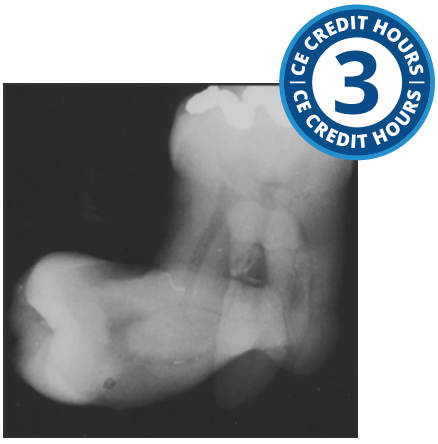
Anomalies of Tooth Structure



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Conflict of Interest Disclosure Statement

- Prof. Williamson reports no conflicts of interest associated with this course. She has no relevant financial relationships to disclose.

Short Description – Anomalies of Tooth Structure

Anomalies of Tooth Structure is a free dental continuing education course that covers a wide range of topics relevant to the oral healthcare professional community.

Course Contents

Overview

This course will present the radiographic appearance of the various components of tooth structure as well as the development and eruption of the primary and permanent dentitions. Subsequently, dental anomalies associated with the eruption, number, size, and shape of the teeth along with acquired alterations of tooth structure will be discussed. This information will facilitate differentiation between normal and abnormal tooth structure and enhance the ability to recognize and describe various tooth related anomalies.

This course will focus on the normal radiographic appearance of the teeth as viewed on intraoral and panoramic radiographic images followed by the presentation of anomalies associated with the teeth. Recognition of the normal radiographic appearance of the teeth serves as a baseline to differentiate normal from abnormal. Anomalies or alterations of the teeth can be congenital, developmental, or acquired and include a variety of changes associated with the eruption, number, size, and shape of the teeth as well as acquired variations.

Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Recognize and describe the radiographic appearance of healthy tooth structure in both the primary and permanent dentitions including eruption patterns.
- Recognize and describe the radiographic appearance of healthy supporting structures of the teeth.
- Describe the various origins of dental anomalies; congenital, developmental, and acquired.
- Recognize and describe processes that can interfere with normal tooth eruption.
- Identify and classify tooth impactions by their position.
- Recognize and describe processes that can cause loss of tooth structure.
- Discuss and identify alterations in tooth structure and the dental pulp.
- Identify and describe alterations in the number of teeth.
- Recognize and discuss alterations in the size of teeth.
- Identify and describe alterations in the shape of teeth.
- Recognize and discuss developmental alterations in the structure of teeth; amelogenesis imperfecta, dentinogenesis imperfecta, and dentin dysplasia.
- Recognize and describe processes that can cause loss of tooth structure.
- Given an intraoral radiograph, radiographic survey and/or a panoramic image, identify and describe anomalies that may be recorded on those images.

Introduction

Fundamental to basic radiographic interpretation of intraoral and panoramic radiographic images is the ability to recognize normal tooth structure and how teeth appear radiographically during various stages of growth and development. This discussion will begin with a review of the various components of tooth structure and the supporting structures of the teeth, followed by presentation of tooth structure anomalies.

Normal Radiographic Appearance of Teeth

Enamel, Dentin, Cementum, Pulp

The teeth are comprised of four basic anatomic structures: enamel, dentin, cementum, and the pulp (Figure 1). The enamel is the layer of mineralized tissue covering the crown of the tooth, which has the highest level of calcified content, approximately 95%.[1] As a result, the enamel appears highly radiopaque (white) on dental images. Underlying the dense enamel layer is the dentin. The dentin, less calcified than enamel with about 75% mineralized content, composes the majority of the tooth.[1] It appears less radiopaque than the enamel due to its lesser mineral content. The dentinoenamel junction (DEJ) is the distinct interface between the enamel and dentin in the coronal aspect of the tooth, while the cementoenamel junction (CEJ) is the interface at the cervical region of the tooth where the crown intersects with the root(s). The cementum covers the root of the tooth but cannot be distinguished from dentin radiographically.[2] Finally, the pulp, located in the center of the tooth, contains the nerve and blood supply. The pulp cavity consists of the pulp chamber and pulp canal(s). The canals are narrow linear structures that extend from the broader pulp chamber and terminate at the end of the root(s). Pulpal anatomy varies, appearing more linear in anterior teeth, while in posterior teeth, the pulp appears like a miniature tooth within the tooth itself. As a non-calcified structure, the pulp appears radiolucent (black) on radiographic images. The size of the dental pulp varies with maturity, appearing larger in children (Figure 2) and more diminished in adults as they age.[3]

Normal Radiographic Appearance of the Supporting Structures of the Teeth

Cancellous Bone

The maxilla and mandible are composed of cancellous or trabecular bone located between the dense cortical outer plates. The cancellous bone consists of trabeculae, thin bony rods, and plates, surrounded by areas of bone marrow, presenting a mixed radiopaque and radiolucent radiographic appearance.[2] The bone pattern varies among individuals as well as in the different regions of the dental arches.[3] The maxilla, especially in the anterior region, has numerous thin trabeculae with many small marrow spaces, displaying a fine granular appearance radiographically.[2] The posterior maxilla demonstrates a similar pattern with larger marrow spaces (Figures 3, 4).[2] In comparison, the anterior mandible shows thicker trabeculae with a coarser horizontal pattern with fewer plates and larger marrow spaces than the maxilla.[2] The posterior regions

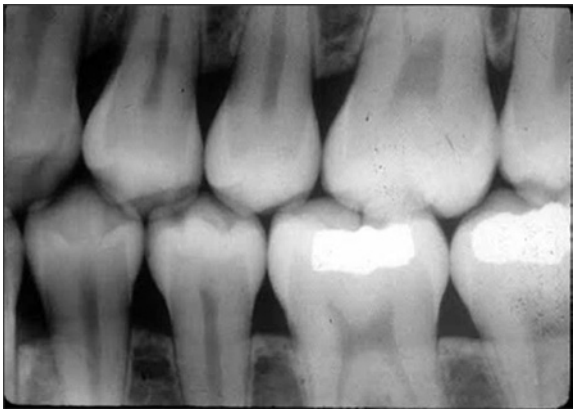


Figure 1. Bitewing radiograph displaying enamel, dentin, and pulp structures.

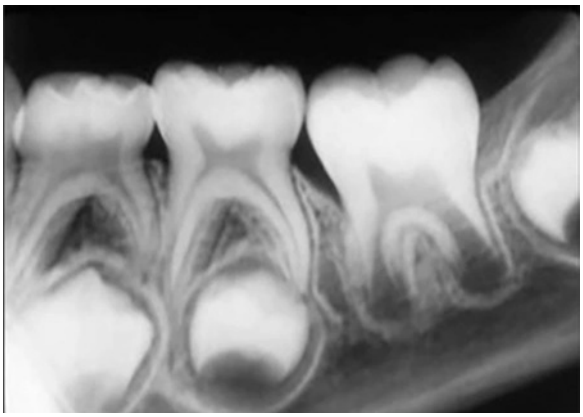


Figure 2. Periapical radiograph of posterior primary and developing permanent teeth.

of the mandible exhibit a pattern similar to the anterior region, but the trabeculae and marrow spaces tend to be larger and sparser (Figures 5, 6).[2] A ladder-like effect may be visualized due to the large size of the posterior marrow spaces. Apical to the molar teeth, the trabeculae may be very sparse or appear to be absent, resulting in a very radiolucent radiographic appearance which may be misinterpreted as pathologic. (Figure 7)[2]

Alveolar Bone and Alveolar Crest

The alveolar bone is the teeth-bearing bony process of the jaws. The alveolar crest is the gingival margin of the alveolar process and extends between and around the tooth root(s).[4,5] The crest is covered by a thin layer of cortical bone, appearing radiopaque radiographically (Figure 7).[3,4] The crestal alveolar bone level ranges from 0.5 to 2.0 millimeters from the CEJ, in health, and follows the plane of adjacent CEJs.[4] The shape of the crestal bone varies from narrow and pointed between the anterior teeth and flat and angular between the posterior teeth.[3] The crestal shape is dictated by the space or distance between the teeth.[2]

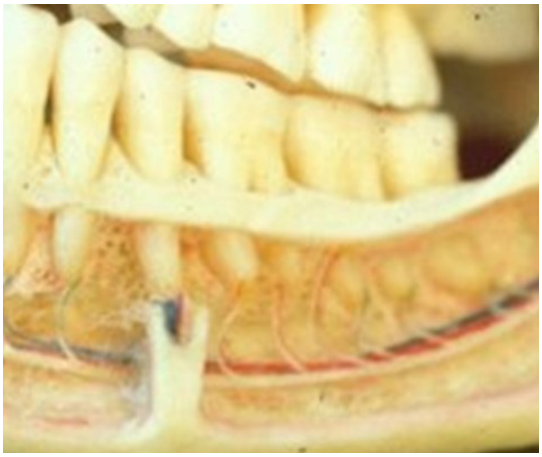


Figure 5. Mandibular posterior cancellous bone.

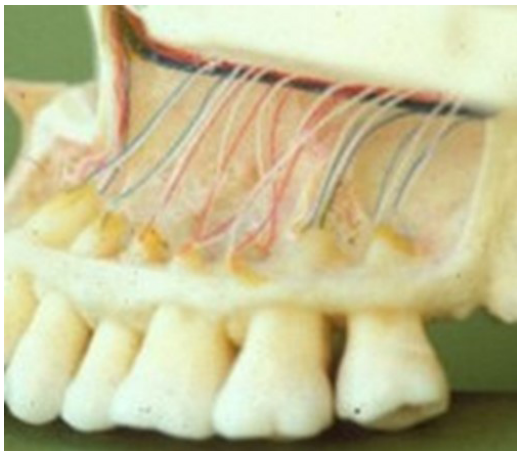


Figure 3. Maxillary posterior cancellous bone.

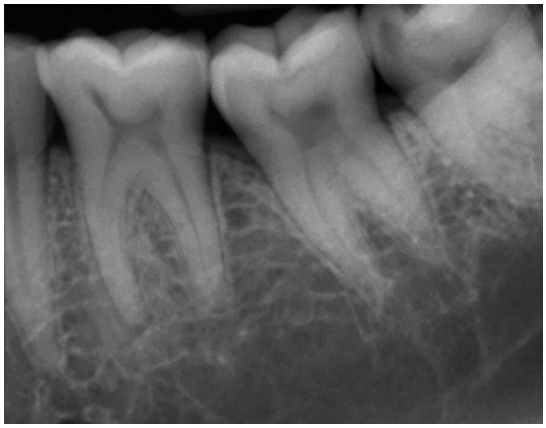


Figure 6. Periapical radiograph of mandibular posterior bone pattern.

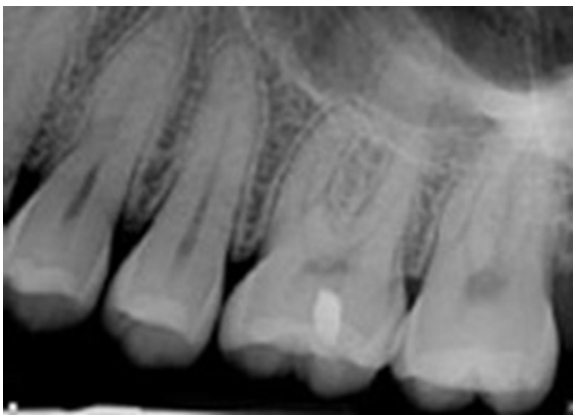


Figure 4. Periapical radiograph of maxillary posterior bone pattern.

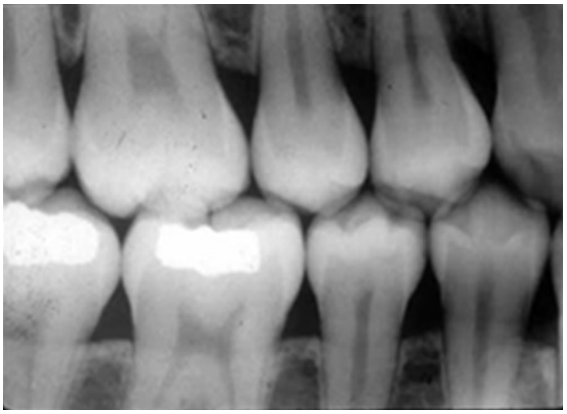


Figure 7. Bitewing radiograph of posterior alveolar bone crests.

Lamina Dura and Periodontal Ligament Space

Surrounding the tooth root(s) is a layer of dense bone which lines the tooth socket called the lamina dura (LD) (Figures 8, 9).[2] This radiopaque structure is contiguous with the alveolar crests.[3] The thickness and degree of radiopacity of the lamina dura varies with occlusal function.[2] It is thicker and more radiopaque with heavy function and thinner and less radiopaque with loss of function.[2] Immediately adjacent to the lamina dura is the periodontal ligament space (PDLS) which contains flexible collagen fibers surrounding and cushioning the tooth root in its bony socket.[1] It is a radiolucent structure that begins at the alveolar crest, surrounds the root, and returns back to the crest on the other side of the tooth (Figures 8,9).[2] The periodontal ligament varies in width, demonstrating differences among patients and the dentition itself.[2] In normal function, the periodontal ligament space appears thinner mid-root and wider at the crest and root apex.[2-4]

Tooth Eruption

Eruption is the physiologic movement of the teeth from the place of development within the jawbone to its functional position inside the oral cavity.[5] Typically, the teeth erupt into the mouth when the first part of the tooth crown peeks through the gingival tissue.[5] This usually happens once the tooth root is approximately 2/3 of its maximum length.[5] Tooth eruption occurs over a wide age range and can differ by both gender and race.[5]

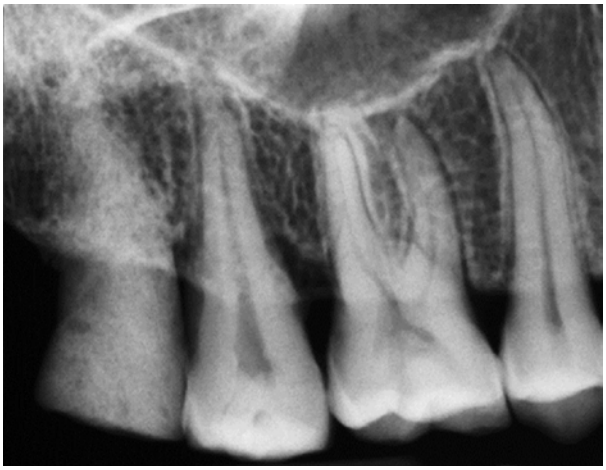


Figure 8. Periapical radiograph of the posterior maxilla showing the PDLS and LD.

Eruption of Primary, Mixed, and Permanent Dentitions

The teeth develop and erupt in three different stages: primary (deciduous), mixed, and permanent dentitions. All teeth begin their development in a tooth follicle or dental sac with coronal development occurring first followed by progressive apical formation until the root end is closed. As mentioned previously, the dental pulp is larger in immature teeth and smaller in mature teeth.[6] As primary or permanent teeth develop, the root apex/apices remain open until the root is fully formed.

Primary and Permanent Dentition Eruption Sequences

Primary Dentition Eruption Sequence

The primary dentition starts to erupt at around 6 months of age, beginning with the mandibular central incisors and ending with the eruption of the maxillary second primary molars between 25-33 months (2-2.75 years).[7] Table 1 outlines the eruption sequence for the primary dentition.[7] The primary dentition

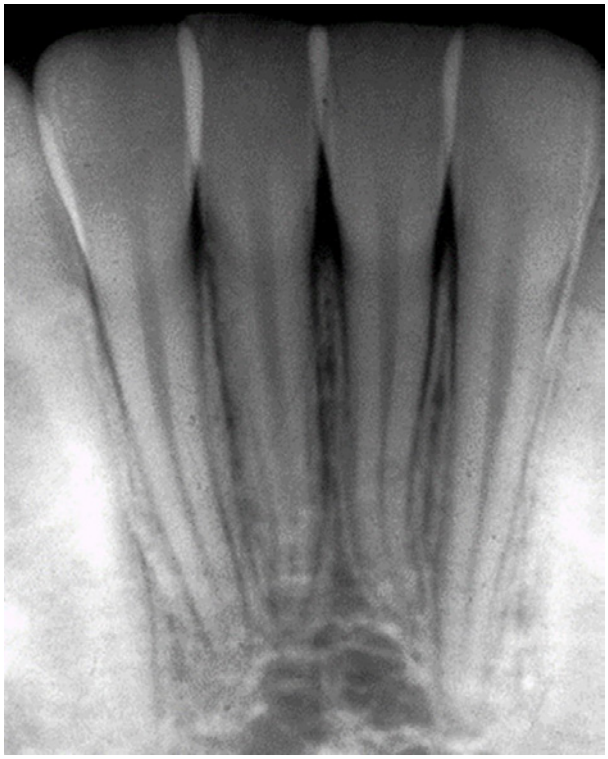


Figure 9. Periapical radiograph of the PDLS and LD surrounding the mandibular incisor roots.

consists of 20 teeth including 4 central incisors (E, F, O, P), 4 lateral incisors (D, G, N, Q), 4 canines (C, H, M, R), 4 first molars (B, I, L, S), and 4 second molars (A, J, K, T) distributed equally between the two dental arches (Figures 10-11).

The mixed dentition is composed of a combination of primary and permanent teeth (Figure 12). As the child matures, the primary teeth begin to exfoliate and are replaced by the permanent dentition. The mandibular central incisors and the first molars are the first to erupt, replacing their primary predecessors while the third molar teeth are the last to erupt in the late teen to early adult years.[7] The permanent dentition consists of 32 teeth including 4 central incisors (8, 9, 24, 25), 4 lateral incisors (7, 10, 23, 26), 4 canines (6, 11, 22, 27), 4 first premolars (5, 12, 21, 28), 4 second premolars (4, 13, 20, 29), 4 first molars (3, 14, 19, 30), 4 second molars (2, 15, 18, 31), and 4 third molars (1, 16, 17, 32) distributed uniformly between the maxilla and mandible. The primary first and second molars are replaced by the premolar teeth and the permanent molar teeth erupt posterior to the primary molars. Table 2 outlines the eruption sequence of the permanent dentition.[7]

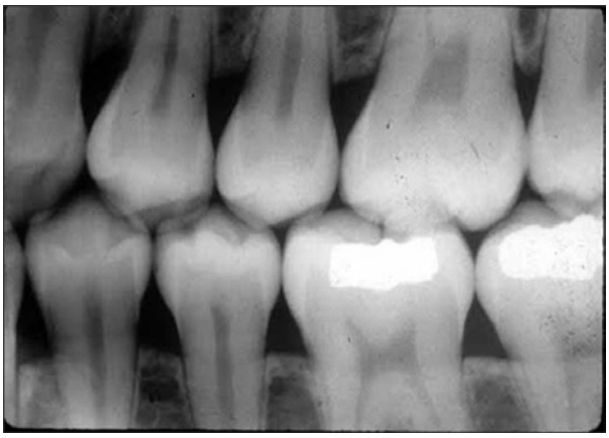


Figure 10. Periapical radiograph of primary mandibular anterior teeth.

