Developmental alterations of teeth

4. Developmental alterations in the structure of the teeth

Defects of Enamel

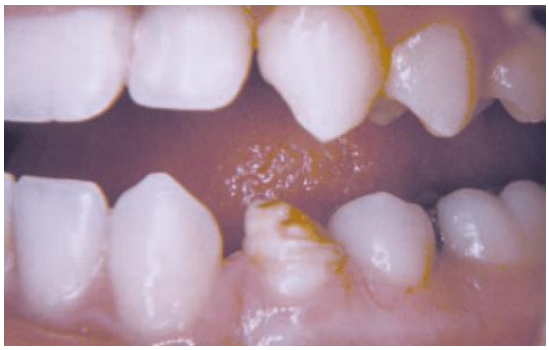
During enamel formation, ameloblast cells (cells which are responsible for the formation of enamel) are susceptible to various external factors that may be reflected in erupted teeth. Sever and long duration metabolic injury can cause defects in the (quantity and shape) of enamel or in the (quality and 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* (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.

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. For systemic factors to have an effect on developing permanent teeth, they generally must occur after

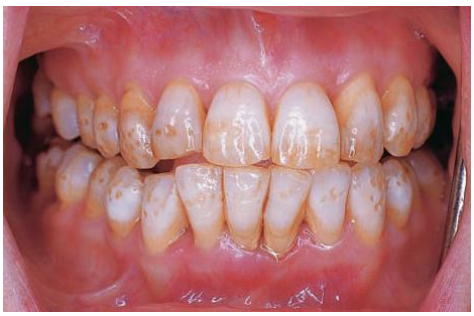
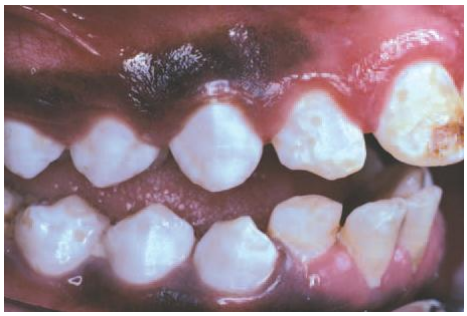
birth and before the age of 6 years. During this time, the crowns of all permanent teeth (with the exception of the third molars) develop.



Turner's hypoplasia. Extensive enamel hypoplasia of mandibular first premolar secondary to previous inflammatory process associated with overlying first deciduous molar

There are many causes of systemically induced enamel defects:

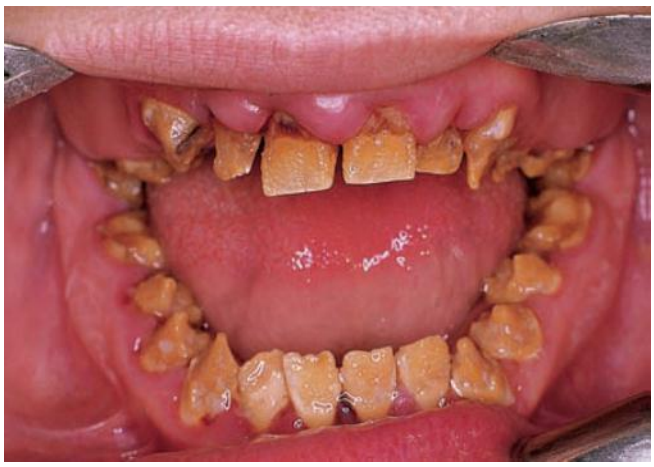
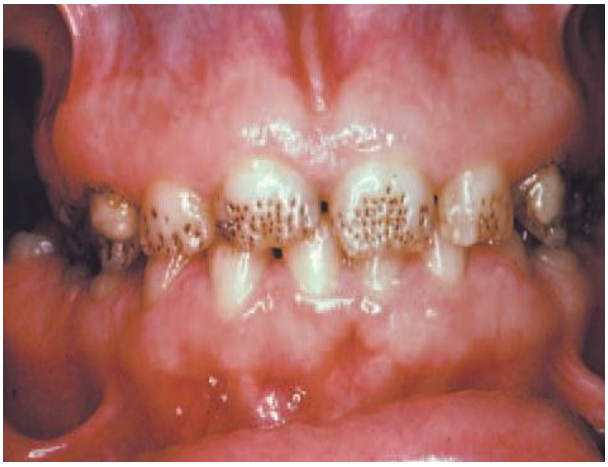
1. Infectious diseases
2. Nutritional defects such as rickets
3. Congenital syphilis
4. Birth trauma (neonatal line in primary teeth)
5. Fluoride: Ingestion of drinking water containing fluoride at levels greater than 1 part per million during the time crowns are being formed may result in enamel hypoplasia or hypocalcification, also known as dental fluorosis. Endemic fluorosis is known to occur in areas where the drinking water contains excessive naturally occurring fluoride. As with other causative agents, the extent of damage is dependent on duration, timing, and intensity or concentration. Mild to moderate fluorosis ranges clinically from white enamel spots to mottled brown-and-white discolorations. Severe fluorosis appears as pitted, irregular, and discolored enamel. Although fluoride-induced enamel hypoplasia or hypocalcification is caries resistant, it may be cosmetically objectionable, making esthetic dental restorations desirable.
6. Idiopathic factor

