

chapter 2

PATHOLOGY OF THE TEETH

Fig. 2.1

When fluoride in the drinking water exceeds 1 ppm, changes can be seen in the structure of developing teeth secondary to defective or deficient enamel formation. In addition, clinically evident dental fluorosis has been detected in children living in areas using water with less than 1 ppm of fluoride. It has become evident that total fluoride intake comes from a combination of fluoride supplements, food sources, naturally released components, and man-made commercial emissions into the environment. In many instances, the actual fluoride intake greatly exceeds the recommended level and results in detectable dental changes. Significant ingestion of toothpaste by children younger than age 4 years has been correlated directly with fluorosis, even in nonfluoridated communities.

The dental changes vary significantly even at similar fluoride levels because of individual variations in fluoride intake. Changes from white opaque teeth, with or without brown-stained areas, to teeth that are extremely pitted and corroded in appearance are seen. The changes are usually generalized to all of the teeth; in mild cases, however, not all of the tooth surface may be involved. Although the teeth may be severely pitted, they are still resistant to caries. Cosmetic therapies include vital bleaching, veneers, or crowns.

LOCALIZED ENVIRONMENTAL ENAMEL HYPOPLASIA (TURNER'S TOOTH)

Figs. 2.2 and 2.3

Localized infection or trauma to a deciduous tooth can affect the enamel formation of the underlying permanent tooth. It may involve either a disturbance in the matrix formation or calcification, depending on when the damage occurred. These teeth may exhibit only a slight area of discoloration, areas of pitting, or a totally irregular hypoplastic crown. The degree of malformation varies according to the severity of the injury and the stage of development of the permanent tooth. Coronal enamel maturation continues until eruption of the tooth; therefore, traumatic enamel alterations can arise long after cessation of ameloblastic activity. Coronal or radicular di-laceration also may occur.

The most commonly involved teeth are the maxillary central incisors secondary to physical trauma to the overlying deciduous teeth. Traumatic intrusion of the deciduous tooth is correlated with the highest prevalence of damage to the underlying permanent tooth and usually affects the facial surface of the permanent tooth due to the anatomic relationship between the deciduous apices and the adjacent crown. Similar coronal damage may occur in bicuspid secondary to periapical inflammatory disease of the overlying deciduous molars. These damaged permanent teeth are referred to as "Turner's teeth," and this type of environmental dental malformation is called "Turner's hypoplasia."

► **Figure 2.1**
DENTAL FLUOROSIS

Dentition exhibiting diffuse white and opaque enamel, with demonstrated areas of brown discoloration, in a patient with chronic ingestion of excess fluoride.



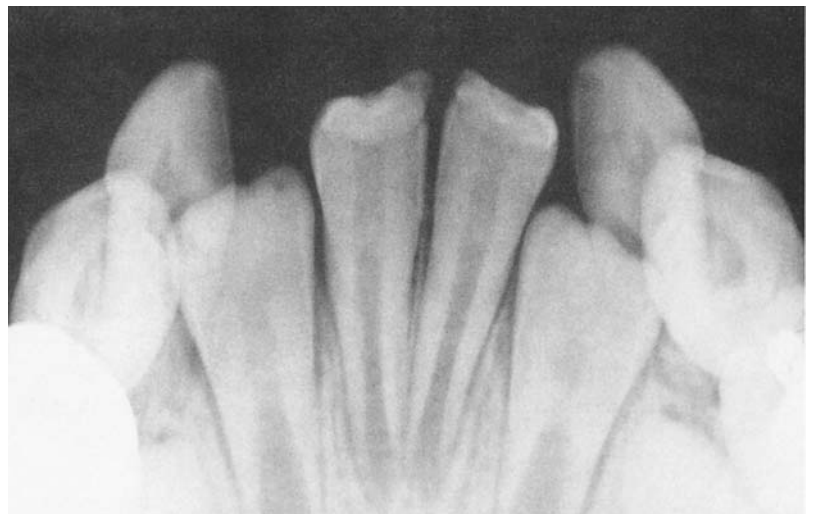
► **Figure 2.2**
LOCALIZED ENVIRONMENTAL ENAMEL HYPOPLASIA

Hypoplasia of the erupting permanent mandibular central incisors as a result of previous trauma to the deciduous incisors.



► **Figure 2.3**
LOCALIZED ENVIRONMENTAL ENAMEL HYPOPLASIA

Radiograph of incisors depicted in Figure 2.2. Note the developmental abnormality of the incisal enamel.



Figs. 2.4, 2.5, and 2.6

Certain systemic problems can affect the sensitive ameloblasts and cause enamel hypoplasia more widespread than that seen in Turner's hypoplasia. Affected teeth may reveal pits or grooves as well as partial or total lack of enamel. In deciduous teeth, the most common pattern is a horizontal groove positioned at the level of the crown that was being developed at the time of injury. In the permanent dentition, one of the frequent patterns presents as areas of opacity, horizontal rows of pits, or linear defects present on the anterior teeth and the first molars. These teeth are formed during the first year of life; if the sensitive ameloblasts are damaged, hypoplasia or hypomaturation will be present in the enamel that was being formed at that time. The damage is bilaterally symmetrical and correlates well with the developmental pattern of the involved teeth. Less commonly, similar alterations of enamel may be present in the bicuspid and second molars if the inciting event occurs around the age of 3 years. A large number of factors are associated strongly with subsequent environmental enamel alterations. Before birth, a maternal history of smoking, higher prepregnancy weight, and lack of appropriate prenatal care are associated with an increased prevalence. Prematurity, low-birth weight, and prolonged intubation of the infant also are implicated. After birth, infections (especially the exanthematous diseases), malnutrition, congenital cardiac and renal disorders, hypocalcemia, hyperbilirubinemia, and significant exposure to tetracycline, antineoplastic therapy, or excessive fluoride can lead to enamel hypoplasia. Treatment of the altered teeth consists of repair with aesthetic resins, until the teeth fully erupt, and then evaluation to determine whether further restoration is required for cosmetic reasons and function.

- **Figure 2.4**
ENVIRONMENTAL ENAMEL HYPOPLASIA
Horizontal rows of pits on the anterior teeth. The maxillary central incisors have been restored with acrylic resin. The affected areas are symmetrical and coincide with the portion of the teeth formed at the time of ameloblastic damage.



- **Figure 2.5**
ENVIRONMENTAL ENAMEL HYPOPLASIA
Posterior teeth of the patient depicted in Figure 2.4. The bicuspid are free of hypoplasia. Although not seen well in this view, all of the first molars exhibited a horizontal row of scattered pits in the middle one-third of the crown.



- **Figure 2.6**
ENVIRONMENTAL ENAMEL HYPOPLASIA
Horizontal enamel defects occurring in the bicuspid and second molars in a patient whose damage occurred at age 3 years.



RADIATION EFFECTS ON THE TEETH

Fig. 2.7

Review of childhood cancer survivors has demonstrated that therapeutic radiation and, to a lesser extent, chemotherapy can induce developmental abnormalities of the dentition and craniofacial skeleton. The tooth germs are sensitive to such external stimuli and are easily damaged. The pattern and severity of the dental alterations are related to the child's age at the time of treatment, the form of therapy, and the dose and field of radiation. The most extensive findings are seen in patients receiving therapy between the ages of 3 and 5.5 years, the time of initial root formation for all permanent teeth except the second and third molars.

It is well documented that radiotherapy may produce both qualitative and quantitative dental abnormalities, whereas chemotherapy alone typically, but not always, creates only qualitative defects in enamel. The most frequently noted quantitative defects are white-to-cream-colored areas of enamel hypomaturation. The pattern of qualitative defects varies according to the timing of the injury. If crown calcification has not been completed, the tooth may be absent totally or be rudimentary. If the crown was formed but root development was not complete at the time of radiation, the tooth may exhibit short, pointed roots. Individual differences in the degree of developmental alterations between same-age patients receiving identical therapeutic regimens are evident.