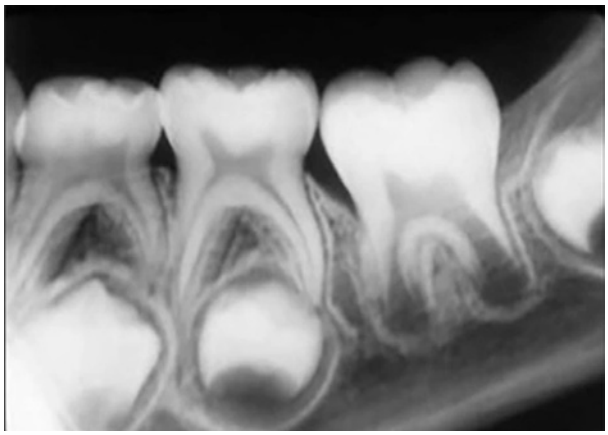


Figure 11. Bitewing radiograph of primary posterior teeth.

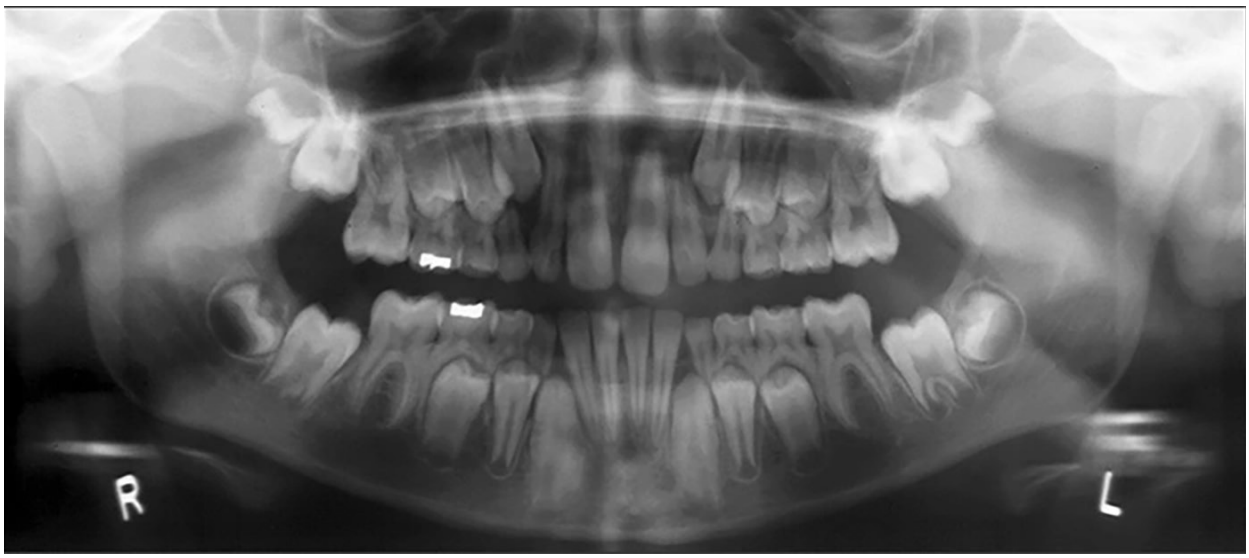


Figure 12. Panoramic radiograph of a mixed dentition.

Table 1. Primary Dentition Eruption.[7]

Tooth Type	Months
Mandibular Centrals	6-10
Maxillary Centrals	8-12
Maxillary Laterals	9-13
Mandibular Laterals	10-16
Maxillary First Molars	13-19
Mandibular First Molars	14-18
Maxillary Canines	16-22
Mandibular Canines	17-23
Mandibular Second Molars	23-31
Maxillary Second Molars	25-33

Table 2. Permanent Dentition Eruption.[7]

Tooth Type	Years
Mandibular Centrals/ First Molars	6-7
Maxillary Centrals/ Mandibular Laterals	7-8
Maxillary Laterals	8-9
Mandibular Canines	9-10
Maxillary First Premolars	10-11
Mandibular First Premolars/ Maxillary Second Premolars	10-12
Maxillary Canines/ Mandibular Second Premolars	11-12
Mandibular Second Molars	11-13
Maxillary Second Molars	12-13
Third Molars	17-21

Universal Tooth Numbering System

There are several tooth numbering systems used around the world. In the United States, the Universal System is the most commonly employed.[1] The primary dentition is identified by the letters of the alphabet, A-T, while the permanent dentition is identified by the numbers 1-32. Table 3 outlines the Universal Tooth Numbering System.

Delayed Eruption

Eruption is considered to be delayed if the tooth has not surfaced 12 months after the normal eruption time or when the tooth root is ¾ complete.[5] There are a number of reasons why teeth might be delayed in their eruption. The problem can be localized in which the eruption path is obstructed (Figure 13) or more widespread when a systemic disorder is implicated.[5] Teeth continue to erupt after emergence to offset masticatory wear and jaw growth.[5]

Ankylosis

Ankylosis is the cessation of eruption after tooth emergence.[5] This is caused by fusion of the tooth dentin or cementum with the alveolar bone.[7] The pathogenesis of this process is not known.[5] While ankylosis can occur at any age, it is most common in children 8 to 9 years of age.[7] The teeth that are most frequently involved include the primary mandibular first molar, followed respectively by the primary mandibular second molar (Figure 14), the primary maxillary first molar, and the primary maxillary second molar.[5] The involved ankylosed tooth usually has a submerged occlusal plane compared to the adjacent teeth and may, upon percussion, produce a sharp, solid sound.[5] The periodontal ligament space may be absent radiographically.[7] Permanent teeth rarely become ankylosed.[5]

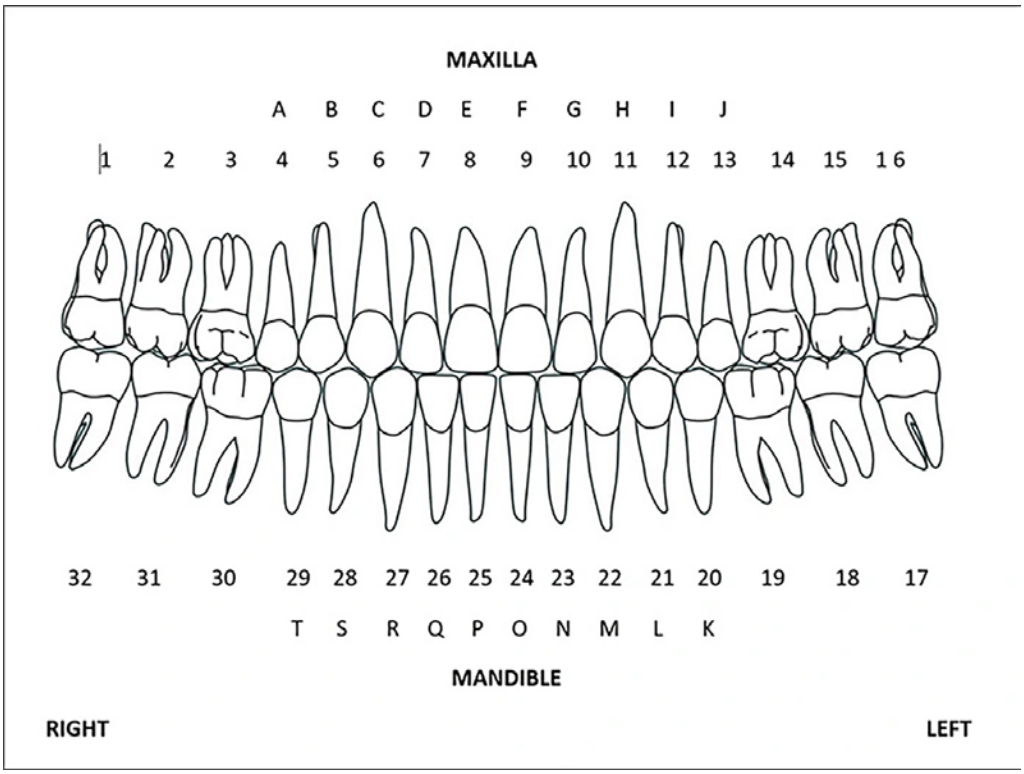


Table 3. Universal Tooth Numbering System.

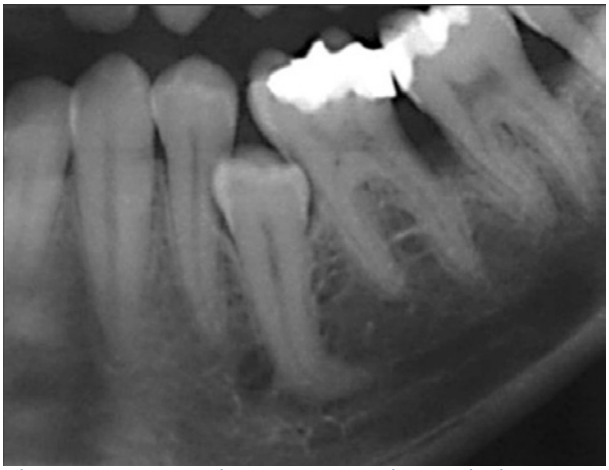


Figure 13. Cropped panoramic radiograph depicting the obstructed eruption path of mandibular left premolar tooth #20.

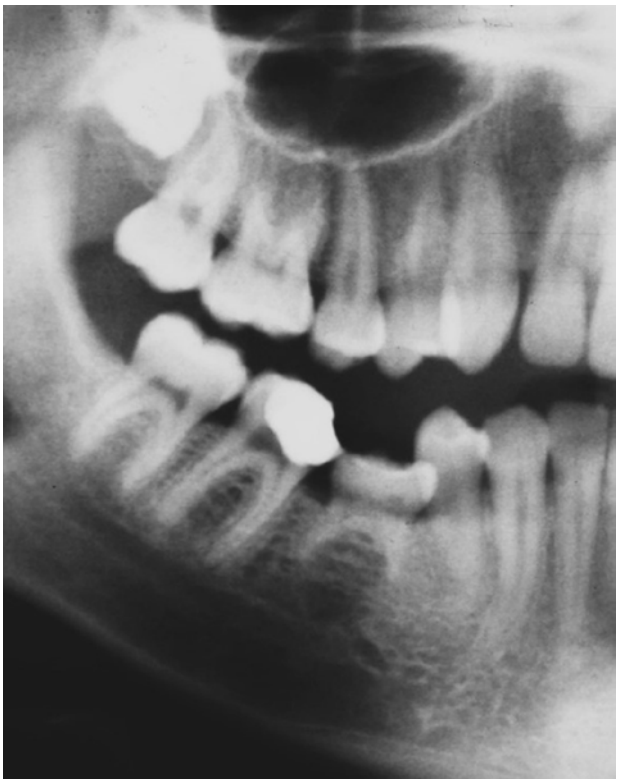


Figure 14. Cropped panoramic radiograph demonstrating a submerged occlusal plane associated with ankylosed primary molar tooth T and congenitally missing permanent premolar tooth #29.

Impacted Teeth

The term used to describe teeth that fail to erupt is impacted.[8] Primary teeth impactions are uncommon but when they do happen, the primary second molar is usually involved.[5] In the permanent dentition, the most commonly impacted teeth in order by frequency are the mandibular third molars, the maxillary third molars, and the maxillary canine teeth.[5] See Figures 15-17 for examples of impacted teeth. Impacted teeth are classified according to their position angulation relative to the rest of the erupted dentition such as: mesioangular, distoangular, horizontal, vertical, inverted or a combination thereof.[7] They can be completely encased in the bone or partially erupted.

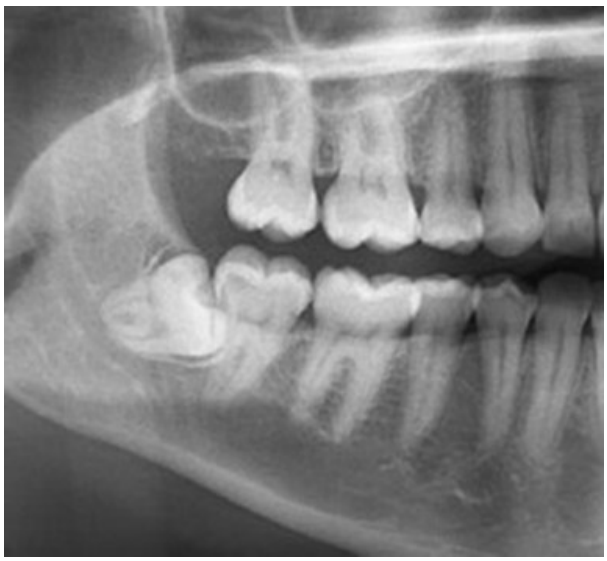


Figure 15. Cropped panoramic image showing a horizontal impaction of mandibular right third molar tooth #32 in a mesioangular orientation.

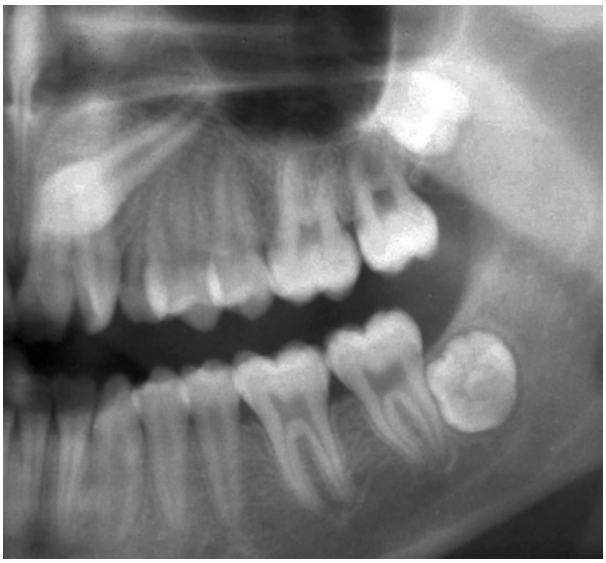


Figure 16. Cropped panoramic image of horizontal mesioangular impaction of maxillary left canine tooth #11 and buccolingual impaction of mandibular left third molar #17.

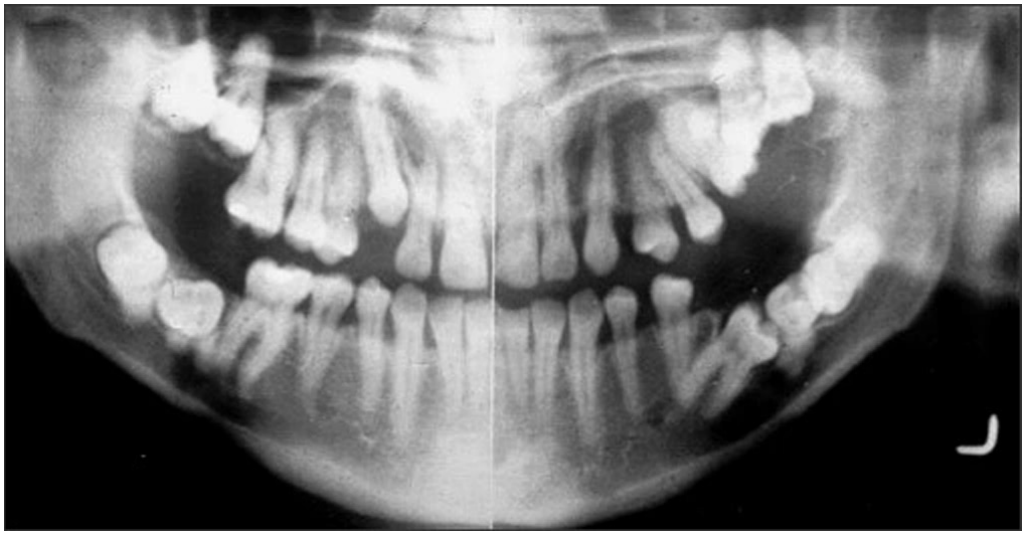


Figure 17. Panoramic radiograph of multiple impacted teeth with differing orientations. Note the kissing molars, mandibular left first molar tooth #19 in a horizontal distoangular position with the second molar tooth #18 trapped between the third molar tooth #17 in a horizontal mesioangular position.

Transposition - Transposed Teeth

When two adjacent teeth have exchanged their normal sequential order, the condition is called transposition.[9] The teeth most frequently involved are the permanent canine and first premolar in which the premolar erupts anterior to the canine tooth (Figures 18, 19).[9] This condition is observed in the permanent dentition but is not seen in the primary dentition.[9]

Congenital, Developmental, and Acquired Anomalies

The anomalies of tooth structure can be categorized as congenital, developmental, and acquired.[9] Congenital anomalies are those that are present or exist at the time of birth. Such anomalies may result from genetic, infectious, environmental, or nutritional factors.[5,9] Developmental anomalies are abnormalities that result from flawed growth and differentiation during tooth formation.[9] By contrast, acquired anomalies are caused by external factors which affect the teeth subsequent to normal development.[9]

Alterations in the Number of Teeth

There are a number of developmental alterations that may occur which influence the number of teeth that are present. Several different terms are used to describe the absence or presence of teeth.[5,8,9] Anodontia is the proper term for the total lack of tooth development involving the entire dentition.[5] This is uncommon, particularly in the primary dentition. Several other terms are used to define missing teeth such as hypodontia, one or more missing teeth, and oligodontia, six or more missing teeth excluding the third molars.[5] In contrast, hyperdontia is the development of more than the usual complement of teeth.5 These extra teeth are sometimes referred to as supernumerary teeth.[5,8,9]

Hypodontia

While rare in the primary dentition, hypodontia is not uncommon in the permanent dentition. In fact, it is one of the most common developmental abnormalities affecting nearly 20% of individuals with a slight female predominance.[5] The most frequently missing teeth are the third molars, followed by the

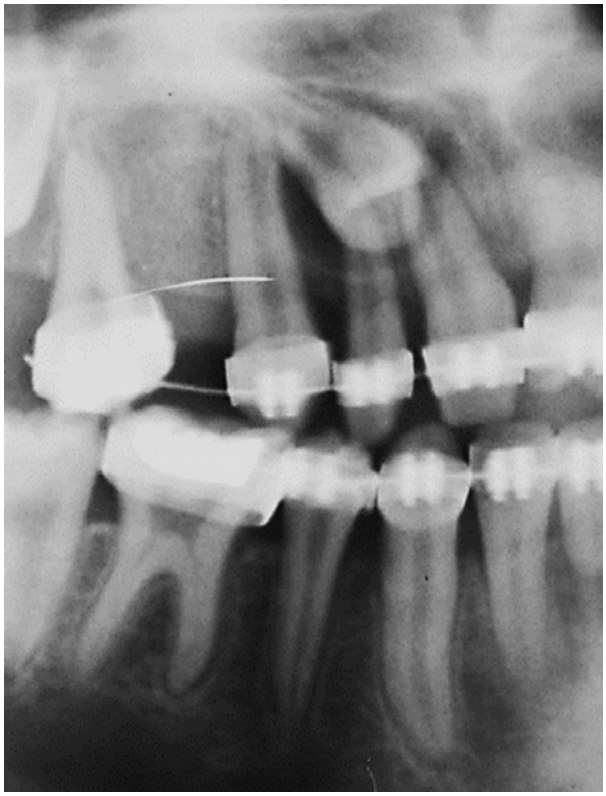


Figure 18. Cropped panoramic radiograph showing transposition of impacted maxillary right first premolar tooth #5 and maxillary right canine tooth #6.