Dental fluorosis

Amelogenesis Imperfecta

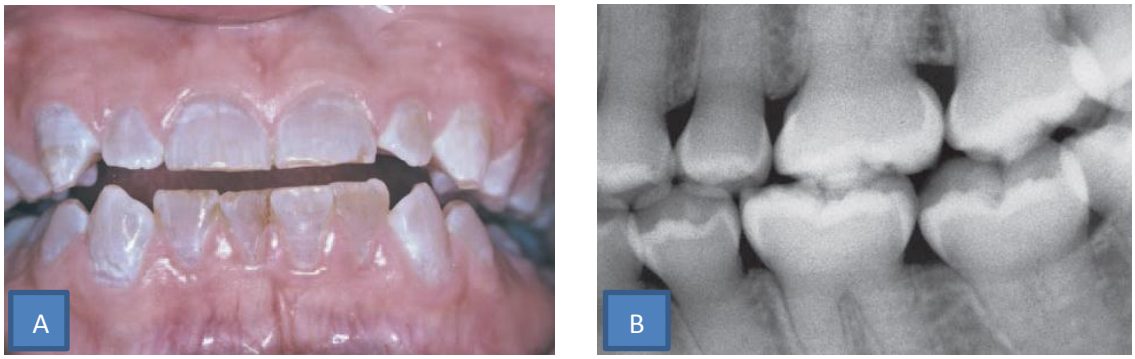
Amelogenesis imperfecta is a clinically and genetically heterogeneous group of disorders of enamel formation that affect both dentitions. Most cases of amelogenesis imperfecta fall into one of two clinical types: hypoplastic or hypocalcified. A third type, known as hypomaturation, has been added to the list. Numerous subtypes of the three major groups are also recognized; these are based on different inheritance patterns, clinical appearances, and radiographic features. Most cases of amelogenesis imperfecta are inherited as an autosomal dominant trait.

Defects of Dentin

1. Dentinogenesis Imperfecta

Dentinogenesis imperfecta is an autosomal-dominant trait. 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. In 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. In type II, patients have only dentin abnormalities and no bone disease. In type III, or the Brandywine type (discovered in a triracial population in Brandywine, Maryland), only dental defects occur. Features of type III that are not seen in types I and II include multiple pulp exposures, periapical radiolucencies.



Dentinogenesis imperfecta. A. Dentition exhibiting diffuse brownish discoloration and slight translucence B. Radiograph of dentition exhibiting bulbous crowns, cervical constriction, and obliterated pulp canals and chambers.

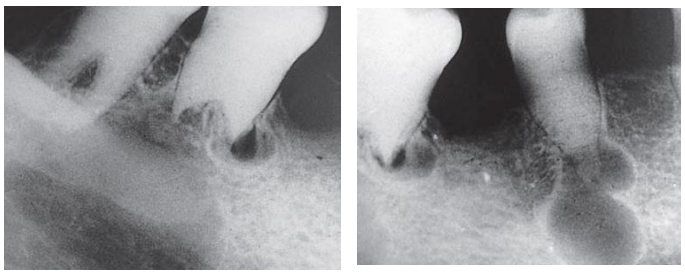
Clinically, all three types share numerous features. In both dentitions, the teeth exhibit an unusual translucent, with color variation from yellow-brown to gray. The entire crown appears discolored because of the abnormal underlying dentin. Although the enamel is structurally and chemically normal, it fractures easily, resulting in rapid wear. The enamel

fracturing is believed to be due to the poor support provided by abnormal dentin. Overall tooth morphology is unusual for its excessive constriction at the cementoenamel 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.

