disease or are secondary. It rarely develops in healthy children in developed countries, with a prevalence in the United States of <1%, but is seen more frequently in children and adolescents from developing areas of Africa, Asia, and South America. In certain African countries, where affected children usually have protein malnutrition, the lesion may extend into adjacent tissues, causing necrosis of facial structures (cancrum oris, or noma).

Clinical manifestations of necrotizing periodontal disease include (1) necrosis and ulceration of gingiva between the teeth, (2) an adherent grayish pseudomembrane over the affected gingiva, (3) oral malodor, (4) cervical lymphadenopathy, (5) malaise, and (6) fever. The condition may be mistaken for acute herpetic gingivostomatitis. Dark-field microscopy of debris obtained from necrotizing lesions will demonstrate dense spirochete populations.

Treatment of necrotizing periodontal disease is divided into an acute management with local debridement, oxygenating agents (direct application of 10% carbamide peroxide in anhydrous glycerol qid), and analgesics. Dramatic resolution usually occurs within 48 hr. If a patient is febrile, antibiotics (penicillin or metronidazole) may be an important adjunctive therapy. A 2nd phase of treatment may be necessary if the acute phase of the disease has caused irreversible morphologic damage to the periodontium. The disease is not contagious.

**INJURIES TO TEETH.** Approximately 10% of children between 18 mo and 18 yr of age will sustain significant tooth trauma. There appear to be three age periods of greatest predilection: (1) toddlers (1–3 yr), usually due to falls or child abuse; (2) school-aged (7–10 yr), usually from bicycle and playground accidents; and (3) adolescents (16–18 yr), often the result of fights, athletic injuries, and automobile accidents. Injuries to teeth are more frequent among children with protruding front teeth. Children with craniofacial abnormalities or neuromuscular deficits are also at increased risk for dental injury. Injuries to teeth may involve the hard dental tissues, the dental pulp (nerve), and injuries to the periodontal structure (surrounding bone and attachment apparatus) (Fig. 311-1 and Table 311-1).

Fractures of teeth may be uncomplicated (confined to the hard dental tissues) or complicated (involving the pulp). Exposure of the pulp will result in its bacterial contamination, which can lead to infection and pulp necrosis. Such pulp exposure complicates therapy and may lower the likelihood of a favorable outcome.

The teeth most often affected are the maxillary incisors. Uncomplicated crown fractures are treated by covering exposed dentin and by placing an aesthetic restoration. Complicated crown fractures usually require **endodontic therapy** (root canal). Crown-root fractures and root fractures usually require extensive dental therapy. Such injuries in the primary dentition can interfere with normal development of the permanent dentition, and, therefore, significant injuries of the primary incisor teeth are usually managed by extraction.

Traumatic oral injuries should be referred to a dentist as soon as possible. Even when the teeth appear intact, a dentist should promptly evaluate the patient. Baseline data (radiographs, mobility patterns, responses to specific stimuli) enable the dentist to assess the likelihood of future complications.

**INJURIES TO PERIODONTAL STRUCTURES.** Trauma to teeth with associated injury to periodontal structures that hold the teeth usually presents as mobile or displaced teeth. Such injuries are more frequent in the primary than in the permanent dentition. Categories of trauma to the periodontium include (1) concussion, (2) subluxation, (3) intrusive luxation, (4) extrusive luxation, and (5) avulsion.

**Concussion.** Injuries that produce minor damage to the periodontal ligament are termed concussions. Teeth sustaining such injuries are not mobile or displaced but react markedly to percussion (gentle hitting of the tooth with an instrument). This type of injury usually requires no therapy and resolves without com-
Avulsion. If avulsed permanent teeth are replanted within 20 min after injury, good success may be achieved; if the delay exceeds 2 hr, however, failure (root resorption, ankylosis) is frequent. The likelihood that normal reattachment will follow replantation of the tooth is related to the viability of the periodontal ligament. Parents confronted with this emergency situation can be instructed to do the following:

1. Find the tooth.
2. Rinse the tooth. (Do not scrub the tooth. Do not touch the root. After plugging the sink drain, hold the tooth by the crown and rinse it under running tap water.)
3. Insert the tooth into the socket. (Gently place it back into its normal position. Do not be concerned if the tooth extrudes slightly. If the parent or child is too apprehensive for replantation, the tooth should be placed in cold cow’s milk or other cold isotonic solution.)
4. Go directly to the dentist. (In transit, the child should hold the tooth in its socket with a finger. The parent should buckle a seatbelt around the child and drive safely.)

