

## Abnormalities of the Teeth

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## Abnormalities of teeth

- Environmental alterations
- Developmental alterations

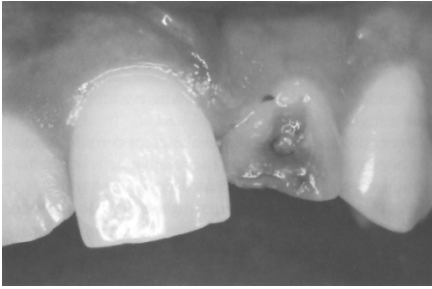
## Environmental alterations

- Effects on tooth structure development
  - Localized
  - Systemic
- Postdevelopmental structure loss
- Discoloration of teeth
- Localized disturbances of eruption

## Local factors associated with enamel defects

- Trauma
- Local infection
- Irradiation

## Enamel hypoplasia



## Enamel hypocalcification



## Systemic factors associated with enamel defects

- Infections
- Medications
- Inherited diseases
- Metabolic disorders
- Malnutrition
- Birth-related trauma

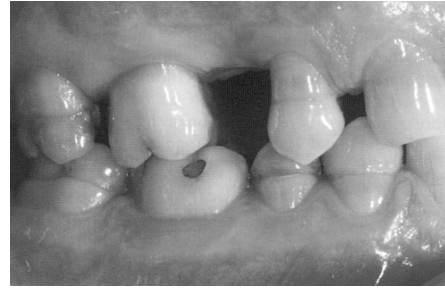
## Enamel hypoplasia



Enamel hypoplasia



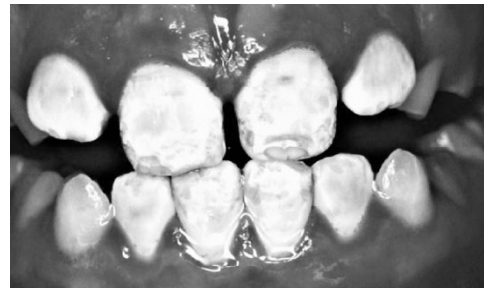
Enamel hypoplasia



Enamel hypoplasia-Rickets



Fluorosis



## Fluorosis



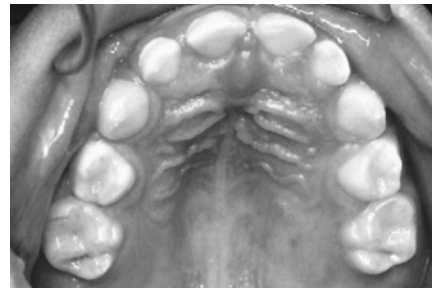
## Postdevelopmental tooth loss

- Tooth wear
  - Attrition
    - Caused by tooth to tooth contact
  - Abrasion
    - Caused by external agent
  - Erosion
    - Caused by chemical process
- Internal resorption
- External resorption

