DEVELOPMENTAL DISTURBANCES IN STRUCTURE OF TEETH

Dr K.Karpagaselvi

Prof & Head

Department of Oral Pathology & Microbiology

Enamel
Dentin

ENAMEL HYPOPLASIA

Hereditary

- Amelogenesis Imperfecta

Environmental

- Nutritional deficiencies (Vitamins A, C & D)
- Exanthematous diseases (measles, chicken pox, scarlet fever)
- Congenital syphilis
- Hypocalcemia
- Birth injury, Prematurity, Rh haemolytic disease
- Local infection or trauma
- Ingestion of chemicals/Fluoride
- Idiopathic causes

Hereditary

- Both deciduous & permanent dentition involved
- Only Enamel affected

Environmental

- Either dentition involved, also single tooth
- Both Enamel & Dentin affected to some degree

AMELOGENESIS IMPERFECTA (hereditary enamel dysplasia, hereditary brown enamel, hereditary brown opalescent teeth)

Amelogenesis Imperfecta (AI)

- A structural defect of the tooth enamel
- A complex inheritance pattern gives rise to amelogenesis imperfecta (AI)
- Depending on
  - Stage of enamel formation that is primarily affected
  - Clinical presentation of the defects
  - Mode of inheritance

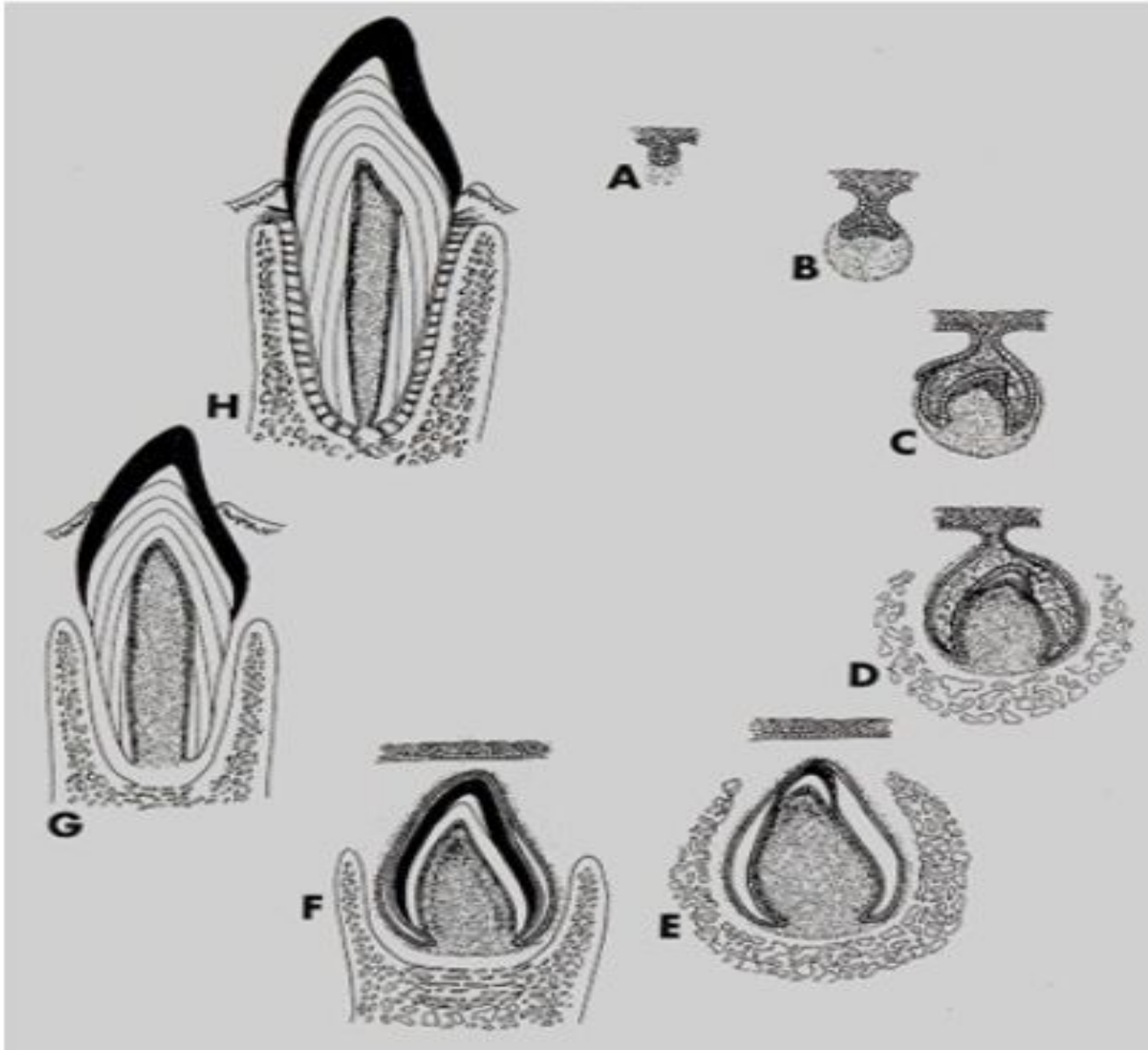
Amelogenesis Imperfecta - Prevalence

- 1 in 718 to 1 in 14,000 depending on population studied
  - Hypoplastic AI - 60–73%
  - hypomaturation AI - 20–40%
  - hypocalcification AI - 7%
- Disorders of enamel epithelium also can cause alterations in eruption mechanism, resulting in anterior open bite

Etiology of AI

- AMELX mutations – X-linked AI – DSX85 at Xp22 – Amelogenin – principle protein in developing enamel
- ENAM mutations – Hypoplastic AI - Enamelin protein mutations
- Enamelysin (*MMP20*) & Kallikrein 4 (*KLK4*) mutations – Hypomaturation AI
- FAM83H gene mutation on 8q24 - Autosomal dominant hypocalcified AI

Tooth Development



A. Bud Stage
B. Cap Stage
C. Bell Stage

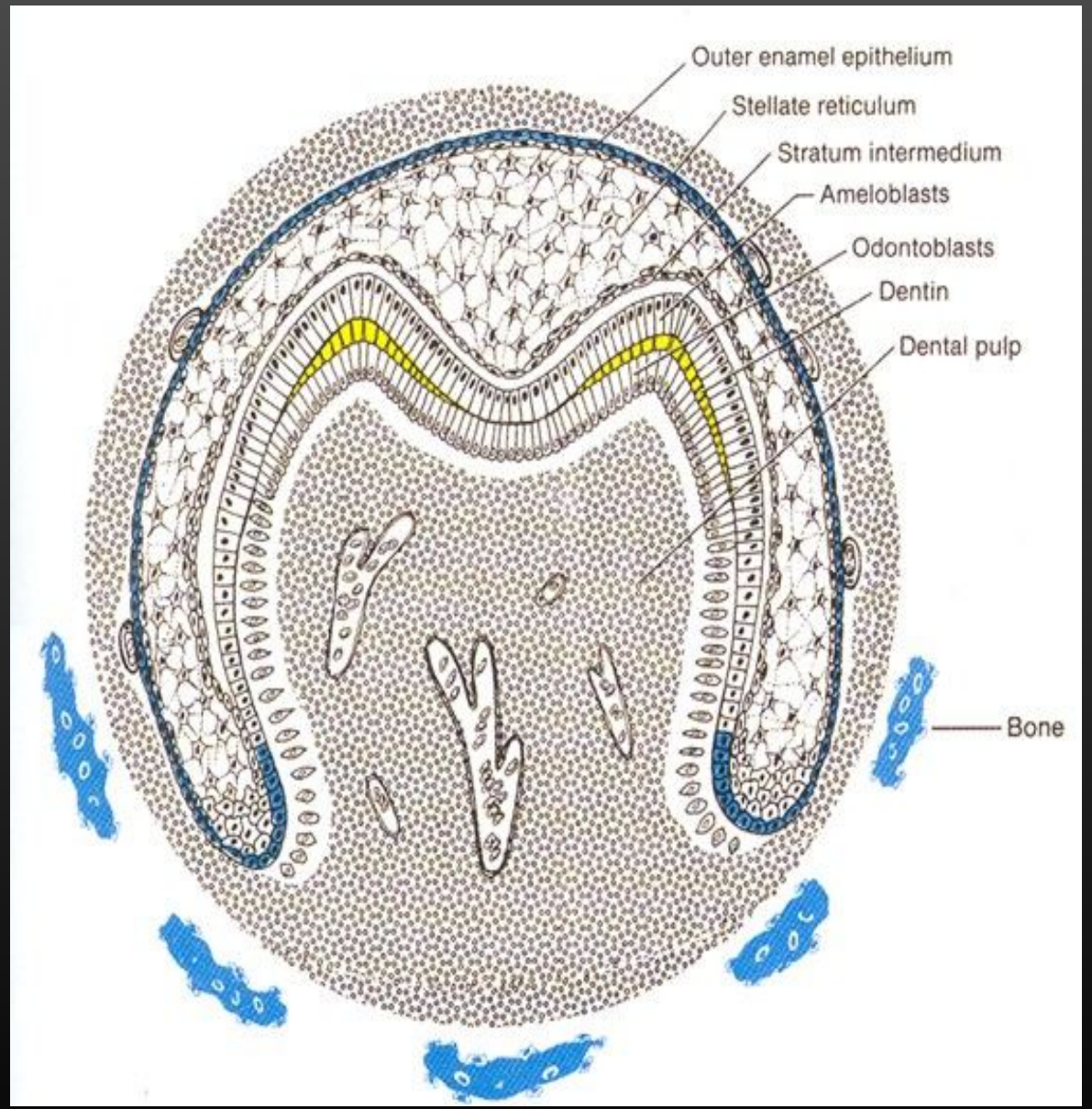
D and E. Dentinogenesis and amelogenesis

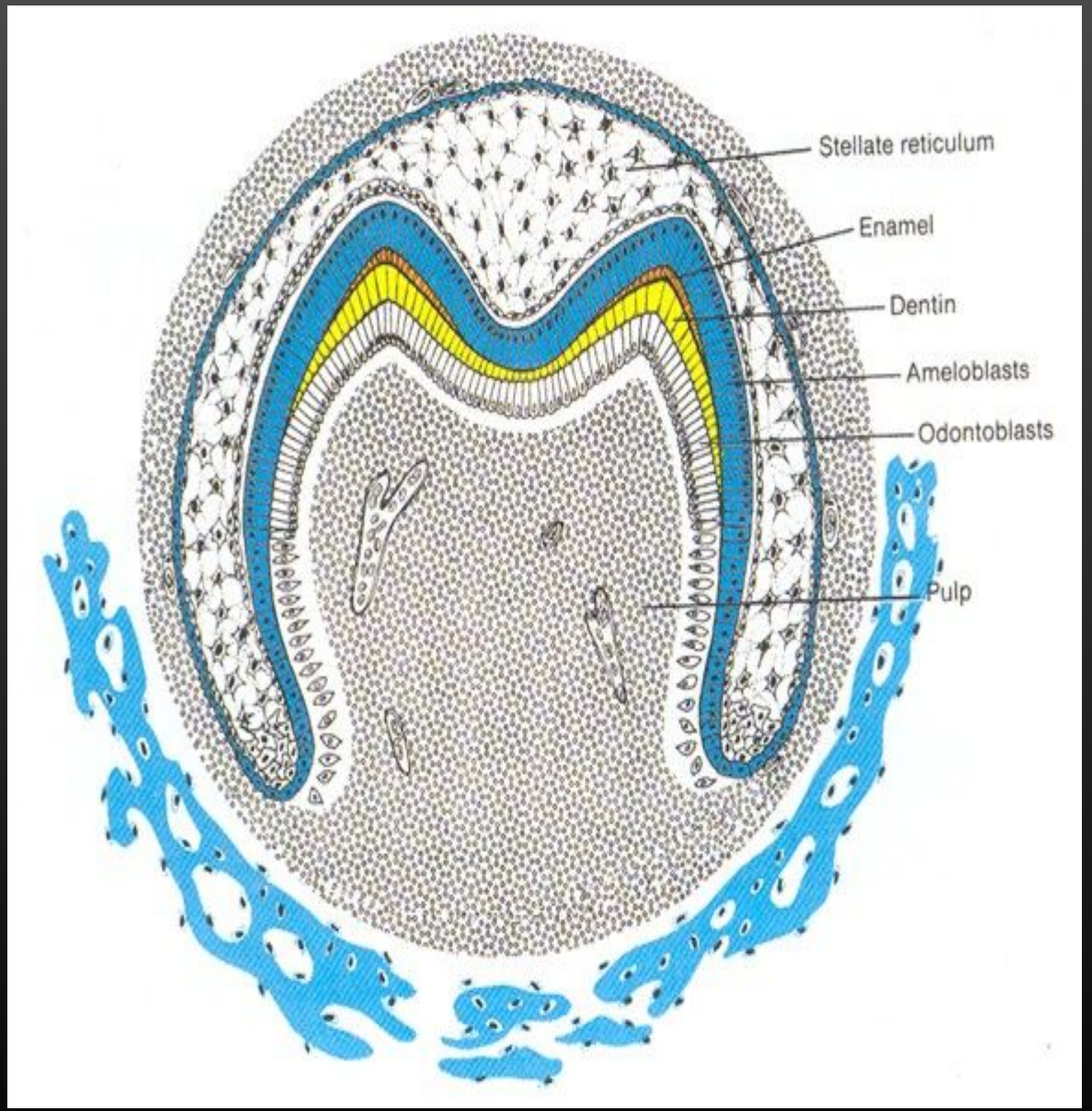
F. Crown formation

G. Root Formation and eruption

H. Function

Essentials of Oral Histology and Embryology, Ed: James Avery, 2nd edition. 2000.


Outer enamel epithelium
Stellate reticulum
Stratum intermedium
Ameloblasts
Odontoblasts
Dentin
Dental pulp
Bone


Stellate reticulum
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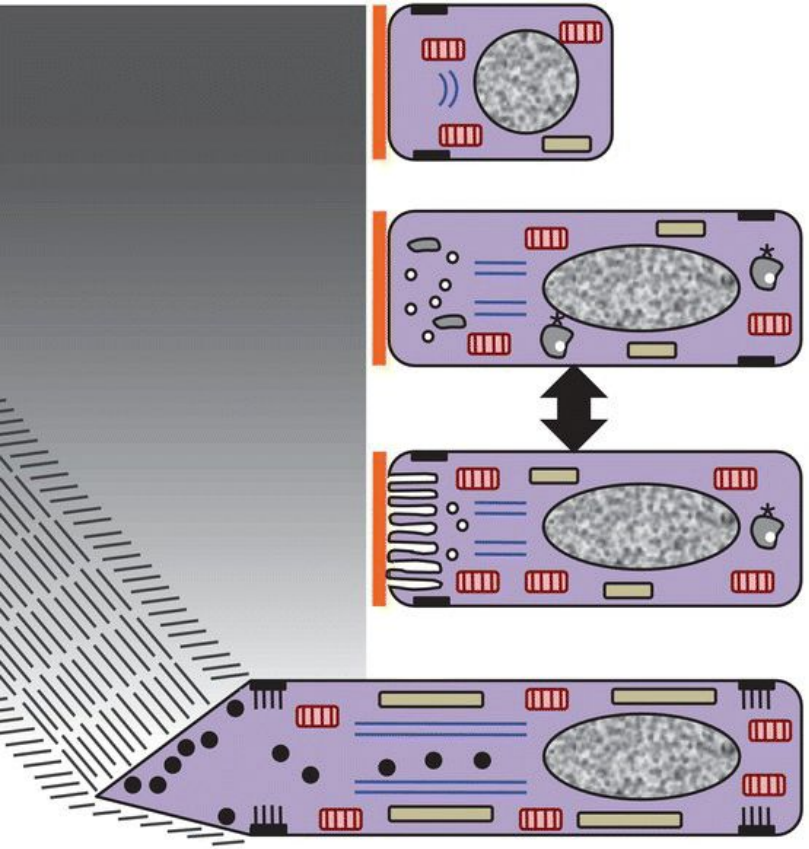


Protective (Reduced)

Smooth-ended maturation

(modulation)

Ruffle-ended maturation

(transition)