ABFRACTION OF TEETH

Fig. 2.8

Abfraction (stress-induced cervical lesions) is the newest documented cause of noncarious cervical lesions in teeth and is added to the list that also includes abrasion, attrition, and erosion. As such, the diagnosis is being used in many situations, some of which may be inappropriate. Abfraction refers to the loss of cervical tooth structure secondary to repeated flexure of the tooth caused by occlusal stresses. It is thought that lateral occlusal forces bend the tooth with tensile stress concentrated in the cervical fulcrum area, leading to disruption of the enamel and dentin bonds. Once damaged, the area may be enlarged secondarily by abrasion, attrition, or erosion.

The typical lesion is a narrow and deep U- or wedge-shaped cervical concavity in enamel or dentin that exhibits sharp line angles and is located on the facial surface of the tooth at the fulcrum. There is a higher prevalence in mandibular teeth, most likely because of the lingual orientation of the arch. Definitive diagnosis requires not only compatible tooth defects but also demonstration of lateral occlusal forces during mastication or parafunctional movements.

Although restoration is not mandatory, most teeth benefit from appropriate interventions. Of primary importance is elimination of the abnormal occlusal forces; without such intervention, the restorative failure rate is high. Due to their ability to flex with teeth rather than debond, microfilled resins and composite glass ionomer restorative materials have been used in the restoration of these defects.

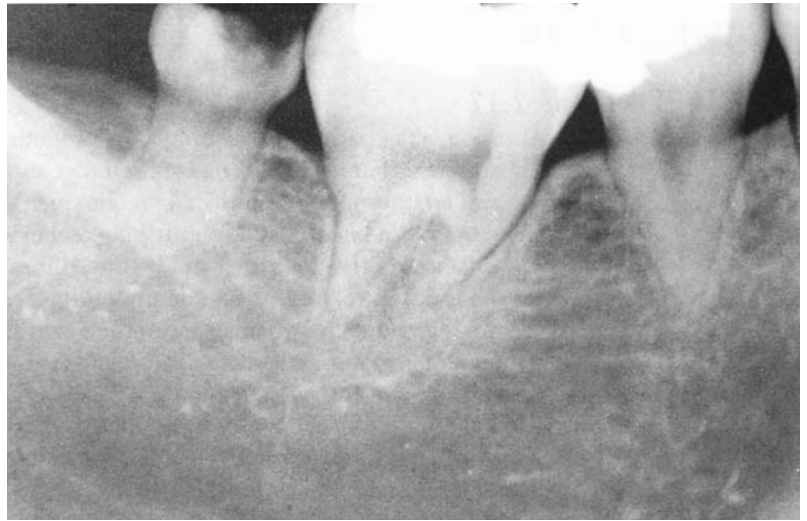
ATTRITION OF TEETH

Fig. 2.9

Attrition is the physiologic wearing away of tooth structure as a result of contact between adjacent teeth or with the teeth in the opposing arch. This wear can be found on the occlusal, incisal, or interproximal surfaces. With time, it may lead to a reduction in both the height of the teeth and the length of the arch. Both deciduous and permanent teeth may exhibit significant attrition. Severe degrees of attrition must be considered a pathologic process. At times, the distinction between attrition, abrasion, erosion, and abfraction is blurred.

Bruxism and other similar habits may accelerate the process. Increased tooth wear has been associated with increased bite force, decreased occlusal tactile sensitivity, and a decreased number of teeth. Males exhibit greater degrees of wear, and the process is more prevalent in late adulthood. Pathologic conditions such as dentinogenesis imperfecta and amelogenesis imperfecta also may result in increased attrition. Pulp exposures typically are not seen because the process proceeds slowly enough to allow for pulpal recession.

- **Figure 2.7**
RADIATION EFFECTS ON THE TEETH
Short, pointed hypoplastic root development in a patient who received therapeutic radiation to the area during the time of final root formation.



- **Figure 2.8**
ABFRACTION OF TEETH
Isolated and narrow U-shaped cervical concavities of the posterior mandibular dentition.



- **Figure 2.9**
ATTRITION OF TEETH
Dentition demonstrating diffuse and extensive loss in tooth height. Note the anterior mandibular incisors with exposed dentin and secondary dentin filling the coronal pulp chambers.



Figs. 2.10, 2.11, and 2.12

Abrasion is the pathologic wearing away of tooth structure by some abnormal mechanical force. This most commonly occurs on exposed root surfaces and usually is related to a combination of age and an improper toothbrushing technique. Most of these patients practice very good oral hygiene but use a horizontal toothbrushing technique that has resulted in abnormal tooth wear. Abrasiveness of the dentifrice and bristle stiffness do not seem to be correlated strongly.

The exposed cementum and dentin of the facial root surfaces typically exhibit notching that is oriented horizontally and adjacent to the gingiva. In about 20% of the cases, it is most severe on the quadrants opposite the dominant hand because of a tendency to brush longer or to use greater force on that side. The pulp typically is not exposed because the process proceeds slowly enough to allow for pulpal recession. The tertiary dentin filling the old pulp canal often can be visualized. On rare occasions, the crown can be undermined so severely by the deep notching that all or part of the crown fractures off.

Abrasion also may occur on the incisal or proximal surfaces. These patterns of abrasion most often are related to habits or occupations that involve biting or habitual placement of foreign objects between the teeth. Nails, toothpicks, bobby pins, and tobacco pipes are common culprits. In some cases, abrasion may be diffuse and closely resemble attrition. These examples typically arise secondary to abrasives being present constantly in the mouth. Such lesions are associated with use of tobacco products (containing silica contaminants) and industrial exposure to sand, cement, or stone dust. In one ironic case, a dental technician developed severe anterior abrasion from chronic oral exposure to porcelain powder. In these cases, pulp exposure also is rare.

► **Figure 2.10**
ABRASION OF TEETH

Dentition demonstrating significant wear of the exposed facial root surfaces of the left side of both arches. The patient was right-handed and used a horizontal brushing technique.



► **Figure 2.11**
ABRASION OF TEETH

Facial mirror view of the dentition on the left side of the patient described in Figure 2.10. Note the exposed dentin and pulp canals, which have filled with tertiary dentin.



► **Figure 2.12**
ABRASION OF TEETH

Incisal wear present on the mandibular incisors from chronic placement of pipe stem while smoking.



Figs. 2.13, 2.14, and 2.15

Erosion is the pathologic loss of tooth structure resulting from chemical action that does not include a bacterial process. The causes of erosion can be subdivided into intrinsic, extrinsic, and idiopathic. Intrinsic factors are related to prolonged exposure of the oral cavity to gastric acid. Patients who regurgitate their stomach contents can exhibit significant dental erosion that often is prominent on the lingual surfaces of the teeth. This is not rare in bulimics, individuals with a hiatal hernia, and those receiving chemotherapy. One pattern of erosion that may occur in bulimics is known as “perimolysis.” This pattern of erosion demonstrates loss of enamel on the occlusal surfaces with eventual relative elevation of any occlusal amalgam above the surface of the remaining tooth structure.

Extrinsic causes can be grouped into environmental, diet, medications, and lifestyle. Many cases related to extrinsic exposure can be explained easily by exposure to a highly acidic substance. Dental erosion is a well-recognized hazard associated with several occupations involving significant exposure to acidic chemicals. Swimmers have exhibited significant erosion secondary to low pH levels in their pools. Individuals who exhibit a large intake of highly acidic beverages or suck on citrus fruits frequently exhibit loss of tooth structure.