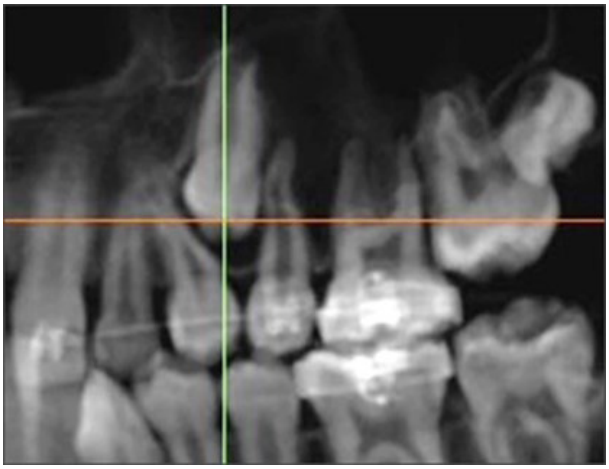


Figure 19. Cropped Cone Beam Computed Tomography (CBCT) panoramic reformat showing transposition of maxillary left first premolar #12 and impacted maxillary left canine #11.

Figure 19 courtesy of Dr. Christos Angelopoulos.

second premolars (Figure 20), the maxillary lateral incisors (Figure 22), and mandibular central incisors (Figure 21)[9,10] The maxillary central incisors, the mandibular first molars, and canines are the least likely teeth to be absent.[5]

In instances in which multiple teeth are missing, especially key teeth like maxillary central incisors or the first molars, a hereditary condition known as ectodermal dysplasia should be considered.[9,10] In this entity, the teeth that are present have an abnormal crown shape (Figure 23).[9] Clinically, dermal adnexal structures like hair, eyebrows and eyelashes are also affected.[5,10]

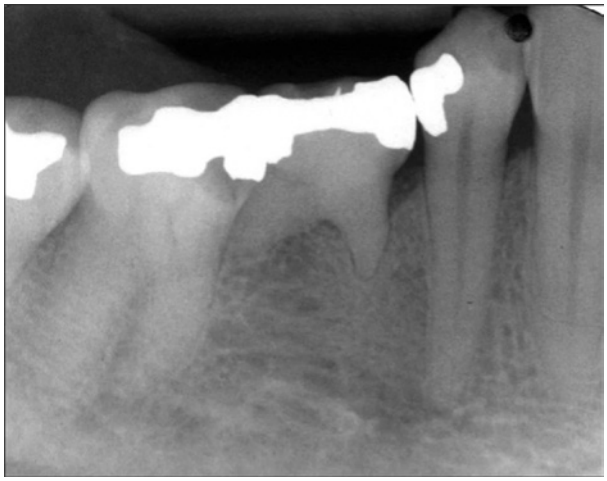


Figure 20. Periapical radiograph of congenitally missing mandibular right premolar tooth #29 with retained primary molar T.

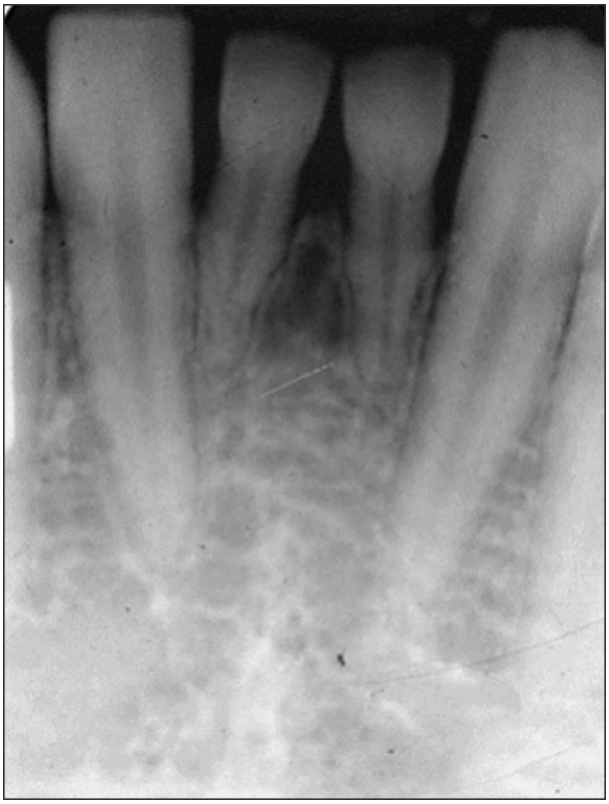


Figure 21. Periapical radiograph of congenitally missing mandibular central incisor teeth #24 and #25.

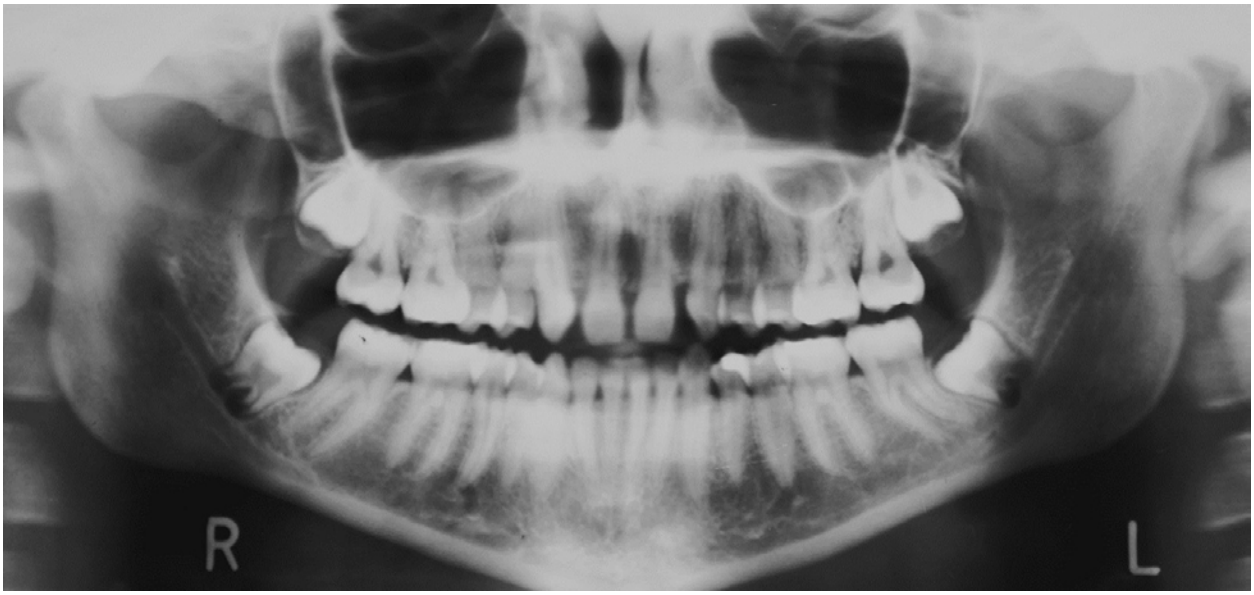


Figure 22. Panoramic radiograph of congenitally missing maxillary lateral incisor teeth #7 and #10.

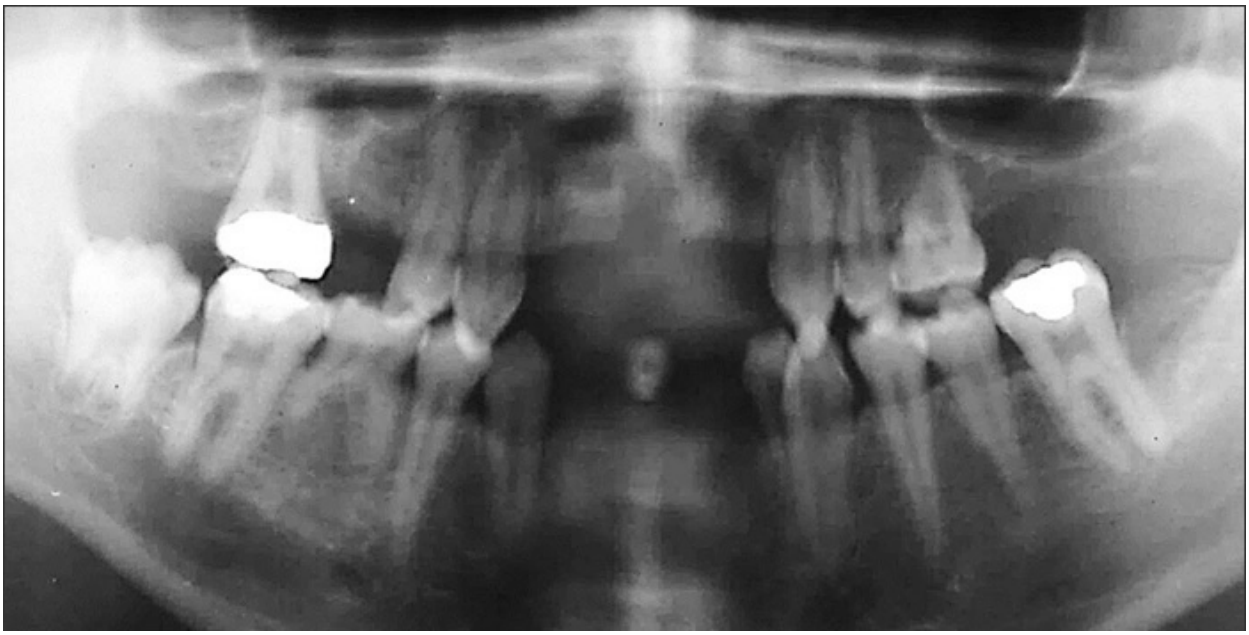


Figure 23. Cropped panoramic radiograph of child patient with ectodermal dysplasia demonstrating multiple missing key teeth. *Image courtesy of Dr. Géza T. Terézhalmy.*

Hyperdontia

Extra or supernumerary teeth usually involve a single tooth in the permanent dentition within the maxilla, particularly in the anterior segment.[5] Other common sites for supernumerary teeth include the maxillary molar area, and the mandibular molar, premolar, canine, and lateral incisor areas.[5,8,9] A supernumerary tooth in the anterior maxilla is a mesiodens (Figure 24A and 24B), while a fourth molar tooth is a distodens or distomolar (Figure 25), and an extra tooth in the premolar area is a peridens (Figure 26).[5,8-10] The term paramolar is used for a posterior extra tooth positioned buccally or lingually to a molar tooth.[9,10] Supernumerary teeth can be of normal size and shape or abnormally shaped and diminished in size. (Figures 25, 27)[5] The latter type can display various shapes including conical, barrel-like or a petite version of a molar or premolar tooth.[5] These teeth can be erupted or unerupted.

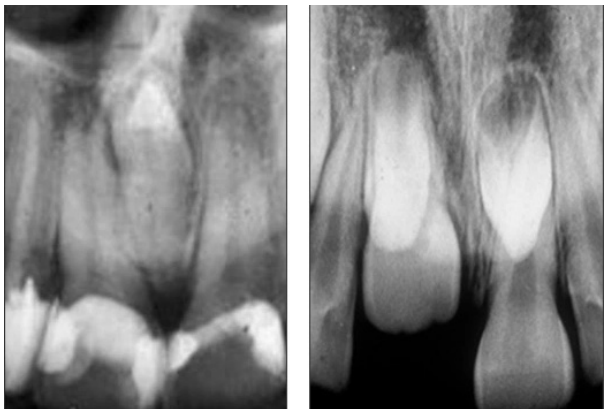


Figure 24A and 24B. Periapical radiographs of single inverted (A) and multiple mesiodens (B).

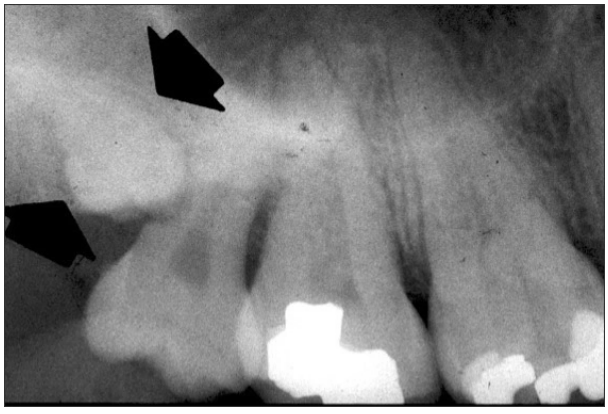


Figure 25. Periapical radiograph of a maxillary distomolar.

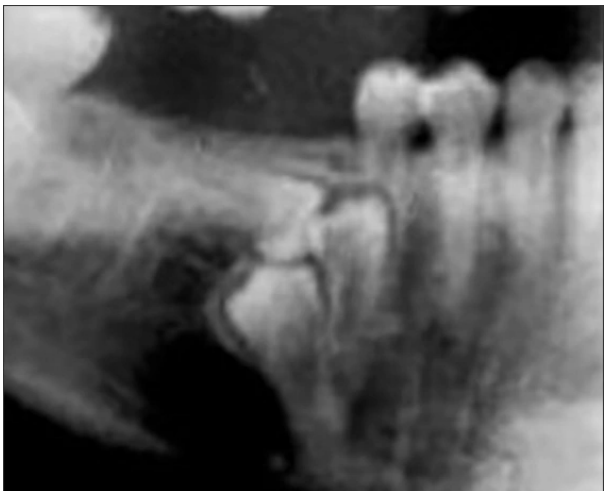


Figure 26. Cropped panoramic radiograph of supernumerary peridens.

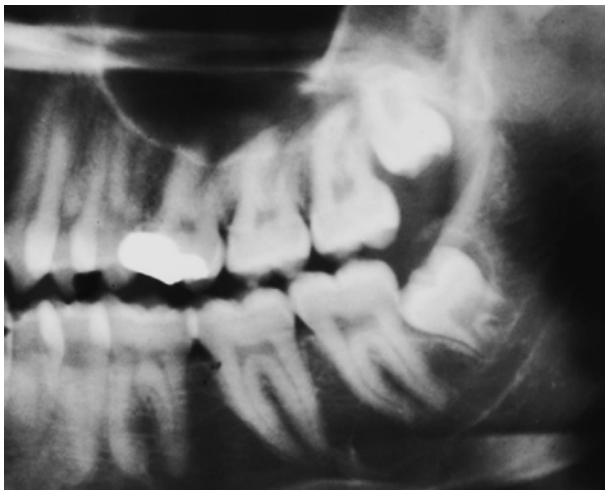


Figure 27. Cropped panoramic radiograph of supernumerary 4th molar teeth of normal tooth size.

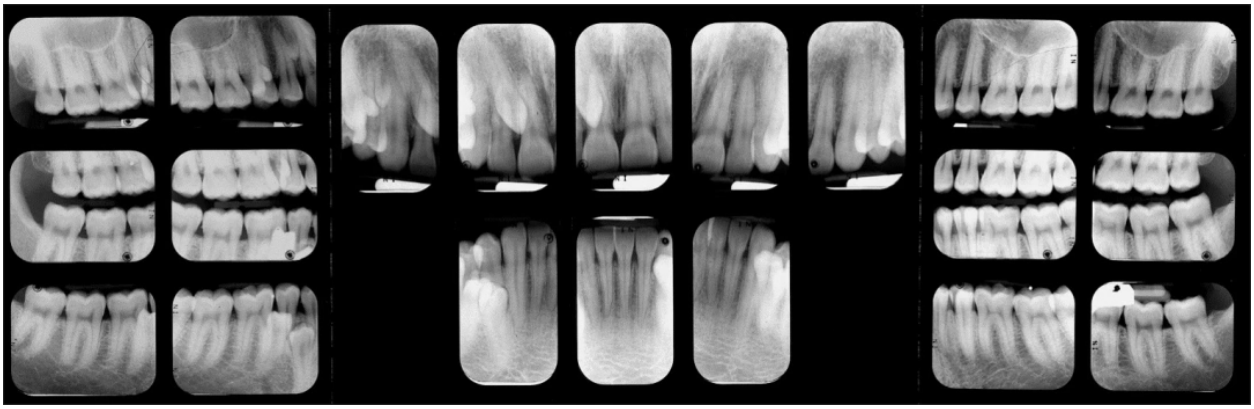


Figure 28. Full mouth radiographic survey of multiple supernumerary teeth. Note the size, shape, and location of the numerous supernumerary teeth present.

It is possible that supernumerary teeth will present in multiple manners. When multiple extra teeth occur, the most common location is in the mandibular premolar region (Figure 26), but molar and anterior areas also can be involved (Figure 24B, 26, 28).[5]

Alterations in the Size of Teeth

Tooth size is variable among racial groups and between genders. Generally speaking, males have larger teeth than females.[5,9] Both genetic and environmental factors can impact the size of the developing teeth.[5,9] Microdontia is the term used for teeth that are smaller than normal while macrodontia is the presence of teeth that are larger than usual.[5,9,10] Typically, these conditions do not affect the entire dentition.[9]

Microdontia

In microdontia, the teeth are visibly smaller and may have an altered shape.[7] Supernumerary teeth may present as microdonts as well.[9] The maxillary lateral incisor tooth (Figure 29) is the most frequently involved and typically demonstrates a peg-shaped crown.[5,9,10] Microdontia is associated with hypodontia and occurs more frequently in females(Figure 30).[5] Third molar teeth are often affected by microdontia as seen in Figure 31.[9] Of note, these same teeth are often congenitally missing.

Macrodontia

Macrodontia is sometimes called megalodontia or megadontia.[5,9,10] Macrodont teeth appear larger and may be associated with crowding,

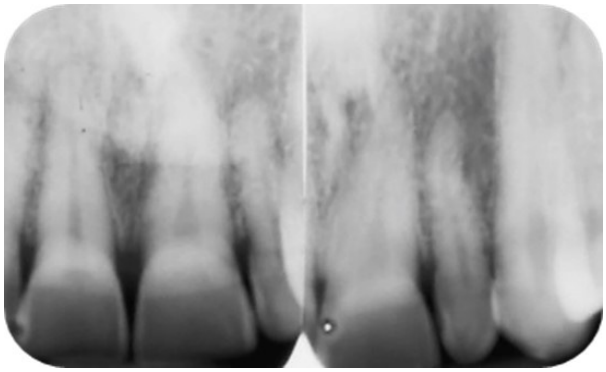


Figure 29. Periapical radiographs of a peg lateral, maxillary left lateral incisor tooth #10.

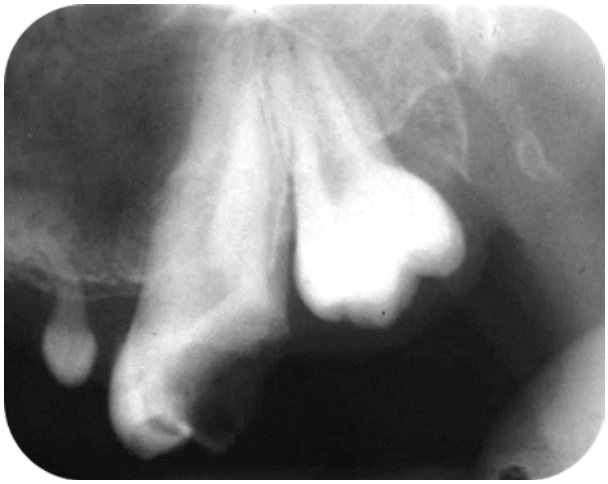


Figure 30. Periapical radiograph of a microdont supernumerary tooth on the maxilla.