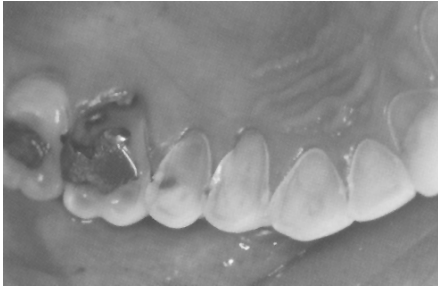
## Attrition



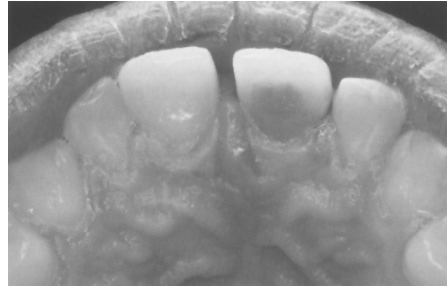
## Attrition



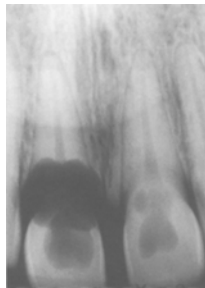
Erosion-Bulemia



Internal resorption



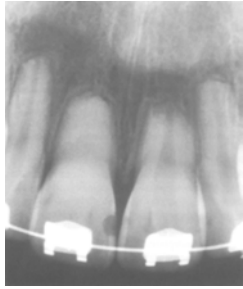
Internal resorption



External resorption



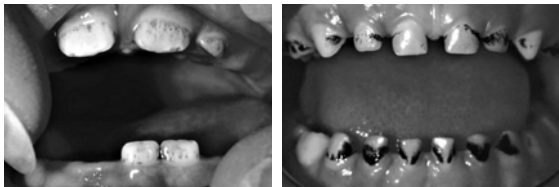
## External resorption



## Environmental discoloration

- Extrinsic
  - Bacteria
  - Iron
  - Tobacco
  - Food and beverage
  - Restorative materials
  - Medications

## Iron stain



## Environmental discoloration

- Intrinsic
  - Erythropoietic porphyria
  - Hyperbilirubinemia
  - Trauma
  - Medications

## Erythropoietic porphyria

- Autosomal recessive disorder of porphyrin metabolism that results in increased synthesis and excretion of porphyrins
- Diffuse discoloration of dentition results
- Teeth appear red-brown and exhibit a red fluorescence when exposed to UV light
- Porphyrin present in enamel and dentin of deciduous teeth so discoloration worse
- Only dentin of permanent teeth affected

## Erythropoietic porphyria



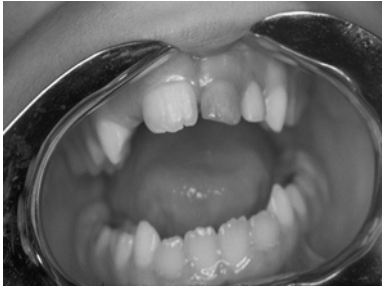
## Hyperbilirubinemia

- Excess levels of bilirubin in blood
- Bilirubin can accumulate in interstitial fluid, mucosa, skin and developing teeth
- Causes include-
  - Erythroblastosis fetalis
    - A hemolytic anemia of newborns secondary to blood incompatibility
  - Biliary atresia
    - A sclerosing process of the biliary tree
  - Premature birth
  - Internal hemorrhage

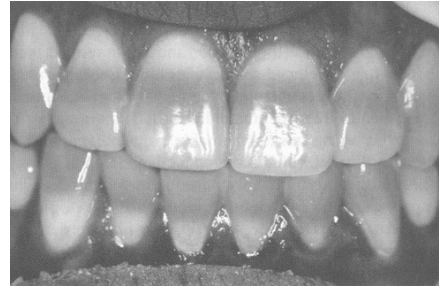
## Hyperbilirubinemia



Trauma



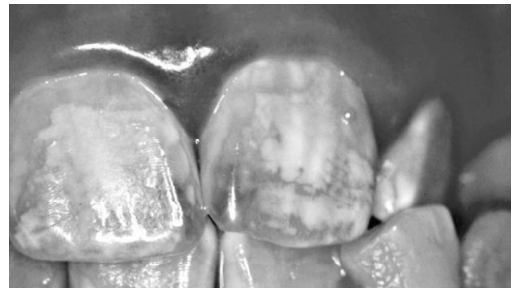
Tetracycline stain



Tetracycline stain



Fluorosis





## Localized disturbances of eruption

- Ankylosis
- Natal teeth

## Ankylosis

- Cessation of eruption after emergence occurring from an anatomic fusion of tooth cementum or dentin to alveolar bone
- Etiology unknown-trauma, local change of metabolism, thermal irritation, and genetic predisposition have been suggested
- Can occur at any age but is clinically most evident when it develops during first two decades of life
- Peak prevalence- 8-9 years of age

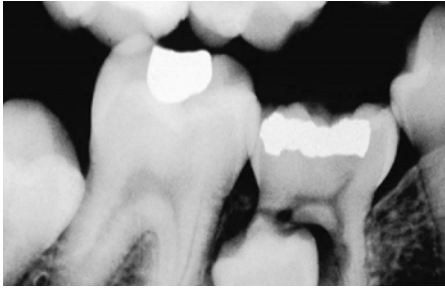
## Ankylosis

- Reported prevalence of clinically detectable ankylosis- 1.5% to 9%
- Primary molars are most commonly involved teeth with most cases in mandible
- Radiographic findings
- Sound on percussion
- Treatment considerations