Secretory

Early secretory

Presecretory

Inner enamel epithelium

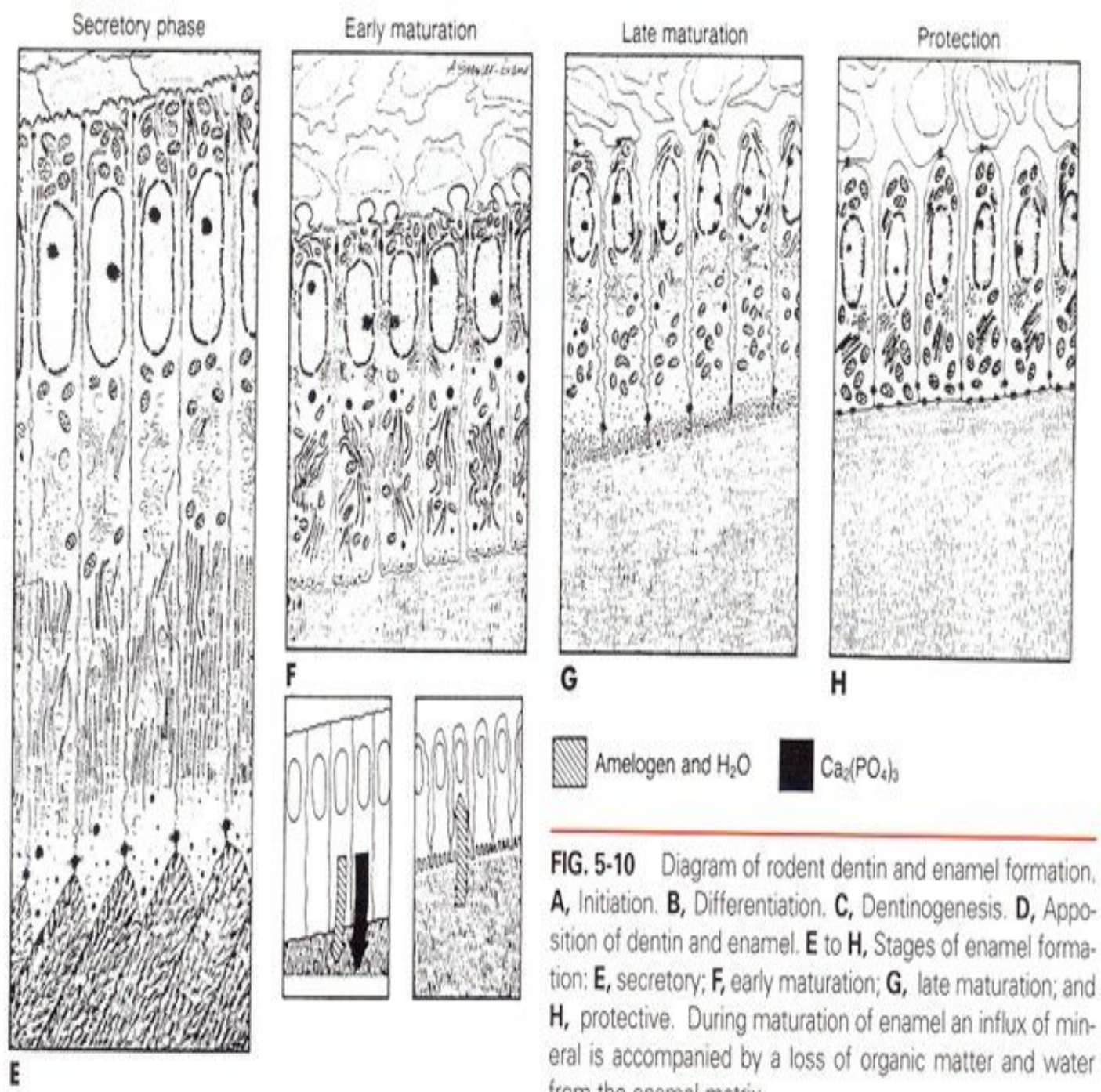
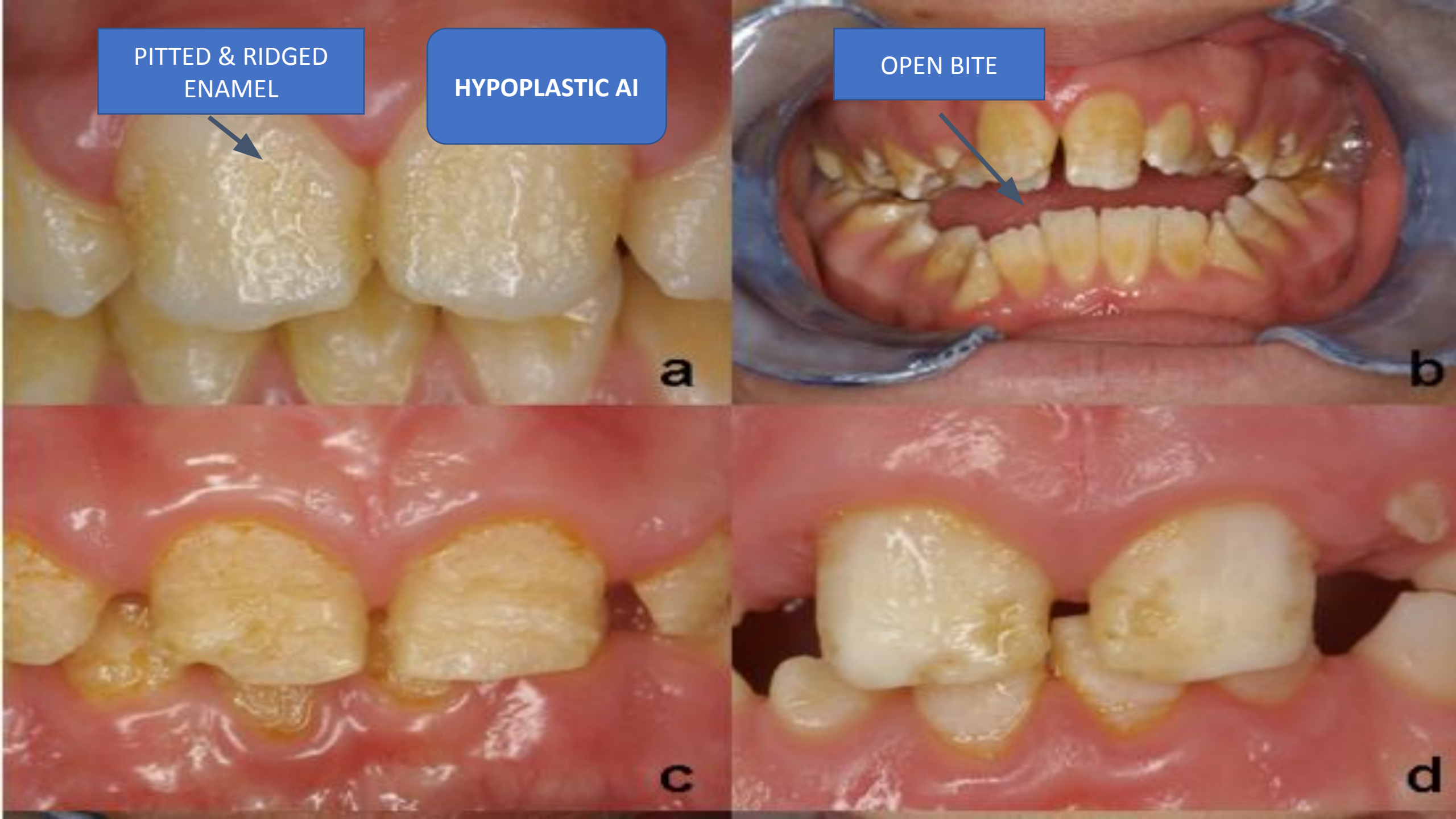


FIG. 5-10 Diagram of rodent dentin and enamel formation. **A,** Initiation. **B,** Differentiation. **C,** Dentinogenesis. **D,** Apposition of dentin and enamel. **E** to **H,** Stages of enamel formation: **E,** secretory; **F,** early maturation; **G,** late maturation; and **H,** protective. During maturation of enamel an influx of mineral is accompanied by a loss of organic matter and water from the enamel matrix.

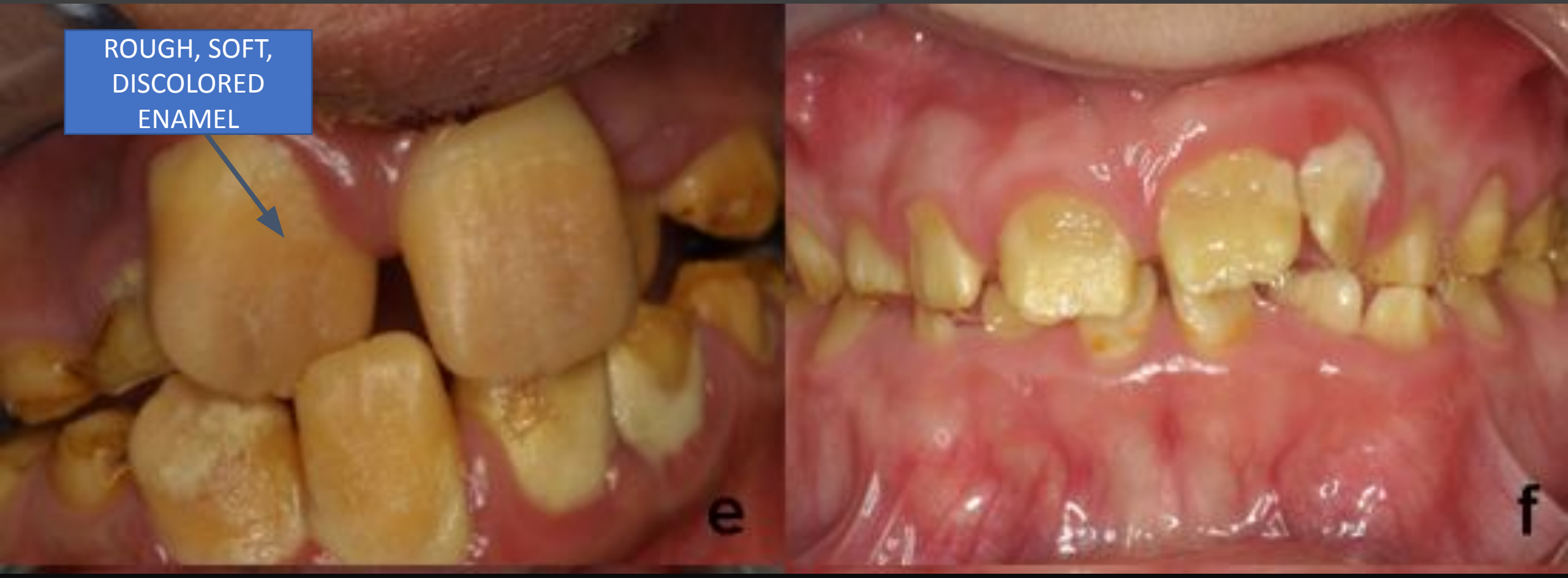
- *Hypoplastic AI* - thin but mineralized enamel, or in extreme cases, complete absence of enamel, that results from failure during secretory stage
- *Hypomineralized AI* - maturation stage failure, giving rise to enamel that is of full thickness but is weak & fails prematurely
  - *hypomaturation* - incomplete removal of protein from the enamel matrix and produces brittle enamel
  - *hypocalcified AI* - insufficient transport of calcium ions (Ca^{2+}) into developing enamel & produces soft enamel

CLINICAL AND HEREDITARY CHARACTERISTICS OF FOUR MAIN AI TYPES (TABLE 1) [8]

Type	Clinical Appearance	Enamel Thickness	Radiographic Appearance	Inheritance
Hypoplastic (Type 1)	Crowns size varies from small to normal, small teeth may lack proxmial contacts, color varies from normal to opaque white – yellow brown	Varies from thin and smooth to normal thickness with grooves, furrows and/or pits	Enamel has normal to slightly reduced contrast/thin	Autosomal dominant, recessive or X-linked
Hypomaturation (Type II)	Varies from creamy opaque to marked yellow/brown, surface of teeth soft and rough, dental sensitivity and open bite common	Normal thickness with enamel that often chips and abrades easily	Enamel has contrast similar to or > than dentin, unerupted crowns have normal morphology	Autosomal dominant, recessive or X-linked
Hypocalcified (Type III)	Opaque white to yellow-brown, soft rough enamel surface, dental sensitivity and open bite common, heavy calculus formation common	Normal thickness with enamel that often chips and abrades easily	Enamel has contrast similar to or < dentin, unerupted crowns have normal morphology	Autosomal dominant, recessive
Hypomaturation/ Hypoplasia/ Taurodontism (Type IV)	White/Yellow- Brown mottled, teeth can appear small and lack proximal contact	Reduced, hypomineralized areas and pits	Enamel contrast normal to slightly > dentin, large pulp chambers	Autosomal dominant

