Lec. 8-A Oral Pathology

DEVELOPMENTAL DEFECTS OF THE ORAL AND MAXILLOFACIAL REGION

Developmental disturbances of the oral region are of these broad categories:

(1) Developmental disturbances affecting teeth.

(2) Developmental disturbances limited to soft tissue.

(3) Developmental disturbances affecting bone.

Disorders of development of teeth

The development of teeth is regulated by genes, but the genetic program is very sensitive to disturbances in the environment such as infection, or toxic chemicals. The causes of developmental disorders of teeth are multifactorial, involving the interaction of genetic and environmental factors.

These disorders may be prenatal or postnatal in origin and may inherit or acquired. Disorders of development of teeth may be due to abnormalities in the differentiation of the dental lamina and the tooth germs, causing anomalies in number, size, form of teeth and abnormalities of morph differentiation or abnormalities in the formation of the dental hard tissue resulting in disturbances in tooth structure ((abnormalities of histo differentiation)).

Developmental alterations in the number of teeth

Anodontia

Absence of teeth is known as *anodontia*. This condition is further qualified as *complete anodontia,* when all teeth are missing; as *partial anodontia* or *hypodontia,* when one or several teeth are missing; as *pseudoanodontia,* when teeth are absent clinically because of impaction or delayed eruption; or as *false anodontia,* when teeth have been exfoliated or extracted. Partial anodontia is relatively common. Congenitally missing teeth are usually third molars, followed by second premolars and maxillary lateral incisors.

1- several regulatory genes are involved in tooth formation and development which may be mutated in hypodontia e.g. (MSX1 and Pax9). These genes are involved in the control of development of the face and of many other tissues and organs in the embryo. Thus, hypodontia may be associated with other craniofacial anomalies and syndromes.

2- Hypodontia is more common in the permanent dentition, occurring in 2-10% in different population (excluding absent third molar) compared to the primary dentition where the prevalence is less than 1%. It is more common in females in racial differences Hypodontia may be symmetrical when particular teeth or groups of teeth are involved or haphazard when the patient is discoverable. When the deciduous teeth to be congenitally absent, which is very unusual is likely that is such cases the permanent teeth will be also fail to form.

Complete anodontia is rare but is often associated with a syndrome known as *hereditary ectodermal dysplasia,* which usually is transmitted as an X-linked recessive disorder. Partial anodontia is more typical of this syndrome, however. The few teeth that are present are usually conical. Hair, cutaneous appendages, and nails are also poorly developed in this syndrome.

Supernumerary Teeth

Extra, or supernumerary, teeth in the dentition most probably result from continued proliferation of the permanent or primary dental lamina to form a third tooth germ. The resulting teeth may have a normal morphology or may be rudimentary and miniature.

Most are isolated events, although some may be familial and others may be syndrome associated (Gardner's syndrome and cleidocranial dysplasia). Supernumerary teeth are found more often in the permanent dentition than in the primary dentition and are much more commonly seen in the maxilla than in the mandible. Mostly in the females than males .The anterior midline of the maxilla is the most common site, in which case the supernumerary tooth is known as a *mesiodens*. The maxillary molar area (fourth molar or paramolar) is the second most common site. The significance of supernumerary teeth is that they occupy space. When they are impacted, they may block the eruption of other teeth, or they may cause delayed eruption or mal-eruption of adjacent teeth. If super-numerary teeth erupt, they may cause malalignment of the dentition and may be cosmetically objectionable.

Teeth appearing at the time of birth are known as *natal teeth,* and those appearing within 6 months following birth are called *neonatal teeth.* Most of these teeth represent prematurely erupted deciduous teeth, usually mandibular central incisors. A small percentage represent supernumerary teeth. Prematurely erupted primary teeth should be preserved (provided they cause no injury to the infant or the mother), and supernumeraries should be extracted. Not to be confused with either of these phenomena is the appearance of common gingival or dental lamina cysts of the newborn.