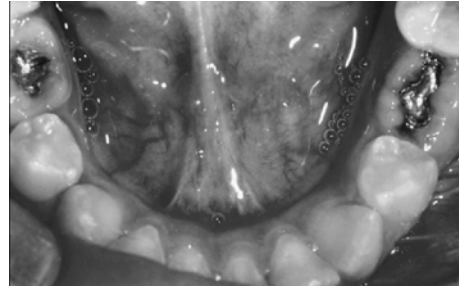
## Ankylosis



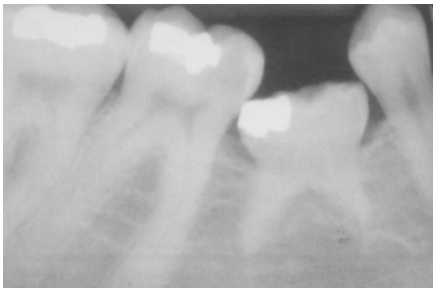
## Ankylosis



## Ankylosis



## Ankylosis



## Natal teeth

- Usually prematurely erupted primary teeth
- Present at birth
- Prevalence- 1 in 2000
- Neonatal teeth erupt within first month
- 85% are lower incisors, 11% maxillary incisors
- Treatment

Natal teeth



Natal teeth



Natal teeth



Natal teeth



## Developmental alterations

- Number
- Size
- Shape
- Structure

## Developmental alterations

- Number
  - Hypodontia
    - Lack of development of one or more teeth
  - Anodontia
    - Total lack of tooth development
  - Hyperdontia
    - Development of an increased number of teeth

## Hypodontia

- Common dental anomaly
  - 3.5%-8% (excluding third molars)
- Female predominance about 1.5:1
- Uncommon in primary dentition (<1%)
- About 20-23% of population missing third molars
- After third molars, second premolars and laterals most frequent

## Syndromes associated with hypodontia

- Ectodermal dysplasia
- Chondroectodermal dysplasia (Ellis-van Creveld)
- Incontinentia pigmenti
- Progeria
- Down
- Hallermann-Streiff
- Rieger
- Crouzons
- Albright hereditary osteodystrophy

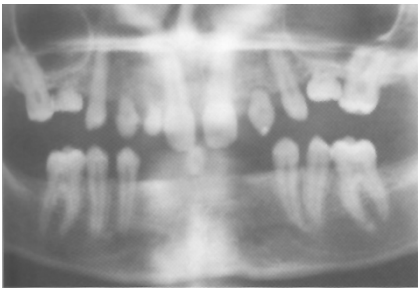
Hypodontia



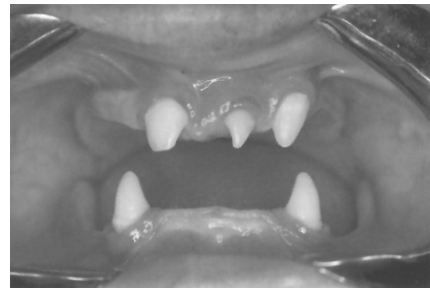
Hypodontia



Hypodontia



Hypodontia-Ectodermal dysplasia



## Hyperdontia

- Prevalence of supernumerary teeth is about 1%-3% (higher rate in Asians)
- Single tooth hyperdontia represent 75%-85% of cases
- More common in permanent dentition
- Almost 90% in maxilla
- Maxillary incisor region most common site then 4th molars, premolars and canines
- If multiples, usually in mandibular premolar region

## Syndromes associated with hyperdontia

- Cleidocranial dysplasia
- Oral-Facial-Digital
- Craniometaphyseal dysplasia
- Apert