Many cases diagnosed as erosion are idiopathic. This category is becoming a “diagnostic dumping ground” for unexplained tooth loss that does not fit the criteria for the diagnosis of attrition or abrasion. A number of examples of cervical idiopathic erosion have been reassessed and appropriately diagnosed as abfraction. In some cases, definitive diagnosis is not possible; chemical action may be involved, but defensible proof is lacking.

Many patients with erosion exhibit a distinctive pattern of tooth loss in which the facial surfaces of the anterior teeth are affected predominantly. The area of loss is noted in the gingival one-third as a shallow, spoon-shaped depression of the enamel adjacent to the cemento-enamel junction. Although the loss usually is located in these areas, posterior teeth and occlusal surfaces may be involved, with great variation in the size and shape of the damage. Often, because of dentin being destroyed more rapidly than enamel, the defects present as concave depressions of dentin surrounded by elevated rims of enamel. In some cases, this begins as a small pit at the cusp tip that may enlarge to destroy much of the coronal anatomy.

► **Figure 2.13**
EROSION OF TEETH

Loss of facial enamel on the maxillary central incisors as a result of chronic lemon sucking.



► **Figure 2.14**
EROSION OF TEETH

Loss of occlusal enamel and dentin on the mandibular teeth of a bulimic patient. Note the amalgams slightly above the occlusal surface.



► **Figure 2.15**
EROSION OF TEETH

Idiopathic loss of enamel and dentin on the occlusal and facial surfaces of the mandibular bicuspid. Widespread loss of tooth structure was noted on either or both the occlusal and facial surfaces of most of the teeth. There were no signs of abrasion or attrition.



TETRACYCLINE STAINING OF TEETH

Fig. 2.16

If tetracycline is administered to pregnant women or to children younger than age 8 years, it may be incorporated into the dentin of developing teeth. Initially, this produces a yellow or gray-to-brown discoloration of the affected teeth. When exposed to ultraviolet light, the teeth will fluoresce a bright yellow. The color of the teeth is more brilliant immediately after eruption and becomes more brown with exposure to light. Long courses of the antibiotics normally are required to produce the staining. Occasionally, enamel hypoplasia also is seen; it is uncertain if these changes are caused by the drug or are secondary to the disorder that required use of the antibiotic. When the drug is required for a life-threatening problem, oxytetracycline or doxycycline should be used because they tend to produce less severe tooth staining.

Tooth discoloration is not restricted to the developing dentition of children. In patients using forms of tetracycline (especially minocycline) for long periods of time, significant discoloration of the teeth is possible in adults. This is thought to be due to incorporation into physiologic secondary dentin that continues to be deposited throughout life. In addition to tooth discoloration, minocycline use has also been shown to cause pigmentation of the bone, skin, nails, sclera, conjunctiva, and thyroid.

Mild cases of discoloration may be bleached externally. Although more severe cases may be bleached internally, most clinicians choose facial veneering with composite resins or porcelain. In teeth with significant enamel hypoplasia, complete crown coverage often is beneficial.

EXTRINSIC STAINING OF TEETH

Fig. 2.17

Abnormal colorations of teeth may be due to intrinsic factors (tetracycline therapy, hyperbilirubinemia, etc.) or extrinsic influences. Frequently encountered extrinsic causes include bacterial stains, tobacco, foods/beverages, gingival hemorrhages, restorative materials, and medications. Common foods associated with significant staining of teeth include coffee, tea, and foods that contain abundant chlorophyll. Use of tobacco products often results in widespread discoloration of the pits and fissures, with more diffuse coverage occasionally noted. When the smooth surfaces are affected, the lingual portion of the mandibular incisors typically is involved initially and most extensively.

Recently, extrinsic staining has been associated with use of several popular oral antimicrobial mouthwashes. Chlorhexidine-containing and, to a lesser extent, essential oil/phenolic rinses have been associated with extrinsic staining. Accumulated evidence strongly suggests a dietary influence in the staining. Although many foods may be responsible, beverages such as tea, coffee, and red wine synergistically influence strongly the staining capability of these mouthwashes. Attempts to negate the staining characteristics of the antimicrobial rinses have been successful but have resulted in diminished antiplaque qualities.

PINK TOOTH OF MUMMERY

Fig. 2.18

Pink tooth of Mummery refers to a rosy pink discoloration of the crown of a tooth, usually at the neck. Most frequently, this arises secondary to internal resorption in which the hyperplastic pulp tissue within the chamber enlarges at the expense of the coronal dentinal walls. On thinning of these walls, the pink, vascular pulp tissue may show through the thin enamel.

On occasion, a pink tooth may be seen secondary to undermining external resorption. One pattern of aggressive external resorption is termed “invasive or cervical resorption” and typically begins along the root surface in the cervical area below the epithelial attachment. Once initiated, the resorption can proceed in several directions and can extend coronally under the enamel, creating a pink discoloration of the crown.

- **Figure 2.16**
TETRACYCLINE STAINING OF TEETH
Dentition exhibiting diffuse grayish discoloration of the crowns. In addition, note the presence of environmental enamel hypoplasia on the cuspids and first bicuspids.



- **Figure 2.17**
EXTRINSIC STAINING OF TEETH
Dentition exhibiting diffuse brown surface staining secondary to long-term use of betel quid.



- **Figure 2.18**
PINK TOOTH OF MUMMERY
Diffuse red discoloration of the maxillary left central incisor. (Case courtesy of Dr. Sheila Porter.)



INTERNAL RESORPTION OF TEETH

Fig. 2.19

Internal resorption of teeth refers to removal of tooth structure that involves the inner dentinal walls by cells originating in the dental pulp. Most instances occur during adulthood and exhibit no significant sex predilection; prevalence is less than 1% of patients. Most commonly, initiation of the process is associated with some form of trauma or dental caries. In a few patients, the resorption is idiopathic, with no obvious predisposing factor identified. The resorption may occur in the root or crown and appears as an area of radiolucency overlying the affected area. Typically, the margins of the lucency are smooth and well defined. The walls of the canal appear to balloon out and cannot be followed through the center of the lesion. Rarely is more than one tooth involved. Root canal therapy may prove beneficial if the resorption has not perforated into the periodontal ligament and if the area of resorption can be properly instrumented.

EXTERNAL RESORPTION OF TEETH

Figs. 2.20 and 2.21

External resorption of the teeth begins along the root surface and is the result of activity of cells within the periodontal ligament. Radiographically, the lucency typically demonstrates symmetrical, ill-defined borders, often superimposed over an intact root canal system. This pattern of resorption has many known causes, but the most frequently encountered is periapical inflammation. Reimplanted teeth that are not able to reestablish their vascular supply exhibit significant resorption. Impacted teeth, tumors, and cysts may produce resorption of the adjacent teeth, mostly secondary to pressure. Orthodontic therapy also may be related to significant resorption, regardless of the duration, type of appliance, or degree of force. Not infrequently, impacted or embedded teeth also may exhibit external resorption. In the past, many cases of external resorption were thought, on radiographic examination, to represent anachoretic caries; but microscopically, the teeth exhibited no caries, only resorption.

In contrast to internal resorption, external resorption is almost universal, with all patients having at least a minor degree of resorption on one or more teeth. The resorption potential is inherent in each individual and is the single most important factor that determines the degree of resorption that occurs secondary to stimulus. When pretreatment radiographs reveal an abnormally high degree of external resorption, treatment that promotes resorption, such as orthodontics, should be undertaken with great hesitation.

Some cases of extensive external resorption are idiopathic and may involve numerous teeth. Without any known cause, these idiopathic examples are frustrating and often difficult to stop. When the resorption occurs in the cervical area, exposure and removal of the soft tissue within the lacuna and restoration of the defect may stop the destruction. On occasion, external resorption has been treated successfully through placement of calcium hydroxide into the pulp canals; this may have been possible because of small communications between the defect and the pulp.