Supernumerary teeth appearing after loss of the permanent teeth are known as *postpermanent dentition*. This is generally regarded as a rare event. Most teeth appearing after extraction of the permanent teeth are believed to arise from eventual eruption of previously impacted teeth.

Developmental Alterations in the Size of Teeth

Microdontia

In generalized microdontia, all teeth in the dentition appear smaller than normal. Teeth may actually be measurably smaller than normal, as in pituitary dwarfism, or they may be relatively small in comparison with a large mandible and maxilla.

In focal, or localized, microdontia, a single tooth is smaller than normal. The shape of these microdonts is often altered with the reduced size. This phenomenon is most commonly seen with maxillary lateral incisors in which the tooth crown appears cone or peg shaped, prompting the designation *peg lateral*. An autosomal-dominant inheritance pattern has been associated with this condition. Peg laterals are of no significance other than cosmetic appearance. The second most commonly seen microdont is the maxillary third molar, followed by supernumerary teeth .

Macrodontia

Generalized macrodontia is characterized by the appearance of enlarged teeth throughout the dentition. This may be absolute, as seen in pituitary gigantism, or it may be relative owing to a disproportionately small maxilla and mandible. The latter results in crowding of teeth and possibly an abnormal eruption pattern caused by insufficient arch space.

Focal, or localized, macrodontia is characterized by an abnormally large tooth or group of teeth. This relatively uncommon condition usually is seen with mandibular third molars. In the rare condition known as *hemifacial hypertrophy,* teeth on the affected side are abnormally large compared with the unaffected side.

Localized Disturbances in Eruption

Primary Impaction and Ankylosis

Impaction: Impaction of teeth is a common event that most often affects the mandibular third molars and maxillary canines. Less commonly, premolars, mandibular canines, and second molars are involved. It is rare to see impactions of incisors and first molars. Impaction occurs because of obstruction from crowding or from some other physical barrier. Occasionally, it may be due to an abnormal eruption path, presumably caused by unusual orientation of the tooth germ. *Ankylosis,* the fusion of a tooth to surrounding bone, is another cause of impaction. This usually occurs in association with erupted primary molars. It may result in impaction of a subjacent permanent tooth. The reason

for ankylosis is unknown, but it is believed to be related to periapical inflammation and subsequent bone repair. With focal loss of the periodontal ligament, bone and cementum become inextricably mixed, causing fusion of the tooth to alveolar bone.

Ankylosis:

Eruption continues after the emergence of the teeth to compensate for masticatory wear and the growth of the jaws. The cessation of erupt ion after emergence is termed ankylosis and occurs from an anatomic fusion of tooth cementum or dentin with the alveolar bone. Although the areas of union may be too subtle to be detected clinically and radiographically. histopathologic examination will demonstrate fusion between the affected toot h and the adjacent bone in almost all cases. Other terms for this process within the literature include infra occlusion, secondary retention submergence. Re impaction. and re inclusion

Developmental Alteration s in the Shape of Teeth

Disturbances in tooth form may involve the crown, the roots or both. The most frequent variations of the crowns of the teeth affect maxillary permanent lateral incisors which may be peg-shaped. Premolars and molars with an increased or decreased number of cusps are also frequently seen

Double teeth:- a descriptive term used to describe a developmental anomaly where two teeth appear joined together. This is variable and may involve the crown, roots, or both. The teeth are united by dentine or pulp. It occurs mostly in primary than permanent dentition especially anterior teeth.

1-Gemination

is the fusion of two teeth from a single enamel organ. The typical result is partial cleavage, with the appearance of two crowns that share the same root canal. Complete cleavage, or twinning, occasionally occurs, resulting in two teeth from one tooth germ.

Although trauma has been suggested as a possible cause, the cause of gemination is unknown. These teeth may be cosmetically unacceptable and may cause crowding.

2-Fusion

is the joining of two developing tooth germs, resulting in a single large tooth structure. The fusion process may involve the entire length of the teeth, or it may involve the roots only, in which case cementum and dentin are shared. Root canals may also be separate or shared. It may be impossible to differentiate fusion of normal and supernumerary teeth from gemination. The cause of this condition is unknown, although trauma has been suggested. The etiology of double teeth remains unclear. A genetic basis has been suggested also double teeth should be differentiated from concrescence which is an acquired condition.