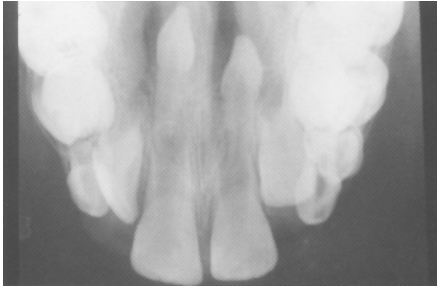
## Hyperdontia



## Mesiodens



Hyperdontia



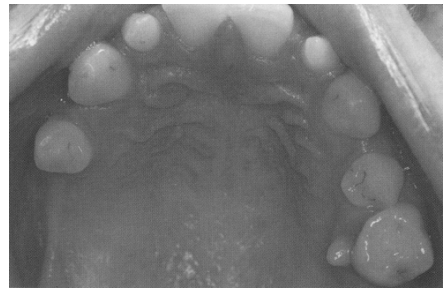
Hyperdontia



Cleidocranial dysplasia



Paramolar



## Developmental alterations

- Size
  - Microdontia
  - Macrodontia

## Microdontia

- Teeth are smaller than usual
- Relative microdontia=macrognathia
- Diffuse true microdontia is uncommon but may occur in Down syndrome and pituitary dwarfism
- Prevalence of isolated microdontia is between 1% and 8%
- Maxillary lateral incisor most frequently affected

## Microdont- peg lateral

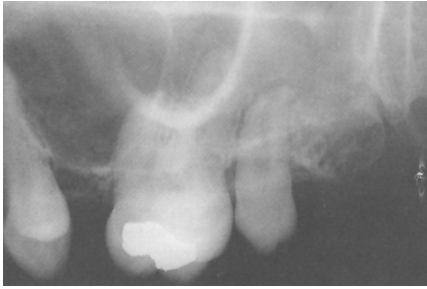


## Microdont- peg lateral





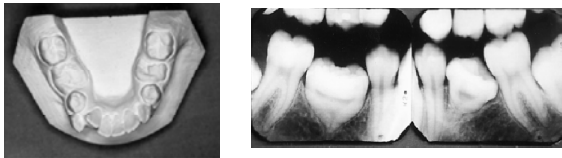
## Microdont



## Macrodontia

- Teeth are larger than usual
- Relative macrodontia=micrognathia
- Diffuse involvement very rare
- Has been noted in association with pituitary gigantism and hemifacial hyperplasia

## Macrodontia



## Developmental alterations

- Shape
  - Generation
  - Fusion
  - Concrescence
  - Talon cusp
  - Dens evaginatus
  - Dens invaginatus
  - Taurodontism
  - Dilaceration

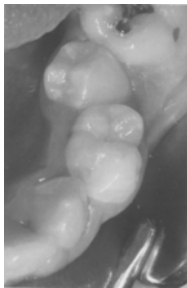
## Double teeth

- Gemination and fusion
  - May have very similar clinical appearance
- Higher frequency in anterior and maxillary regions
- Rate is about 0.1% in permanent dentition and 0.5% in deciduous
- Bilateral cases more infrequent
- Etiology unknown but trauma has been suggested
- Treatment considerations

## Gemination

- Single joined or enlarged tooth in which tooth count is normal when anomalous tooth is counted as one
- Result from single tooth bud????

## Gemination



## Gemination



## Gemination



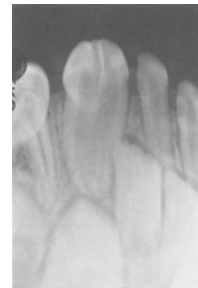
## Fusion

- Single joined or enlarged tooth in which tooth count reveals missing tooth when anomalous tooth is counted as one
- Union of two separate tooth buds?

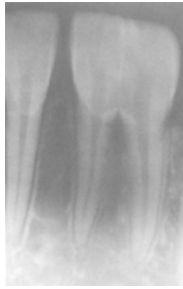
## Fusion



## Fusion



## Fusion



## Concrescence

- Union of two adjacent teeth by cementum alone
- May occur before or after eruption
- Seen most commonly posterior and maxillary regions
- Etiology believed to be trauma or overcrowding
- Treatment considerations

## Concrescence



## Talon cusp

- Well-delineated additional cusp on the surface of an anterior tooth and extends 1/2 the distance from CEJ to incisal edge
- Vast majority on lingual surface
- Prevalence studies vary from <1% to 8%
- 3/4 found in permanent dentition, most commonly maxillary lateral then central
- In deciduous dentition, maxillary central most common site
- Has been associated with other dental anomalies
- Treatment considerations