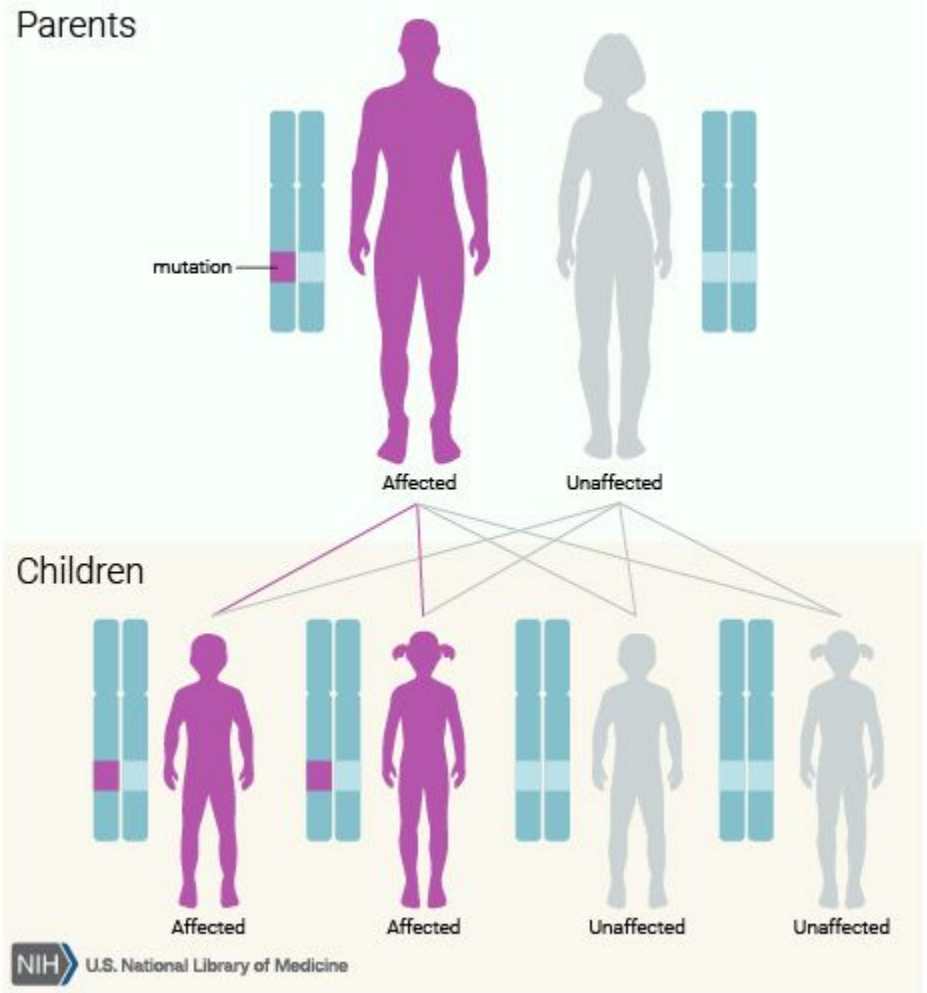

PITTED & RIDGED ENAMEL
HYPOPLASTIC AI
OPEN BITE
a
b
c
d

HYPOCALCIFIED AI

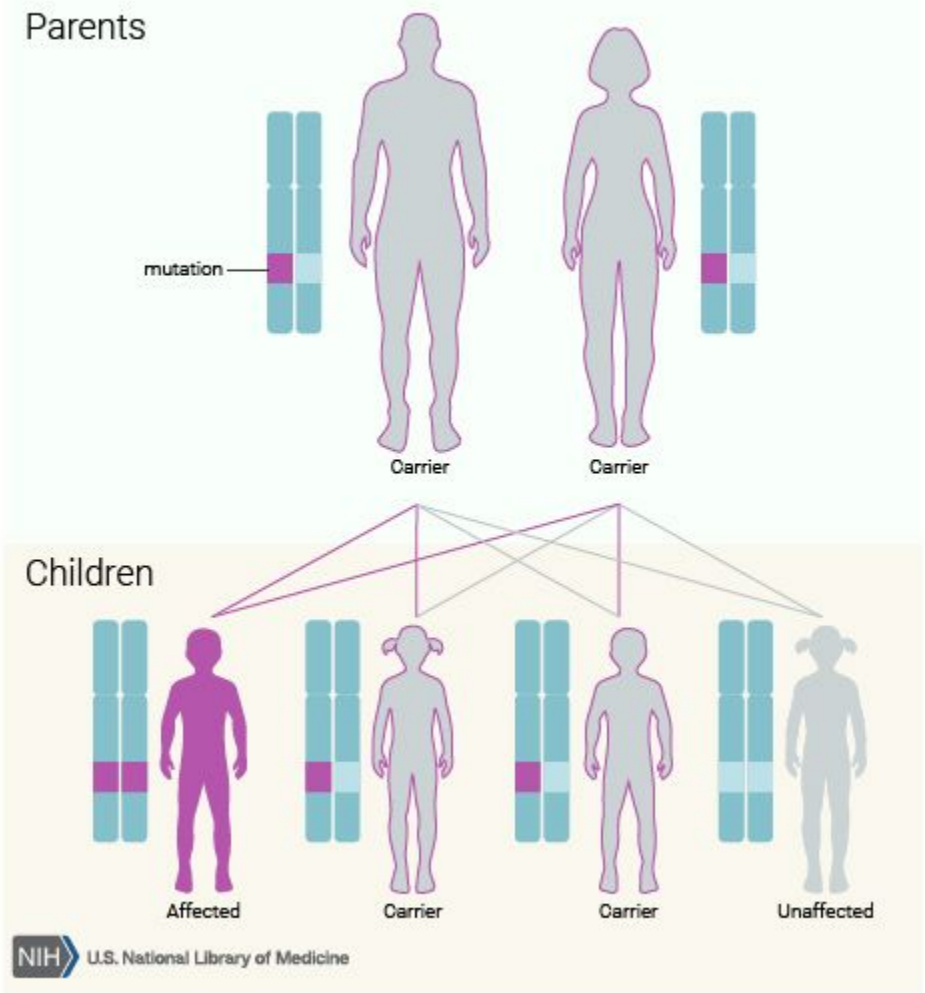


HYPOMATURATION TYPE AI

Autosomal Dominant



Autosomal Recessive



MODES OF MENDELIAN INHERITANCE ASSOCIATED WITH AI

Autosomal Dominant Inheritance	• Can have male to male transmission. • On average, half of the offspring of an affected individual will be affected. There is a 50 percent chance for the child of an affected individual to be affected. • Affected males and females have similar clinical presentation.
Autosomal Recessive Inheritance	• Unaffected parents will have affected offspring. • On average, one in four offspring of carrier parents will be affected. • More likely to occur when parents are related (consanguineous relationship).
X-linked Recessive Inheritance	• Do not have male to male transmission. • All daughters of an affected male are carriers. • Half of the sons born to a carrier female will be affected. • Affected males have more severe manifestations than females. • Females can show no manifestations to severe manifestations due to lyonization. Females express only one X chromosome per cell with the other X chromosome becoming the bar body. If adequate numbers of cells express the X chromosome carrying the mutant allele, they will have varying degrees of the enamel defect.

Radiographic Features – AI

- Enamel - totally absent or when present - very thin layer, chiefly over cusp tips & on interproximal surfaces
- Overall shape of tooth may or may not be normal depending upon amount of enamel present & amount of occlusal & incisal wear
- Affected enamel - same radiodensity as dentin, making differentiation between the two difficult

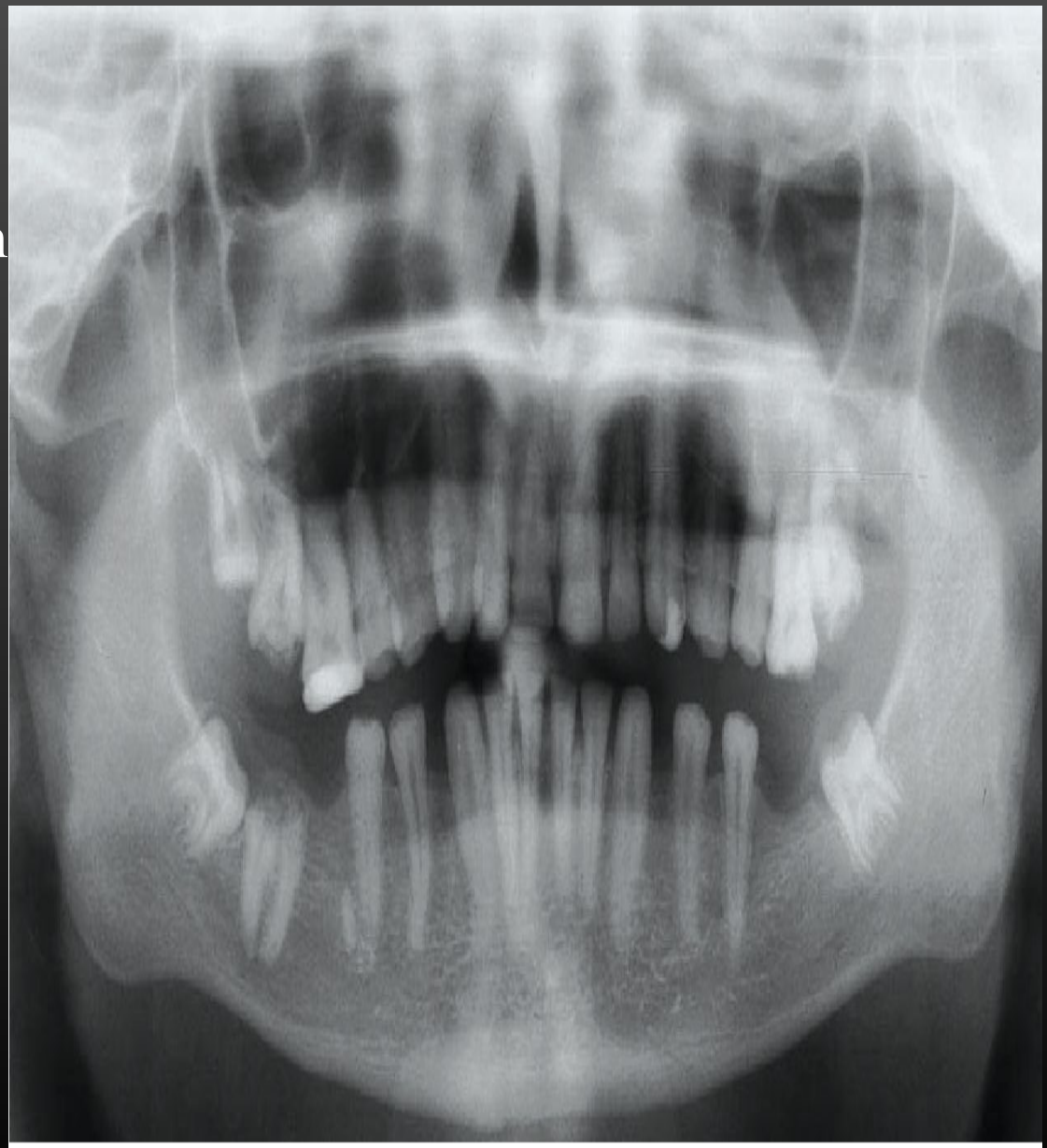
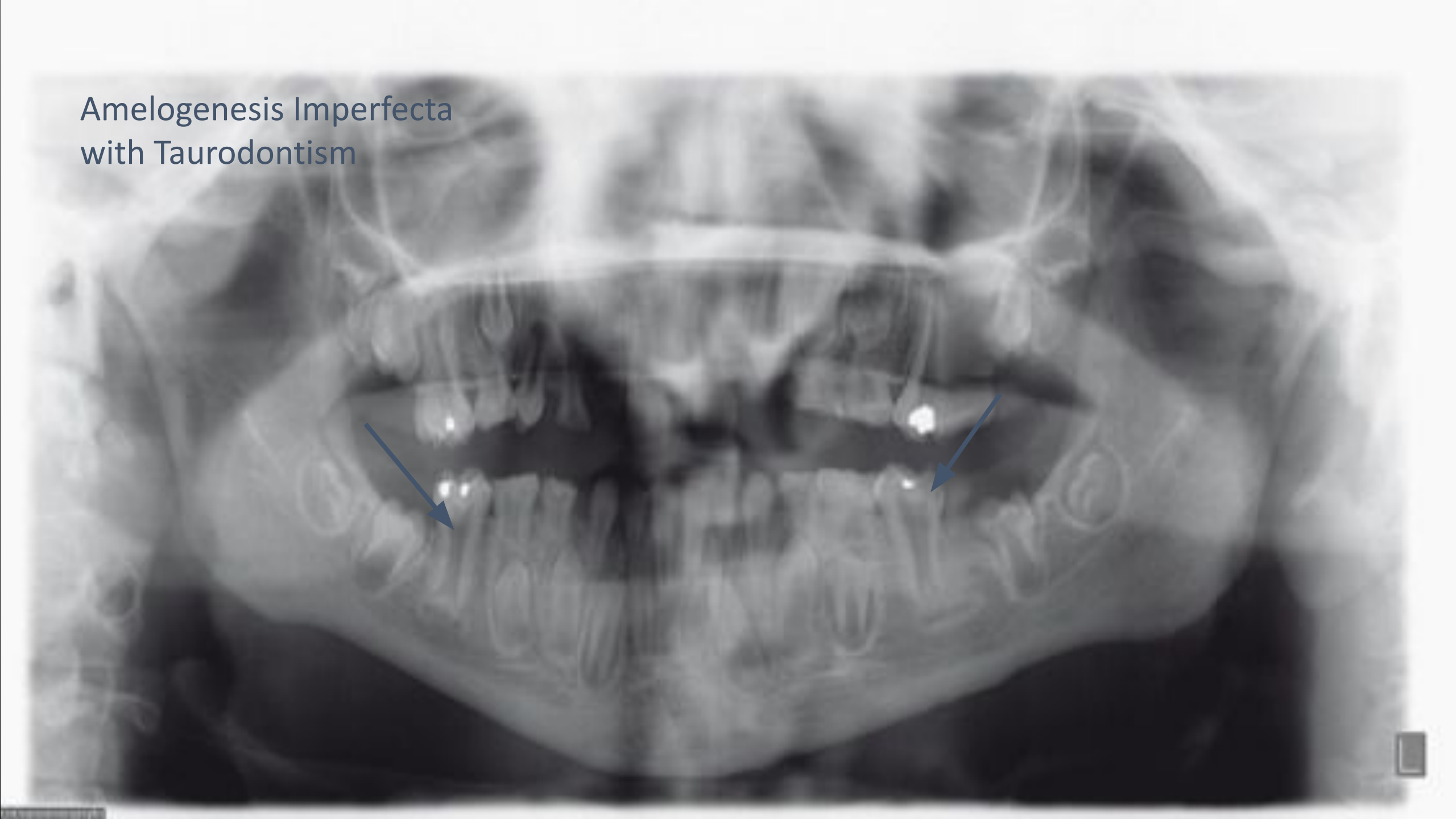


Figure 2. Panoramic radiograph of patient after 1 year

Amelogenesis Imperfecta
with Taurodontism



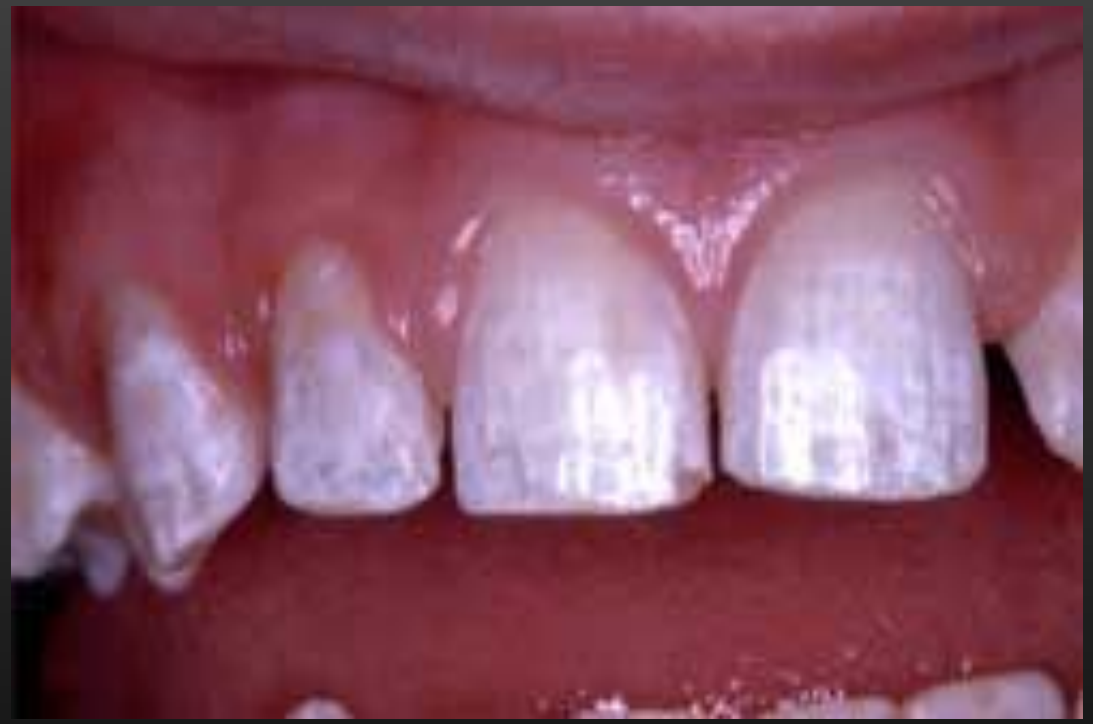
Histologic Features - AI

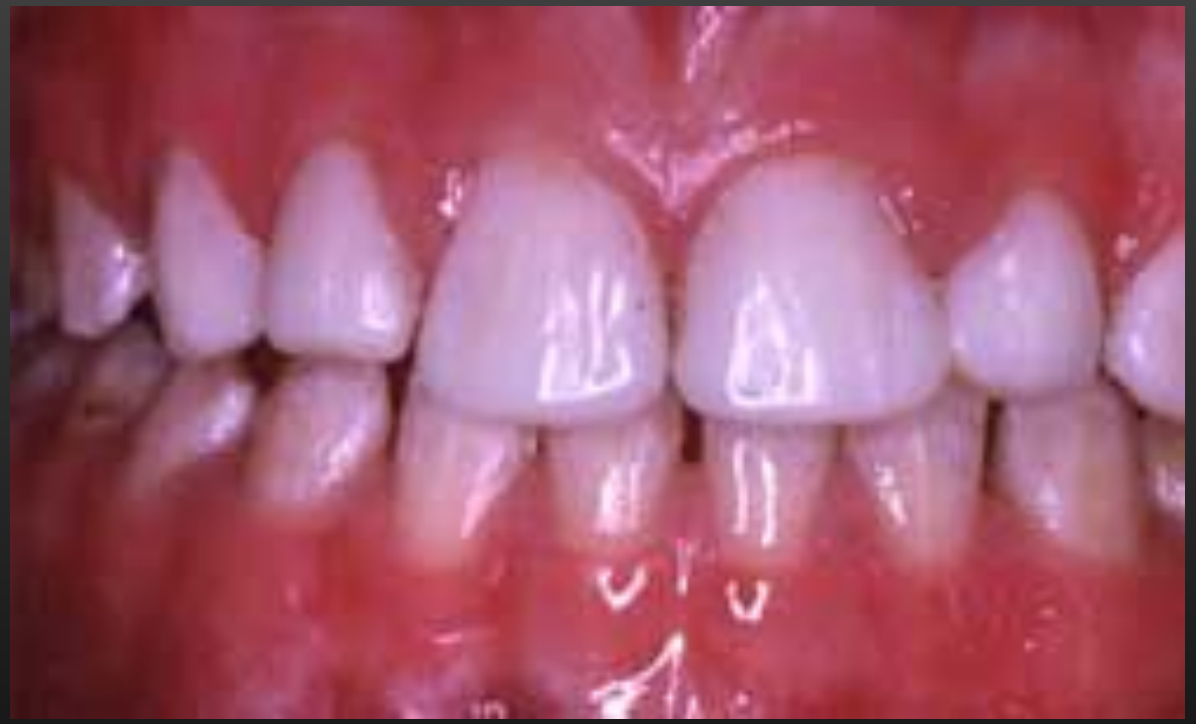
- General enamel histologic features
- Hypoplastic types - defects in matrix formation up to & including total absence of matrix (disturbance in the differentiation or viability of ameloblasts)
- Hypocalcification types - defects of matrix structure & of mineral deposition
- Hypomaturation types - alterations in enamel rod & rod sheath structures