Concrescence:

Concrescence is an acquired union form in which adjacent, already formed teeth are joined by cementum only. This may take place before or after eruption of teeth and is believed to be related to trauma or overcrowding. Mostly seen in permanent than primary dentition. Concrescence is most commonly seen in association with the maxillary second and third molars. This condition is of no significance, unless one of the teeth involved requires extraction. Surgical sectioning may be required to save the other tooth.

ACCESSORY CUSPS

The cuspal morphology of teeth exhibits minor variations among different populations; of these. Three distinctive patterns deserve further discussion:

(1) Cusp of Carabelli.

(2) Talon cusp.

When an accessory cusp is present, the other permanent teeth often exhibit a slightly increased tooth size.

Clinical and Radiographic Features

1-The cusp of Carabelli is an accessory cusp located on the palatal surface of the mesio lingual cusp of a maxillary molar. The cusp may be seen in the permanent or deciduous dentitions and varies from a definite cusp to a small indented pit or fissure. When present. The cusp is most pronounced on the first molar.

2-*Talon cusp* A talon cusp (dens evaginatus of anterior tooth) is a well-delineated additional cusp that is located on the surface of an anterior tooth and extends at least half the distance from the cementoenamel junction to the incisal edge. Three fourths of all reported talon cusps are located in the permanent dentition.

Odontoma like lesions:

1-Dens Invaginatus : Also known as dens in dente or tooth within a tooth arises as a result of invagination of a portion of enamel organ into the dental papilla at an early stage in odontogenesis before the formation of calcified tissue, dens invaginatus is an uncommon tooth anomaly that represents an exaggeration or accentuation of the lingual pit. This defect ranges in severity from superficial, in which only the crown is affected, to deep, in which both the crown and the root are involved. The permanent maxillary lateral incisors are most commonly involved, although any anterior tooth may be affected. Bilateral involvement is commonly seen. The cause of this developmental condition is unknown. Genetic factors are believed to be involved in only a small percentage of cases.

Because the defect cannot be kept free of plaque and bacteria, dens invaginatus predisposes the tooth to early decay and subsequent pulpitis. Prophylactic filling of the pit is recommended to avoid this complication. Because the defect may often be identified on radiographic examination before tooth eruption, the patient can be

prepared in advance of the procedure. In cases in which pulpitis has led to non-vitality, endodontic procedures may salvage the affected tooth.

2-Dens Evaginatus :- is a relatively common developmental condition affecting predominantly premolar teeth (Leung's premolars). It has been reported almost exclusively in Asians, Inuits, and Native Americans. The defect, which is often bilateral, is an anomalous tubercle, or cusp, located at the center of the occlusal surface. Because of occlusal abrasion, the tubercle wears relatively quickly causing early exposure of an accessory pulp horn that extends into the tubercle. This may result in periapical pathology in young, caries-free teeth, often before completion of root development and apical closure, making root canal fillings more difficult. Judicious grinding of the opposing tooth or the accessory tubercle to stimulate secondary dentin formation may prevent the periapical sequelae associated with this defect. Sealants, pulp capping, and partial pulpotomy have been suggested as measures to allow complete root development.

3-Enamel Pearls (enameloma): Droplets of ectopic enamel, or so-called enamel pearls, may occasionally be found on the roots of teeth. They occur most commonly in the bifurcation or trifurcation of teeth but may appear on single-rooted premolar teeth as well. Maxillary molars are more commonly affected than mandibular molars. These deposits are occasionally supported by dentin and rarely may have a pulp horn extending into them. This developmental disturbance of enamel formation may be detected on radiographic examination. It generally is of little significance except when located in an area of periodontal disease. In such cases, it may contribute to the extension of a periodontal pocket, because a periodontal ligament attachment would not be expected and hygiene would be more difficult.