Figure 2.19

INTERNAL RESORPTION OF TEETH

A right maxillary central incisor exhibiting large, centrally placed radiolucency of the middle portion of the root. Note the loss of pulp canal boundaries in the area of destruction. (Case courtesy of Dr. L. R. Bean.)



Figure 2.20

EXTERNAL RESORPTION OF TEETH

A maxillary left central incisor demonstrating extensive root destruction. The tooth was traumatically avulsed 2 years ago. At that time, root canal therapy was performed and the tooth was reimplanted.



Figure 2.21

EXTERNAL RESORPTION OF TEETH

Diffuse extensive root resorption that began during orthodontic therapy.



Fig. 2.22

Historically, natal teeth are deciduous teeth that are erupted at birth; neonatal teeth are those that erupt within the first 30 days of life. This is an artificial separation, and it seems appropriate to designate both forms as natal teeth. These prematurely erupted deciduous teeth have been termed “predeciduous dentition,” but only rarely do they represent supernumerary teeth. Although other teeth may be involved, most are mandibular central incisors. Natal teeth may produce nursing difficulties because the infant may place the tongue over these teeth while nursing. This often results in the formation of a traumatic ulceration on the ventral surface of the tongue known as “Riga-Fede disease” (see page 174). In many of these cases, extraction is not necessary and resolution of the ulceration often occurs after smoothing of rough incisal edges or placement of round smooth composite over the exposed portion of the tooth.

On occasion, newly erupted natal teeth may be loose and mobile. Concern over possible aspiration seems overblown, and inhalation of a natal tooth has never been reported. Typically, natal teeth become less mobile by 1 month of age. Natal teeth should be left alone if not causing significant difficulty to the infant or mother. If removed, the soft tissue at the surgical site should be curetted to prevent further tooth development from odontogenic remnants.

ANKYLOSIS

Fig. 2.23 and 2.24

Eruption is a continuous process of occlusal movement by a tooth from its developmental position to its functional position. Although the process typically continues throughout life, cessation in eruption occasionally occurs and is termed “submergence” or “infraocclusion.” A tooth is considered submerged when its marginal ridges (previously in normal position) are more than 0.5 mm below the marginal ridges of the adjacent normal teeth. In rare cases, the submerged tooth may become completely encased by the growing alveolar ridge, a process termed “reimpaction.” Although permanent teeth may be involved, deciduous teeth are affected most commonly.

Most cases of submerged teeth are due to ankylosis in which there is a focus of fusion between the cementum and the adjacent bone. In a few cases, the affected tooth exhibits a sharp, solid sound on percussion or radiographic absence of the periodontal ligament space in a focal area. Submergence may result in drift of adjacent teeth, super-eruption of its antagonist, plaque accumulation, gingival inflammation, and decreased alveolar bone growth.

Submerged deciduous teeth with an underlying successor usually exfoliate normally, although the process may be delayed for a few months. Appropriate therapy consists of close follow-up or, in severe cases, space maintenance or restoration to rebuild the height of the tooth. Those without successors and affected permanent teeth may be restored to proper height or extracted and replaced with a prosthesis. In addition, permanent teeth may be luxated and orthodontically moved into position.

► **Figure 2.22**
NATAL TEETH

A mandibular left central deciduous incisor that was erupted at birth. The infant developed a traumatic ulceration of the ventral tongue secondary to nursing (Riga-Fede disease). A protective shield was constructed and the tooth was retained.



► **Figure 2.23**
ANKYLOSIS

Mirror view of a retained maxillary second deciduous molar that failed to maintain a functional level of occlusion.



► **Figure 2.24**
ANKYLOSIS

Radiograph of dentition depicted in Figure 2.23.



MESIODENS

Figs. 2.25 and 2.26

Supernumerary teeth are additional teeth present within the dental arch. These teeth are thought to arise from accessory buds of the dental lamina that develop into fully formed teeth. These extra teeth may or may not resemble the adjacent teeth.

Approximately 90% of supernumerary teeth develop in the maxilla, with the most common being the mesiodens. These supernumerary teeth occur in the anterior maxilla in the midline and may present alone or bilaterally. Since few achieve eruption, most mesiodentes are discovered secondary to lack of eruption of the adjacent maxillary incisor or on radiographic examination. Often the crowns are cone-shaped with short roots and occasionally may be inverted.

Extraction during childhood is recommended due to a significant number of possible complications related to their continued presence. Delayed eruption, dilaceration, crowding or displacement of adjacent teeth, abnormal diastema formation, tumor or cyst formation, and eruption into the nasal cavity have been associated with mesiodentes. Impacted supernumerary teeth also may create problems if they are exposed secondary to bone resorption and trauma associated with denture use. Early removal often results in spontaneous eruption and alignment of the adjacent permanent teeth within 6 months to 3 years. Removal in older adults often is difficult because of dense bone, further justifying early intervention. Heredity exerts a strong influence on the presence of supernumerary teeth, but a consistent Mendelian pattern is not evident.

SUPERNUMERARY TEETH

Fig. 2.27

Although the mesiodens is the most common supernumerary tooth, numerous other supernumerary teeth may occur. Fourth molars, bicuspid, maxillary lateral incisors, mandibular central incisors, and maxillary paramolars are not rare. A paramolar is a small supernumerary maxillary molar located buccal, palatal, or interproximal to a permanent maxillary molar.

In contrast to most supernumerary teeth, supernumerary bicuspid most frequently arise in the mandible rather than the maxilla. These accessory premolars typically are discovered late, with most visible initially between 12 and 14 years of age. If eruption occurs, they often produce crowding, hygiene difficulties, malalignment of teeth, and resorption of adjacent teeth.

► **Figure 2.25**
MESIODENS

Bilateral supernumerary teeth overlying the crowns of the unerupted maxillary central incisors.



► **Figure 2.26**
MESIODENS

Multiple erupted supernumerary teeth of the anterior maxilla. (Courtesy of Dr. Kurt Studt.)



► **Figure 2.27**
SUPERNUMERARY TEETH

Two erupted supernumerary mandibular bicuspid.



SUPERNUMERARY TEETH

Fig. 2.28

Although most supernumerary teeth occur in the maxilla, supernumerary mandibular teeth are not rare. Of these, accessory mandibular incisors are least common and represent less than 2% of supernumerary teeth. These teeth can be overlooked because they are not counted easily and often exhibit crowding and malalignment.

Most supernumerary teeth occur in the permanent dentition, but supernumerary deciduous teeth do occur. Typically, removal of these teeth is not recommended because most shed normally on eruption of the permanent dentition and removal may danger the underlying developing permanent tooth. Other more unusual locations for supernumerary teeth include the gingiva, soft palate, nasal cavity, incisive suture, ophthalmic conchae, sphenomaxillary fissure, maxillary tuberosity, maxillary sinus, and between the orbit and the brain.

Multiple supernumerary teeth may occur, and many are associated with one of many syndromes. More than 20 syndromes have been associated with supernumerary teeth; the most strongly correlated is cleidocranial dysplasia (see Figs. 11.4 to 11.6). Reports of impacted and supernumerary teeth associated with Gardner syndrome (see Figs. 11.11 and 11.12) are known, but many cases exhibit only permanent teeth embedded within the dense bone of the jaw osteomas.

HYPODONTIA

Figs. 2.29 and 2.30

Anodontia is the congenital absence of all teeth. Impaction or previous extraction of the teeth must be ruled out and is not considered true anodontia. Generalized true anodontia is rare and most often is associated with hereditary hypohidrotic ectodermal dysplasia. Absence of one or more teeth is relatively common, and is termed hypodontia. Oligodontia is a subdivision of hypodontia and refers to six or more absent teeth. Missing teeth are uncommon in the deciduous dentition, and the most commonly involved permanent teeth are the third molars, maxillary lateral incisors, and bicuspid. When present, an increased prevalence of ankylosis, disturbances of spacing, tooth eruption, and exfoliation are noted. Hypodontia is correlated positively with microdontia and exhibits a greater prevalence in females. Radiation therapy to the jaws during tooth formation can destroy the tooth buds and produce a pattern similar to hypodontia. A strong genetic influence is noted, with hypodontia demonstrating an association with close to 40 different syndromes.