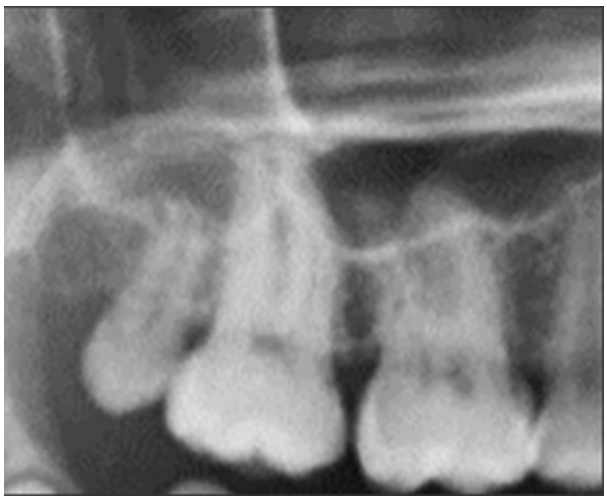


Figure 31. Cropped panoramic image of microdont maxillary right third molar tooth #1.

malocclusion, or impaction.[9] Often a single tooth or several teeth are involved rather than affecting the entire dentition.[5] Macrodontia is associated with hyperdontia and occurs more frequently in males.[7] Macrodontia tends to occur in incisor or canine teeth but may also occur in second premolars and third molars.[5] Macrodontia terminology should not be applied to teeth that have been altered by gemination or fusion processes which will be discussed next.[5]

Alterations in the Shape of Teeth

There are a number of conditions in which the usual morphology of the tooth is altered, and as a result presents differently both clinically and/or radiographically. These alterations can affect various aspects of the tooth structure and tooth anatomy.

Conjoined Teeth

Conjoined or double teeth can occur through different processes. Traditionally, conjoined teeth fall into three categories: gemination, fusion, and concrescence.[5] In some cases it is quite difficult to discern the true pathogenesis of the particular anomaly.[5] Some of the traditionally accepted parameters discussed in the following text do not apply to every occurrence of conjoined teeth.

Gemination

Gemination, or twinning, is a rare abnormality in which a single tooth bud tries to divide.[9] Partial division may produce a bifid crown (Figure 32) with a shared pulp canal and root.[9] Complete division, while rare, produces a normal tooth along with a supernumerary tooth (Figure 33).[9] Gemination usually affects the primary teeth but the permanent dentition can be involved as well.[5,9] The incisor region is the most commonly affected area with no apparent gender predilection.[5] In gemination, the number of teeth is usually normal.[10]

Fusion

Fusion is defined as two adjacent tooth buds that join together to form one normal looking tooth or a much larger tooth (Figure 34).[5,9,10] The degree of union may be total or partial and often presents with a coronal cleft.[10] The normal complement of teeth is reduced by

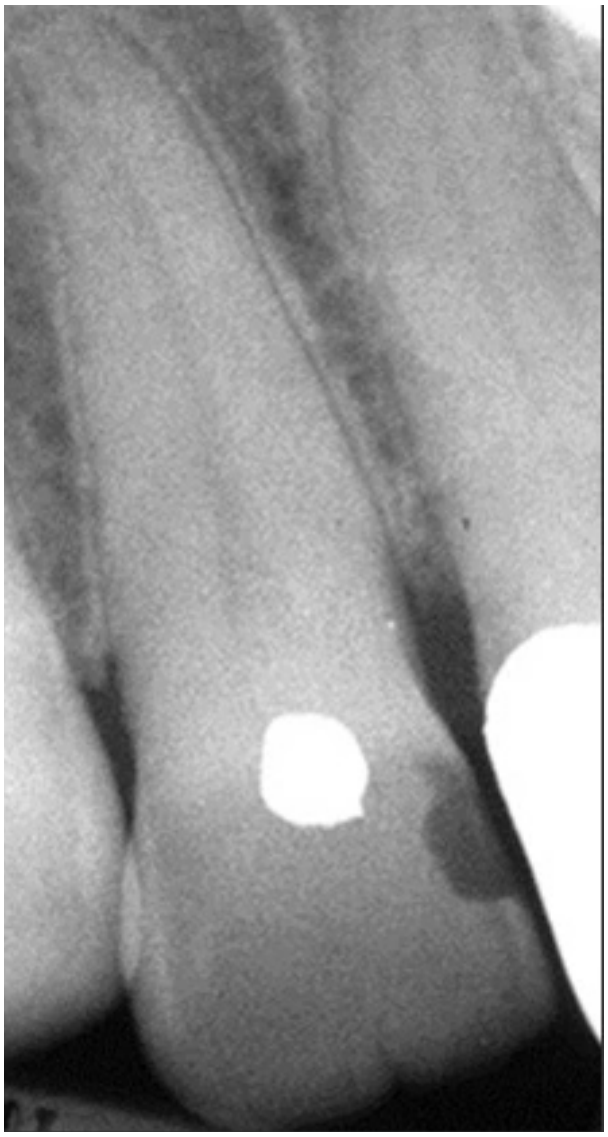


Figure 32. Periapical radiograph of maxillary lateral incisor tooth #7 with coronal cleft.

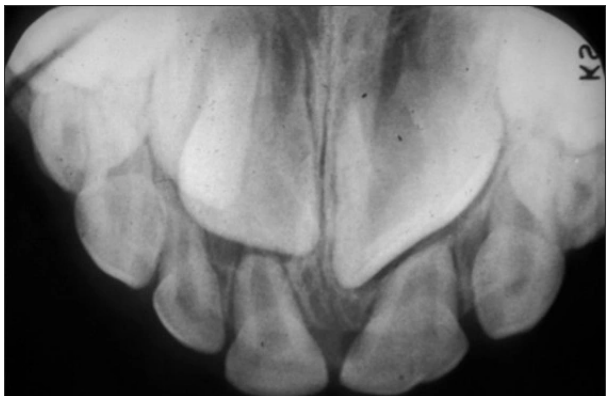


Figure 34. Periapical radiograph of fused primary teeth F & G and permanent teeth #9 & #10. Note the larger fused crown size.

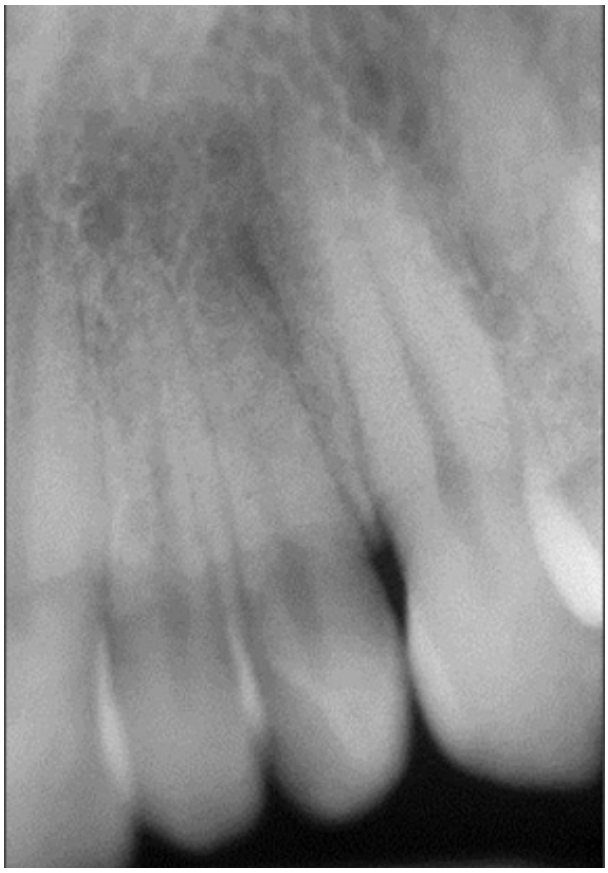


Figure 33. Periapical radiograph of maxillary lateral incisor tooth #10 with complete division.

one.[5,9] Like gemination, fusion is more common in the primary dentition but can occur in the permanent dentition as well.[5,9,10] It also leans toward involvement in the anterior region of the dental arch.[5,9,10] While there is no gender predilection, incidence is higher among Native American, Asian, and other indigenous peoples.[5,9]

Concrescence

Concrescence is the joining of two completely formed teeth by the cementum along the root surface (Figures 35, 36).[5,9] It favors posterior teeth and the maxillary arch, often involving a second molar tooth closely approximating the roots of an impacted third molar.[5] The affected teeth may fail to erupt or only partially erupt.[9] The cause of this altered morphology is not known.[9] In instances in which concrescence causes eruption difficulties, surgical extraction may be necessary and result in loss of both teeth.[9,10]

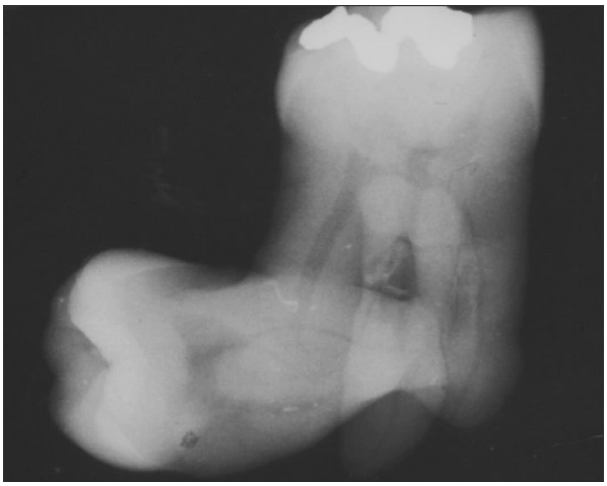


Figure 35. Periapical radiograph demonstrating molar concrescence.

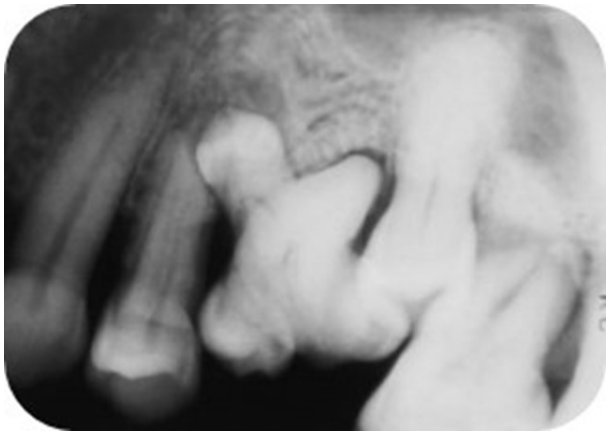


Figure 36. Periapical radiograph showing concrescence of premolar supernumerary teeth.

Accessory Cusps

Teeth with accessory cuspal anatomy can impact the overall size of the teeth.[5] When accessory cusps are present, it is not uncommon for the other teeth in the dentition to be slightly larger.[5] Examples of accessory cusps include the cusp of Carabelli, the talon cusp and dens evaginatus.

Cusp of Carabelli

The cusp of Carabelli is located on the mesiolingual cusp of maxillary molar teeth and can occur in both primary and permanent dentitions.[5] The cusp tend to be the most prominent on the first molars and lesser so or absent on the second and third molars.[7] When present, particularly in the permanent dentition, the other teeth frequently appear wider mesiodistally.[5] The cusp of Carabelli is the most prevalent in white populations.[5]

Talon Cusp

The talon cusp, another supernumerary or accessory cusp, affects maxillary or mandibular incisor teeth.[10] This extra cusp occurs on the cingulum of maxillary lateral incisor teeth (Figure 37), most commonly, followed respectively by maxillary central incisors, mandibular incisors, and maxillary canine teeth.[5] More common in the permanent dentition, the cusp extends from the lingual aspect of the tooth with a formation that resembles an eagle's talon.[10] Facial talon cusps have been reported but are rare.[10] The cusp is well-formed and extends at least half the distance from the cementoenamel junction to the incisal edge, often containing a pulpal extension.[5,7,10] A deep developmental groove may exist between the talon cusp and the underlying tooth structure.[5,10] Radiographically, the cusp anatomy will appear superimposed over the other tooth structure components.[5,10] The pulpal extension, if present, may not be demonstrated radiographically.[10]

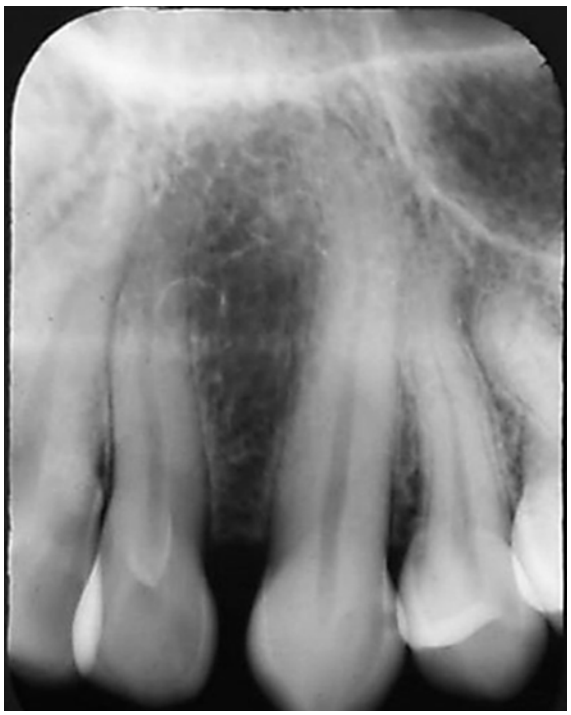


Figure 37. Periapical radiograph of a talon cusp on maxillary left lateral incisor tooth, #10.

Talon cusps tend to occur more frequently in Native American, Asian, Inuit and Arab populations with prevalence ranging from 1-8%.[5,10] This cuspal anomaly can occur in either gender and may present unilaterally or bilaterally.[5,9] The talon cusp is often associated with other dental anomalies such as supernumerary teeth, impacted teeth and dens invaginatus.(Figure 38)[9,10]

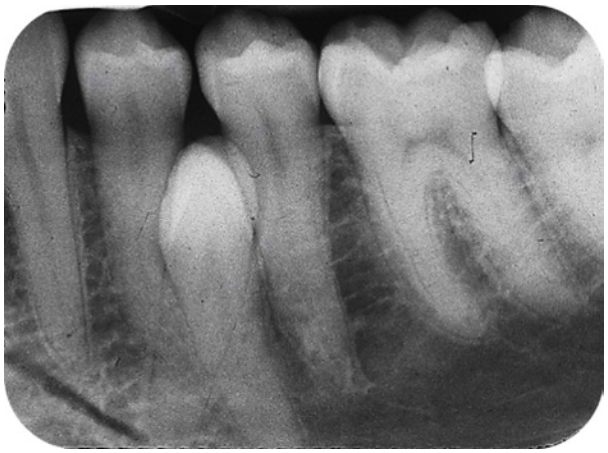


Figure 38. Periapical radiograph of talon cusp anatomy on a peridens impacted between mandibular left premolar teeth #20 and #21

Dens Evaginatus

Dens evaginatus is an external outcropping of tooth structure in the form of a cusp-like projection of enamel on the tooth crown.[5,9] Dens evaginatus is usually found in the central groove or on the lingual ridge of the buccal cusp of a molar or premolar tooth (Figure 39).[7,9] Most often, the mandibular premolar teeth are involved bilaterally.[5,9] This extra cusp or tubercle is composed of enamel and dentin, and in many instances pulp tissue as well. This particular anomaly occurs in less than 5% of the population, most commonly in Native American, Asian, and aboriginal racial groups.[5,9] Exposure and necrosis of the pulp can result from cuspal wear or fracture.[9]

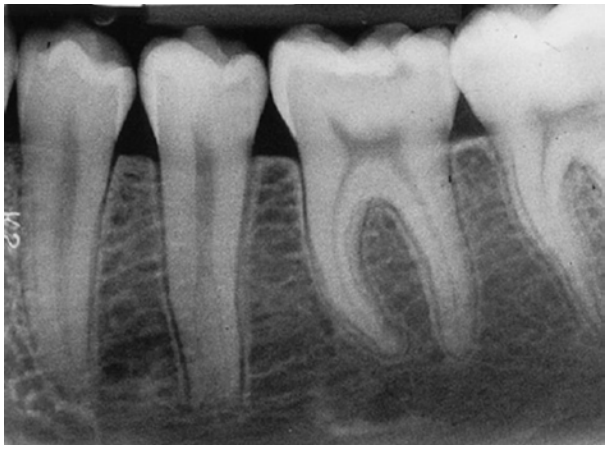


Figure 39. Periapical radiograph showing dens envaginatus mandibular left premolar teeth #20 and #21.

Often teeth with dens evaginatus occur in association with incisor teeth with shovel-shaped anatomy.[5] This variation in coronal anatomy is seen predominantly in Native American and Inuit populations and most commonly affects the maxillary central and lateral incisor teeth.[5] The hallmarks of the affected incisor teeth include heavy lateral marginal ridges and dished out lingual surfaces that resemble the shape of a shovel (Figure 40).[5] The ridges coalesce at the cingulum often forming a deep pit or a dens invaginatus defect. The later condition will be discussed next.

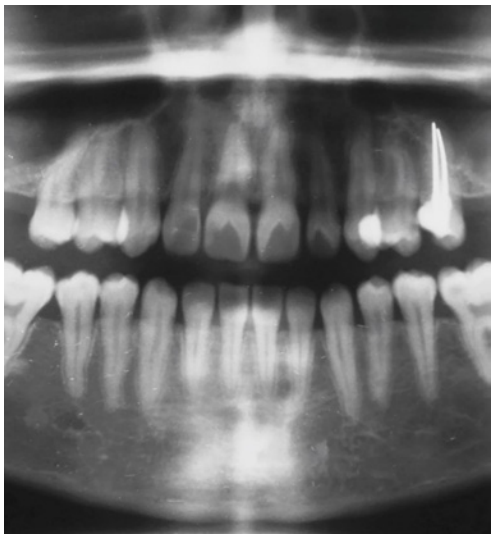


Figure 40. Panoramic radiograph demonstrating shovel-shaped anatomy of the maxillary incisors.

Dens Invaginatus

Dens invaginatus is an inversion or enfolding of enamel into the crown, sometimes extending beyond the CEJ or into the root.[5,9] Comparatively, dens invaginatus is an internal involution of tooth structure while dens evaginatus is an external outcropping of tooth structure.[5,9]

Dens invaginatus varies in the degree of tooth structure enfolding. Three types are recognized

per Oehlers Classification, the most widely used nomenclature. (Table 4).[10,11] These coronal invaginations usually involve the permanent maxillary lateral incisors but other permanent teeth can be affected including central incisors, premolar, canine, and molar teeth with the maxillary arch more frequently involved than the mandibular arch.[5,9,10] The most common and least severe form of coronal dens invaginatus, Type I, is limited to the crown.[10] Typically, the cingulum exhibits a small pit but an invagination of enamel can be present as well (Figure 41).[5,9] The pit is prone to develop caries or possible explorer perforation into the pulp due to the thin layer of enamel.[8]

Type II exhibits more severe coronal invagination extending from the incisal edge to below the CEJ ending in a blind sac or involution of enamel.[5,9] Radiographically, it may have the appearance of a radiolucent teardrop with a radiopaque outline (Figure 41).[9] If the defect is more extensive, often the tooth crown is malformed and the invagination dilated giving the appearance of a dens in dente or tooth within a tooth.[5,9] In cases where there is communication between the invagination and the pulp, necrosis will occur resulting in periapical disease.[9] This may occur before the apex is completely formed and closed.[5,9] The permanent maxillary lateral incisor teeth are most commonly affected with a tendency for bilateral occurrence.[9]

Type III dens invaginatus extends through the root either perforating it laterally or apically communicating with the PDL (Figure 42A and B).