Taurodontism

Taurodontism is a variation in tooth form in which teeth have elongated crowns or apically displaced furcations, resulting in pulp chambers that have increased apical-occlusal height. Because this abnormality resembles teeth in bulls and other ungulates, the term *taurodontism* was coined. Various degrees of severity may be seen, but subclassifications that have been developed to describe them appear to be of academic interest only.

Taurodontism may be seen as an isolated incident, in families, and in association with syndromes such as Down syndrome and Klinefelter's syndrome; it was also seen in the now-extinct Neanderthals. Although taurodontism is generally an uncommon finding, it has been reported to have a relatively high prevalence in Eskimos, and incidence has been reported to be as high as 11% in a Middle Eastern population. Other than a possible relationship to other genetically determined abnormalities, taurodontism is of little clinical significance unless the tooth becomes nonvital, in which case it becomes a challenging endodontic problem. No treatment is required.

Dilaceration

A term used to describe a deformity in which the crown is displaced from its normal alignment with the root, so that the tooth is severely bent along its long axis. The cause of this condition has been related to trauma during root development. Movement of the crown or of the crown and part of the root from the remaining developing root may result in sharp angulation after the tooth completes development. Hereditary factors are believed to be involved in a small number of cases. Eruption generally continues without problems. However, extraction may be difficult. Obviously, if root canal fillings are required in these teeth, the procedure is challenging.

Supernumerary Roots

Accessory roots are most commonly seen in mandibular canines, premolars, and molars (especially third molars). They are rarely found in upper anterior teeth and mandibular

incisors. Radiographic recognition of an extraordinary number of roots becomes important when extractions or root canal fillings are necessary.

Developmental Alterations in the Structure of Teeth

Amelogenesis Imperfecta

It is a clinically and genetically heterogeneous group of disorders of enamel formation that affect both dentitions.

Most cases of amelogenesis imperfecta fall into one of two clinical types: ***hypoplastic*** or ***hypocalcified***. A third type, known as ***hypomaturation***, has been added to the list. Numerous subtypes of the three major groups are also recognized; these are based on different inheritance patterns, clinical appearances, and radiographic features.

Several genes that are involved in enamel formation (amelogenin, enamelin, kallikrein 4, MMP20, others) are mutated in various forms of this condition.

In the ***hypoplastic*** type of amelogenesis imperfecta, teeth erupt with insufficient amounts of enamel, ranging from pits and grooves in one patient to complete absence (aplasia) in another. Because of reduced enamel thickness in some cases, abnormal contour and absent interproximal contact points may be evident. In the ***hypocalcified*** type, the quantity of enamel is normal, but it is soft and friable, so that it fractures and wears readily. The color of the teeth varies from tooth to tooth and from patient to patient—from white opaque to yellow to brown. Teeth also tend to darken with age as a result of exogenous staining.

Radiographically, enamel appears reduced in bulk, often showing a thin layer over occlusal and interproximal surfaces. Dentin and pulp chambers appear normal. Although the enamel is soft and irregular, teeth are not caries prone.

Treatment focuses on esthetics and protection of tooth tissue. Restorative dental procedures at an early age not only preserve teeth but have a significant effect on the patient’s self-esteem.

DEFECTS OF DENTIN

Dentinogenesis Imperfecta

It is an autosomal-dominant trait with variable expressivity. Mutations in the dentin sialo phosphoprotein gene have been described. It typically affects the dentin of both primary and permanent dentitions. Because of the clinical discoloration of teeth, this condition has also been known as (hereditary) ***opalescent dentin.***

Dentinogenesis imperfecta has been divided into three types.

1-type I or syndrome-associated, in which the dentin abnormality occurs in patients with concurrent osteogenesis imperfecta, primary teeth are more severely affected than permanent teeth.

2- type II, patients have only dentin abnormalities and no bone disease.

3-type III or the Brandywine type, only dental defects occur. This type is similar to type II, but has some clinical and radiographic variations.

Features of type III that are not seen in type I and II include multiple pulp exposures, periapical radiolucencies, and a variable radiographic appearance.