► **Figure 2.28**

SUPERNUMERARY TEETH

An erupted, supernumerary left mandibular central incisor. (Courtesy of Dr. Janet Edwards.)



► **Figure 2.29**

HYPODONTIA

Dentition demonstrating retained deciduous teeth and the absence of numerous permanent teeth that never developed. Note the cone-shaped appearance of the posterior teeth.



► **Figure 2.30**

HYPODONTIA

Occlusal view of the dentition described in Figure 2.29. The cuspids and the bicuspid were the only mandibular permanent teeth to develop. The patient did not exhibit any other signs or symptoms of ectodermal dysplasia.



MICRODONTIA AND MACRODONTIA

Fig. 2.31

Microdontia refers to teeth that are smaller than normal, and macrodontia refers to teeth that are larger than normal. These disorders may be generalized to all of the teeth or they may be localized to one or more teeth. The generalized forms are rare and normally associated with pituitary dysfunction. Localized microdontia is not uncommon and most often is seen involving the maxillary lateral incisors or third molars. Affected laterals often exhibit cone-shaped crowns and are termed “peg laterals.” Localized macrodontia is less common and must be separated from fusion and gemination.

Hereditry exhibits a strong influence, but seems to be combined with environmental factors in many cases. Microdontia is correlated positively with hypodontia, and macrodontia is associated with an increased prevalence of hyperdontia (supernumerary teeth). Females demonstrate a higher frequency of microdontia, and males exhibit a greater degree of macrodontia.

GEMINATION

Figs. 2.32 and 2.33

Double teeth refers to two teeth that are totally or partially joined by dentin and maybe their pulps. These abnormal teeth are the result of gemination or fusion (see Figs. 2.34 and 2.35). Gemination is defined as a single enlarged tooth or joined (double) tooth in which the tooth count is normal when the anomalous tooth is counted as one. Classically, gemination is thought to represent the incomplete attempt of one tooth germ to divide into two. In many instances, the origin is arguable; the double tooth could have arisen from a split of a single tooth bud or fusion between a normal tooth bud and that of a supernumerary tooth. This argument is academic, and the definition of gemination need not consider this controversial aspect. Gemination is distinguished from fusion by the presence of a full complement of teeth; if no tooth is missing and an incompletely divided tooth exists, it usually is termed gemination.

Geminated teeth typically demonstrate two crowns or one large partially separated crown sharing a single root or root canal. The most commonly affected teeth are the permanent maxillary incisors and the deciduous mandibular incisors. Most examples present as isolated anomalies, but bilateral cases have been reported. Treatment is problematic and often requires extraction and prosthetic replacement of the affected teeth. Recently, investigators described sectioning and removal of the excess coronal tooth structure while retaining vitality and not performing conventional endodontics.

► **Figure 2.31**
MICRODONTIA

A maxillary right lateral incisor that is smaller than normal.



► **Figure 2.32**
GEMINATION

A patient who has a full complement of teeth and demonstrates bilateral enlarged maxillary central incisors. Note the incisal groove in the left central incisor. (Case courtesy of Dr. John Mink.)



► **Figure 2.33**
GEMINATION

Radiograph of a lateral incisor that has partially divided to form two incompletely separated crowns.



FUSION

Figs. 2.34 and 2.35

As mentioned in the discussion of gemination, double teeth may result from either fusion or gemination. Fusion is defined as a single enlarged tooth or joined (double) tooth in which the tooth count reveals a missing tooth when the anomalous tooth is counted as one. Classically, fusion is thought to arise from the union of two normally separated tooth germs. The diagnosis of fusion is best reserved for two completely or incompletely fused teeth that have arisen in the place of two normal teeth. A hereditary pattern occasionally is observed.

These teeth may appear as one large tooth, as one incompletely fused crown, or as two crowns sharing completely or incompletely fused roots. No matter what the pattern, by definition, the fusion must involve the dentin. The occurrence is seen in both deciduous and permanent teeth and has been reported more frequently in the deciduous dentition.

CONCRESCENCE

Fig. 2.36

Concrecence is the union of two adjacent teeth by excessive cementum production. This most commonly occurs in the molar region and may involve two normal teeth or a supernumerary tooth and a normal tooth. Concrecence normally is developmental in origin, but some cases may arise after repair of a previous inflammatory lesion. If a nonvital molar has periapical damage from inflammation, the damage may be repaired with cementum production once the inflammatory lesion resolves. Adjacent teeth may become fused after the cementum repair. This most frequently is seen with nonvital maxillary second molars and impacted third molars. Concrecence can produce extraction difficulties and problems in vitality testing.

► **Figure 2.34**
FUSION

Dentition demonstrating union of the crowns of the adjacent right mandibular lateral and central incisors.



► **Figure 2.35**
FUSION

Radiograph demonstrating union of the crowns of the adjacent right mandibular lateral and central incisors.



► **Figure 2.36**
CONCRESCENCE

Gross photograph of maxillary third molar that is fused by cementum to the adjacent supernumerary fourth molar.



Figs. 2.37, 2.38, and 2.39

Dens invaginatus is a deep surface invagination of the crown or, rarely, the root of a tooth that is lined by enamel and dentin; it has been termed “dens-in-dente” because it may appear to be a tooth within a tooth. The invagination may be shallow and confined to the crown of the tooth or it may extend all the way to the apex.

In the coronal variants, the defect typically is isolated but may occur in multiple teeth. Bilateral and symmetrical cases occasionally are seen. The most commonly affected tooth is the maxillary lateral incisor. The invagination normally opens at the cingulum area. Radiographically, the invagination is seen easily since it is lined by a thin but distinct layer of enamel. The invagination is constricted adjacent to the opening but often widens as it approaches the base. Occasionally, there is a bulbous enlargement of the invagination at the base that contains an abundance of dysplastic enamel.

Radicular examples of dens invaginatus are rare and arise in areas of ectopic enamel that are similar to that associated with enamel pearls (see Fig. 2.43). Rather than forming an exophytic projection, the ectopic enamel lines a surface invagination into the root. Typically, no clinical problems arise unless the radicular concavity is exposed to the oral cavity.

Coronal invaginations are prone to development of caries; without treatment, pulpal necrosis can occur rapidly. These openings should be restored prophylactically as soon as possible after eruption to prevent carious pulpal necrosis. Because of the open apex, successful endodontic therapy is difficult but has been accomplished.

Occasionally, coronal invaginations rupture out the lateral aspect of the root without communication with the associated dental pulp. Due to the direct communication between the oral cavity and the underlying bone, these teeth develop lateral radicular inflammatory lesions while often maintaining pulpal vitality. Endodontic-like restoration of the coronal invagination may result in resolution of the lateral inflammatory lesion.



Figure 2.37

DENS INVAGINATUS

Maxillary right lateral incisor demonstrating enamel-lined invagination originating in the area of the cingulum.



Figure 2.38

DENS INVAGINATUS

Radiograph of an extracted permanent maxillary lateral incisor that exhibits a deep surface invagination lined by enamel. The invagination originated close to the cusp tip, and the apical portion was filled with an abundance of dysplastic enamel. (Courtesy of Dr. Michael Ridley.)



Figure 2.39

DENS INVAGINATUS

Gross photograph of a decalcified specimen demonstrating deep surface invagination that extends close to the apex.