The most severe form, dilated odontome, involves the tooth root with a doughnut-shaped invaginated defect lined with cementum.[5,9] Radiographically, the affected tooth is significantly malformed displaying a radiopaque round or oval periphery with a radiolucent center as seen in Figure 43.[5,9,12] This anomaly is rare and appears to be independent of the traditional classification system outlined in Table 4.[12] The clinical relevance of these defects is the possibility of pulpal involvement and necrosis.[5,9,10]

Table 4. Oehlers Classification.[10,11]

Type	Description
Type I	Defect is enamel-lined and confined to the tooth crown
Type II	Defect is enamel-lined and extends into the pulp chamber but remains within the root canal
Type III	Defect extends into the tooth root and perforates the root laterally or through the apical foramen subtypes A and B)
Type IIIA	Perforates the root laterally with PDLS communication; usually without pulp involvement
Type IIIB	Communicates with the PDLS at the apical foramen; usually without pulp foramen; usually without pulp involvement

Enamel Pearls

Enamel pearls are tiny nodules of ectopic enamel that occur most typically on the root surface of permanent maxillary molar teeth, although mandibular molars can be affected.[5,10] These projections typically consist of enamel, but dentin and pulp tissue may be present underneath the enamel layer posing a potential risk for pulp exposure. Radiographically, enamel pearls are singular, well-defined circular radiopacities located near the cervical aspect of the tooth (Figure 44) or in the root furcation area (Figure 45).[5,8-10] The prevalence of enamel pearls ranges from ≈ 1% to 10% with Asians demonstrating the greatest incidence.[5,10] In most circumstances, enamel pearls are asymptomatic, but their presence may impact oral hygiene care and periodontal health.[9,10]

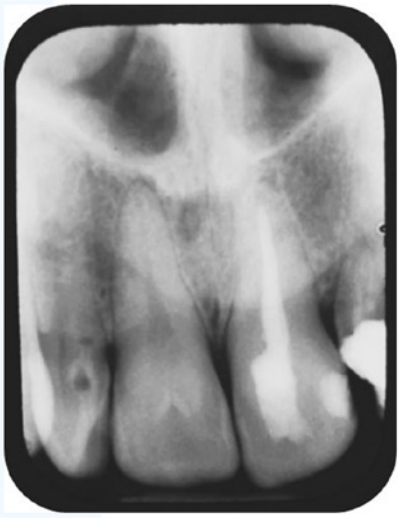
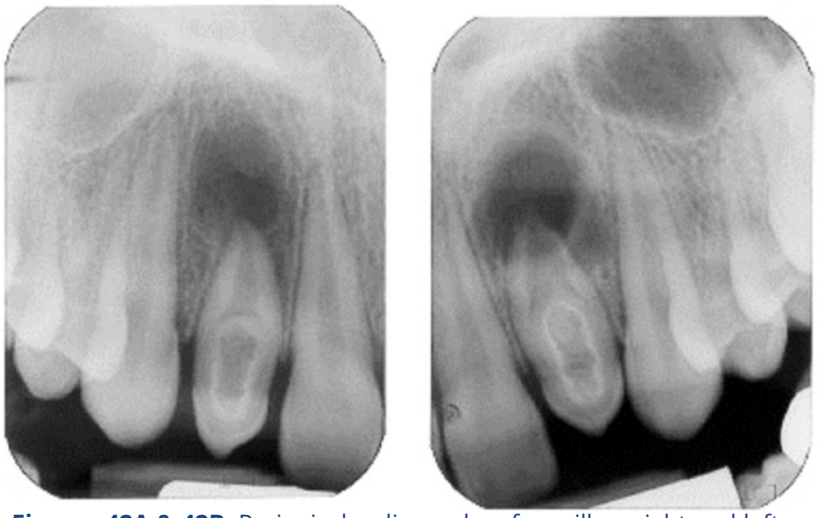


Figure 41. Periapical radiograph of maxillary right central incisor tooth #8 with dens invaginatus Type I and lateral incisor tooth #7 with dens invaginatus Type II.



Figures 42A & 42B. Periapical radiographs of maxillary right and left lateral incisor teeth #7 (A) and #10 (B) with dens invaginatus Type IIIB.

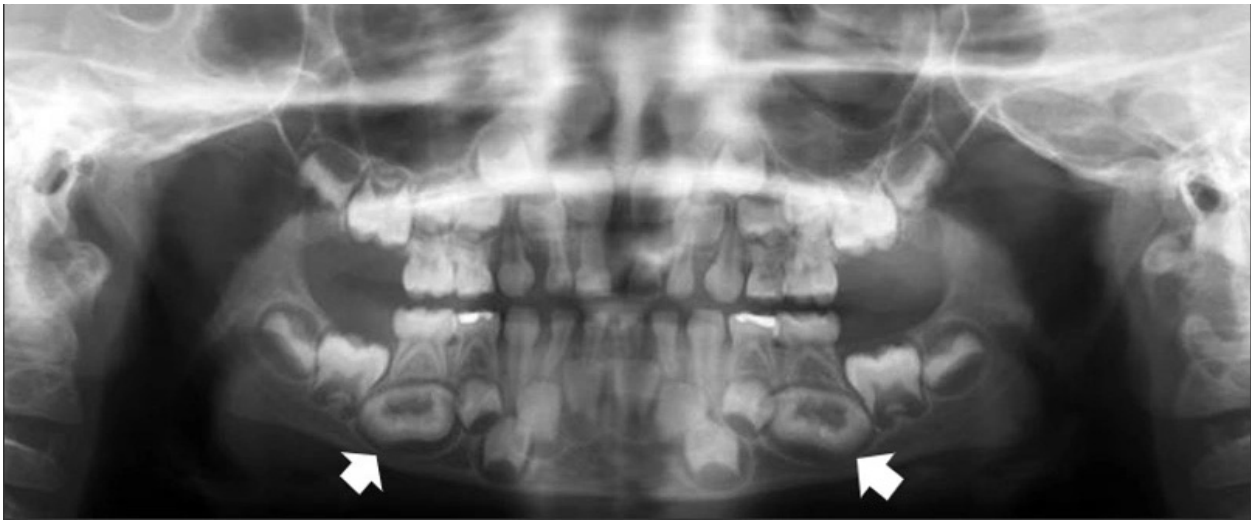


Figure 43. Panoramic radiograph with developing dilated odontomes of mandibular premolar #20 & #29.

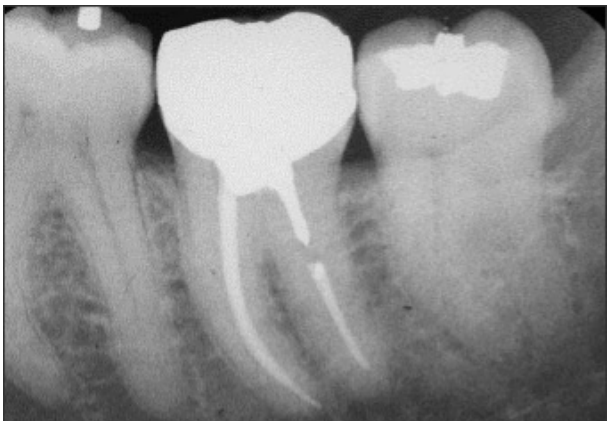


Figure 44. Periapical radiograph of a distal enamel pearl on mandibular left third molar tooth #17.

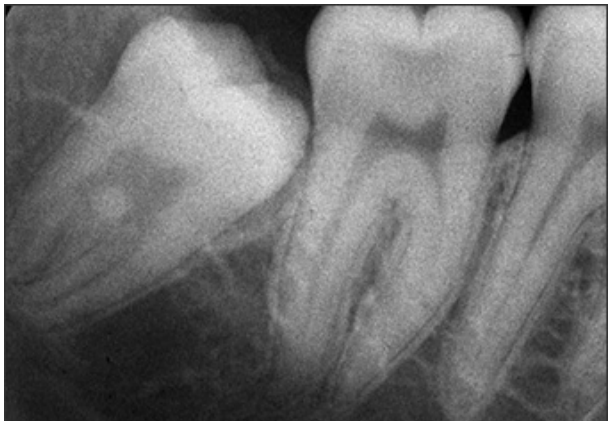


Figure 45. Periapical radiograph of a furcal enamel pearl on mandibular right third molar tooth #32.

Taurodontism

Taurodonts are molar teeth that present unusual tooth morphology, resembling the teeth of bulls (Figure 46).[10,13,14] This may be a variation of normal tooth formation. Radiographically, taurodonts display elongated pulp chambers and short stubby roots that look like bull's horns.[8-10] Table 5 outlines three categories used to classify the severity of the deformity, originally described by Shaw in 1928: Hypotaurodontism (Mild), Mesotaurodontism (Moderate), and Hypertaurodontism (Severe).[13-15] The severity is determined by the increasing apical displacement of the pulpal floor.[5,13-15] Permanent molar teeth are more frequently involved than primary teeth with either unilateral or bilateral presentation.[5] The prevalence is variable but approximates 3% of the U.S. population.[5] Taurodontism is associated with a number of syndromes indicating possible linkage to chromosomal aberrations in tooth development.[5,13,14]

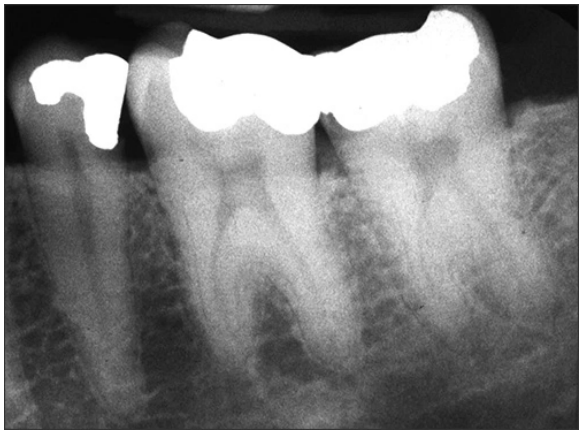


Figure 46. Periapical radiograph of mesotaurodont teeth #18 and #19.

Hypercementosis

Hypercementosis is overproduction or increased proliferation of cementum on the tooth root (Figure 47). This can result in mild to marked misshaping of the tooth root with a bulbous-like or lobular appearance.[9,10] The excess cementum will appear slightly less radiopaque than the dentin with the lamina dura and periodontal ligament space evident around the excess cementum.[9] Usually the additional cementum demonstrates a smooth outline, but it can also appear irregular.[9,10] The exact cause is unknown but hypercementosis may be a reaction to inflammation or loss of function of a supraerupted tooth due to absence of an opposing tooth.[8-10] Hypercementosis is known to be associated with fractured teeth or teeth in heavy occlusion.[9,10]

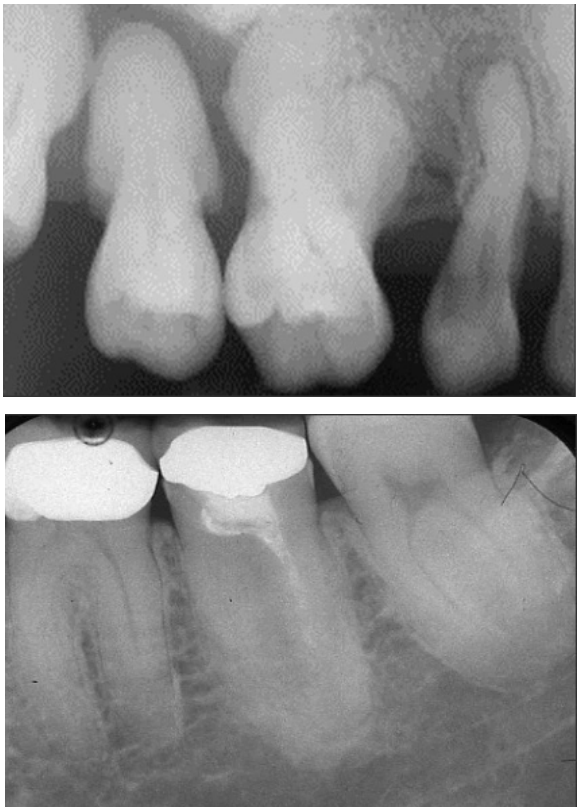


Figure 47A and 47B. Periapical radiographs of maxillary right (A) and mandibular left (B) posterior teeth demonstrating hypercementosis.

Table 5. Taurodontism Classification.[5,13,14]

Category	Degree	Description
Hypotaurodontism	Mild	Moderate enlargement of the pulp chamber compared to the roots
Mesotaurodontism	Moderate	Large pulp chamber with short separate roots
Hypertaurodontism	Severe	Pulp chamber nearly reaches root apices before dividing into stubby separate roots

Dilaceration

Dilaceration, a disturbance in tooth development, generates a sharp angular bend or distinct curvature in the root (Figures 48-50) or less often, in the crown.[5,8-10] While coronal curvatures may be evident clinically, radicular dilacerations are best revealed radiographically. The most commonly involved teeth tend to be mandibular third molars, maxillary second premolars and mandibular second molars with the maxillary premolars demonstrating the greatest incidence.[5,9,10] Although most cases are idiopathic (without a known cause) in nature, this alteration can occur as a result of trauma.[5,8-10] Injury induced dilaceration usually involves anterior teeth or less frequently occurs secondary to impingement of an adjacent anatomic structure or pathologic entity (Figure 50).[10] Usually, dilaceration is not problematic but treatment difficulties may be encountered if endodontic treatment or tooth extraction are necessary.[7-10]

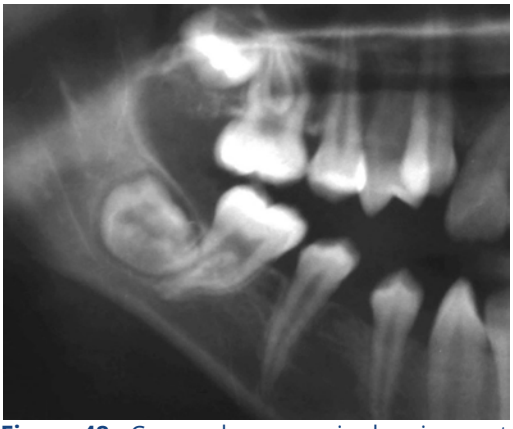


Figure 48. Cropped panoramic showing root dilaceration of maxillary and mandibular right second molar roots, teeth #2 and #31.

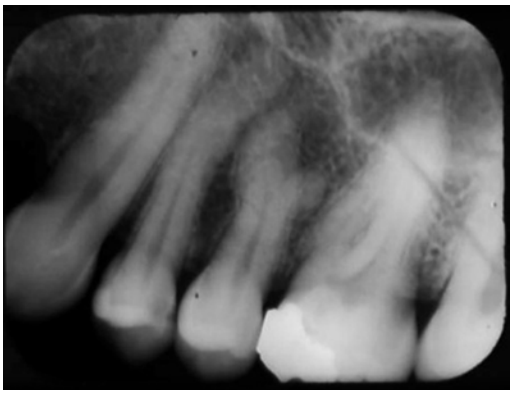


Figure 49. Periapical radiograph showing dilaceration of the maxillary left second premolar tooth #13 with supernumerary roots.

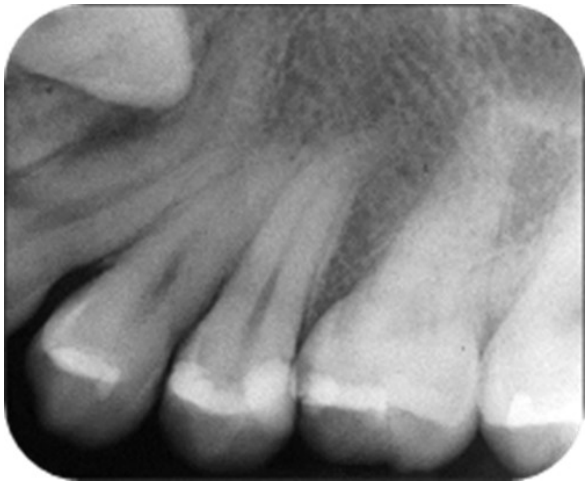


Figure 50. Periapical radiograph showing root dilaceration of the maxillary left canine tooth #11 from impingement adjacent impacted tooth.

Supernumerary Roots

Similar to supernumerary teeth, an extra root or several more can develop beyond the normal number of root(s) typical for the specific tooth type (Figures 51-53). Whether primary or permanent, any tooth can form an accessory root.[5] Third molars are especially prone to supernumerary roots, but the other molars from either arch may be involved as well as mandibular canine and premolar teeth.[5,10] At times, the extra root is easily seen radiographically while in other instances it may be fused to the other root(s) or so small as not to be visible.[5,10] Although not typically a problem, the extra root may present a challenge if endodontic treatment or extraction is indicated.[5,10]

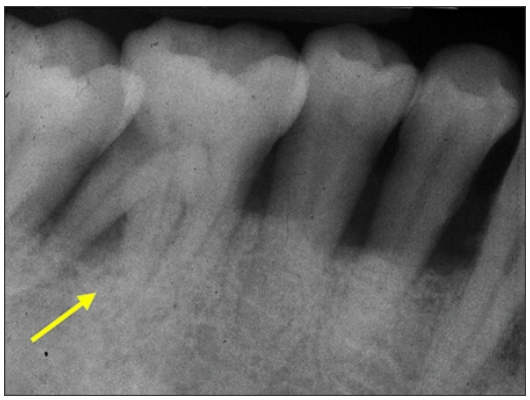


Figure 51. Periapical radiograph of a supernumerary tooth root on mandibular right first molar, #30

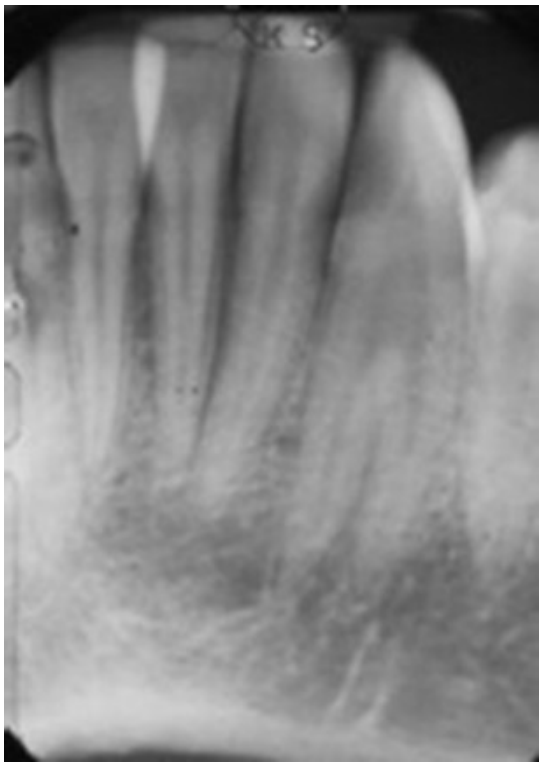


Figure 52. Periapical radiograph of a supernumerary root on mandibular left canine tooth #22.

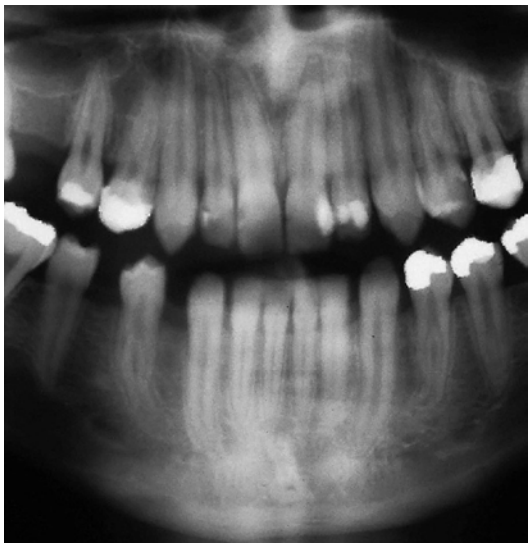


Figure 53. Cropped panoramic radiograph showing supernumerary roots on the mandibular premolar teeth, #21 and #28.