Dentinogenesis imperfecta type I (syndromal dentinogenesis imperfecta) is caused by mutations in the genes that encode collagen type I. Dentinogenesis imperfecta types II and III, on the other hand, have been shown to be related to mutations in a gene known as dentin sialophosphoprotein that encodes noncollagen proteins of dentin. Other genes that encode dentin proteins, such as osteopontin, do not appear to be mutated in dentinogenesis imperfecta.

Clinically

All three types share numerous features. In both dentitions, the teeth exhibit an unusual translucent, opalescent appearance, with color variation from yellow-brown to gray. The entire crown appears discolored because of the abnormal underlying dentin. Although the enamel is structurally and chemically normal, it fractures easily, resulting in rapid wear. The enamel fracturing is believed to be due to the poor support provided by abnormal dentin, and possibly in part to the absence of the microscopic scalloping

normally seen between dentin and enamel, which is believed to help mechanically lock the two hard tissues together. Overall tooth morphology is unusual for its excessive constriction at the cement-enamel junction, giving the crowns a tulip or bell shape. Roots are shortened and blunted. The teeth do not exhibit any greater susceptibility to caries, and they may in fact show some resistance because of the rapid wear and absence of interdental contacts.

Radiographically

Types I and II exhibit identical changes. Opacification of dental pulps occurs as the result of continued deposition of abnormal dentin. The short roots and the bell-shaped crowns are also obvious on radiographic examination. In type III, the dentin appears thin and the pulp chambers and root canals extremely large, giving the appearance of thin dentin shells—hence the previous designation of ***shell teeth*.**

Microscopically

The dentin of teeth in dentinogenesis imperfecta contains fewer, but larger and irregular, dentinal tubules. The pulpal space is nearly completely replaced over time by irregular dentin. Enamel appears normal, but the dentinoenamel junction is smooth instead of scalloped.

Treatment

Directed toward protecting tooth tissue from wear and toward, thereby improving the esthetic appearance of the teeth. Generally, fitting with full crowns at an early age is the treatment of choice. Despite the qualitatively poor dentin, support for the crowns is adequate. These teeth should not be used as abutments because the roots are prone to fracture under stress.

Dentin Dysplasia

Dentin dysplasia, subdivided into types I and II, is another autosomal-dominant condition that affects dentin. The incidence of this rare disorder is approximately 10 times less than that of dentinogenesis imperfecta. As in dentinogenesis imperfecta II and III, genetic mutations occur in the dentin sialophosphoprotein gene in dentin dysplasia type II. Genetic lesions have yet to be elucidated in dentin dysplasia type I.

In dentin dysplasia type II, the color of the primary dentition is opalescent and the permanent dentition is normal; in type I, both dentitions are of normal color. The coronal pulps in type II are usually large (thistle tube appearance) and are filled with globules of abnormal dentin. Also, periapical lesions are not a regular feature of type II, as they are of type I.

Clinically

The crowns in dentin dysplasia type I appear to be normal in color and shape. Premature tooth loss may occur because of short roots or periapical inflammatory lesions. Teeth show greater resistance to caries when compared with normal teeth.

Radiographically

In dentin dysplasia type I, roots appear extremely short and pulps are almost completely obliterated. Residual fragments of pulp tissue appear typically as horizontal lucencies (chevrons). Periapical lucencies are typically seen; they represent chronic abscesses, granulomas, or cysts. In dentin dysplasia type II, deciduous teeth are similar in radiographic appearance to those in type I, but permanent teeth exhibit enlarged pulp chambers that have been described as thistle tube in appearance.

Microscopically

The enamel and the immediately subjacent dentin appear normal. Deeper layers of dentin show atypical tubular patterns, with amorphous, a tubular areas and irregular organization. On the pulpal side of the normal-appearing mantle of dentin, globular or nodular masses of abnormal dentin are seen.

Treatment

Directed toward retention of teeth for as long as possible. However, because of the short roots and periapical lesions, the prognosis for prolonged retention is poor. This dental condition has not been associated with any systemic connective tissue problems.