Treatment of AI

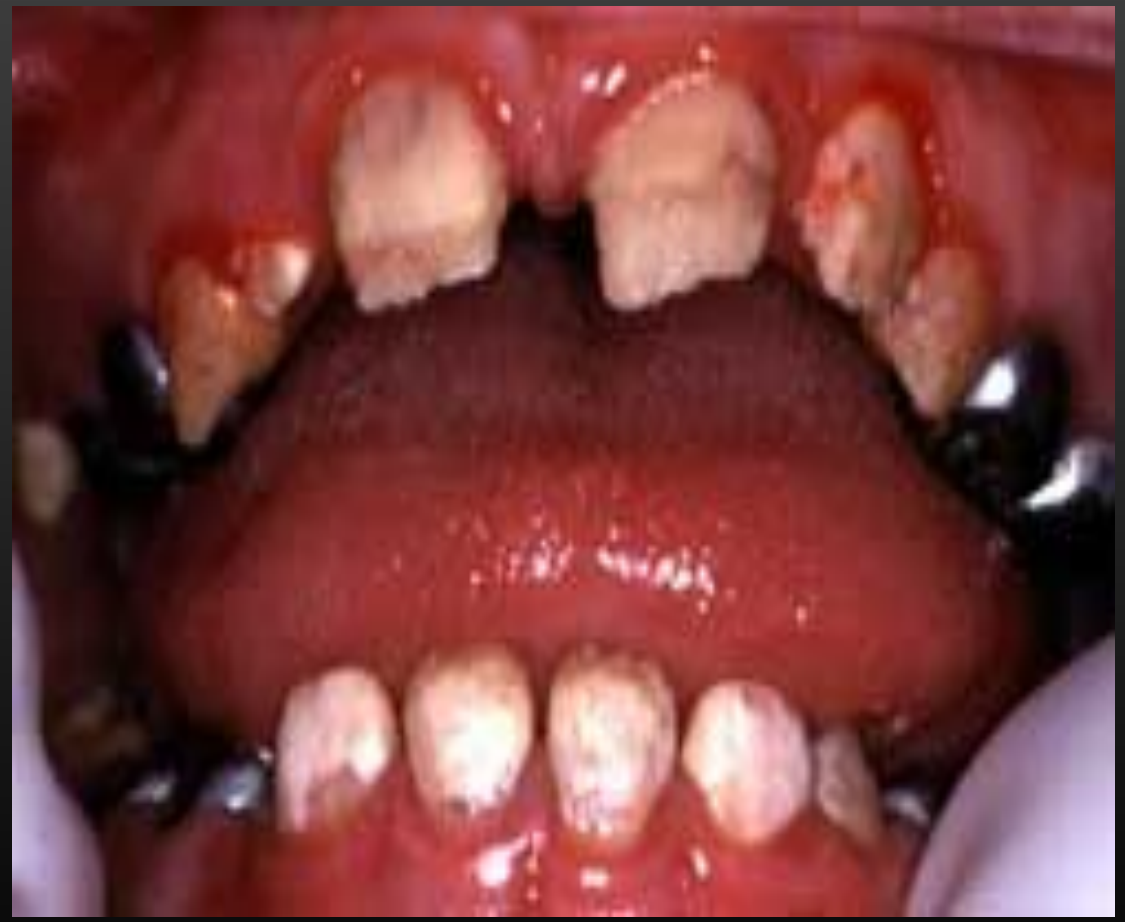
- Improvement of cosmetic appearance
- Multidisciplinary approach through out both dentitions in phases
    - General or paediatric dentist
    - Restorative dentist
    - Orthodontist
    - Prosthodontist
    - Oral surgeon – orthognathic surgery to correct skeletal open bite
- Genetic counselling

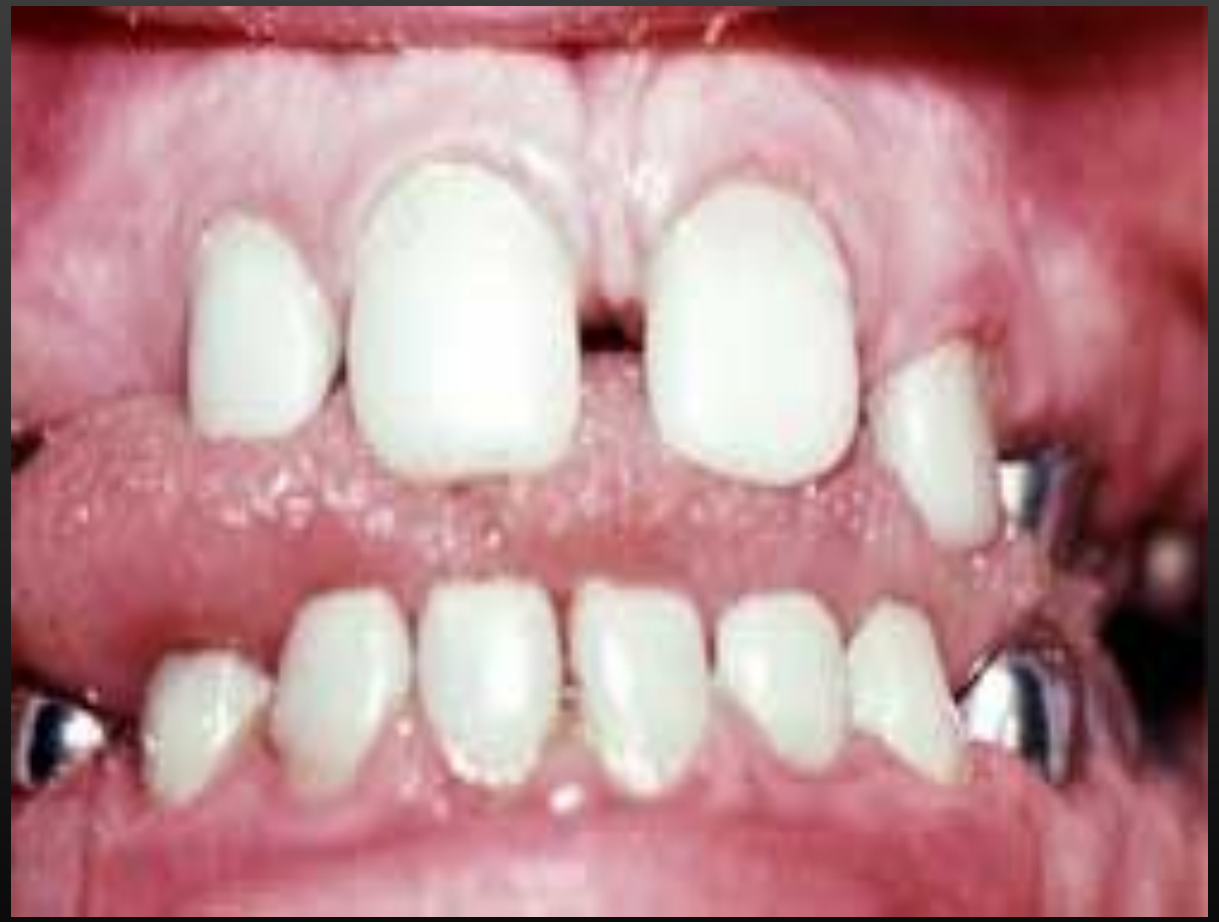
Hypoplastic AI before & after treatment with porcelain veneers



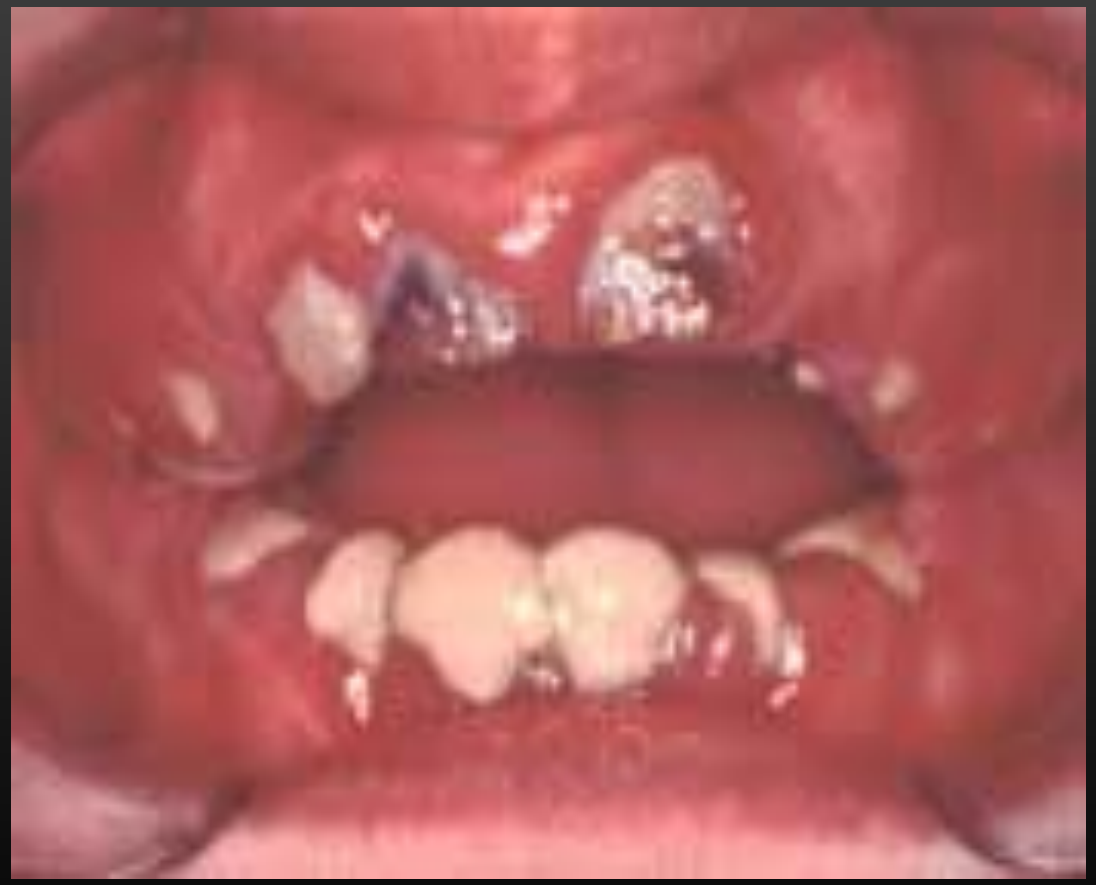


Hypomaturation & Hypocalcified AI before & after composite crowns





AR Hypomaturation AI treated over several years with stainless steel crowns, orthodontics, orthognathic surgery & eventually porcelain fused to metal crowns to achieve this excellent result



Enamel Hypoplasia

- Incomplete or defective formation of the organic enamel matrix of teeth
- Two basic types
  - A hereditary type, described previously under amelogenesis imperfecta
  - A type caused by environmental factors

Factors producing injury to Ameloblasts

1. Nutritional deficiency (vitamins A, C, and D)
2. Exanthematous diseases e.g. Measles, chickenpox, scarlet fever
3. Congenital syphilis
4. Hypocalcemia
5. Birth injury, prematurity, Rh hemolytic disease
6. Local infection or trauma
7. Ingestion of chemicals (chiefly fluoride)
8. Idiopathic causes

- Ameloblasts - one of the most sensitive groups of cells in body in terms of metabolic function
- Hypoplasia results only if injury occurs during the time teeth are developing formative stage of enamel development
- Once enamel has calcified, no such defect can be produced
- Thus, knowing chronologic development of deciduous & permanent teeth, it is possible to determine from location of defect on teeth the approximate time at which injury occurred

Enamel as a record

- Enamel defects in children have potential to detail specific stressors that may be responsible for their formation, particularly on deciduous teeth
- Enamel matrix secretion is disrupted due to elevated cortisone levels that inhibit protein synthesis as a result of a stress episode
- Defects on first deciduous incisors may indicate stress 1 month after birth
- Defects on lateral deciduous incisors could result from stress between 3 & 4 months after birth & hence, could be considered perinatal

Clinical features related to formation of deciduous & permanent dental enamel defects

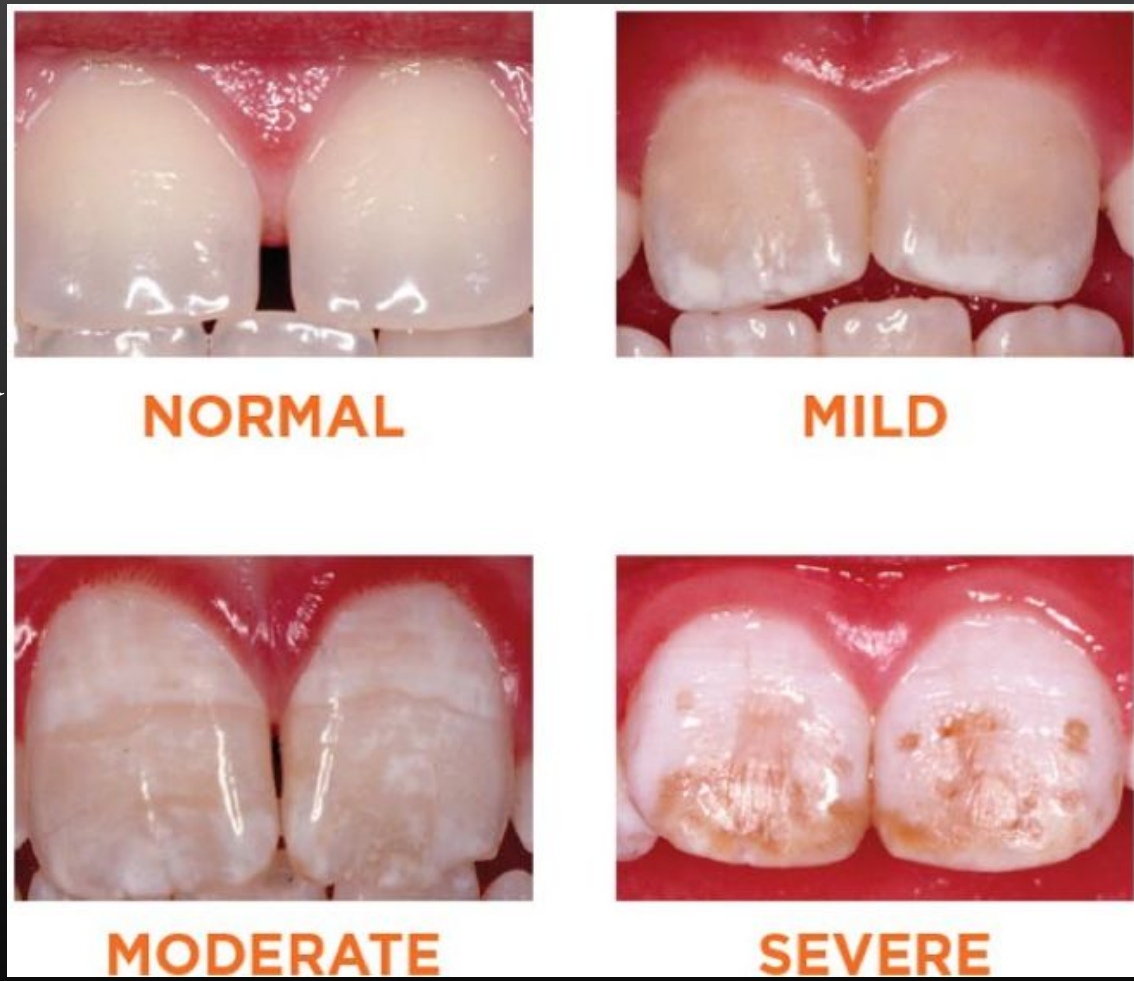
- ✔ Fever - Kronfield and Schour (1939)
- ✔ Birth trauma - Kronfield and Schour (1939)
- ✔ Congenital syphilis - Hillson et al. (1998)
- ✔ Tuberculosis - Knick (1982)
- ✔ Low birth weight - Seow (1992)
- ✔ Severe childhood malnutrition - Sweeney et al. (1971)
- ✔ Rickets and hypocalcaemia - Kreshover (1960), Levine and Keen (1974), Seow et al. (1984)
- ✔ Zinc deficiency - Dolphin and Goodman (2002)
- ✔ Intrauterine undernutrition due to poor maternal diet - Acosta et al. (2003), Noren (1983), Noren et al. (1978)
- ✔ Prematurity - Aine et al. (2000)