After the tooth is replanted, it must be immobilized to facilitate reattachment; endodontic therapy is always required. The initial signs of complications associated with replantation may appear as early as 1 wk post trauma or as late as several years later. Close dental follow-up is indicated for at least 1 yr.

PREVENTION. To minimize the likelihood of dental injuries:

1. Every child or adolescent who engages in contact sports should wear a mouth guard, which may be constructed by a dentist or purchased at any athletic goods store.
2. Helmets with face guards should be worn by children or adolescents with neuromuscular problems or seizure disorders to protect the head and face during falls.
3. Helmets should also be used during biking, roller blading, and skateboarding.
4. All children or adolescents with protruding incisors should be evaluated by a pediatric dentist or orthodontist.

ADDITIONAL CONSIDERATIONS. Children who experience dental trauma may also have sustained head or neck trauma, and, therefore, neurologic assessment is warranted. Tetanus prophylaxis should be considered with any injury that disrupts the integrity of the oral tissues. The possibility of child abuse should always be considered.
Oropharyngeal candidiasis (See Chapter 231.1). Oropharyngeal infection with Candida albicans (thrush, moniliasis) is common in neonates from contact with the organism in the birth canal or breast. The lesions of oropharyngeal candidiasis (OPC) appear as white plaques covering all or part of the oropharyngeal mucosa. These plaques are removable from the underlying surface, which is characteristically inflamed with pinpoint hemorrhages. The diagnosis is confirmed by direct microscopic examination on potassium hydroxide smears and culture of scrapings from lesions. OPC is usually self-limited in the healthy newborn, but treatment with nystatin will hasten recovery and reduce the risk of the infection spreading to other infants. Persistent infections should be treated with fluconazole therapy. OPC is also a major problem during myelosuppressive therapy. Systemic candidiasis (SC), a major cause of morbidity and mortality during myelosuppressive therapy, develops almost exclusively in patients who have had prior oropharyngeal, esophageal, or intestinal candidiasis. This observation implies that prevention of OPC should reduce the incidence of SC. The use of a multigent regimen, 0.2% chlorhexidine solution and fluconazole, may be effective in preventing OPC, SC, or candidal esophagitis.

Aphthous ulcers. The aphthous ulcer (canker sore) is a distinct oral lesion, prone to recurrence. The differential diagnosis is noted in Table 312-1. Aphthous ulcers are reported to develop in 20% of the population. Their etiology is unclear, but infectious agents such as Helicobacter pylori, herpes simplex virus, and Behçet's syndrome resembles aphthous lesions; associated with genital ulcers, geographic tongue (migratory glossitis) is a benign and asymptomatic lesion and is characterized by one or more smooth, bright red patches, often showing a yellow, gray,
or white membranous margin on the dorsum of an otherwise normally roughened tongue. The condition has no known cause and no treatment is indicated (see Chapter 663).

**Fissured Tongue.** The fissured tongue (scrotal tongue) is a malformation manifested clinically by numerous small furrows or grooves on the dorsal surface (see Chapter 663). If the tongue is painful, brushing the tongue or irrigating with water can reduce the bacteria in the fissures.

**Chapter 313 Diseases of the Salivary Glands and Jaws**

With the exception of mumps (see Chapter 245), disease of the salivary glands is rare in children. Bilateral enlargement of the submaxillary glands can occur in AIDS, cystic fibrosis, Epstein-Barr virus infection, and malnutrition and, transiently, during acute asthmatic attacks. Chronic vomiting can be accompanied by enlargement of the parotid glands. Benign salivary gland hypertrophy has been associated with endocrinopathies: thyroid disease, diabetes, and disorders of the pituitary-adrenal axis.