DEFECTS OF ENAMEL AND DENTIN

Regional Odontodysplasia

Regional odontodysplasia is a dental abnormality that involves the hard tissues derived from both epithelial (enamel) and mesenchymal (dentin and cementum) components of the tooth-forming apparatus. The teeth in a region or quadrant of the maxilla or mandible are affected to the extent that they exhibit short roots, open apical foramina, and enlarged pulp chambers. The thinness and poor mineralization quality of the enamel and dentin layers have given rise to the term ***ghost teeth***. One or both dentitions may be affected. The permanent teeth are more affected than the primary teeth, and the maxillary anterior teeth are more affected than other teeth. Eruption of the affected teeth is delayed or does not occur.

The cause of this rare dental abnormality is unknown, although numerous causative factors have been suggested, including trauma, nutritional deficiency, infection, metabolic abnormality, systemic disease, local vascular compromise, and genetic influences. Because of the poor quality of the affected teeth, their removal is usually indicated. The resulting edentulous zone can then be restored with a prosthesis or implant.

ENVIRONMENTAL EFFECTS ON TOOTH STRUCTURE DEVELOPMENT

DEFECTS OF ENAMEL

Environmental Defects of Enamel

During enamel formation, ameloblasts are susceptible to various external factors that may be reflected in erupted teeth. Metabolic injury, if severe enough and long enough, can cause defects in the quantity and shape of enamel or in the quality and color of enamel. Quantitatively defective enamel, when of normal hardness, is known as ***enamel hypoplasia***. Qualitatively defective enamel, in which normal amounts of enamel are produced but are hypomineralized, is known as ***enamel hypocalcification.*** In this defect, the enamel is softer than normal. The extent of the enamel defect is dependent on three conditions:

1) The intensity of the causative factor.
2) The duration of the factor's presence.
3) The time at which the factor occurs during crown development. Factors that lead to ameloblast damage are highly varied, although the clinical signs of defective enamel are the same.

Causative factors may occur locally, affecting only a single tooth, or they may act systemically, affecting all teeth in which enamel is being formed. Local trauma or abscess formation can adversely affect the ameloblasts overlying a developing crown, resulting in enamel hypocalcification or hypoplasia. Affected teeth may have areas of coronal discoloration, or they may have actual pits and irregularities. This is most commonly seen in permanent teeth in which the overlying deciduous tooth becomes abscessed or is physically forced into the enamel organ of the permanent tooth. The resulting hypoplastic or hypocalcified permanent tooth is sometimes known as ***Turner's tooth*** (Turners hypoplasia).

Hypoplasia caused by antineoplastic therapy. As modern medicine increases its prevalence of successful therapy against childhood cancer. it has become evident that a number of developmental alterations arise secondary to use of therapeutic radiation or chemotherapy. As would be expected. developing teeth are affected most severely. With these therapies producing clinically obvious alterations most commonly in patients under the age of 12 and most extensively in those under 5 years of age. The degree and severity of the developmental alterations are related to the patient's age at treatment. Form of therapy and the dose and field of radiation .

Dental fluorosis. The ingestion of excess amounts of fluoride also can result in significant enamel defects known as dental fluorosis. Although the fluoride produced an unusual permanent dental stain, a resistance to caries also was noted. it was discovered that fluoride in the water at 1.0 ppm reduced caries by 50% to 70%. In

addition, this level of fluoride in the water supply was associated with a low and mostly mild prevalence of mottled enamel.

Syphilitic hypoplasia. Congenital syphilis results in a pattern of enamel hypoplasia that is well known but currently so rare that lengthy discussion is not warranted. Anterior teeth altered by syphilis are termed **Hutchinson's incisors** and exhibit crowns that are shaped like straight-edge screw drivers. With the greatest circumference present in the middle one third of the crown and a constricted incisal edge. The middle portion of the incisal edge often demonstrates a central hypoplastic notch. Altered posterior teeth are termed **mulberry molars** and demonstrate constricted occlusal tables with a disorganized surface anatomy that resembles the bumpy surface of a mulberry.