Loss of Tooth Structure

Alterations in the Structure of Teeth – Enamel and Dentin

There are several genetic disorders that manifest as developmental alterations in the structural formation of enamel and dentin.[9] These hereditary conditions are the result of gene mutations that control these key structural components.[9] The conditions that will be presented include amelogenesis imperfecta, dentinogenesis imperfecta, and dentin dysplasia.

Enamel

Amelogenesis Imperfecta

Amelogenesis imperfecta (AI) encompasses a group of inherited genetic disorders that disrupt enamel development.[16] Multiple gene mutations result in enamel aberrations such as pitted, grooved, and discolored teeth that are prone to fracture and severe wear.[16,17] Amelogenesis imperfecta affects both the primary and permanent dentitions and can occur alone without other signs or symptoms or in association with a syndrome that affects other parts of the body.[5,16-18] Amelogenesis imperfecta is also associated with other dental issues such as malocclusion, anterior open bite, altered eruption, and attrition[19-21] The clinical and radiographic manifestations can be classified into four general types: hypoplastic, hypomaturation, hypomineralization, and hypomaturation/hypoplastic with taurodontism as summarized in Table 6.[10,16,22,23] While the clinical implications vary depending on type and severity, the main treatment challenges involve esthetics, tooth sensitivity, and loss of vertical dimension.[10,19]

Hypoplastic Amelogenesis Imperfecta – Type I

Hypoplastic amelogenesis imperfecta features inadequate deposition of enamel with yellow-brown tooth coloration from the dentin below the thin enamel layer.[5,10,16] The generalized pattern of this condition demonstrates pitted enamel, often stained, usually affecting the buccal aspect of the teeth.[10,16] However, the enamel may also appear rough or smooth.[16] The crowns of the teeth look square-shaped with open contacts and flat occlusal surfaces.[5,9,10] In localized patterns, the affected teeth have horizontal rows of pits, a linear depression, or a single large area of hypoplastic enamel on the buccal middle third of the tooth.[5,10,16] The incisal edges or occlusal surfaces are usually unaffected.[5] In rare instances, there

Table 6: Amelogenesis Imperfecta – Types and Descriptions Summary.[10,16,23]

Amelogenesis Imperfecta: Types and Descriptions
Hypoplastic - Type I • Quantitative alterations in enamel deposition to complete absence of enamel • Generalized/localized patterns with varied enamel textures (pitted, smooth, rough) affecting buccal surfaces • Clinically, teeth have yellow-brown coloration with square-shaped crowns, open contacts, and flattened occlusal surfaces • Radiographically, the enamel appears as a thin radiopacity
Hypomaturation - Type II • Defective enamel maturation of normal thickness • Enamel is soft, puncturable, and fractures from the crowns • Clinically the teeth display a range of colorations: mottled brown, yellow, snow-capped • Radiographically, the enamel and dentin display the same radiodensity
Hypomineralization - Type III • Faulty enamel mineralization of normal thickness • Enamel is soft and easily fractures from the crown after eruption • Clinically, teeth are of normal shape with a range of colorations: white, yellow, orange, brown with rapid abrasion, staining, and calculus deposition • Radiographically, the enamel and dentin display the same radiodensity with the presence of unerupted teeth
Hypomaturation/Hypoplastic with Taurodontism - Type IV • Inadequate and defective deposition of enamel • Clinical features vary according to the predominant variant o Hypomaturation-hypoplastic – normal enamel thickness, mottled yellow to yellow-brown enamel with buccal pitting o Hypoplastic-hypomaturation – thin enamel with large hypoplastic areas • Radiographically, the enamel and dentin display the same radiodensitys • Taurdontism of varying degrees may be present

is total agenesis of enamel while the teeth have the shape and color of dentin.[10,16] Both the primary and permanent dentitions may be affected or the primary dentition only.[5,10,16] The hypoplastic type is the easiest to identify radiographically.[9]

Hypomaturation Amelogenesis Imperfecta – Type II

Hypomaturation amelogenesis imperfecta is a defect in the quality of the enamel with normal thickness but mottled appearance.[10,16] The enamel is soft, easily pierced, and fractures away from the crown.[5,9,10,16] There are several types of enamel mottling that are associated with the underlying inheritance pattern.[10,16,22] The mottling ranges from pigmented brown, translucent to opaque white, yellow white, and snow-capped enamel.[5,9,10,16] The enamel mottling can mimic dental fluorosis, but the pattern is different in that AI affects all teeth similarly rather than time-based incidence.[9,22] Radiographically, the enamel appears to have the same radiodensity as dentin.[9,10,16]

Hypomineralization Amelogenesis Imperfecta – Type III

Hypomineralization amelogenesis imperfecta, prior to eruption, shows that the teeth are of normal size and shape with usual enamel thickness.[10,16] Upon eruption, however, the inadequately mineralized, soft enamel quickly fractures away from the crown resulting in rapid wearing of the exposed dentin, stain uptake causing darkening, tooth sensitivity, and excessive calculus deposition.[10,16,22] The enamel color can range from white to creamy yellow.[24] Anterior open bite and the presence of unerupted teeth can be observed.[10,16] Radiographically, the enamel appears to have the same degree of radiodensity as dentin.[9]

Hypomaturation/Hypoplastic Amelogenesis Imperfecta with Taurdontism – Type IV

This type of amelogenesis imperfecta displays variant patterns of hypomaturation combined with hypoplasia affecting both primary and permanent dentitions diffusely.[5,16] The manifestations of the hypomaturation/hypoplastic variants are slight and depend on the predominance of one pattern over the other.[16] In the hypomaturation-hypoplastic variant with taurodontism, the teeth have normal enamel thickness but appear mottled yellow white to yellow brown coloration with pitted buccal surfaces.[16] Taurodontism with large pulp chambers and thin radiopaque enamel are observed on radiographic images.[10,16] The hypoplastic-hypomaturation variant is similar but the enamel is thinner with large areas of hypoplasia.[10,16,23] Radiographically, the enamel has the same radiodensity as dentin with single-rooted teeth displaying enlarged pulp chambers and posterior teeth varying degrees of taurodontism.[10,16]

Dentin

Genetic disorders of the dentin are broadly classified into two main categories, dentinogenesis imperfecta (DI) and dentin dysplasia (DD).[25] Two classification systems for dentin disorders based on phenotypic presentation have been broadly accepted, one proposed by Shields and the other by Witkop.[5,23,26] Most widely used, the Shields classification divides dentinogenesis imperfecta into three types and dentin dysplasia into two.[26,28] Ciola modified the classification by adding a third dentin dysplasia type, a combination of DD I and II and Carroll et al. further updated the classification with subgroups based on radiographic features.[28,30] More recent advances in molecular genetic research have proven that mutations in dentin sialphosphoprotein (DSPP) cause dentiongenesis imperfecta types II & III and dentin dysplasia type II, all variations in the severity of the same pathology.[31,32] As a result, a new classification focused on isolated forms of DI and a modification of Shields classification based on the causative mutation and genetic testing have been proposed to improve diagnoses.[31,32]

Dentinogenesis Imperfecta

Dentiogenesis imperfecta (DI) is a genetic abnormality primarily of dentin with characteristic clinical and radiographic features. The aberrations are caused by autosomal dominant mutations in several different genes associated with collagen synthesis.[31] Dentinogenesis imperfecta is the most frequent dentinal disorder which demonstrates varying degrees of severity: mild, moderate, and severe.[31,32]

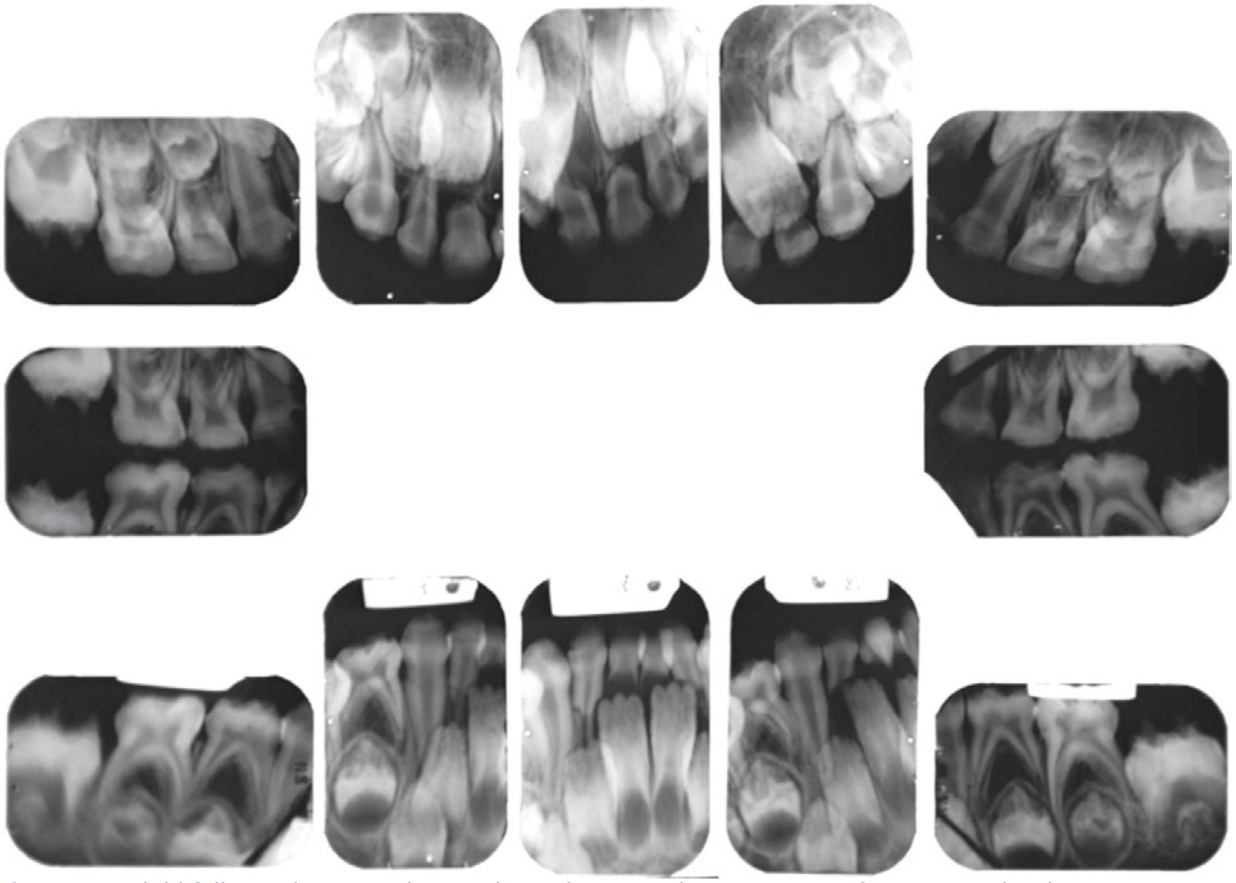


Figure 54: Child full mouth survey showing hypoplastic amelogenesis imperfecta, generalized pattern. Note the loss of enamel and flattened occlusal surfaces in the primary dentition. A thin layer of enamel can be seen radiographically.

The mild form of dentiogenesis imperfecta, DD-II in the Shields Classification, affects the primary dentition.[25,26,31] The permanent teeth are normal color, shape, and height clinically while radiographically the pulp has a thistle-tube or flame configuration with a large pulp chamber, thin root canals and multiple pulp stones (Figure 55).[9,25,33]

The moderate form of dentinogenesis imperfecta, DI-II or DGI-II in the Shields Classification, is the most common with the primary dentin more severely affected than the permanent dentition.[26,31] Clinically, the teeth display a color range from grey blue, brown, and opalescent but the enamel becomes displaced resulting in rapid attrition of the underlying dentin.[25,31,32] The radiographic appearance is pathognomonic, demonstrating bulbous crowns, cervical constriction, short, thick roots and pulp obliteration (Figure 56).[31,32]

The severe form of dentinogenesis imperfecta, DI-III or DGI-III in the Shields Classification, known as the Brandywine isolate, is very rare.[23,26,31,32] It is associated with a sub-population in Maryland which has the highest incidence of dental genetic disorders; 1:15.[34] The severe form of DI affects both dentitions with bluish amber opalescent teeth, bulbous crowns, and attrition, much like the moderate form.[31,32] The main differentiation is significant enlargement of the pulp causing reduction of the dentin and multiple pulp exposures in the primary dentition described as shell teeth.[26,31,32,35] The permanent dentition demonstrates obliterated pulp chambers as do individuals that have shell teeth in their primary dentition.[23]

Dentin Dysplasia

Dentin dysplasia (DD) is a rare developmental anomaly of unknown etiology that affects

both the primary and permanent dentitions with a prevalence of 1:100,000.[31,32,36,37] Dentin dysplasia or radicular dentin dysplasia (Shields Classification DD-I) demonstrates clinically normal teeth in color and morphology but with short, abnormally shaped, or absent roots.[9,26,29,31,36] Teeth can be misaligned and mobile resulting in premature exfoliation with little or no trauma.[9,38] Partial or complete obliteration of the pulp chambers and canals often with a remnant crescent-shaped pulp parallel to the CEJ and periapical radiolucencies without associated pathology are observed in some cases (Figure 57).[10,25]

Acquired Alterations of Tooth Structure

Loss of tooth structure typically occurs through tooth wear, a normal physiological function associated with aging. Wear of the teeth can be caused by several processes including attrition, abrasion, abfraction, and erosion. These processes are acquired alterations that occur subsequent to tooth development and eruption.

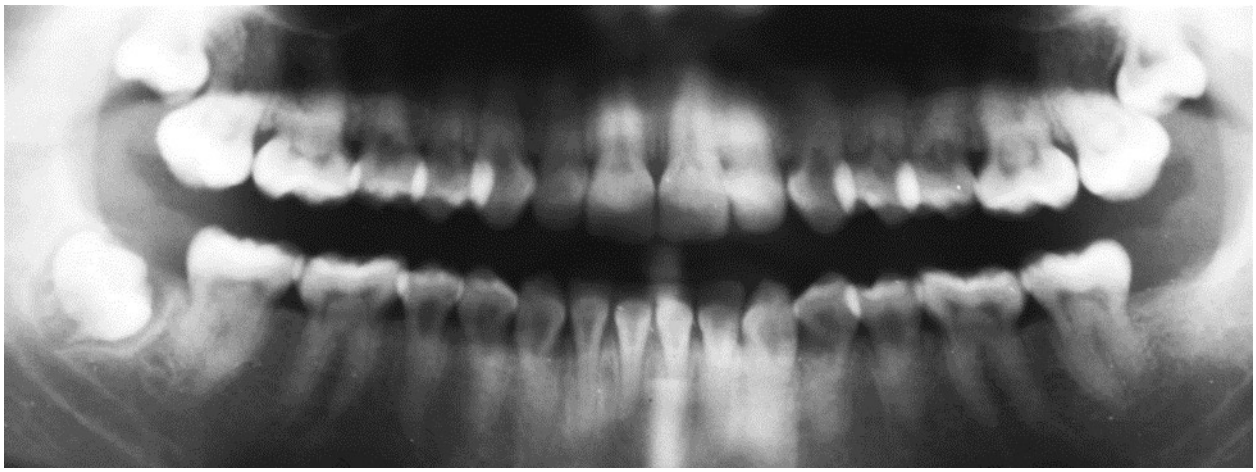


Figure 55: Cropped panoramic image showing mild DI with thistle-tube or flame pulp configurations with enlarged chambers, thin canals and pulp stones.

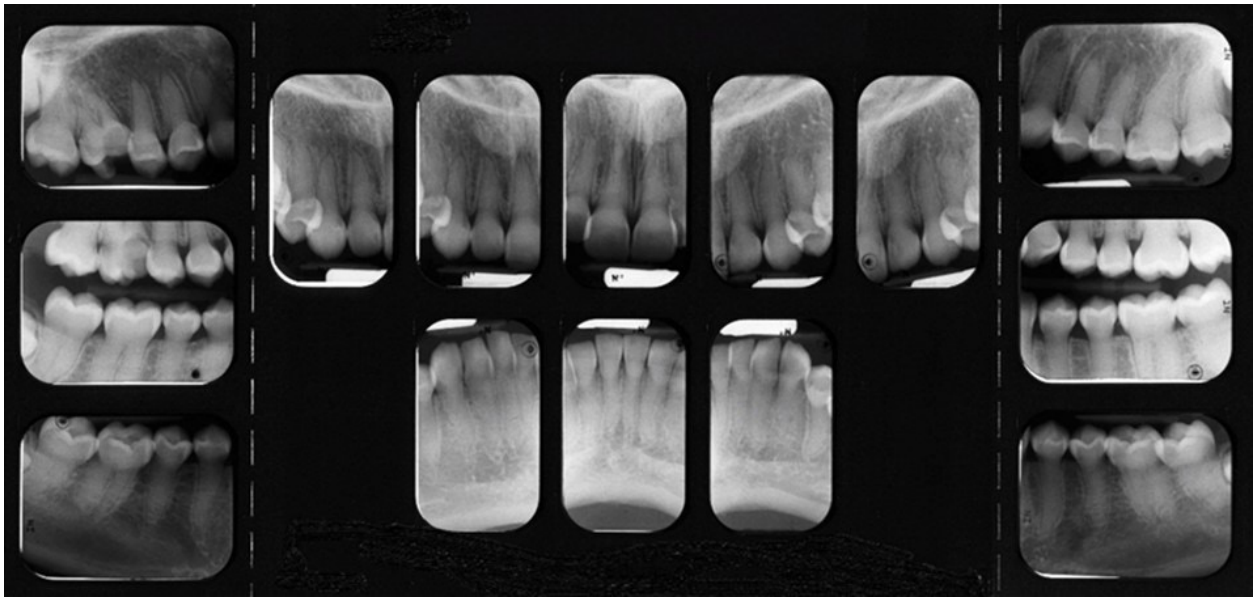


Figure 56: Full mouth survey demonstrating moderate dentinogenesis imperfecta. Note the bulbous crowns, cervical constriction, and pulp obliteration.