**Recurrent Parotitis.** Recurrent idiopathic swelling of the parotid gland can occur in otherwise healthy children. The swelling is usually unilateral, but both glands can be involved simultaneously or alternately. There is little pain; the swelling is usually unilateral, but both glands can be involved simultaneously or alternately. The incidence appears to be higher in the spring.

**Suppurative Parotitis.** This is usually due to *Staphylococcus aureus* and can be primary or a complication of parotitis from another cause. It is usually unilateral and may be accompanied by fever. The gland becomes swollen, tender, and painful. Suppurative parotitis responds to appropriate antibacterial therapy based on culture obtained from the Stensen duct or by surgical drainage, which is infrequently required.

**Ranula.** This is a cyst associated with a major salivary gland in the sublingual area. A ranula is a large, soft, mucous-containing swelling in the floor of the mouth. It occurs at any age, including infancy. The cyst should be excised, and the severed duct should be exteriorized.

**Mucocoele.** This is a salivary gland lesion caused by a blockage of a salivary gland duct. It is most common on the lower lip and has the appearance of a fluid-filled vesicle or a fluctuant nodule with the overlying mucosa normal in color. Treatment is surgical excision, with removal of the involved accessory salivary gland.

**Congenital lip pits.** These are caused by fistulous tracts that lead to embedded mucous glands in the lower lip. They leak saliva, especially with salivary stimulation. Lip pits can be isolated anomalies, or they can be found in patients with cleft lip or palate. Treatment is surgical excision of the glandular tissue.

**Eruption cyst.** This is a smooth, painless swelling over the erupting tooth. If bleeding occurs in the cyst space, it may appear blue or blue-black. In most cases, no treatment is indicated and the cyst resolves with the full eruption of the tooth.

**Xerostomia.** Also known as dry mouth, xerostomia may be associated with fever, dehydration, anticholinergic drugs, chronic graft versus host disease, Mikulicz disease (leukemia infiltrates), Sjogren syndrome, or tumoricidal doses of radiation when the salivary glands are within the field. Long-term xerostomia is a high-risk factor for dental caries.

**Salivary gland tumors.** See Chapter 500.

**Histiocytosis X (see Chapter 507).** The etiology and pathogenesis of histiocytosis remains obscure. In the severe form, there are oral lesions with pain, swelling, gingival necrosis, and destruction of alveolar bone, resulting in premature exfoliation of teeth. Treatment varies according to the extent of the disease, with surgical curettage or radiation therapy being used to treat the focal disease. Multagent chemotherapy and bone marrow transplantation may be needed to treat disseminated multiorgan disease.

**Tumors of the jaw.** Ossifying fibroma is a common benign tumor of the jaw. It is often asymptomatic, being discovered on routine radiographic examinations. Treatment is enucleation or curettage. Central giant cell granuloma is another common lesion thought to be reactive rather than neoplastic. Although usually asymptomatic, it can be expansile, with or without divergence of teeth. Treatment is complete curettage or surgical excision. Dentigerous cysts are common lesions associated with the crown of an impacted or unerupted tooth. Although usually asymptomatic, they can become large and destructive. Treatment is surgical removal.

The malignant primary tumors of the jaw in children include Burkitt lymphoma, osteogenic sarcoma, lymphosarcoma, ameloblastoma, and, more rarely, fibrosarcoma.

**Chapter 314 Diagnostic Radiology in Dental Assessment**

The panoramic radiograph provides a single tomographic image of the upper and lower jaw, including all the teeth and supporting structures. The x-ray tube rotates about the patient’s head with reciprocal movement of the film or image receptor during the exposure. The panoramic image shows the mandibular bodies, rami, and condyles; maxillary sinuses; and a majority of the facial buttresses. Such images are used to show abnormalities of tooth number, development and eruption pattern, cystic and neoplastic lesions, bone infections, fracture, as well as dental caries and periodontal disease (Fig. 314-1).