Post developmental loss of tooth structure (enamel, dentin and cementum)

Attrition, Abrasion, Erosion and Abfraction

Attrition is the physiologic wearing of teeth as a result of mastication. It is an age-related process that varies from one individual to another. Factors such as diet, dentition, jaw musculature, and chewing habits can significantly influence the pattern and extent of attrition.

Abrasion is the pathologic wearing of teeth caused by an abnormal habit or abnormal use of abrasive substances orally. Pipe smoking, tobacco chewing, aggressive tooth brushing, and use of abrasive dentifrices are among the more common causes. The location and pattern of abrasion are directly dependent on the cause, with so-called toothbrush abrasion along the cementoenamel junction an easily recognized pattern.

Erosion is the loss of tooth structure through a nonbacterial chemical process. Most commonly, acids are involved in the dissolution process from an external or an internal source. Externally, acid may be found in the work environment (e.g., battery manufacturing) or in the diet (e.g., citrus fruits, acid-containing soft drinks). The internal source of acid is most probably regurgitation of gastric contents. This may be seen in any disorder of which chronic vomiting is a part. Self-induced vomiting, as a component of bulimia or, less commonly, anorexia nervosa, has become an increasingly important

cause of dental erosion and other oral abnormalities. The pattern of erosion associated with vomiting is usually generalized tooth loss on the lingual surfaces of maxillary teeth. However, all surfaces may be affected, especially in individuals who compensate for fluid loss by excessive intake of fruit juices. In many cases of tooth erosion, no cause is found.

Abfraction loss of tooth from occlusal stress that create repeated tooth flexure with failure of enamel and dentin at a location away from the point of locking, it appears as wedge-shaped defects limited to the cervical area of the teeth and may closely resemble cervical abrasion or erosion. Clues to the diagnosis include defects that are deep, narrow, and V-shaped (which do not allow the tooth brush to contact the base of the defect) and often affect a single tooth with adjacent unaffected teeth. In addition, occasional lesions are subgingival, a site typically protected from abrasion and erosion. The lesions are seen almost exclusively on the facial surface and exhibit a much greater prevalence in those with bruxism. A higher frequency is noted in the mandibular dentition, presumably because the lingual or indentation makes them more susceptible to the concentration of tensile stresses at the cervical regions.

Internal and External Resorption

Internal Resorption

Resorption of the dentin of the pulpal walls may be seen as part of an inflammatory response to pulpal injury, or it may be seen in cases in which no apparent trigger can be identified. The resorption occurs as a result of activation of osteoclasts or dentinoclasts on internal surfaces of the root or crown. Resorption lacunae containing these cells and chronic inflammatory cells are seen. Reversal lines may also be found in adjacent hard tissue, indicating attempts at repair. In time, the root or crown is perforated by the process, making the tooth useless.

Any tooth may be involved, and usually only a single tooth is affected, although cases in which more than one tooth is involved have been described. In advanced cases, teeth

may appear pink because of the proximity of pulp tissue to the tooth surface. Until root fracture or communication with a periodontal pocket occurs, patients generally have no symptoms.

The treatment of choice is root canal therapy before perforation. Once communication between pulp and periodontal ligament occurs, the prognosis for saving the tooth is very poor. Occasionally, the process may spontaneously stop for no apparent reason.

External Resorption

Resorption of teeth from external surfaces may have one of several causes. This change may be the result of an adjacent pathologic process, such as (1) chronic inflammatory lesions, (2) cysts, (3) benign tumors, or (4) malignant neoplasms. The pathogenesis of external resorption from these causes has been related to the release of chemical mediators, increased vascularity, and pressure. External resorption of teeth may also be seen in association with (1) trauma, (2) reimplantation or transplantation of teeth, or (3) impaction. Trauma that causes injury to or necrosis of the periodontal ligament may initiate resorption of tooth roots. This trauma may result from a single event, from malocclusion, or from excessive orthodontic forces. Because reimplanted and transplanted teeth are nonvital and have no surrounding viable periodontal ligament, they eventually are resorbed and replaced by bone. This is basically a natural physiologic process in which the calcified collagen matrix of the tooth serves as a framework for the deposition of new, viable bone. Impacted teeth, when they impinge or exert pressure on adjacent teeth, may cause root resorption of the otherwise normally erupted tooth. Impacted teeth themselves occasionally may undergo resorption. The cause of this phenomenon is unknown, although it is believed to be related to partial loss of the protective effect of the periodontal ligament or reduced enamel epithelium.