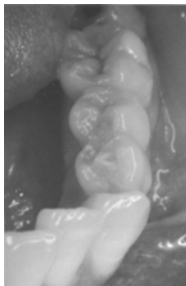
## Talon cusp



## Dens evaginatus

- Also known as a central tubercle
- A cusplike elevation located in the central groove
- Typically occurs in permanent mandibular premolars
- Usually bilateral
- Rare in whites with higher prevalence in Asians, native Americans and Alaskans
- Treatment considerations

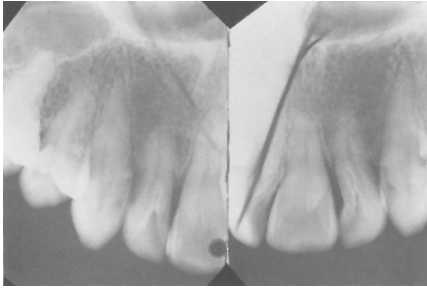
## Dens evaginatus



## Dens invaginatus

- Dens in dente
- Deep surface invagination of crown that is lined by enamel
- Represents an accentuation of the lingual pit
- Depth varies
- Prevalence studies vary from <1% to 10%
- Lateral incisors most commonly affected
- Bilateral involvement common
- Treatment considerations

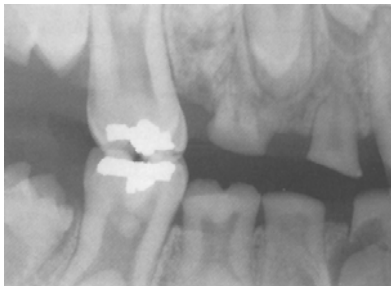
## Dens invaginatus



## Taurodontism

- Enlargement of the body and pulp chamber of a multirooted tooth with apical displacement of the pulpal floor
- More commonly seen in permanent dentition
- Prevalence is highly variable
  - 2%-3% in U.S.
  - Much higher in Eskimos and Middle Eastern populations
- Increased frequency in patients with CL and/or CP, Down, Klinefelter, ectodermal dysplasia, trich-dento-osseous

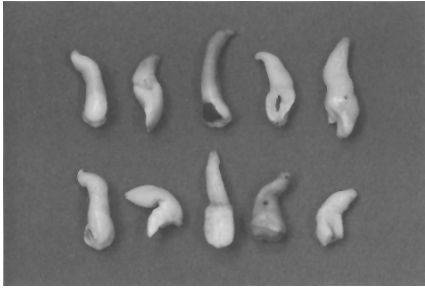
## Taurodontism



## Dilaceration

- Abnormal angulation or bend in the root
- Thought to be related to trauma during root development
- Permanent maxillary incisors most commonly affected followed by mandibular incisors
- Rare in primary dentition
- Treatment depends on severity

## Dilaceration



## Amelogenesis imperfecta

- A heterogeneous group of hereditary disorders that demonstrate developmental alterations in the structure of enamel in the absence of a systemic disorder
- Many subtypes
- Numerous patterns of inheritance
- Wide variety of clinical manifestations
- Frequency varies between 1:718 and 1:14,000
- Both dentitions involved

## Amelogenesis imperfecta

- Formation of enamel a multistep process
  - Formation of enamel matrix
  - Mineralization of matrix
  - Maturation of matrix
- Hereditary defects of enamel formation usually classified as:
  - Hypoplastic
  - Hypocalcified
  - Hypomaturative

## Amelogenesis imperfecta Witkop classification

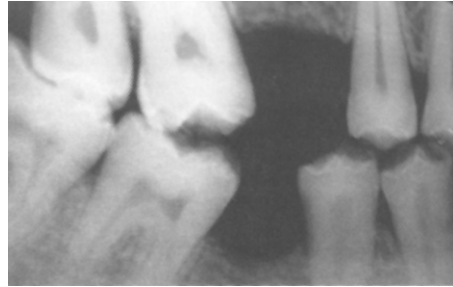
Table 2-1 Classification of Amelogenesis Imperfecta