Treatment is directed toward protecting tooth tissue from wear and tear, 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 less frequent than Dentinogenesis imperfecta. 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. Radiographically: the roots appear extremely short and pulps are almost completely obliterated. Periapical lucencies are typically seen; they represent chronic abscesses, granulomas, or cysts. In dentin dysplasia type II the color of primary dentition is opalescent but the permanent teeth are of normal color. The coronal pulp is usually enlarged and filled with globules (stone) of abnormal dentine. Periapical lesions are not always



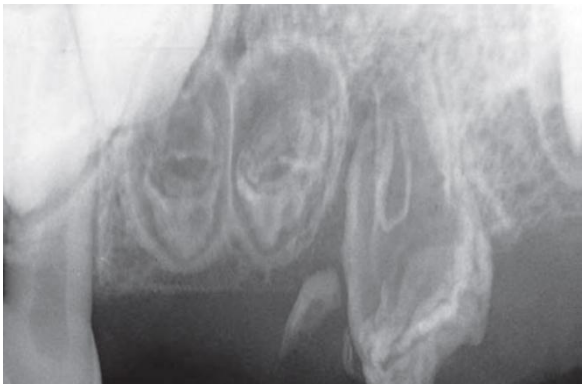
Dentin dysplasia type I. Note obliterated pulps, short roots, and periapical lesions

Treatment is 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. Eruption of the affected teeth is delayed or does not occur. trauma, nutritional deficiency, infection, metabolic abnormality, systemic disease, local vascular compromise, and genetic influences are suggested causes of this abnormality. 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.

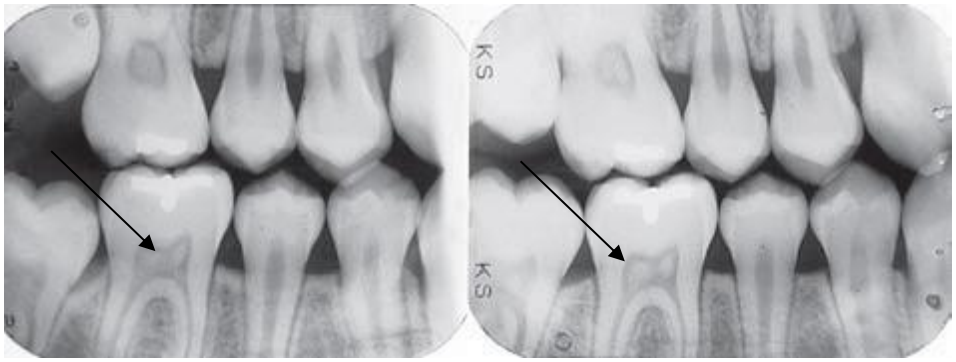


Ghost teeth

Abnormalities of Dental Pulp

Pulp Calcification

Pulp calcification is a rather common phenomenon that occurs with increasing age for no apparent reason. There appears to be no relation to inflammation, trauma, or systemic disease. Pulp calcification may be of microscopic size or may be large enough to be detected radiographically. Calcifications may be diffuse (linear) or nodular (pulp stones). The diffuse, or linear, deposits are typically found in the root canals and generally are parallel to the blood vessels. Pulp stones usually are found in the pulp chamber. When they are composed predominantly of dentin, they are referred to as true denticles; when they represent foci of dystrophic calcification, they are referred to as false denticles. Pulp stones occasionally are subdivided into attached and free types, depending on whether they are incorporated into the dentin wall or are surrounded by pulpal tissue. Pulp stones appear to have no clinical significance. They are not believed to be a source of pain and are not associated with any form of pulpitis. They may, however, be problematic during endodontic therapy of nonvital teeth.



Pulp calcification

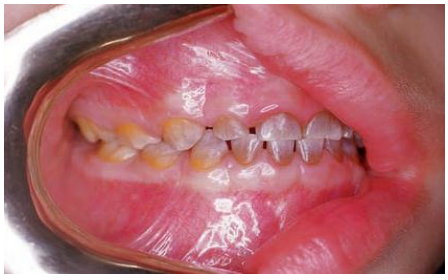
Alterations in Color of teeth

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 substances in the diet (e.g., coffee, tea, wine) or associated with habits (e.g., "betel" areca nut, tobacco products). Colored byproducts of chromogenic bacteria in dental plaque may also cause exogenous staining. Chromogenic bacteria are believed to be responsible for brown, black, green, and orange stains observed predominantly in children. Blood pigments are thought to contribute to the green color.

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. The significance of tetracycline staining lies in its cosmetically objectionable appearance. tetracycline should not be prescribed for children younger than 7 years.



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. Liver disease, biliary atresia, and neonatal hepatitis may produce discoloration of the primary dentition. 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.