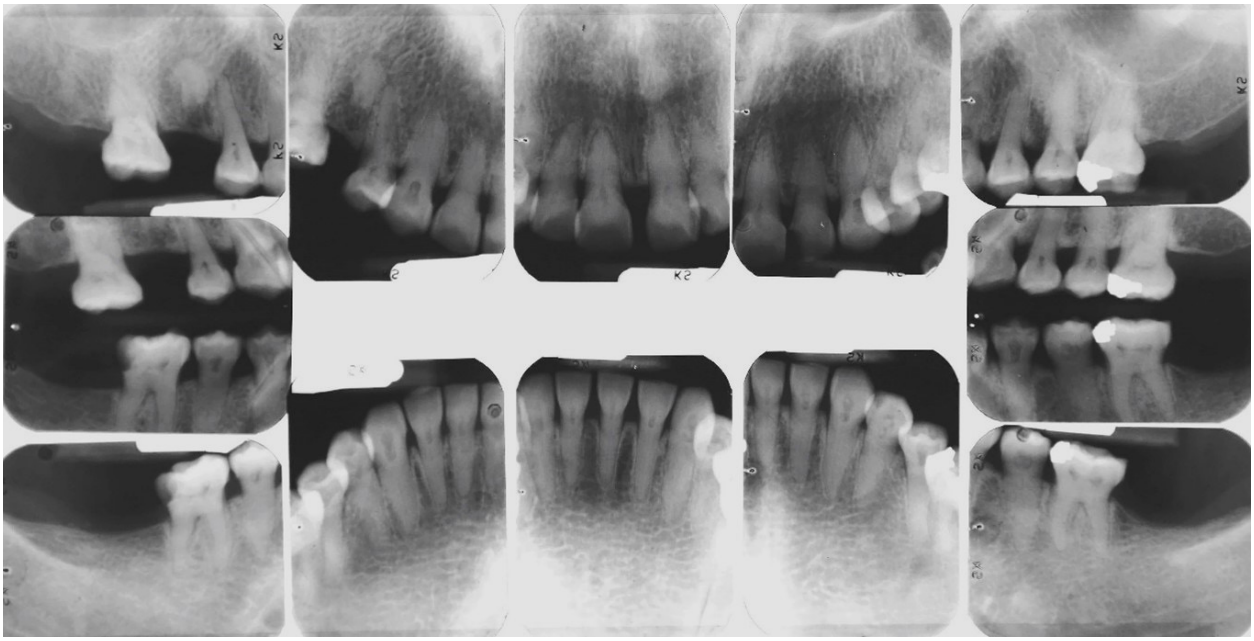


Figure 57: Full mouth survey demonstrating dentin dysplasia. Note short roots, partial and complete pulp chamber and canal obliteration of teeth.

Table 7: Dentiongenesis Imperfecta and Dentin Dysplasia – Key Features.[31,32]

Dentinogenesis Imperfecta and Dentin Dysplasia – Key Features
Dentinogenesis Imperfecta (DI) **Mild** • Primary and permanent dentitions affected • Clinical appearance: teeth normal in color, shape, height • Radiographic appearance: o Pulp configuration thistle-tube or flame shaped o Large pulp chambers with thin canals o Multiple pulp stones **Moderate** • Most common form of DI • Clinical appearance: o Color range - grey blue, brown, opalescent o Enamel lost causing rapid dentin attrition

Table 7: Cont.

- Radiographic appearance:
  - Bulbous crowns with cervical constriction
  - Short thick roots
  - Pulp obliteration

Severe

- Very rare Brandywine isolate
- Affects both primary and permanent dentitions
- Clinical appearance:
  - Teeth blue, amber opalescent color
  - Enamel lost with rapid attrition
- Radiographic appearance:
  - Primary dentition
    - Pulp enlargement, dentin reduction, pulp exposures
    - Shell teeth appearance
  - Permanent dentition
    - Bulbous crown
    - Pulp obliteration

Dentin Dysplasia (DD)

- Primary dentition and permanent dentitions affected
- Clinical appearance:
  - Teeth normal in color and shape
  - May be misaligned, mobile, or lost prematurely
- Radiographic appearance:
  - Short, abnormal or absent roots
  - Partial or complete pulp chamber and canal obliteration
  - Chevron-shaped pulp chamber parallel to the CEJ remnant possible
  - Periapical radiolucencies may be present without associated pathology

Attrition

Attrition of the tooth structure is the result of tooth-to-tooth contact during mastication and occlusion.[5,39] This condition affects the incisal and occlusal surfaces of the teeth and can be exacerbated by contact or ingestion of abrasive materials, abrasive foods, and behaviors like teeth grinding or bruxism.[5,9,39] Attrition is quite prevalent in adults and tends to be more severe in men.[9] Both clinical and radiographic manifestations can be observed. Clinically, the incisal edges demonstrate broadening and wear facets will be evident on the cusps and ridge anatomy of posterior teeth (Figures 58-60).[5,9,39] The dentin can become exposed and the wear facets, which appear flat and shiny, can deepen over time, and become stained.[9] Radiographically, the teeth will appear flattened and shorter coronally with reduction in the size of the pulp chambers and canals due to deposition of secondary dentin.[9,39] Ultimately, the pulpal structures can become completely obliterated.[9,39]

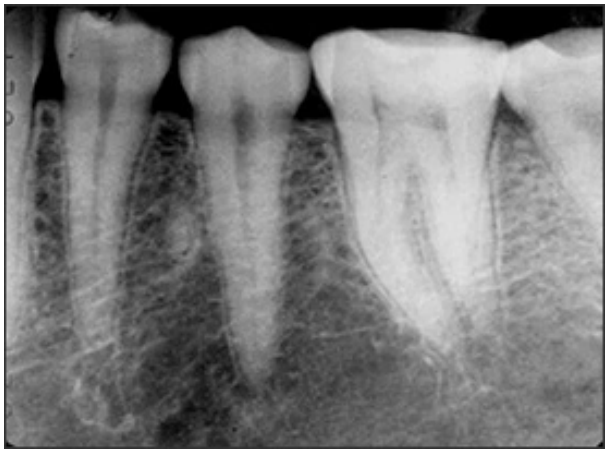


Figure 58. Periapical radiograph of mandibular left posterior teeth with attrition.

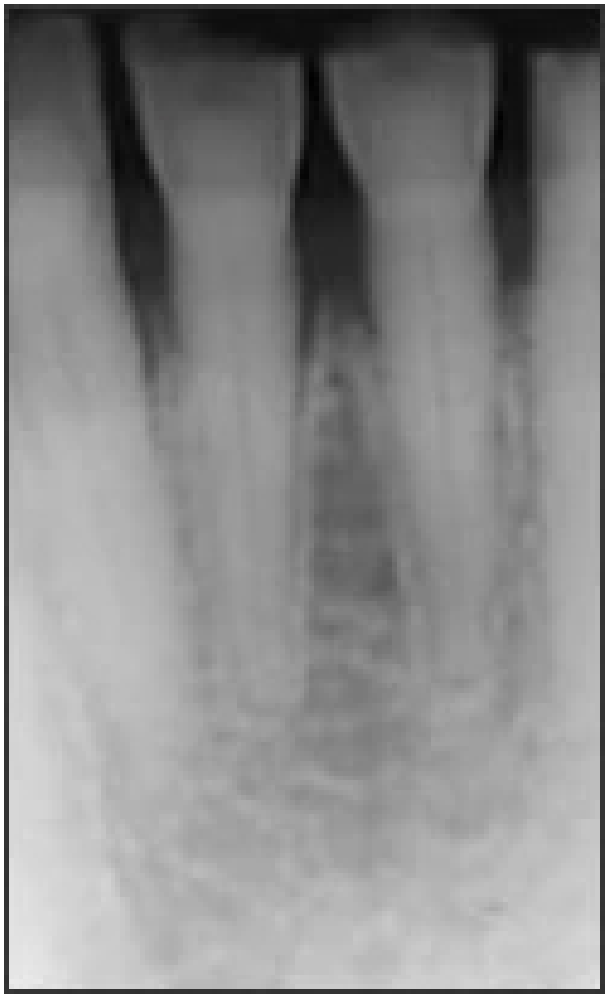


Figure 59. Periapical radiograph of mandibular anterior teeth with attrition.

Abrasion

In abrasion, the loss of tooth structure is not from physiologic wear but rather from contact with substances or devices that cause friction.[9] These frictional forces are often associated with improper toothbrush and flossing habits that wear away the tooth structure.[5,9,39] Toothbrush abrasion can be the result of vigorous scrub-brushing methods and the use of abrasive toothpastes or powders. The back-and-forth scrubbing action produces V-shaped notches or grooves in the cervical aspect of the tooth buccally near the gingival tissue (Figure 61).[9] The side of the dentition that is most severely affected is the side opposite the tooth brusher's dominant hand.[5,9,39] In addition, wear can result from incorrect dental floss usage producing interproximal cervical notches or craters, deeper on the distal aspect than the mesial.[9,39] These areas of reduced tooth structure appear radiolucent on radiographs and may be misinterpreted as cervical or root caries. Parafunctional habits can be causal factors such as improper toothpick usage, hairpin opening, thread-biting, pipe smoking as well as grooves produced by partial denture clasp friction.[9,39]

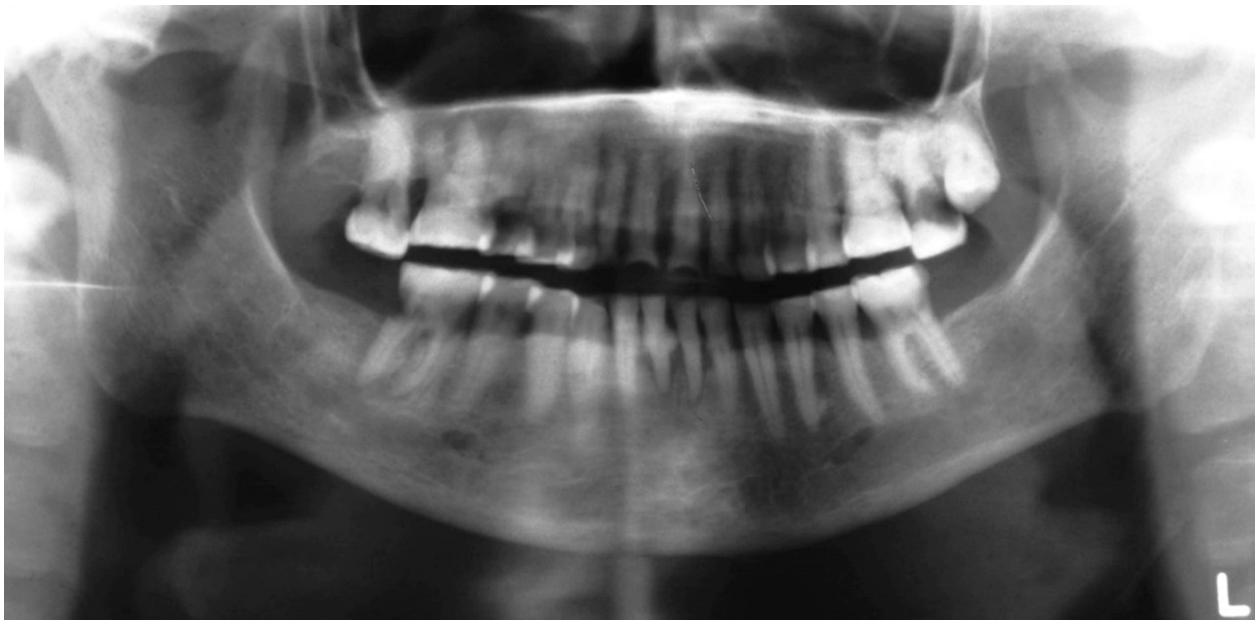


Figure 60. Panoramic radiograph demonstrating universal attrition of the permanent dentition. Note the extensive flattening of incisal and occlusal surfaces.

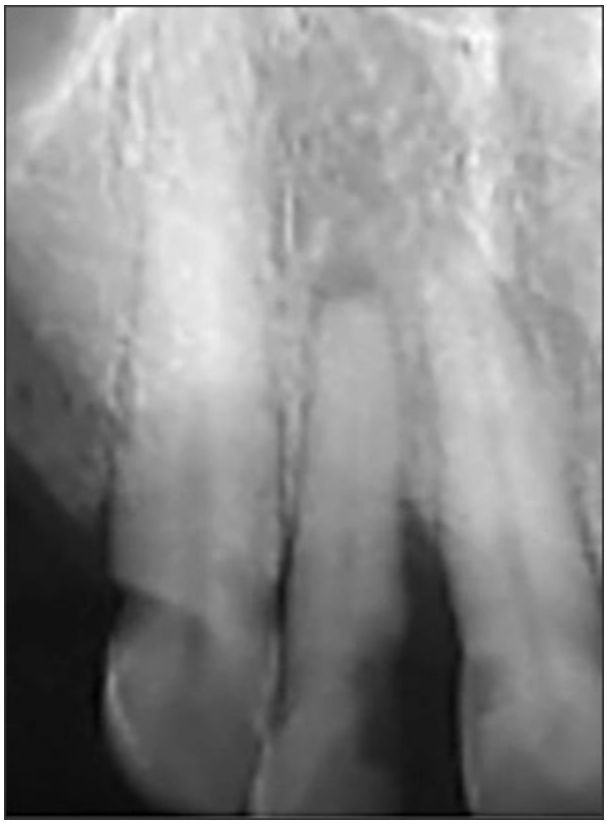


Figure 61. Periapical radiograph of the maxillary right canine tooth #6 with classic toothbrush abrasion.

Abfraction

Heavy occlusal forces that repetitively flex the tooth structure over time, like bruxism, can cause cervical loss of tooth structure.[16] This process, known as abfraction, produces deep, narrow, wedge-shaped defects on the buccocervical aspect of the teeth.[5,39] It usually involves a single premolar or molar tooth with the adjacent teeth remaining unaffected.[5,39] These defects can mimic abrasion, erosion, and cervical caries but solitary tooth involvement and subgingival defect occurrence can help differentiate abfraction from the other entities.[39]

Erosion

Erosion is the loss of tooth structure from a non-bacterial, chemical process.[9,39] Typically, the chemical agent is acid from acid reflux or regurgitation, frequent vomiting (e.g., bulimia-associated), excessive home tooth-whitening, high acidic dietary intake, or occupationally related acid exposure.[5,39,40] The location and pattern of erosion is a key to the likely causal agent. Erosion affecting the facial surfaces of the cervical aspect of maxillary anterior teeth often is indicative of dietary acid from habits such as lemon-sucking.[39] Regurgitated acids

typically affect the palatal and lingual surfaces of the maxillary anterior teeth and the occlusal surfaces of maxillary and mandibular posterior teeth.[5,39,40] Clinically, the areas of erosion look smooth and shiny where the enamel has been removed and the dentin is exposed.[5,9,39,40] However, these exposed areas of dentin can subsequently take-up stain.[7] Radiographically, these eroded coronal depressions appear radiolucent with smooth margins (Figure 62A and 62B).

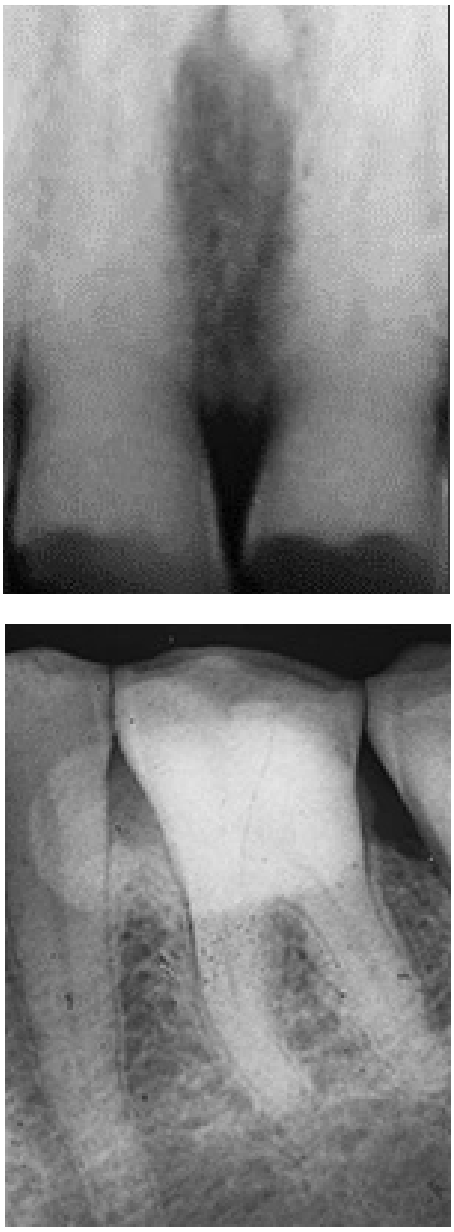


Figure 62A and 62B. Periapical radiographs demonstrating erosion of anterior (A) and posterior (B) teeth.

Resorptive Processes

Normal resorptive processes occur to allow the exfoliation of primary teeth in preparation for the eruption of the permanent teeth. However, there are other resorptive processes that can result in the destruction or removal of tooth structure including external resorption, which affects the outer surface of the tooth, and internal resorption, which affects the pulp chamber and canal(s) housed inside the tooth. Some cases may be referred to as idiopathic when no causal factor can be identified for the resorption.

External Resorption

A common entity, external resorption tends to be mild, affects one or more teeth, and usually is only discovered upon radiographic examination.[5,9] It most commonly involves the dentin and cementum of the root surfaces of the teeth but the crown of an unerupted tooth (Figures 63, 64) can be affected as well.[9] In most situations, it does not present any substantial clinical problems. However, 10% of patients demonstrate significant external resorption.[5] A number of factors are associated with this condition such as excessive mechanical forces, extreme occlusal forces, tooth reimplantation, dental trauma, as well as systemic conditions and pathologic entities.[39] Root resorption may be an aftermath of orthodontic treatment when heavy forces are applied to the teeth, and significant tooth movement is required to correct malocclusion (Figure 65).[5] A genetic component may be at play in situations in which the resorption is severe.[5]

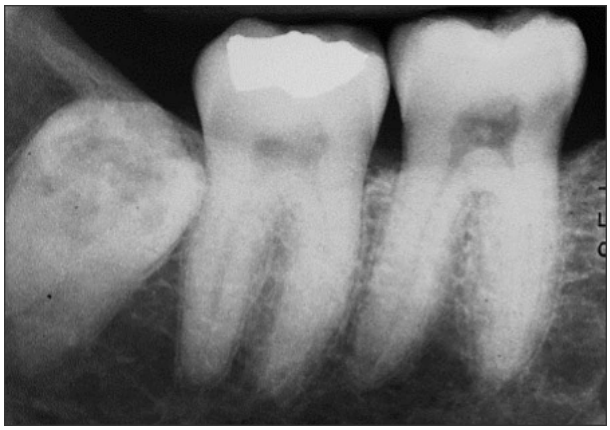


Figure 63. External resorption of impacted mandibular right third molar tooth, #32.

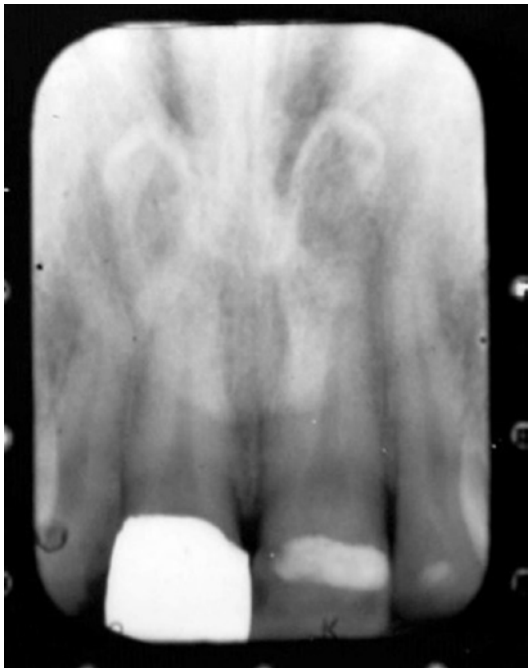


Figure 64. External resorption of vertical inverted impacted mesiodens.