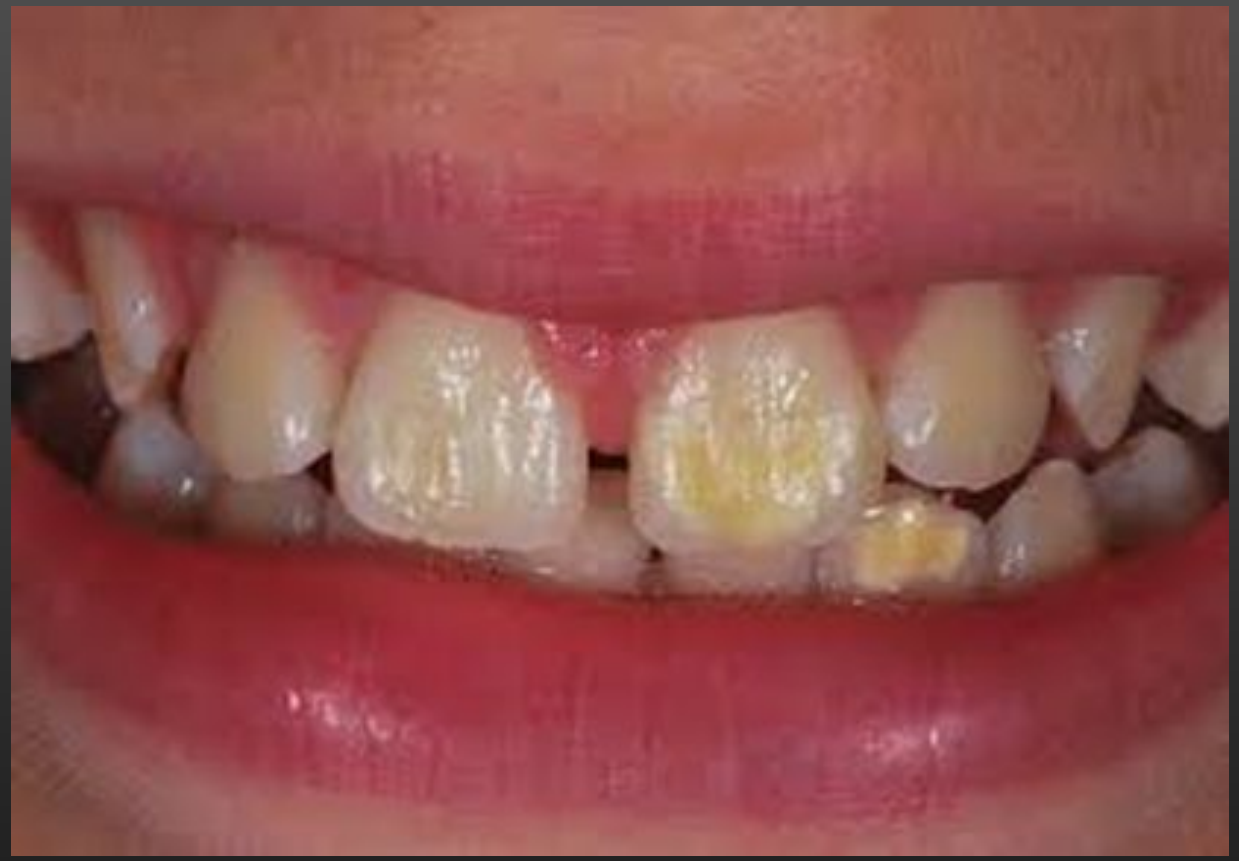
Clinical outcome of various disturbances of Amelogenesis depends on:

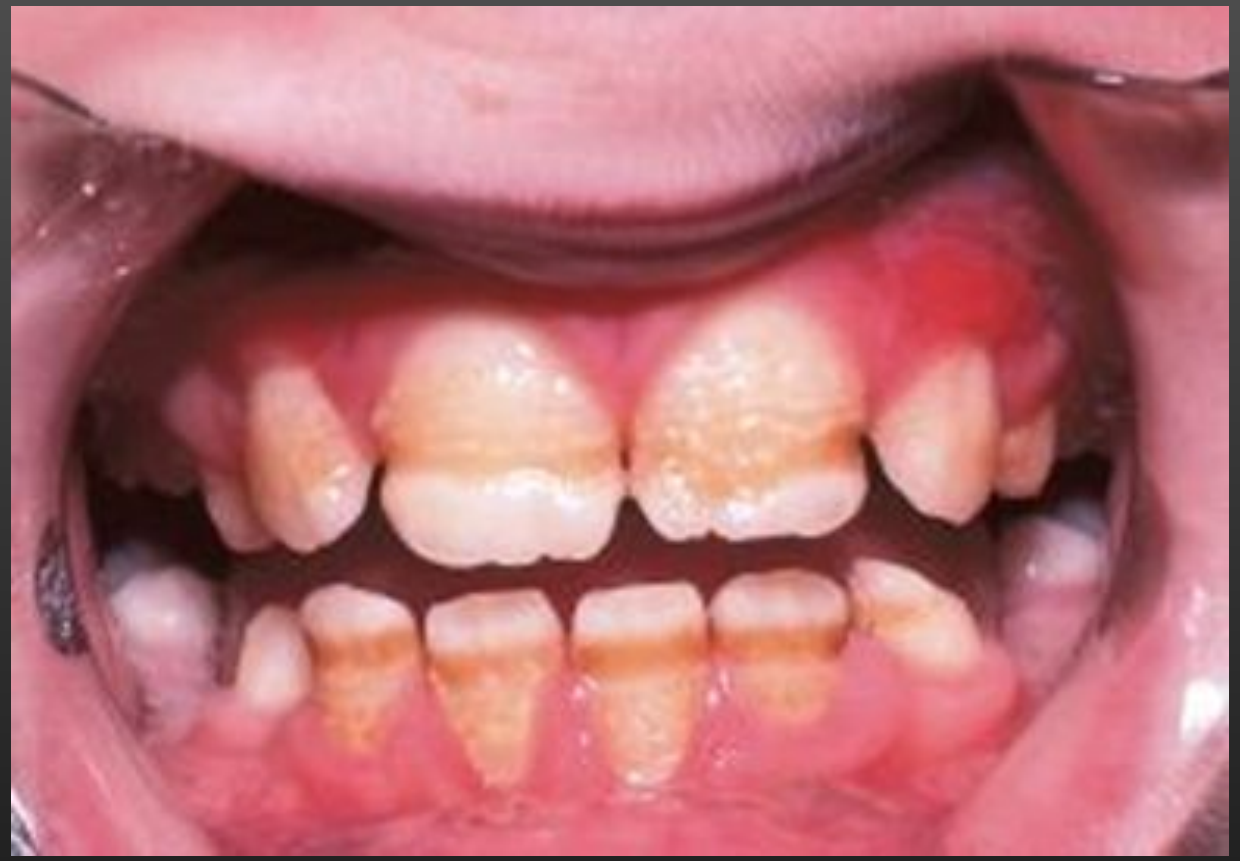
- Timing & stage of development of Enamel
- Severity & duration of the insult

Consequences of Environmental Enamel Hypoplasia

- Mild environmental hypoplasia – few small grooves, pits, or fissures on enamel surface
- Severe conditions - rows of deep pits arranged horizontally across surface of tooth
- Most severe cases, a considerable portion of enamel absent, suggesting a prolonged disturbance in function of ameloblasts







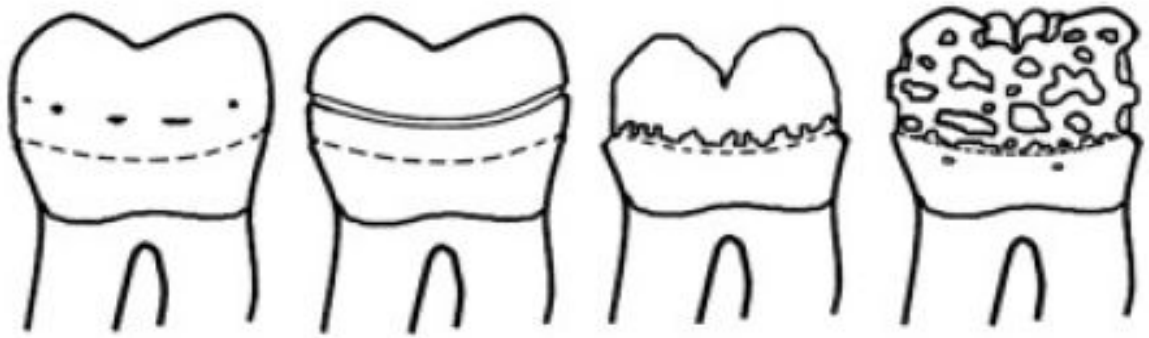
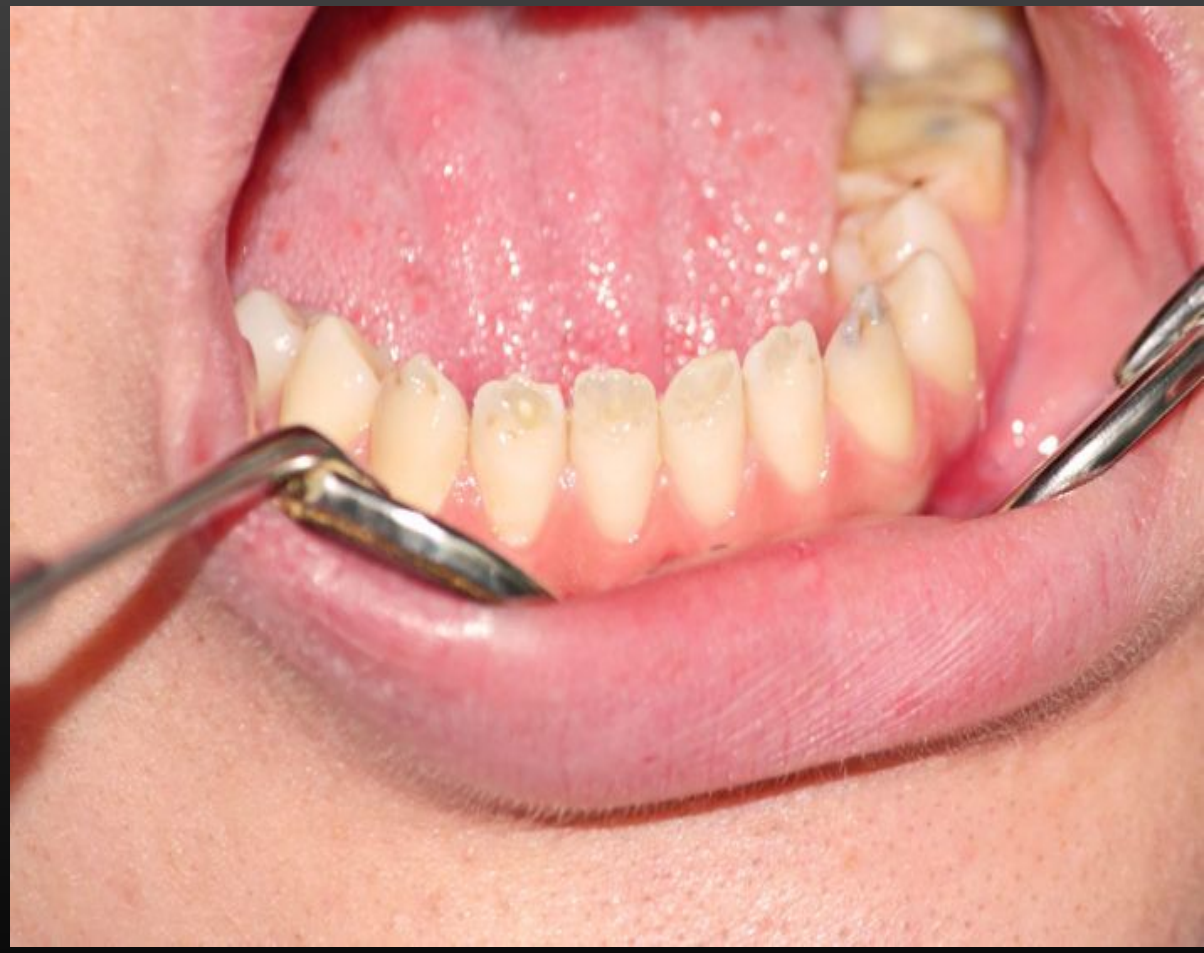


Diagram showing the different expressions of dental defects

Clinical Features

- Teeth that form within the first year after birth
- Teeth most frequently involved - central and lateral incisors, cuspids, and first molars
- Since the tip of the cuspid begins formation before the lateral incisor, some cases involve only the central incisor, cuspid, and first molar

❑ Premolars and second and third molars are seldom affected



Enamel hypoplasia & Dental Caries

- Controversial clinical reports
- Most reasonable to assume that the two are not related
- Hypoplastic teeth do appear to decay at a somewhat more rapid rate once caries has been initiated

Hypoplasia due to Nutritional Deficiency & Exanthematous Fevers

Nutritional deficiencies

- Deficiencies of vitamin A & C
- Rickets during time of tooth formation - most common known cause of enamel hypoplasia

Exanthematous diseases including

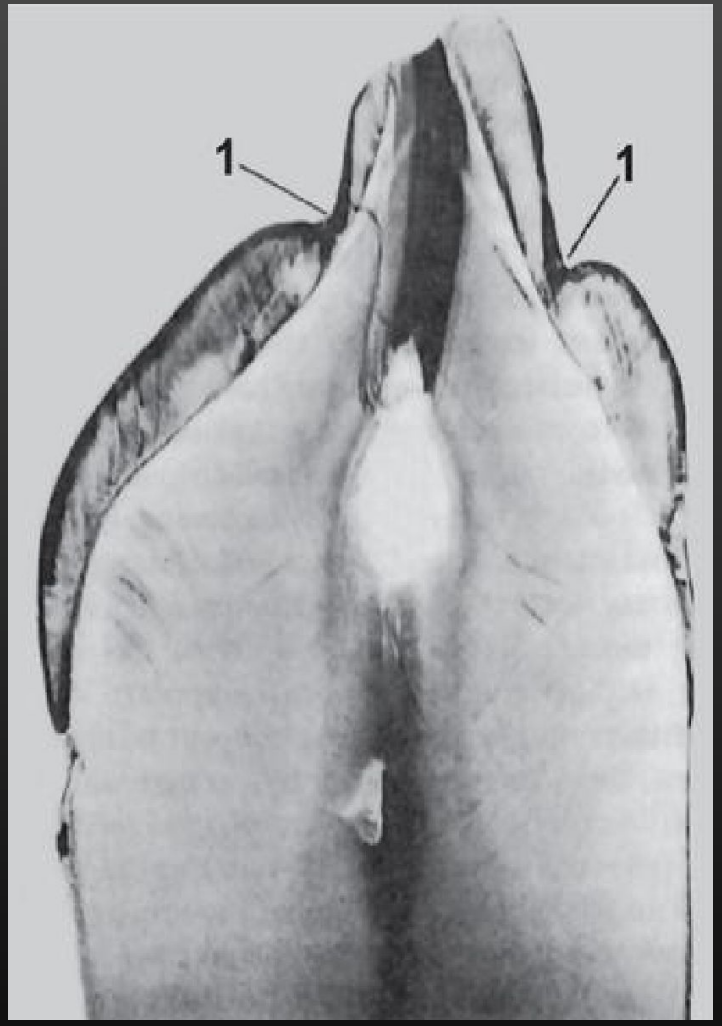
- ✔ Measles
- ✔ Chickenpox
- ✔ Scarlet fever
- ✔ Any serious nutritional deficiency or systemic disease is potentially capable of producing enamel hypoplasia

Tetracycline & its derivatives in Tooth Staining

- Tetracycline, broad spectrum antibiotic & its derivative minocycline is common in treatment of acne
- It chelates calcium ions & is incorporated into teeth, cartilage & bone
- Ingestion during the years of tooth development causes a yellow-green discoloration of dentin, which is visible through the enamel and fluorescent under ultraviolet light
- Later tetracycline oxidizes & staining becomes more brown & no longer fluoresces under UV light
- Because tetracyclines crosses placenta, a child may have tooth staining if drugs are administered during mother's pregnancy