Finally, external resorption of erupted teeth may be idiopathic. This may occur in one or more teeth. Any tooth may be involved, although molars are least likely to be affected.

External resorption eventually causes loss of the affected teeth.

Environmental Discoloration of Teeth

Exogenous Stains

Stains on the surfaces of teeth that can be removed with abrasives are known as *exogenous* or *extrinsic stains*. The color change may be caused by pigments in dietary substances (e.g., coffee, "betel" areca nut, tobacco) or by the colored bi-products of chromogenic bacteria in dental plaque. Chromogenic bacteria are believed to be responsible for brown, black, green, and orange stains observed predominantly in children. Brown and black stains typically are seen in the cervical zone of teeth, either as a thin line along the gingival margin or as a wide band. This type of stain is also often found on teeth adjacent to salivary duct orifices. Green stain is tenacious and usually is found as a band on the labial surfaces of the maxillary anterior teeth. Blood pigments are thought to contribute to the green color. Orange or yellow-orange stains appear on the gingival third of teeth in a small percentage of children. These generally are easily removed.

Endogenous Stains

Discoloration of teeth resulting from deposits of systemically circulating substances during tooth development is defined as *endogenous* or *intrinsic staining*.

Systemic ingestion of *tetracycline* during tooth development is a well-known cause of endogenous staining of teeth. Tetracycline binds calcium and therefore is deposited in developing teeth and bones. The bright yellow color of the drug is reflected in subsequently erupted teeth. The fluorescent property of tetracycline can be demonstrated with an ultraviolet light in clinically erupted teeth. Over time, the tetracycline oxidizes, resulting in a change from yellow to gray or brown with loss of its fluorescent quality. Because tetracycline can cross the placenta, it may stain primary teeth if taken during pregnancy. If it is administered between birth and age 6 or 7 years, permanent teeth may be affected. Only a small minority of children given tetracycline for various bacterial diseases, however, exhibit clinical evidence of discoloration.

Staining is directly proportional to the age at which the drug is administered and the dose and duration of drug usage.

The significance of tetracycline staining lies in its cosmetically objectionable appearance. Because other, equally effective antibiotics are available, tetracycline should not be prescribed for children younger than 7 years except in unusual circumstances.

It should be noted that minocycline, a semisynthetic derivative of tetracycline, can stain the roots of adult teeth. It also may stain skin and mucosa in a diffuse or patchy pattern.

Rh incompatibility (erythroblastosis fetalis) has been cited as a cause of endogenous staining in primary teeth. Because of red blood cell hemolysis resulting from maternal antibody destruction of fetal red blood cells, blood breakdown products (bilirubin) are deposited in developing primary teeth. The teeth appear green to brown. Treatment is not required because only primary teeth are affected.

Congenital porphyria, one of several inborn errors of porphyrin metabolism, is also a potential cause of endogenous pigmentation. This autosomal-recessive trait is associated with photosensitivity, vesiculobullous skin eruptions, red urine, and splenomegaly. Teeth may appear red to brown because of deposition of porphyrin in the developing teeth. Affected teeth fluoresce red with ultraviolet light.

Liver disease, ***biliary atresia***, and ***neonatal hepatitis*** may produce discoloration of the primary dentition. In biliary atresia, the teeth may assume a green discoloration; a yellowish-brown color is noted in cases of neonatal hepatitis. This is a result of the deposition or incorporation of bilirubin in developing enamel and dentin.