Figure 314-1. A panoramic radiograph of a 10 yr old child showing extensive dental caries of the 1st permanent molars (arrows), as well as normal structures: erupting 1st permanent molar, unerupted 2nd molar, and unerupted 3rd molar; erupted incisors (EI), unerupted permolars (UP), and erupted primary canines (pc).
Cephalometric radiographs are posteroanterior and lateral skull films that are taken using a cephalostat (head positioner) and employ techniques that clearly demonstrate the facial skeleton and soft facial tissues. Similar protocols for positioning children are used throughout the world. From these images, cranial and facial points and planes can be determined and compared with standards derived from thousands of images. A child’s facial growth can be assessed serially when cephalometric radiographs are taken sequentially. Relationships among the maxilla, mandible, cranial base, and facial skeleton can be determined in a quantitative manner. Additionally, the alignment of the teeth and the relation of the teeth to the supporting bone can be serially measured.

Intraoral dental radiographs are highly detailed, direct exposure films that demonstrate sections of the child’s teeth and supporting bone structures. The film or image receptor is placed lingual to the teeth, and the x-ray beam is directed through the teeth and supporting structures. The resulting images are used to detect dental caries, loss of alveolar bone (periodontal disease), abscesses at the roots of the teeth, and trauma to the teeth and supporting structures. The resulting images are used to detect dental caries, loss of alveolar bone (periodontal disease), abscesses at the roots of the teeth, and trauma to the teeth and alveolar bone and to demonstrate the developmental status of permanent teeth within the bone.


Section 3 — The Esophagus

Chapter 315  ■ Embryology, Anatomy, and Function of the Esophagus  ■ Sunny Zaheed Hussain

The esophagus is a hollow muscular tube, separated from the pharynx above and the stomach below by two tonically closed sphincters. Its primary function is to convey ingested material from the mouth to the stomach. Largely lacking digestive glands and enzymes, and exposed only briefly to nutrients, it has no active role in digestion.

EMBRYOLOGY. The esophagus develops from the postpharyngeal foregut and can be distinguished from the stomach in the 4 wk old embryo. At the same time, the trachea begins to bud just anterior to the developing esophagus; the resulting laryngotracheal groove extends and becomes the lung. Disturbance of this stage may result in congenital anomalies such as tracheoesophageal fistula. The length of the esophagus is 8–10 cm at birth, and doubles in the 1st 2–3 yr of life, reaching ~23 cm in an adult. The abdominal portion of the esophagus is as large as the stomach in an 8 wk old fetus but gradually shortens to a few mm at birth, attaining a final length of ~3 cm by a few years of age. This intra-abdominal location of both the distal esophagus and the lower esophageal sphincter (LES) is an important antireflux mechanism, because increases in intra-abdominal pressure are also transmitted to the sphincter, augmenting its defense. Swallowing can be seen in utero as early as 16–20 wk of gestation, helping to circulate the amniotic fluid; polyhydramnios is, thus, a hallmark of lack of normal swallowing or of esophageal or upper gastrointestinal tract obstruction. Sucking and swallowing are not fully coordinated before 34 wk of gestation, a contributing factor for feeding difficulties in premature infants.

ANATOMY. The luminal aspect of the esophagus is covered by thick, protective, nonkeratinized stratified squamous epithelium, which abruptly changes to simple columnar epithelium at the stomach’s upper margin at the gastroesophageal junction (GEJ). This squamous epithelium is relatively resistant to damage by gastric secretions (in contrast to the ciliated columnar epithelium of the respiratory tract), but chronic irritation by gastric contents may result in morphometric changes and subsequent metaplasia of the cells lining the lower esophagus from squamous to columnar. Deeper layers of the esophageal wall are composed successively of lamina propria, muscularis mucosae, submucosa, and the two layers of muscularis propria (circular surrounded by longitudinal). The two delimiting sphincters of the esophagus, the upper esophageal sphincter (UES) at the cricopharyngeus muscle and the LES at the GEJ, constrict the esophageal lumen at its proximal and distal boundaries. The muscularis propria of the upper third of the esophagus is predominantly striated, and that of the lower two thirds is smooth muscle. Clinical conditions involving striated muscle (cricopharyngeal dysfunction, cerebral palsy) affect the upper esophagus, whereas those involving smooth muscle (achalasia, reflux esophagitis) affect the lower