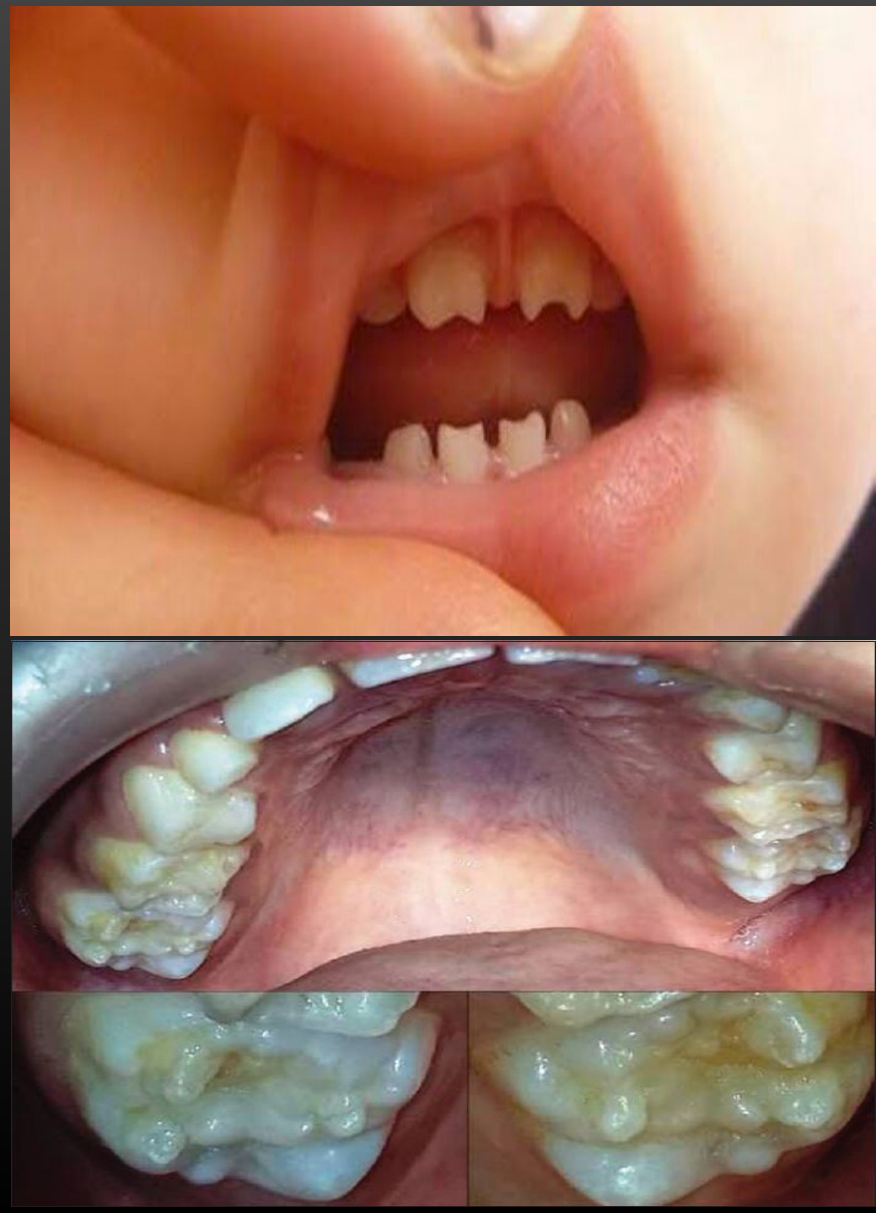
Clinical Features

- Pitting variety of enamel hypoplasia
- Tends to stain
- Clinically unsightly



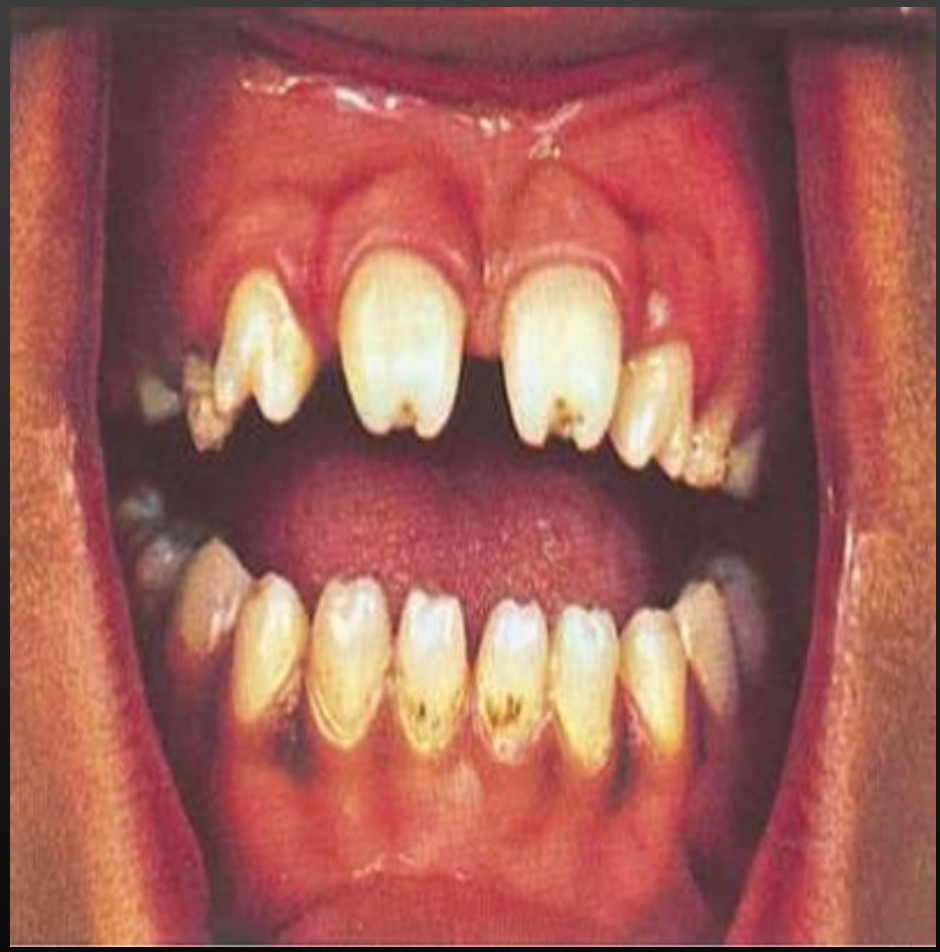
Enamel Hypoplasia due to Congenital Syphilis

- ‘Hutchinson’s teeth’
- ‘Mulberry molars’ (Moon’s molars, Fournier’s molars)
- Hypoplasia is characteristic, pathognomonic appearance
- Maxillary & mandibular permanent incisors & first molars



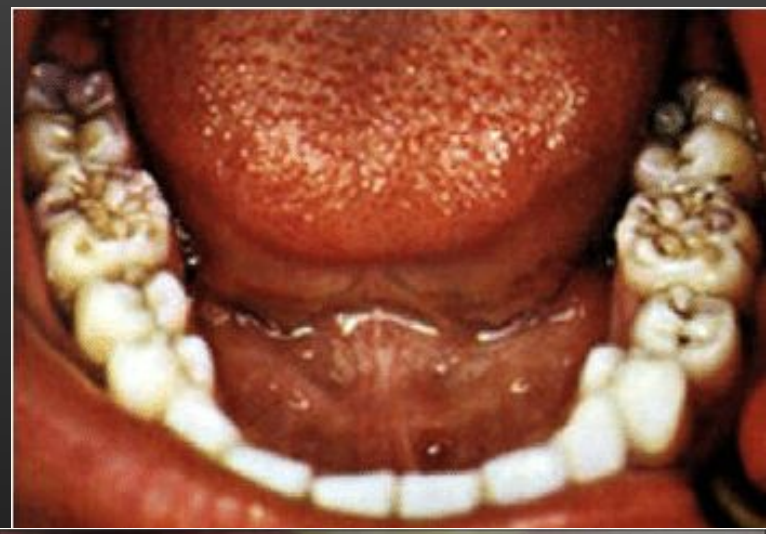
‘Hutchinson’s Incisors’ – Congenital Syphilis

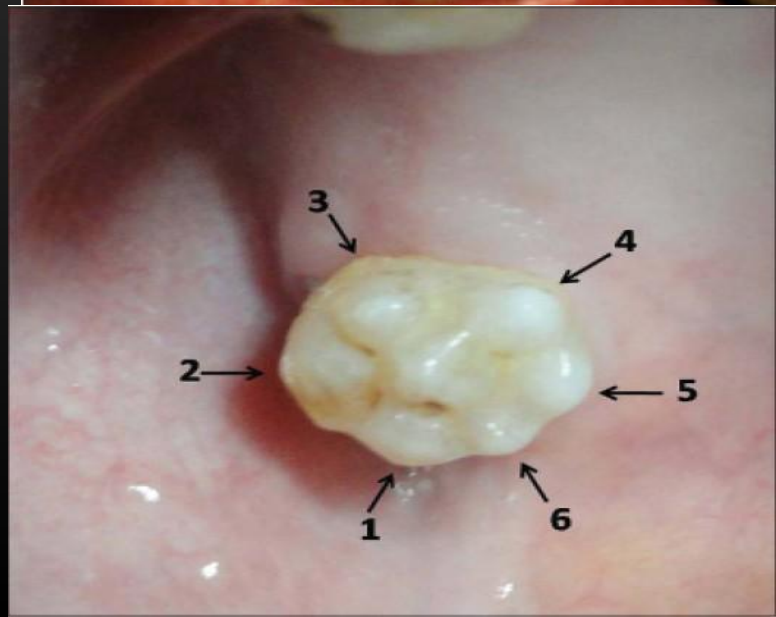
- Characteristically, upper central incisor (also, mandibular central & lateral incisors)- ‘screw-driver’ shaped (mesial & distal surfaces of crown tapering & converging toward incisal edge rather than toward cervical margin
- Incisal edge - usually notched
- Maxillary lateral incisor may be normal
- Cause: absence of central tubercle or calcification center



‘Mulberry Molars’ in Congenital Syphilis

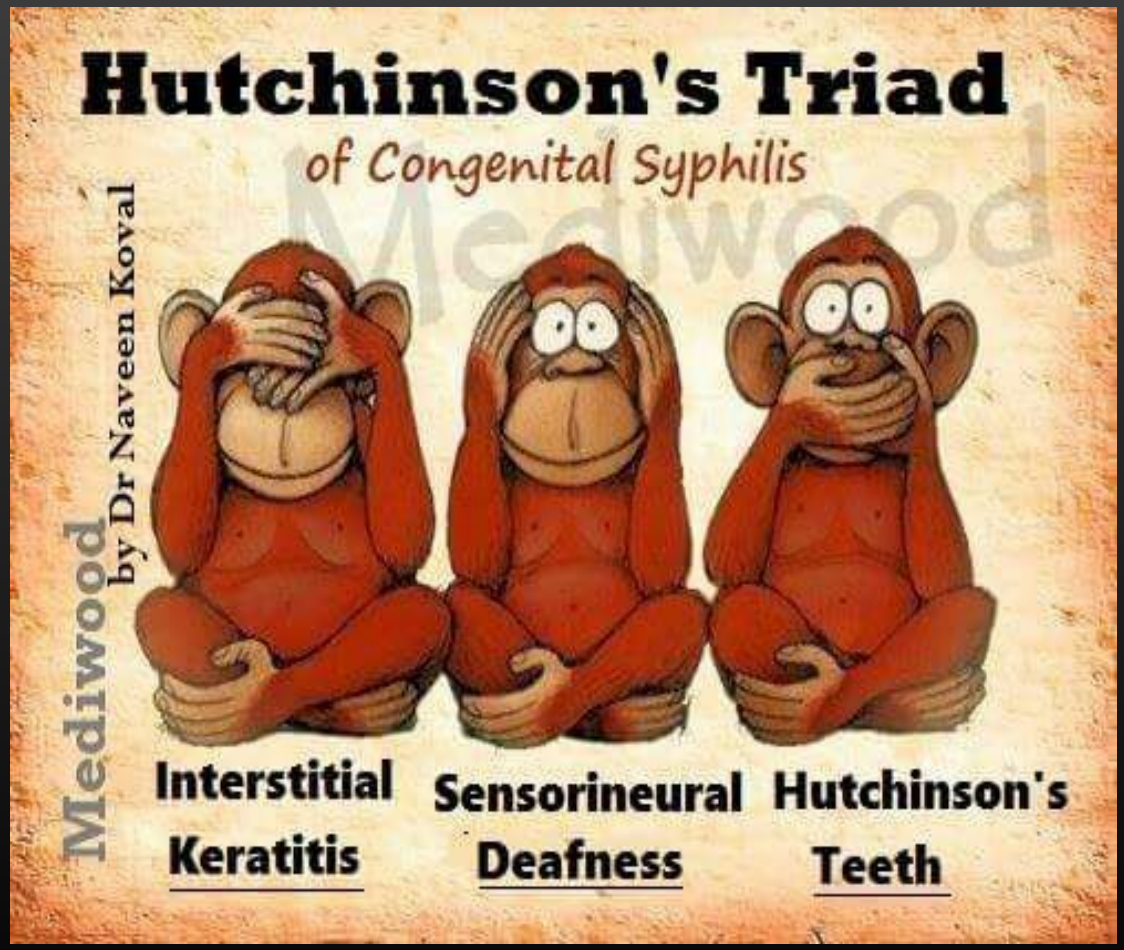
- Crowns of first molars - irregular
- Enamel of occlusal surface & occlusal third of tooth appears to be arranged in an agglomerate mass of globules rather than in well-formed cusps
- Crown narrower on occlusal surface than at cervical margin





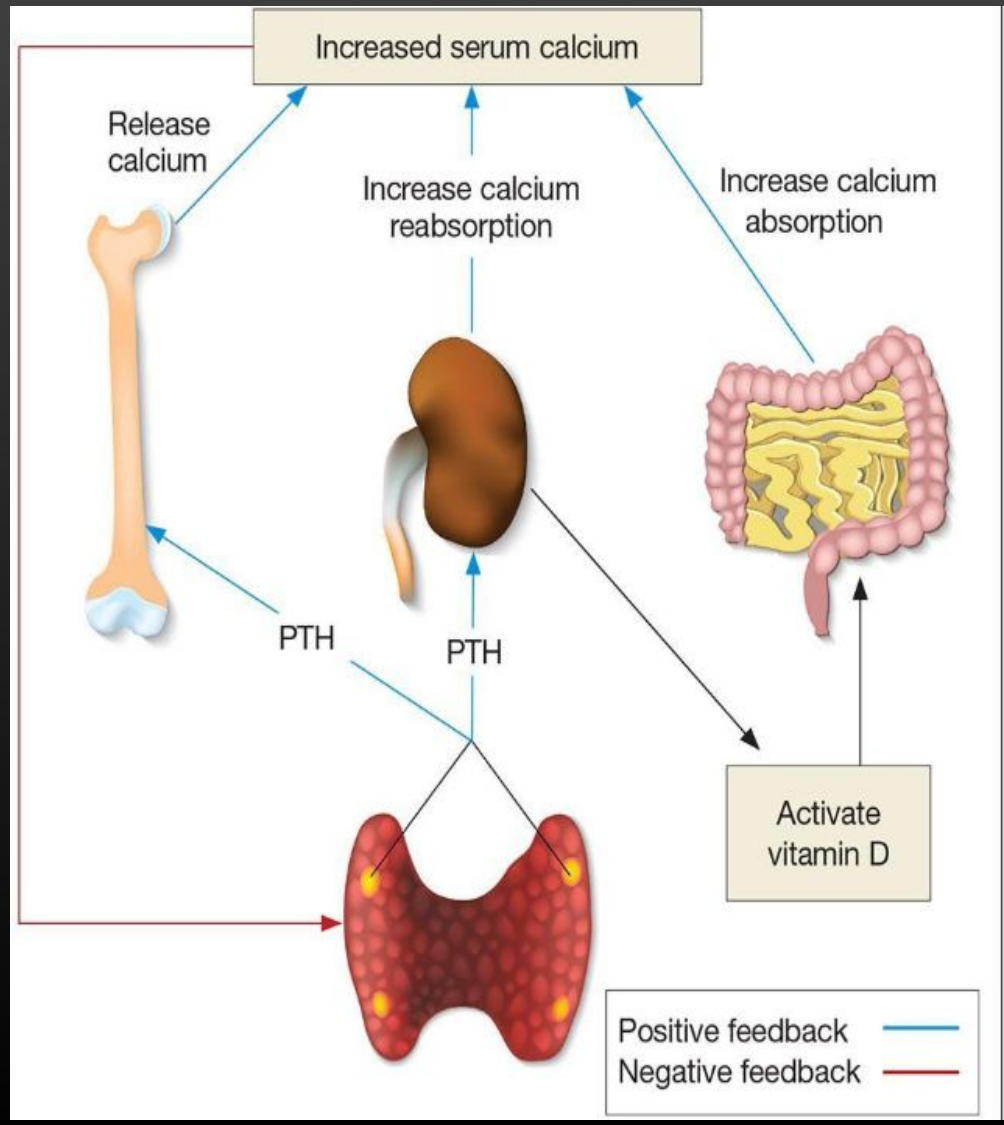
Hutchinson's Triad

- Named after sir Jonathan Hutchinson (1828–1913)
- Common pattern of presentation for Congenital Syphilis
- Consists of three phenomena: interstitial keratitis, malformed teeth (hutchinson incisors & mulberry molars) & eighth nerve deafness
- May also have saddle nose deformity