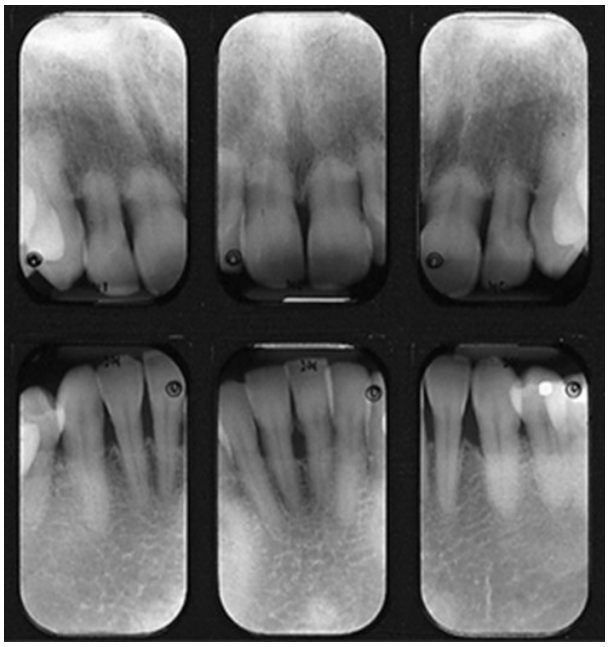


Figure 65. Anterior periapical radiographs of multiple teeth with external resorption, particularly severe on the maxilla.

Internal Resorption

While less common than external resorption, internal resorption can affect both the primary and permanent teeth.[9] When internal resorption does occur, usually it is associated with trauma to the tooth and inflammation of the pulp tissues.[5,9] The resorption involves the pulp chamber or pulp canal and the adjacent dentin, giving rise to an enlarged or expanded pulp structure (Figure 66A and 66B).[5,39] The permanent central incisors and the first and second molar teeth are most frequently involved.[9] Typically asymptomatic, internal resorption can elicit pain if the resorption results in major pulpal inflammation. When the resorptive lesion affects the coronal pulp structure, a pink coloration known as the pink tooth of Mummery may be observed clinically.[5] If the root and pulpal canal are involved and the integrity of the normal canal anatomy is breached, an oval or balloon-shaped radiolucent area of expansion may be visualized radiographically (Figure 67).[5,39] In severe cases, perforation of the lateral root can occur. If the tooth remains sound, endodontic therapy can be rendered to stop the resorptive process, or in cases of root perforation, surgical endodontic retrofill procedures may be necessary.[39] Ultimately, extraction may be required if the tooth is unrestorable.[39]

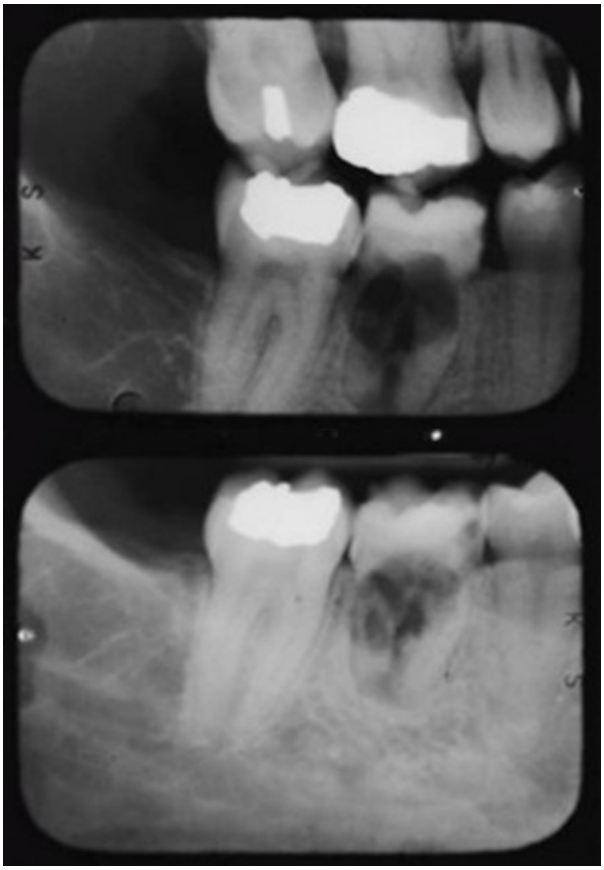


Figure 66A and 66B. Bitewing (A) and periapical (B) radiographs showing internal resorption of mandibular right first molar tooth #30.

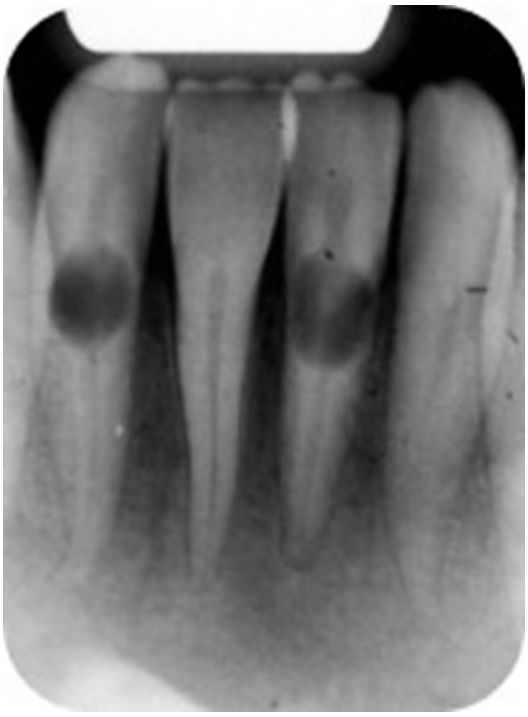


Figure 67. Periapical radiograph demonstrating ballooning of the pulp canal in mandibular central and lateral incisor teeth, #24 and #26.

Dentinal Deposition

Secondary and Tertiary Dentin

Secondary dentin is that which is deposited in the pulp structures. This process occurs after the primary dentin has fully formed, usually as part of aging.[9] Dentinal deposition can be a response to a variety of conditions such as moderate carious lesions, prolonged tooth trauma, deep restorative treatments, and tooth-wearing processes like attrition (Figure 68A and 68B), abrasion, and erosion.[5,9] In such instances, the term tertiary dentin may be applied, suggesting that the additional dentin is deposited in response to conditions unrelated to aging.[5,9]

Age-related secondary dentin deposition diminishes the size of the pulp chamber, especially the pulp horns early in the process, and the pulp canals.[9] These changes can be visualized radiographically. This process may eventually result in obliteration of all pulp structures.[7,9] Tertiary dentinal deposition tends to be a more rapid, localized process by comparison.[5]

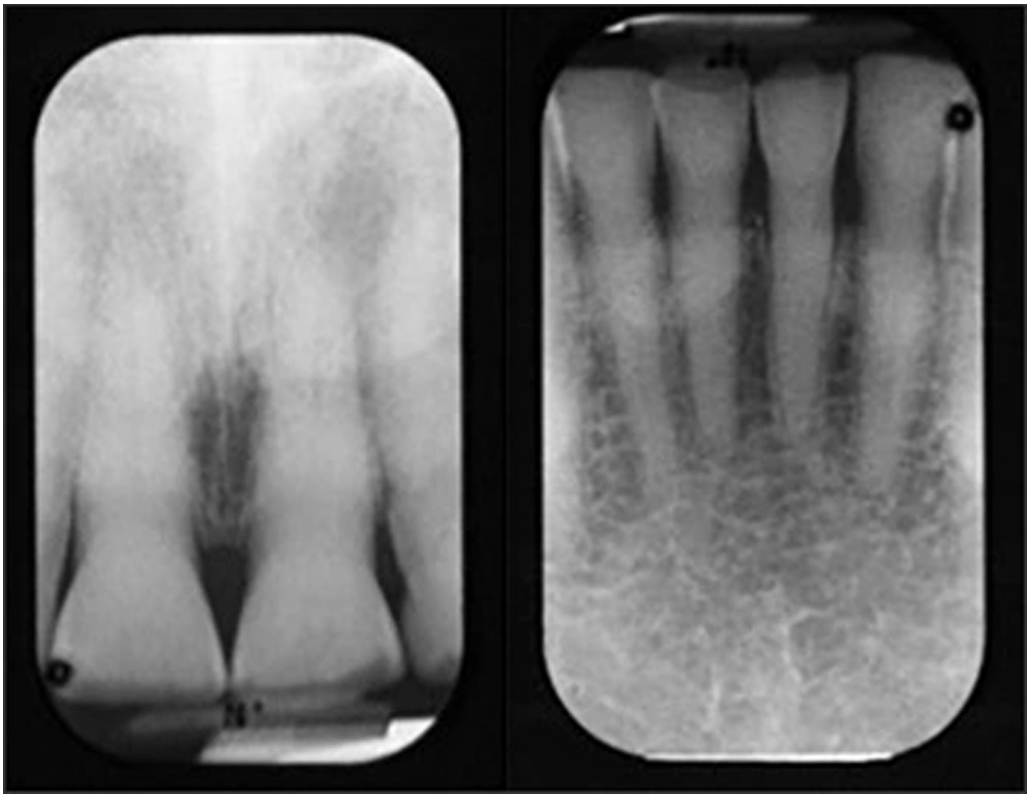


Figure 68A and 68B. Periapical radiographs showing secondary dentin deposition in maxillary (A) and mandibular (B) anterior teeth most likely due to attrition.

Pulpal Calcifications

Several other types of calcifications can affect the pulpal structures of the teeth including pulp stones and pulpal sclerosis. The stones tend to be more localized while pulpal sclerosis is more generalized.

Pulp Stones

Pulp stones are small focal calcifications that occur within the pulp structures of the teeth. Of unknown origin, pulp stones are rather common and affect the teeth of both the young and old.[9] Most stones are so tiny that they cannot be seen radiographically, while others are large and calcified enough to be visualized.[5] Their size, shape, number, and density are variable, and they can be found in every type of tooth, but especially the molar teeth (Figure 69A and 69B).[9] No treatment is required.

Pulpal Sclerosis

Another alteration of the pulp structures, pulpal sclerosis, produces sclerotic changes that can result in complete obliteration of the pulp chamber and canals (Figures 70, 71). Although the etiology is unknown, total obliteration may be a result of trauma to the affected tooth.[5] The calcifications are diffuse and ill-defined radiographically, with a strong association with older age.[9] This process is asymptomatic and requires no treatment. If endodontic therapy is indicated, the calcification may present difficulty in completing the root canal procedure.[9]

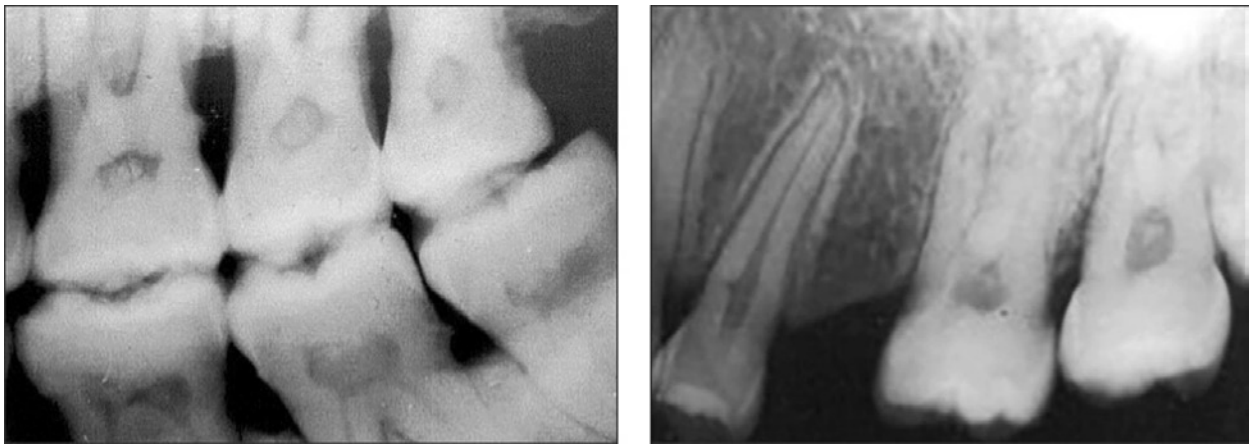


Figure 69A and 69B. Bitewing (A) and periapical (B) radiographs showing posterior pulp stones of varying sizes in the maxillary and mandibular molar teeth.

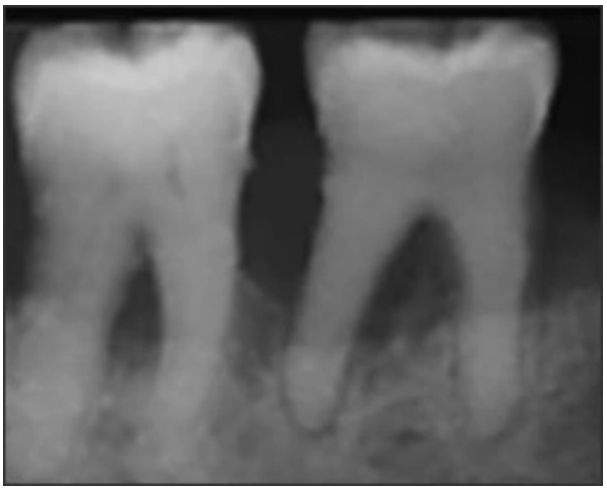


Figure 70. Periapical radiograph of pulpal sclerosis with total obliteration of pulp structures of mandibular right molar, #30.

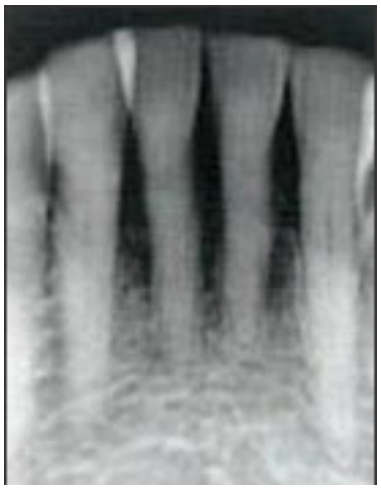


Figure 71. Periapical radiograph of pulpal sclerosis in the mandibular anterior teeth.

Summary

The normal radiographic appearance of the teeth and their supporting structures throughout the various stages of tooth development, eruption, and maturation are important to recognize and understand. This foundational knowledge provides a sound baseline to assist the clinician in identifying and describing deviations from normal. Dental anomalies associated with the eruption, number, size, and shape of the teeth include a variety of entities, some which may have important treatment considerations and implications. Acquired alterations of tooth structure such as attrition, abrasion, abfraction, and erosion may affect the appearance or function of the teeth. In some cases, clinical intervention may be required to preserve the remaining tooth structure and maintain normal function. This information will facilitate differentiation between normal and abnormal tooth structure and enhance the ability to recognize and describe various structural anomalies.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: *www.dentalcare.com/en-us/ce-courses/ce651/test*

1. Which structural component comprises the majority of the tooth?

A. Enamel

B. Cementum

C. Dentin

D. Pulp

2. The cancellous bone in the posterior mandible _____.

A. consists of many thin rods and plates of bone

B. contains numerous tiny islands of bone marrow

C. displays a fine granular appearance radiographically

D. has large sparse trabeculae and marrow spaces

3. Which phrase best describes the periodontal ligament space?

A. Cushions the tooth via flexible collagen fibers

B. Lines the entire periphery of the tooth socket

C. Located approximately 0.5 to 2.0 mm from the CEJ

D. Visualized as a radiopaque structure on dental images Lines the entire periphery of the tooth socket

4. Which primary teeth erupt between nine and thirteen months of age?

A. Mandibular canine teeth

B. Mandibular first molars

C. Maxillary lateral incisors

D. Maxillary second molars

5. The permanent maxillary canine teeth erupt _____.

A. after the mandibular canine teeth

B. after the maxillary second molars

C. before the mandibular first premolars

D. before the maxillary lateral incisors

6. Which feature is characteristic of tooth ankylosis?

A. Dentinal fusion with the coronal enamel

B. Involvement of the permanent dentition

C. Occlusal plane submersion of the crown

D. Widening of the periodontal ligament space

7. What is the definition of the term anodontia?

A. Absence of the third molars

B. Lack of tooth development

C. One or more missing teeth

D. Six or more missing teeth

8. Which is the most common type of supernumerary tooth?

A. Distodens

B. Distomolar

C. Mesiodens

D. Paramolar

9. Which phrase is most closely associated with microdontia?

A. Frequently associated with hyperdontia

B. Linked to teeth crowding and malocclusion

C. Tendency for occurrence in the male gender

D. Usually involves the maxillary lateral incisor

10. Gemination is an anomaly in which _____.

A. a single tooth tries to divide into two

B. a tooth forms larger than normal size

C. two adjacent teeth join to form one tooth

D. two separate teeth fuse along the root

11.Which of the following is NOT associated with dens evaginatus?

A. Coronal cusp-like projection of enamel

B. Mandibular premolar teeth most affected

C. Produces an involution of tooth structure

D. Found commonly in aboriginal racial groups

12.According to Oehlers Classification, the most severe form of dens invaginatus involves the _____.

A. crown cingulum

B. incisal edge

C. pulp chamber

D. tooth root

13.Which phrase accurately describes taurodontism?

A. Demonstrates elongated pulp chamber anatomy

B. Greatest incidence is among Asian populations

C. Is frequently associated with fractured molar teeth

D. Tendency toward a female gender predilection

14.Hypercementosis is characterized by _____.

A. Innumerous tiny nobs of cementum

B. Lobular, misshaped root anatomy

C. Outcroppings of secondary dentin

D. Short stubby root configuration

15.Of the teeth listed, which is most frequently affected by root dilaceration?

A. Mandibular canine teeth

B. Mandibular first molars

C. Maxillary second premolars

D. Maxillary third molar teeth

16. Which type of amelogenesis imperfecta is associated with taurodontism?

A. Type I

B. Type II

C. Type III

D. Type IV

17. Which is a distinguishing radiographic feature of moderate dentinogenesis imperfecta?

A. Flame-shaped pulp

B. Multiple pulp stones

C. Pulp obliteration

D. Shell teeth

18.What causes erosion of the tooth structure?

A. Chemical agent exposure

B. Friction-induced wearing

C. Heavy occlusal forces

D. Tooth-to-tooth contact

19. Each description of external resorption is correct EXCEPT one. Which is this EXCEPTION?

A. Linked to excessive mechanical forces

B. Produces a distinct tooth discoloration

C. Root dentin and cementum are affected

D. Usually involves more than one tooth

20.Which best describes pulp stones?

A. Affected teeth have a long trauma history

B. Correlated strongly with increased age

C. Pulp chamber is diminished in overall size

D. Focal calcifications within chambers/canals

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Additional Resources

- No Additional Resources Available

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Gail F. Williamson is Professor Emerita of Dental Diagnostic Sciences, Department of Oral Pathology, Medicine and Radiology at Indiana University School of Dentistry in Indianapolis, Indiana. She received an AS in Dental Hygiene, BS in Allied Health, and MS in Education, all from Indiana University. A consummate educator, Prof. Williamson has received numerous awards for teaching excellence during her academic career including the 2013 Outstanding Teacher of the Year Award from the Indiana University School of Dentistry and the 2018 Gordon J. Christensen Lecturer Recognition Award from the Chicago Dental Society. She is a co-author of several radiology textbooks and author/co-author of multiple book chapters, journal articles and continuing education monographs. She has held numerous positions in several professional organizations including the American Academy of Oral and Maxillofacial Radiology and the American Dental Education Association. She has presented continuing education courses on topics in oral and maxillofacial radiology nationally.

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