TALON CUSP

Figs. 2.40 and 2.41

The talon cusp is an accessory cusp usually located on the lingual surface and rarely on the facial surface of permanent or deciduous incisors. The cusp arises in the cingulum area, and more than 90% affect maxillary teeth. This projection, in combination with the normal incisal edge, occasionally forms a pattern resembling an eagle's talon. The cusp often demonstrates a deep developmental groove where it joins the lingual surface of the incisor. This groove is prone to caries and should be restored prophylactically to prevent carious exposure of the pulp. Although a talon cusp may occur as an isolated finding, often additional dental abnormalities, such as shovel-shaped incisors, bifid cingula, cusps of Carabelli, and dens invaginatus, also are present.

Involvement of maxillary teeth may result in malalignment of the anterior teeth or occlusal interference with the mandibular teeth. In these instances, the cusp often must be removed to achieve normal function. It must be remembered that the cusp usually consists of enamel, dentin, and pulp; rapid removal often results in pulp exposure. To avoid exposure and the resultant endodontic therapy, the cusp can be removed a small portion at a time. Removal should cease any time the pulp is approached. The exposed dentin should be covered with calcium hydroxide or a desensitizing agent such as a fluoride varnish, and the patient told to return after the pulp has had time to recede from the surface. With time and patience, the cusp can be removed without necessitating root canal therapy. Talon cusps demonstrate a multifactorial inheritance pattern and have been seen in association with the Rubinstein-Taybi and Mohr syndromes.

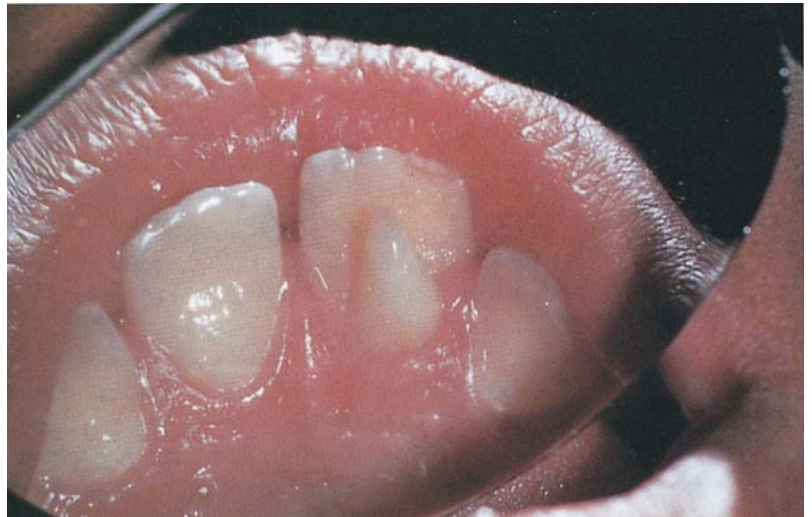
TAURODONTISM

Fig. 2.42

Taurodontism is a developmental abnormality of molar teeth in which the body of the affected tooth is very large and the associated roots are shortened, with the bifurcation near the apex. The tooth tends to be rectangular in shape, with a large pulp chamber dramatically increased in its apico-occlusal height. Variations in severity are seen. Involvement of one or more molars may be seen in one or all four of the quadrants. The permanent teeth are most commonly affected, but occurrence is seen in the deciduous dentition. The condition may occur as an isolated trait or be associated with one of close to 20 syndromes known to be associated with taurodontism. An increased prevalence of this tooth abnormality is noted in patients demonstrating oligodontia.

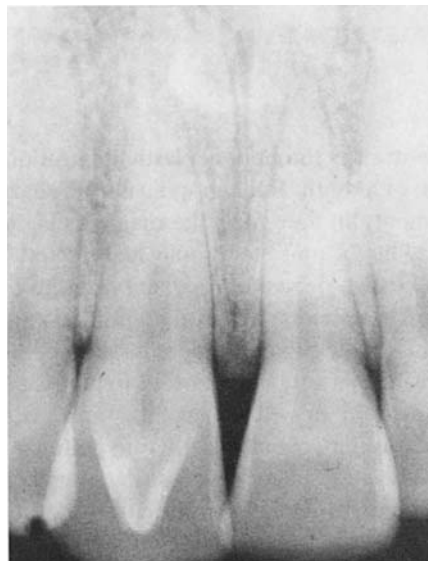
► **Figure 2.40**
TALON CUSP

A maxillary central incisor demonstrating an accessory palatal cusp.



► **Figure 2.41**
TALON CUSP

Radiograph of a maxillary central incisor demonstrating an accessory cusp that consists of enamel, dentin, and pulp.



► **Figure 2.42**
TAURODONTISM

A mandibular first molar exhibiting short roots, bifurcation close to the apex, and a pulp chamber that is dramatically increased in apico-occlusal height.



ENAMEL PEARL

Fig. 2.43

The enamel pearl is an uncommon ectopic mass of enamel located near the bifurcation of molar teeth, usually in the permanent dentition. Rare cases have been documented in teeth anterior to the molars. This is thought to be a developmental abnormality that arises from a cluster of misplaced ameloblasts located in the area. The maxillary molars are affected most frequently, but mandibular cases do occur. The pearls may be seen radiographically and can be confused with calculus. Large pearls may exhibit root formation, and some believe the histogenesis is based on the ability of the carrier root to partially behave as a new tooth germ.

These globules of enamel usually do not present any clinical problems unless periodontal disease develops in the bifurcation. The ectopic enamel precludes normal periodontal attachment and may lead to hygiene problems and rapid loss of attachment. If removal is planned, it must be remembered that they may contain a fine strand of dentin and pulp. In deciduous teeth, enamel pearls may produce delayed exfoliation or deviation of the underlying permanent tooth.

HYPERCEMENTOSIS

Fig. 2.44

Hypercementosis is the nonneoplastic deposition of excessive secondary cementum along the root surface of a tooth. Radiographically, no sharp dividing line can be seen between the dentin and the cementum; therefore, the diagnosis is made from the abnormal blunt shape of the enlarged root. One or more teeth may be affected, and bicuspid are involved most frequently. On occasion, excessive deposition is generalized to the entire dentition.

Typically, the process occurs in adulthood and increases in prevalence with age. Cases discovered in younger patients often demonstrate familial clustering. Hypercementosis is known to be associated with tooth repair, adjacent inflammation, and teeth that are not in occlusion (impacted, embedded, or without an antagonist). A few systemic disorders, including Paget disease, acromegaly, pituitary gigantism, thyroid goiter, rheumatic fever, arthritis, calcinosis, and possibly vitamin A deficiency, have been associated with hypercementosis. No treatment is required; however, such teeth may prove difficult to extract.

DILACERTION

Fig. 2.45

Dilaceration is an abnormal bend in the root or crown of a tooth. The bend occurs much more frequently in the root but may be present anywhere along the length of the tooth. Dilaceration is thought to arise secondary to trauma during tooth formation that alters the angle between the tooth germ and the portion of the tooth already developed. On occasion, the bend is created by pressure from an adjacent cyst, tumor, or odontogenic hamartoma. The cause in some cases is difficult to ascertain.

The most frequently affected teeth are the permanent maxillary incisors, followed by the mandibular anteriors. Occasionally, deciduous teeth are affected from perinatal trauma such as laryngoscopy or intubation. The severity of the angulation seems to be related to the age of the patient and the direction and degree of the force applied. Dilaceration of the roots may produce delayed eruption or difficulties during root canal therapy or extraction; recognition on preoperative radiographs often minimizes these problems.

► **Figure 2.43**
ENAMEL PEARL

Ectopic mass of enamel located in the bifurcation of a maxillary first molar.
(Courtesy of Dr. Joseph Beard.)