Enamel Hypoplasia due to Hypocalcemia

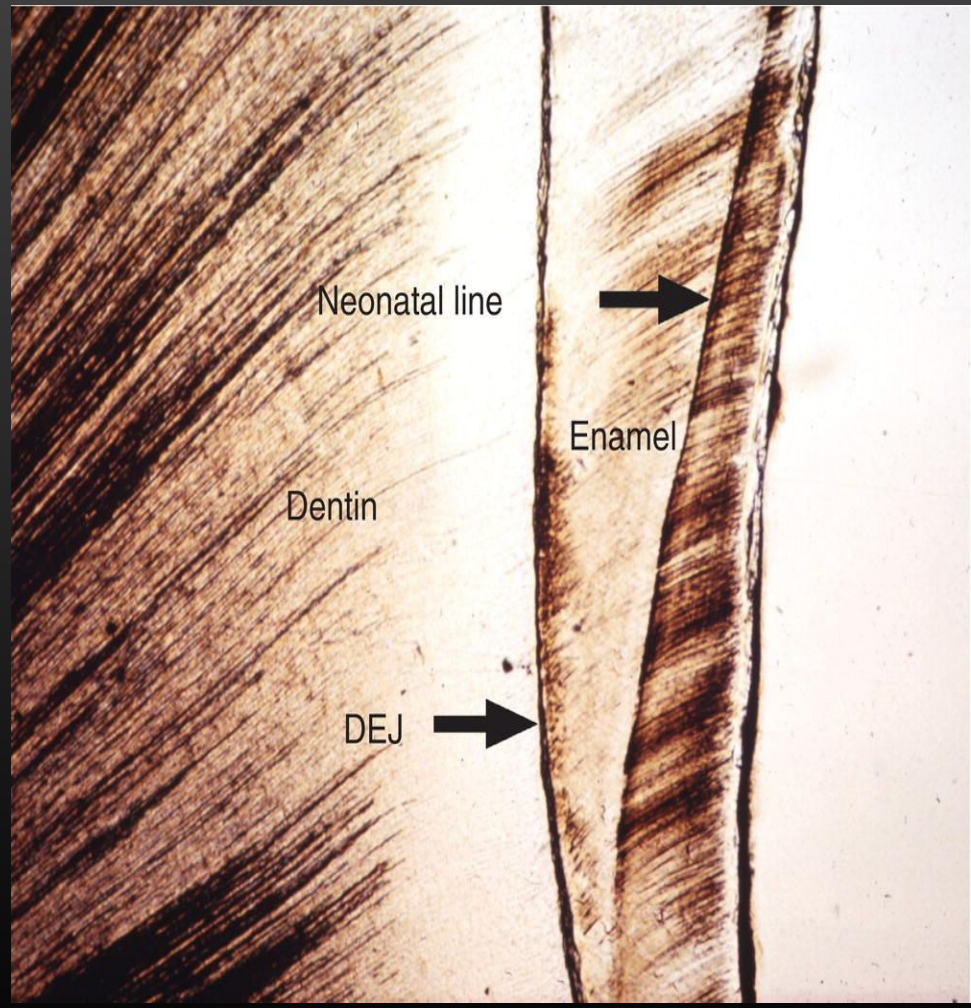
- Tetany, induced by a decreased level of calcium in blood
- May result from several conditions, the most common being vitamin D deficiency & parathyroid deficiency (parathyroprivic tetany)
- In tetany, serum calcium level - as low as 6–8 mg per 100 ml
- At this level, enamel hypoplasia is frequently produced in teeth developing concomitantly
- Type of enamel hypoplasia - pitting variety



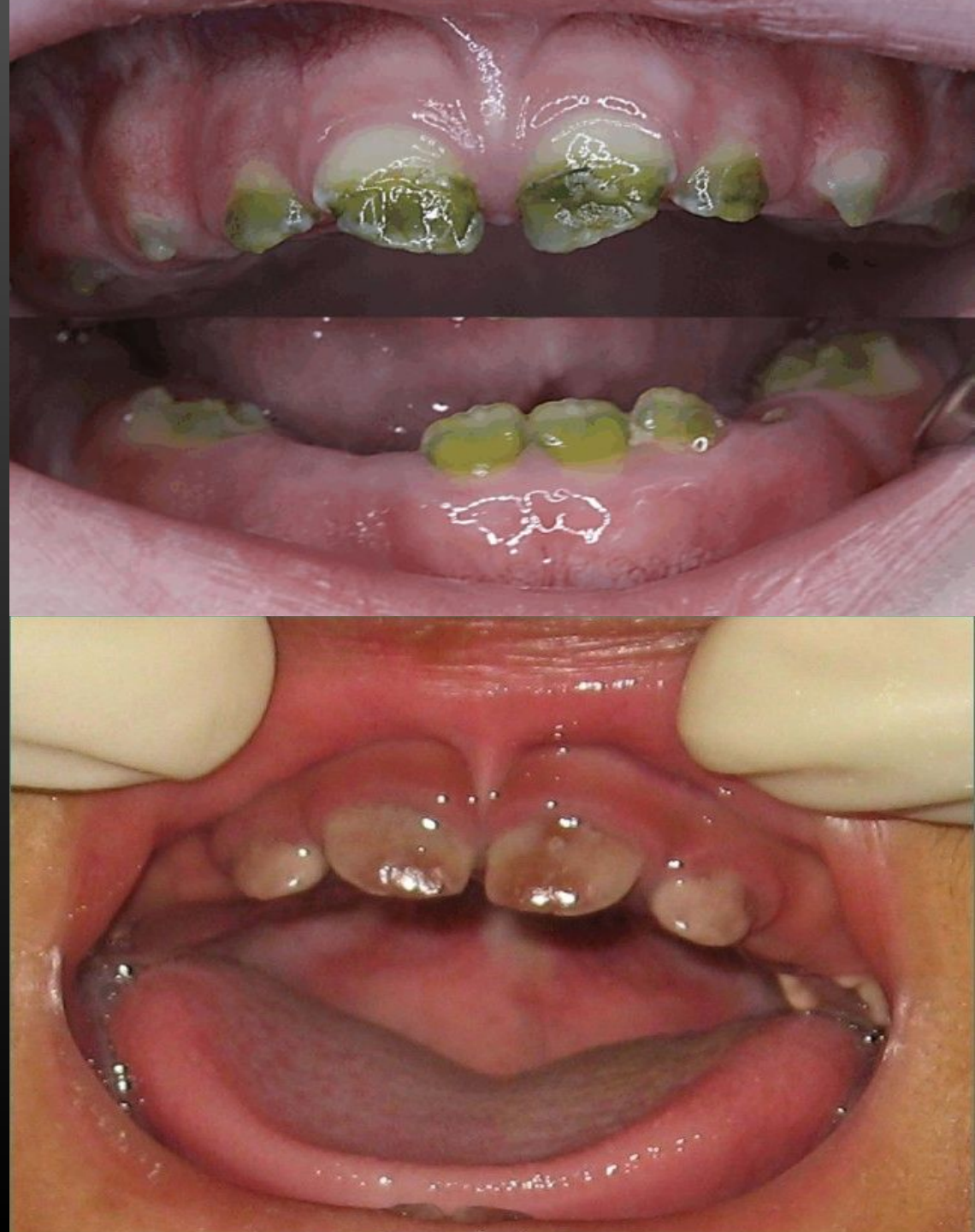
Hypoplasia due to Birth Injuries

The neonatal line or ring - Schour 1936 (THE BIRTH CERTIFICATE IN YOUR MOUTH)

- Present in deciduous teeth & first permanent molars
- May be thought of as a type of hypoplasia
- Disturbance produced in enamel & dentin, which is indicative of trauma or change of environment at time of birth
- In traumatic births, formation of enamel may even cease at this time

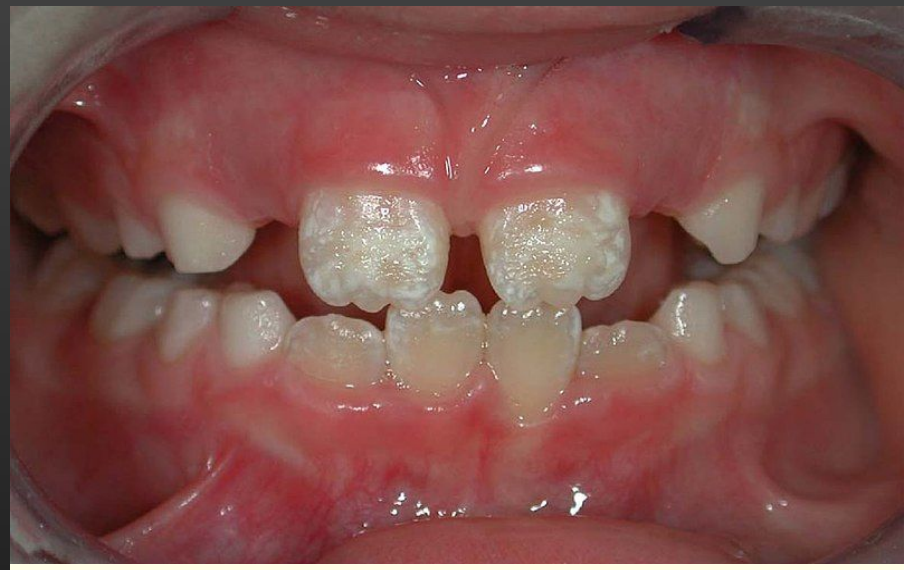


- Enamel hypoplasia - far more common in prematurely born children than in normal term infants
- Staining of teeth in children who had suffered from rh hemolytic disease at birth , also with reported enamel hypoplasia- ring like defect around the crown

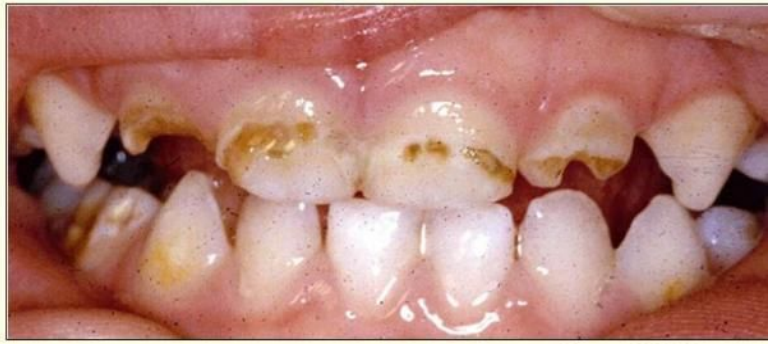


Enamel hypoplasia of deciduous teeth

- Enamel hypoplasia of deciduous teeth –
  - Prenatal or
  - Postnatal enamel
- If prenatal enamel defects are pronounced, a gastrointestinal disturbance (Celiac Disease) or some other illness in the mother may be responsible



Enamel Hypoplasia Due to Maternal Toxemia



Source: TUSDM

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Enamel Hypoplasia due to Local Infection or Trauma - 'Turner's hypoplasia' or 'Turner's Teeth'

- A single tooth is involved – 'Turner's Teeth'
- Most commonly one of the permanent maxillary incisors or a maxillary or mandibular premolar
- Any degree of hypoplasia - mild, brownish discoloration of enamel to a severe pitting & irregularity of tooth crown

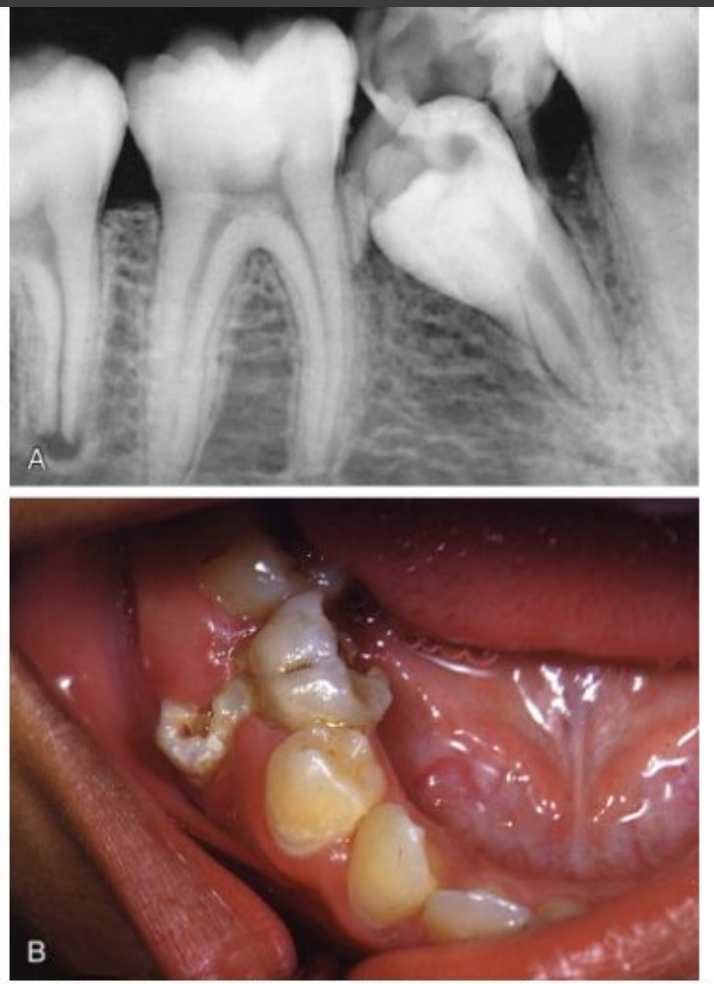


Figure 3-16. **A,** Infected mandibular second primary molar has caused hypoplasia of the second premolar and delayed eruption of the tooth. **B,** Hypoplasia is evident in the occlusal third of the second premolar.

- A similar type of hypoplasia may follow trauma to a deciduous tooth, particularly when deciduous tooth has been driven into alveolus & has disturbed permanent tooth bud
- If this permanent tooth crown is still being formed, resulting injury may be manifested as a yellowish or brownish stain or pigmentation of enamel, usually on the labial surface, or as a true hypoplastic pitting defect or deformity

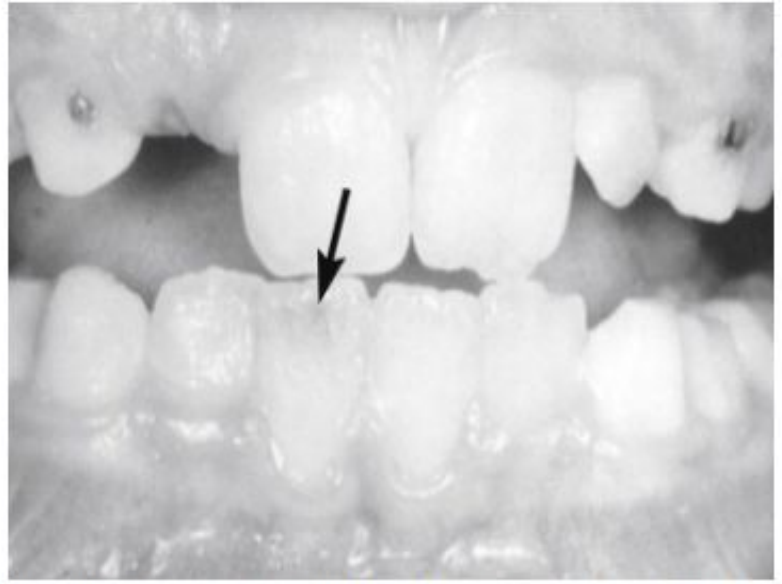


Figure 3-18. Hypoplastic defect on the labial surface of a mandibular permanent central incisor *(arrow)*. There was a history of trauma to the primary tooth.