TYPE	PATTERN	SPECIFIC FEATURES	INHERITANCE
IA	Hypoplastic	Generalized pitted	Autosomal dominant
IB	Hypoplastic	Localized pitted	Autosomal dominant
IC	Hypoplastic	Localized pitted	Autosomal recessive
ID	Hypoplastic	Diffuse smooth	Autosomal dominant
IE	Hypoplastic	Diffuse smooth	X-linked dominant
IF	Hypoplastic	Diffuse rough	Autosomal dominant
IG	Hypoplastic	Enamel agenesis	Autosomal recessive
IIA	Hypomaturative	Diffuse pigmented	Autosomal recessive
IIB	Hypomaturative	Diffuse	X-linked recessive
IIC	Hypomaturative	Snow capped	X-linked
IID	Hypomaturative	Snow capped	Autosomal dominant?
IIIA	Hypocalcified	Diffuse pigmented	Autosomal dominant
IIIB	Hypocalcified	Diffuse	Autosomal recessive
IVA	Hypomaturative-hypoplastic	Taurodontism present	Autosomal dominant
IVB	Hypoplastic-hypomaturative	Taurodontism present	Autosomal dominant

## Amelogenesis imperfecta

- Hypoplastic
  - Teeth erupt with insufficient amounts of enamel
  - Amount of enamel varies greatly
  - Enamel present is mineralized appropriately and contrasts well with dentin on radiographs
  - Teeth may have abnormal shape and open contacts
  - Open bite may be present

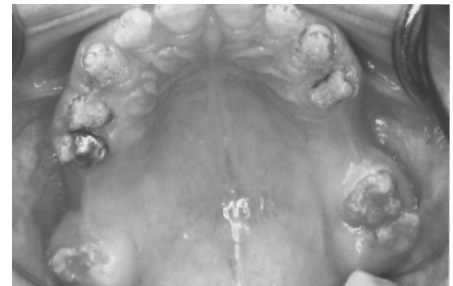
## AI-Hypoplastic smooth



## AI-Hypoplastic pitted



## AI-Hypoplastic pitted





### AI-Hypoplastic rough



### Amelogenesis imperfecta

- Hypocalcified
  - Proper amount of enamel matrix is formed but it doesn't mineralize properly
  - Teeth shaped normally upon eruption but enamel is soft and easily lost
  - Enamel yellow-brown upon eruption but quickly becomes brown to black
  - Accumulate calculus
  - Enamel and dentin have similar density on radiographs

### AI-Hypocalcified



### AI-Hypocalcified



## Amelogenesis imperfecta

- Hypomaturative
  - Enamel matrix is laid down properly and begins to mineralize but there is a defect in maturation of enamel's crystal structure
  - Affected teeth normal in shape
  - Mottled appearance-white, brown or yellow
  - Enamel soft and chips away from dentin
  - Enamel has similar radiodensity to dentin

## AI-Hypomaturative



## AI-Hypomaturative, snowcapped



## Dentinogenesis imperfecta

- Hereditary developmental disturbance of dentin
- Autosomal dominant
- Also known as hereditary opalescent dentin
- Shields classification
- Prevalence is about 1:8000
  - Most cases in whites of English or French ancestry from communities near English Channel

## Dentinogenesis imperfecta

- All teeth in both dentitions affected
- Deciduous teeth affected most severely followed by permanent incisors and first molars
- Yellow-brown to blue-gray translucent, opalescent appearance
- Enamel frequently separates easily from dentin
- Once exposed, dentin exhibits rapid attrition
- Bulbous crowns with cervical constriction
- Thin roots
- Early obliteration of pulp chambers and root canals

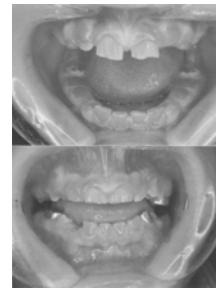
## Dentinogenesis imperfecta



## Dentinogenesis imperfecta



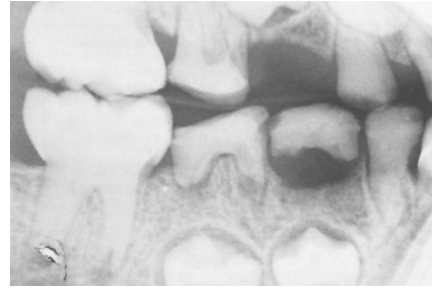
## Dentinogenesis imperfecta



## Dentinogenesis imperfecta



## Dentinogenesis imperfecta



## Dentin dysplasia

- Rare autosomal-dominant condition that affects dentin
- Prevalence about 1:100,000