► **Figure 2.44**
HYPERCEMENTOSIS

Radiograph of the mandibular first molar demonstrating apical enlargement and blunting of the roots secondary to excessive cementum deposition.



► **Figure 2.45**
DILACERATION

Abnormal bend in the apical third of the root of the mandibular second bicuspid.



Figs. 2.46, 2.47, and 2.48

Amelogenesis imperfecta is a complicated group of disorders that encompasses at least 15 different hereditary abnormalities of enamel formation that typically affect all of the teeth in both the deciduous and permanent dentitions in the absence of a related systemic disease. Enamel develops in three stages: first the organic matrix is laid down, then it is mineralized, and, finally, the enamel crystals mature into their final organization. Malfunction of each individual stage may be seen and be responsible for the variation of the clinical presentations. The current classification uses the inheritance pattern and clinical features to group the different forms of amelogenesis imperfecta. Several investigators have begun to formulate a more accurate classification based on the molecular biology of these hereditary enamel defects.

Hereditary defects in enamel matrix formation constitute the category of hypoplastic amelogenesis imperfecta. The affected teeth demonstrate numerous pits, vertical grooving, or a dramatically diminished thickness of enamel. Hypoplastic amelogenesis imperfecta may be inherited as an autosomal-dominant, autosomal-recessive, or X-linked-dominant disorder. When pitted, the defects may be randomly distributed over all of the teeth or localized and arranged in horizontal rows in the middle one third of the crown. In contrast to other forms of amelogenesis imperfecta, the localized pitted form may involve only scattered teeth and affect only the deciduous dentition.

Those with dramatically thin enamel may exhibit a smooth or rough surface texture; in some cases, the enamel is so thin it is termed “enamel agenesis.” These patterns produce teeth that often are small and yellow with open contact points and shaped like the underlying dentin. In the X-linked-dominant pattern, affected males demonstrate diffuse involvement; females reveal vertical furrows of thin hypoplastic enamel alternating between bands of normal-thickness enamel.

Radiographically, the pitted type demonstrates numerous pits within the enamel, whereas those with thinned enamel exhibit teeth covered by a very thin shell of radiopaque enamel. To restore function and improve aesthetics, these enamel defects often are treated best with full coverage; on occasion, however, attrition reduces the crown height to such an extent that overdentures become the treatment of choice.

► **Figure 2.46**
AMELOGENESIS IMPERFECTA, ROUGH
HYPOPLASTIC TYPE

Dentition demonstrating dramatic reduction in thickness of enamel that has resulted in teeth that appear smaller than normal with open contact points. The teeth are the shape and color of the underlying dentin.



► **Figure 2.47**
AMELOGENESIS IMPERFECTA, SMOOTH
HYPOPLASTIC TYPE

Shiny-surfaced dentition covered by a thin layer of hypoplastic enamel (see radiograph in Fig. 2.48). Due to crowding, the typical widely spaced dentition is lacking. Composite resin has been added to the maxillary central incisors. Note the anterior open bite. (Courtesy of Dr. John G. Stephenson.)



► **Figure 2.48**
AMELOGENESIS IMPERFECTA, SMOOTH
HYPOPLASTIC TYPE

Radiograph of patient depicted in Figure 2.47. Note the thin radiopaque band of enamel overlying the cusps of the molars. (Courtesy of Dr. John G. Stephenson.)



AMELOGENESIS IMPERFECTA, HYPOCALCIFIED TYPE

Figs. 2.49 and 2.50

Inheritable defects in enamel formation that prevent mineralization of the enamel matrix are termed “amelogenesis imperfecta, hypocalcified type.” These teeth demonstrate white and opaque or brownish-orange enamel that can be removed easily with hand instruments and has a soft, cheesy, and crumbly texture. Before eruption, the teeth exhibit enamel of normal shape and thickness, but the radiodensity is similar to that of the underlying dentin. On eruption, the enamel can be abraded easily, and it often exhibits significant calculus accumulation. The most common form is inherited as an autosomal-dominant trait, but some cases do exhibit autosomal-recessive inheritance. Full coverage is the treatment of choice to improve aesthetics, restore function, and prevent accelerated periodontitis secondary to the significant calculus accumulation.

AMELOGENESIS IMPERFECTA, DIFFUSE HYPOMATURATION TYPE

Fig. 2.51

Inheritable defects in enamel formation that prevent the final enlargement and maturation of the enamel crystals are termed “amelogenesis imperfecta, hypomaturational type.” The enamel is of normal size and shape but often is lost partially by chipping. The hypomaturational may be diffuse or localized to the incisal/occlusal one third of the teeth (see Fig. 2.52).

In the typical diffuse pattern, the entire dentition is opaque yellow-white, may exhibit mottling, and is inherited as an X-linked-recessive disorder. Secondary to its mode of inheritance, males reveal uniformly diffuse involvement and affected females typically demonstrate alternating vertical bands of white opaque enamel and normal translucent enamel. The vertical pattern in females is not obvious under normal lighting conditions and is best viewed by transillumination. Clinically and radiographically, the teeth in affected males closely resemble the dental changes of fluorosis. In most instances, the dentition is not impaired functionally, and treatment centers around aesthetic improvement of the anterior teeth.

Another variant of hypomaturational amelogenesis imperfecta is termed the “pigmented pattern”; it appears diffusely agar-brown and exhibits autosomal-recessive inheritance. In this pattern, the enamel often separates from the underlying dentin and frequently is soft enough to be pierced by a dental explorer. On occasion, enamel softness similar to that seen in the hypocalcified forms is present. Treatment with full crowns in severe cases can improve aesthetics and function.

► **Figure 2.49**

**AMELOGENESIS IMPERFECTA,
HYPOCALCIFIED TYPE**

Dentition demonstrating brown-to-orange enamel that can easily be removed with hand instruments. (Courtesy of Drs. D. S. Holbrook and S. Altman.)



► **Figure 2.50**

**AMELOGENESIS IMPERFECTA,
HYPOCALCIFIED TYPE**

Radiograph of the dentition depicted in Figure 2.49. Note the significant loss of enamel that has occurred from physiologic use. In addition, the radiodensity of the enamel is similar to that of the underlying dentin. (Courtesy of Drs. S. Altman and D. S. Holbrook.)



► **Figure 2.51**

**AMELOGENESIS IMPERFECTA, DIFFUSE
HYPOMATURATION TYPE**

Dentition demonstrating enamel with diffuse white opaque and yellow discoloration.



Fig. 2.52

In this pattern of amelogenesis imperfecta, areas of opaque white hypomaturation are noted in the incisal and occlusal one quarter of the teeth. The altered areas do not form a pattern consistent with environmental damage and often reveal an anterior-to-posterior distribution that has been compared with the cusp tips of a denture dipped in white paint. Both the deciduous and permanent dentitions are affected, with most cases exhibiting an X-linked pattern of inheritance. An autosomal-dominant pattern also may exist. The affected teeth typically are functionally sound and require no therapy.

DENTIN DYPLASIA, TYPE I

Figs. 2.53 and 2.54

Dentin dysplasia, type I, which also is known as “radicular dentin dysplasia,” or “rootless teeth,” is one of two forms of dentin dysplasia. The second, type II, is related closely to dentinogenesis imperfecta and is presented following that section (see Figs. 2.58 and 2.59). Dentin dysplasia, type I, is autosomal dominant and affects both the deciduous and the permanent dentitions.

The coronal enamel and dentin are well formed and appear normal clinically. The dentin in the roots is extremely disorganized, often resulting in short roots and total obliteration of the pulp canals. The deciduous teeth usually are affected severely and exhibit no pulp chambers. The radiographic pattern in permanent teeth varies according to the degree of dentin disorganization within the root. In some patients, the roots are extremely short with no detectable pulp; in others, the roots are slightly longer and reveal crescent or chevron-shaped pulp chambers. In mildly affected patients, the root length approaches normal, and the canals contain a centrally placed pulp stone. Frequent periapical inflammatory lesions are seen secondary to coronal exposure of superficial microscopic threads of pulpal remnants present within the defective dentin.