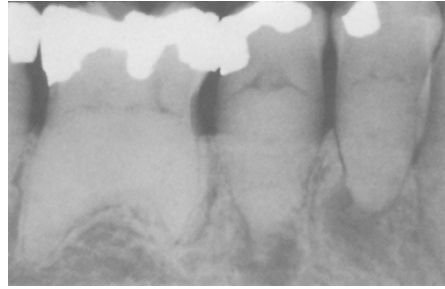
## Dentin dysplasia

- Type I
  - Radicular type or 'rootless teeth'
  - Roots short and pulps almost obliterated
  - Periapical radiolucencies
  - More common type
  - Enamel and coronal dentin are normal
  - Wide variation in root formation because dentinal disorganization may occur at different stages of tooth development
  - Color is normal in both dentitions
  - Radiographically, deciduous teeth more severely affected

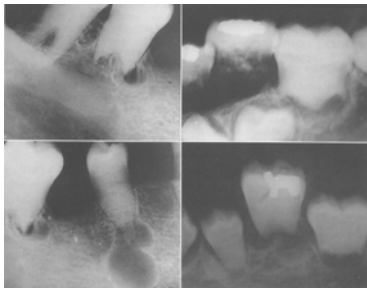
Dentin dysplasia type I



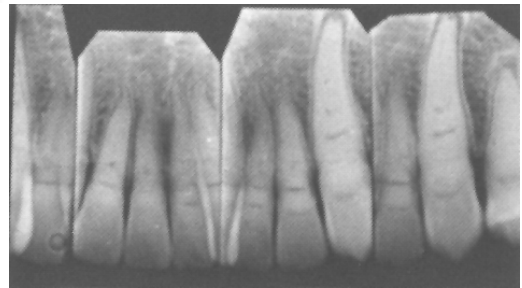
Dentin dysplasia type I



Dentin dysplasia



Dentin dysplasia



## Dentin dysplasia

- Type II
  - Coronal type
  - Root length normal in both dentitions
  - Primary teeth
    - Clinically resemble dentinogenesis imperfecta
    - Radiographically have similar appearance to Type I
  - Permanent teeth
    - Normal coloration
    - Pulp chambers enlarged with apical extension-thistle-tube-shaped or flame-shaped

## Dentin dysplasia type II



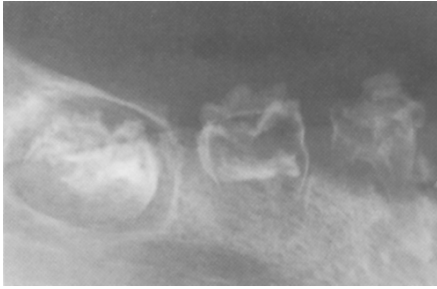
## Dentin dysplasia type II



## Regional odontodysplasia

- 'Ghost teeth'
- Localized, non-hereditary developmental abnormality of teeth with extensive adverse effects on formation of enamel, dentin and pulp
- Occurs in region or quadrant
- Etiology unknown
- Occurs in both dentitions and if present in primary dentition, permanent teeth in area usually affected
- Maxillary predominance- 2.5:1
- Many affected teeth fail to erupt
- Erupted teeth have small irregular yellow-brown crowns
- Short roots, enlarged pulp and open apical foramina

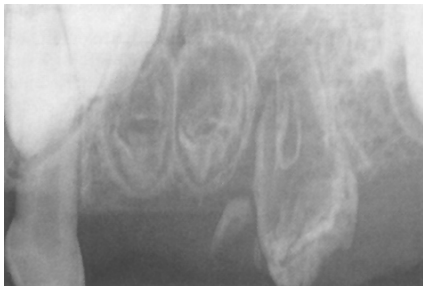
## Regional odontodysplasia



## Regional odontodysplasia



## Regional odontodysplasia



## References

- Oral & Maxillofacial Pathology-Second Edition
  - Neville, Damm, Allen and Bouquot
- Oral Pathology-Clinical Pathologic Correlations
  - Regezi, Sciubba and Jordan

Thank you!