Early tooth loss secondary to the short roots and frequent periapical lesions may occur. A few cases have been treated endodontically, with resolution of some of the periapical lesions. The initial endodontic therapy is most difficult because the obliterated root canals have to be instrumented initially with burs. Lesions that fail to resolve are treated with apical curettage if they enlarge or become symptomatic. Otherwise, without these heroics, there is no treatment.

► **Figure 2.52**
AMELOGENESIS IMPERFECTA, SNOW-CAPPED TYPE

Dentition demonstrating white opaque enamel in the incisal and occlusal one-quarter of the teeth.



► **Figure 2.53**
DENTIN DYSPLASIA, TYPE I

Dentition that demonstrates normal coronal structure. (Courtesy of Dr. Charles J. Cunningham.)



► **Figure 2.54**
DENTIN DYSPLASIA, TYPE I

Radiograph of the posterior mandible, in which the teeth demonstrate normal coronal structure, short roots, absence of pulp canals, and only small crescents of pulp chambers. Note the numerous periapical lesions. (Courtesy of Tidwell E, Cunningham CJ. Dentinal dysplasia: endodontic treatment, with case report. J Endod 1979;5:372.)



Figs. 2.55, 2.56, and 2.57

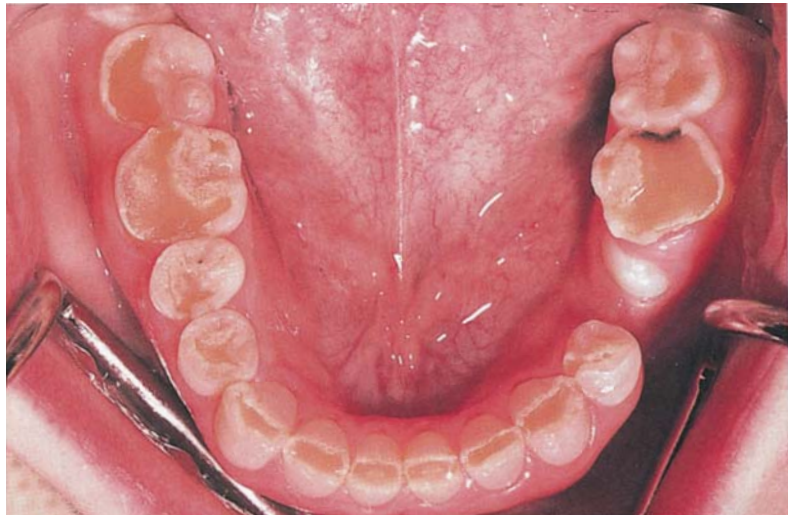
Dentinogenesis imperfecta is an autosomal-dominant disturbance in dentin formation that presents with opalescent teeth in both dentitions and is not associated with a systemic disease. Similar dental findings can be seen in patients with osteogenesis imperfecta, a systemic disorder of bone exhibiting a clearly separate genetic origin. Because these two diseases are distinctly different, when altered teeth are noted in the latter disease, it should be termed “osteogenesis imperfecta with opalescent teeth” and not “dentinogenesis imperfecta.”

The classic presentation involves both deciduous and permanent teeth that are clinically opalescent and radiographically exhibit premature closure of the pulp chambers and canals. Although the overlying enamel is normal, it may be lost early, with resultant severe attrition. The coloration of the teeth varies from blue-gray to yellow to brown. When large kindreds are reviewed, occasional variations in expression are noted and previously have been termed the “Brandywine isolate.” In these cases, the deciduous teeth may exhibit enamel hypoplasia and multiple pulp exposures in addition to the other dentin changes. The teeth are opalescent, and although many pulps may exhibit premature closure, others are normal or greatly enlarged, forming the pattern known as “shell teeth.” A similar variation in expression has been noted in patients with osteogenesis imperfecta and opalescent teeth. Although full crowns may prove beneficial in preserving the teeth, most affected individuals are candidates for full dentures or implants by age 30 years despite numerous interventions. Partial dentures are contraindicated because the roots fracture easily.

- **Figure 2.55**
DENTINOGENESIS IMPERFECTA
Dentition demonstrating translucence and grayish discoloration.



- **Figure 2.56**
DENTINOGENESIS IMPERFECTA
Occlusal view of the dentition depicted in Figure 2.55. Note the loss of enamel and the accelerated attrition.



- **Figure 2.57**
DENTINOGENESIS IMPERFECTA
Radiograph of the patient depicted in Figures 2.55 and 2.56. Note the thin, pointed roots, the bulbous crowns, and the obliteration of the pulp canals.



DENTIN DYSPLASIA, TYPE II

Figs. 2.58 and 2.59

Dentin dysplasia, type II (coronal dentin dysplasia), is an autosomal-dominant abnormality of dentin formation that exhibits numerous features of dentinogenesis imperfecta and has been suggested to be a variant of that disorder. The deciduous teeth are opalescent and exhibit premature closure of the pulp chambers and canals. Clinically, the permanent teeth are normal without opalescence. Radiographically, they exhibit enlarged “thistle-tube” shaped pulp chambers, all of which will eventually develop pulp stones. No significant clinical problems are seen in the permanent teeth, and no treatment is required.

REGIONAL ODONTODYSPLASIA

Fig. 2.60

Regional odontodysplasia or “ghost teeth” is an idiopathic developmental abnormality of enamel and dentin that is not hereditary. Although no consistent cause has been documented, many cases have been related to a variety of syndromes, growth abnormalities, neural disorders, and vascular malformations.

This condition may affect a single tooth or several contiguous teeth in a localized area. Rarely, an unaffected tooth may be interposed within a row of affected teeth. The maxilla is affected more frequently with an anterior predominance. Affected deciduous teeth typically are followed by altered permanent teeth; involvement isolated to the permanent teeth is not rare. Usually, the process is confined to a single segment, but it may be bilateral or involve two ipsilateral quadrants. Frequently, affected teeth demonstrate delayed or failed eruption. On eruption, the teeth are yellow-to-brown with a rough surface. Radiographically, a large lucent pulp is encased by a thin layer of enamel and dentin. Short roots and open apices often are present.

Frequent caries and associated periapical inflammatory disease complicate the therapeutic approach. If at all possible, the altered teeth should be retained to allow for appropriate development and preservation of the associated alveolar ridge. Frequently, persistent infection combined with poor tooth structure mandates extraction. Endodontic therapy and coronal restoration have been performed successfully on teeth with sufficient hard tissue. Retained teeth that have maintained their vitality often eventually demonstrate near-normal root formation with overlying hypoplastic crowns. Removable partial dentures can be used to cover affected embedded teeth until the skeletal growth spurt is complete.

► **Figure 2.58**

DENTIN DYSPLASIA, TYPE II

Radiograph of the left posterior maxillary dentition demonstrating thistle-tube shaped pulp chambers. The clinical appearance of the teeth was within normal limits. (Case courtesy of Dr. David Besser.)



► **Figure 2.59**

DENTIN DYSPLASIA, TYPE II

Radiograph of the right anterior mandibular dentition in the patient depicted in Figure 2.58. Note the thistle-tube shaped pulp canals with stones. (Case courtesy of Dr. David Besser.)



► **Figure 2.60**

REGIONAL ODONTODYSPLASIA

Radiograph of the left mandible exhibiting several contiguous deciduous teeth demonstrating large pulp canals encased by a thin layer of enamel and dentin. Note that the underlying succedaneous teeth are affected similarly. Elsewhere, all other teeth are within normal limits. (Courtesy of Dr. John B. Perry.)



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