Enamel Hypoplasia due to Fluoride: Mottled Enamel

- Frederick McKay in Colorado Springs (1901) discovered a high proportion of the residents had stained teeth, locally termed the "Colorado brown stain”
- Greene Vardiman Black (1916) described the condition as "[a]n endemic imperfection of the enamel of the teeth, heretofore unknown in the literature of dentistry“
- Interestingly, although the mottled enamel was hypomineralized, and therefore should be more susceptible to decay, this was not the case
- Gradually, they became aware of existing & further reports of a similar condition worldwide

- In 1931, 3 different groups of scientists around the world published their discoveries that this condition was caused by fluoride in drinking water during childhood
- The condition then started to become termed "dental fluorosis“
- Through epidemiological studies in the US, Henry Trendley Dean helped to identify a causal link between high concentrations of fluoride in drinking water & mottled enamel
- He also produced a classification system for dental fluorosis that is still used in modern times, Dean's Index
- As research continued, the protective effect of fluoride against dental decay was demonstrated

Etiology of Mottled Enamel

- Ingestion of fluoride-containing drinking water during the time of tooth formation may result in mottled enamel
- Severity of mottling increases with an increasing amount of fluoride in the water
- Little mottling of any clinical significance at a level below 0.9–1.0 part per million of fluoride in water
- Mottling becomes progressively evident above this level (>5 ppm)

Pathogenesis in Dental Fluorosis

- Disturbance of ameloblasts during formative stage of tooth development
- Exact nature of injury not known
- Enamel matrix defective or deficient
- Higher levels of fluoride interferes with calcification process of matrix

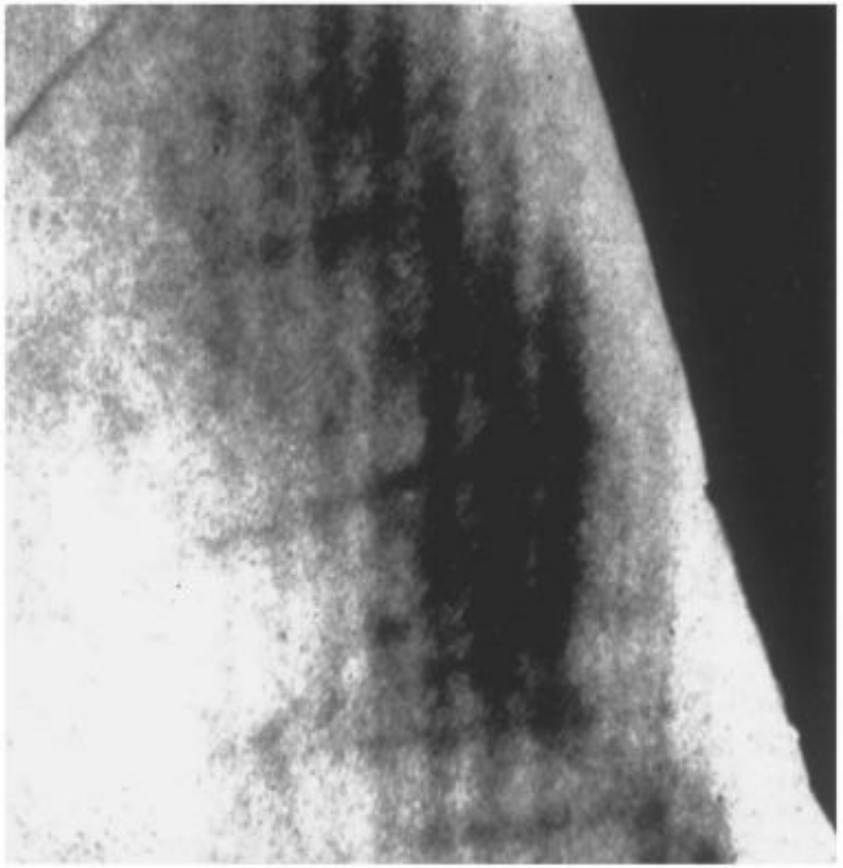
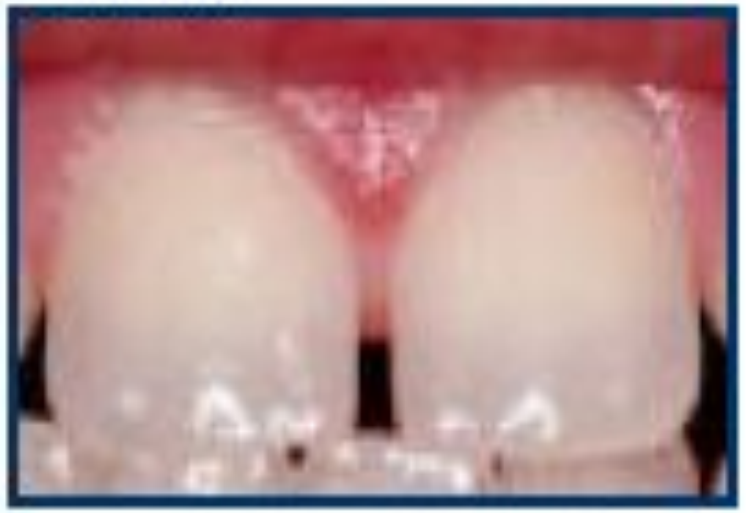


Fig. 4.
Microradiograph of fluorosed enamel from Colorado Springs. Note the radiolucent outer third of the enamel with a well-calcified surface layer. From Newbrun [97], reprinted with permission.

Normal



Questionable

Very mild

Mild

Moderate

Severe

Classification	Code	Criteria – description of enamel
Normal	0	The enamel represents the usual translucent semivitriform (glass-like) type of structure. The surface is smooth, glossy and usually of pale creamy white color
Questionable	1	The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots. This classification is utilised in those instances where a definite diagnosis is not warranted and a classification of 'normal' not justified
Very Mild	2	Small, opaque, paper white areas scattered irregularly over the tooth but not involving as much as approximately 25% of the tooth surface. Frequently included in this classification are teeth showing no more than about 1 – 2mm of white opacity at the tip of the summit of the cusps, of the bicuspids or second molars.
Mild	3	The white opaque areas in the enamel of the teeth are more extensive but do involve as much as 50% of the tooth.
Moderate	4	All enamel surfaces of the teeth are affected and surfaces subject to attrition show wear. Brown stain is frequently a disfiguring feature
Severe	5	All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

Differential Diagnosis for Dental Fluorosis

1. Turner's hypoplasia (although this is usually more localized)
2. Enamel defects caused by an undiagnosed and untreated celiac disease
3. Some mild forms of amelogenesis imperfecta and enamel hypoplasia
4. Enamel defects caused by infection of a primary tooth predecessor
5. Dental caries: Fluorosis-resembling enamel defects are often misdiagnosed as dental caries
6. Dental Trauma: Mechanical trauma to the primary tooth may cause disturbance to the maturation phase of enamel formation, which may result in enamel opacities on the permanent successors

Geographic Distribution of Dental Fluorosis

- Fluorosis is worldwide in distribution & endemic at least in 25 countries. It has been reported from **fluoride belts**: one that stretches from Syria through Jordan, Egypt, Libya, Algeria, Sudan and Kenya, and another that stretches from Turkey through Iraq, Iran, Afghanistan, **India**, northern Thailand and China
- In some parts of Africa, China, the Middle East and southern Asia (India, Sri Lanka), as well as some areas in the Americas and Japan, high concentrations of ionic fluoride have been found in ground waters, vegetables, fruit, tea & other crops, although drinking water is usually the major source of the daily fluoride intake

State/Area	Age-group (Years)	Prevalence (%)	Author
Cuddalore, TN	5-12	31.4	Sarvanan et.al. Indian J Community Med. 2008; 33(3): 146–150.
Alapuzzha, kerala	10-17	35.6	Gopalakrishnan et.al. Natl Med J India. 1999; 12(3):99-103.
Vadodara, Gujarat	Adults	39.2 - 59.3	Kotecha et al. Indian J Med Res. 2012 June; 135(6): 873–877.
Davangere, karnataka	12-15	13-100	Chandrasekhar and Anuradha. Int Dent J. 2004; 54(5):235-9.
Jhajjar, Haryana	7-15	30-94.9	Yadav et al. Environ Geochem Health. 2009; 31(4):431-8.
Birbhum, West Bengal	Adults	61-66.7	Majumdhar. Indian J Public Health 2011; 55:303-8.
Punjab	5-60	91.1	Shashi and Bhardwaj. Biosci. Biotech. Res. Comm. 2011; 2:155-163.
Nalgonda, A.P	12-15	71.5	Shekar et al. Indian J Public Health. 2012; 56(2):122-8.
Durg, Chattisgarh	Adults	8.2	Pandey. Trop Doct. 2010; 40(4):217-9.
Dungarpur, Udaipur (Rajasthan)	All ages	39.2-72.1	Choubisa et al. J Environ Sci Eng. 2010; 52(3):199-204.
Palamau Jharkhand	children	83.2	Srikanth et al. Research report Fluoride. 2008; 41(3)206–211.
Assam	All ages	31.3	Chakraborti et al. Current Science. 2000; 78 (12): 1421-1423.
Uttar Pradesh	All ages	28.6	Srivastava et al. Int J Oral & Maxillofacial Pathology; 2011:2(2):7-12.
Kareka, Shivpuri Madhya Pradesh	13-50	86.8	Saksena and Narwaria. Int j Environ Sci. 2012; 3(3).
Raigad, Maharashtra	0-23	91.7	Bawaskar and Bawaskar. Trop Doct. 2006; 36: 221.
Nalgonda, A.P	Adults	30.6	Nirgude et al. Indian J Public Health. 2010;54(4):194-6.

TOP 5 STATES WORST AFFECTED BY FLUORIDE

Number of habitations affected

Rajasthan 5,143	**West Bengal** 1,263
Bihar 781	**Karnataka** 365
Andhra Pradesh 317	

Source: Union ministry of drinking water and sanitation

DENTAL FLUOROSIS Sparkling white teeth become dull and yellow. White spots appear on tooth surface. Gradually these spots turn into brown streaks. As it worsens, the teeth get pitted, perforated and even chip off over the years

SKELETAL FLUOROSIS
A crippling disease resulting from deposits of fluoride in hard and soft tissues of the body

BELLANDUR WORST HIT IN BENGALURU

In 2009, a team from the department of mines and geology found Bellandur had the highest content of fluoride in water in Bengaluru. It even summoned a property owner who had six borewells. "We were shocked to learn the man was selling water to nearby schools and hospitals. The fluoride content ranged from 5.5 to 6.3ppm," officers said. TOI's efforts to locate the property were in vain as the team of officers who had worked on the case had retired and official records contained no measures taken. While fluoride content has nothing to do with the infamous Bellandur lake, the proliferation of borewells in the area is due to lack of fresh water from the lake.

OTHER EXTREME ON THE COAST

"While some districts in the state have groundwater that is high in fluoride content, in coastal areas it's the other extreme where fluoride is less than 0.5 mg/l. In such places, people need to use fluoride-based toothpastes. But people living in areas where fluoride content is very high are also using such toothpastes which is only making the problem worse

NJ Devaraj Reddy | GEOLOGIST AND RAIN WATER HARVESTING EXPERT, GEO RAIN WATER BOARD, CHITRADURGA

BOREWELL STORY

3.7lakh
borewells in Bengaluru, according to BWSSB

500mtr
Minimum distance between two borewells as per mines and geology department

60% Applications for borewells that are rejected by BWSSB

Source: BWSSB

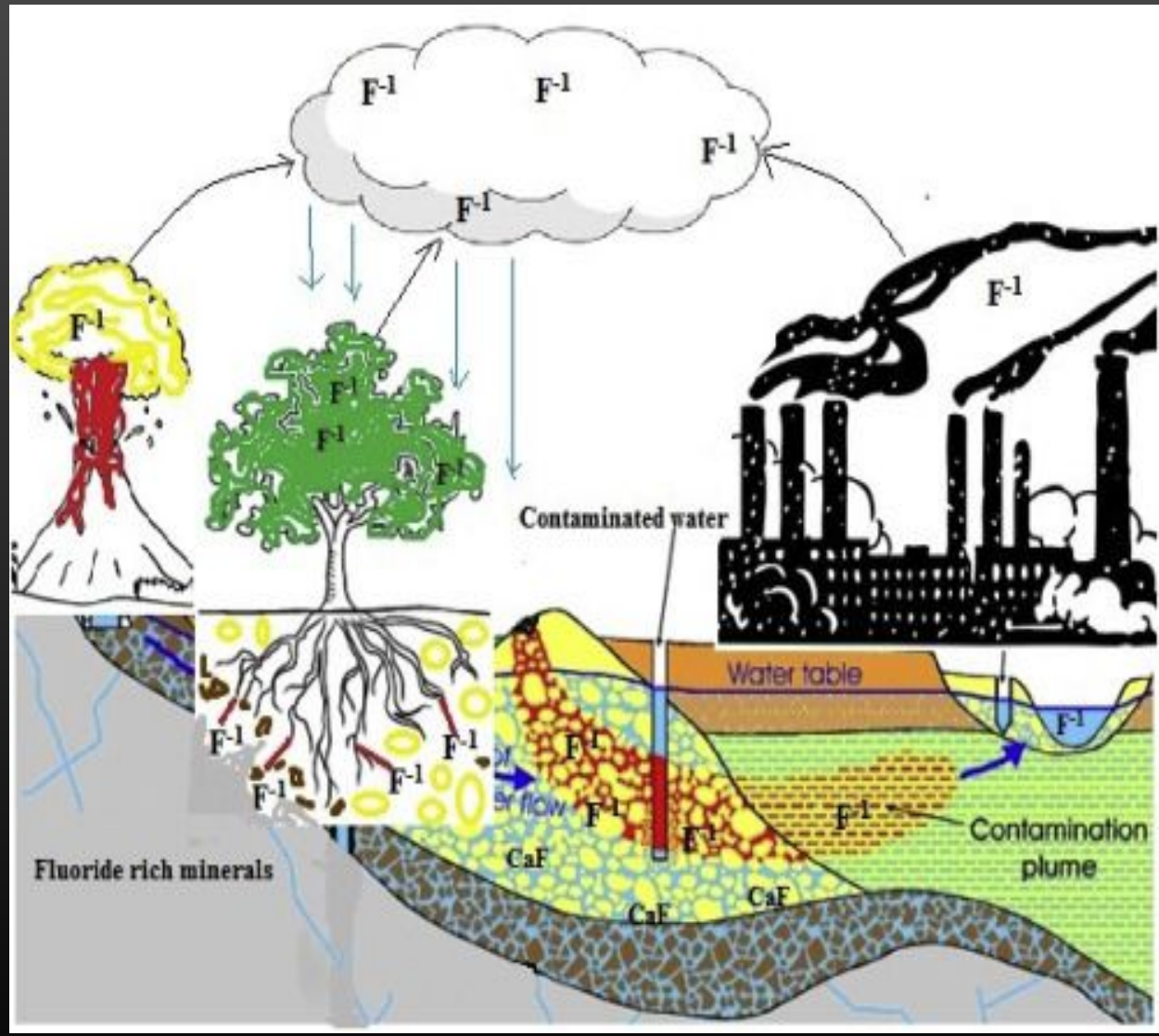
MAJOR REASONS FOR REJECTION
Public borewells nearby, BBWSSB supplying Cauvery water to the area, or the area not falling under BWSSB limits

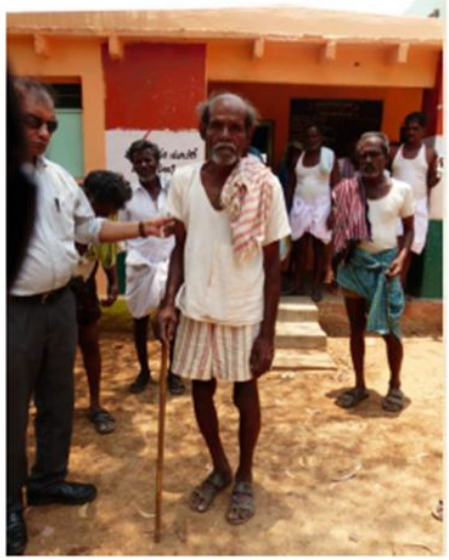
TOP FIVE

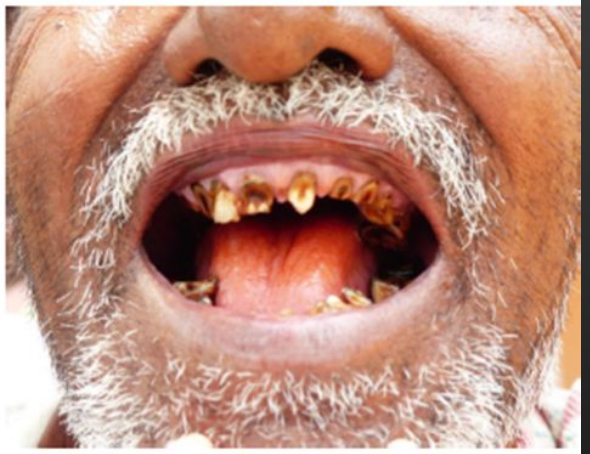
DISTRICTS WITH HIGHEST CONTAMINATION IN GROUNDWATER

Excess fluoride	No. of water sources
Chickballapur	689
Tumkur	689
Bellary	489
Chitradurga	360
Kolar	289

Excess nitrate	No. of water sources
Mandya	767
Kolar	647
Davangere	442
Bangalore Urban	399
Tumkur	359







A B

Figure A and B representing case of Skeletal and Dental Fluorosis due to excess intake of fluoride from drinking water. Generalized attrition and erosion is a common feature among elderly population group. (Curtsey: Professor Chitta Chowdhury & Team. Consent of Photograph was taken)

Two primary sources have been identified as being potentially responsible for prevalence of dental fluorosis:

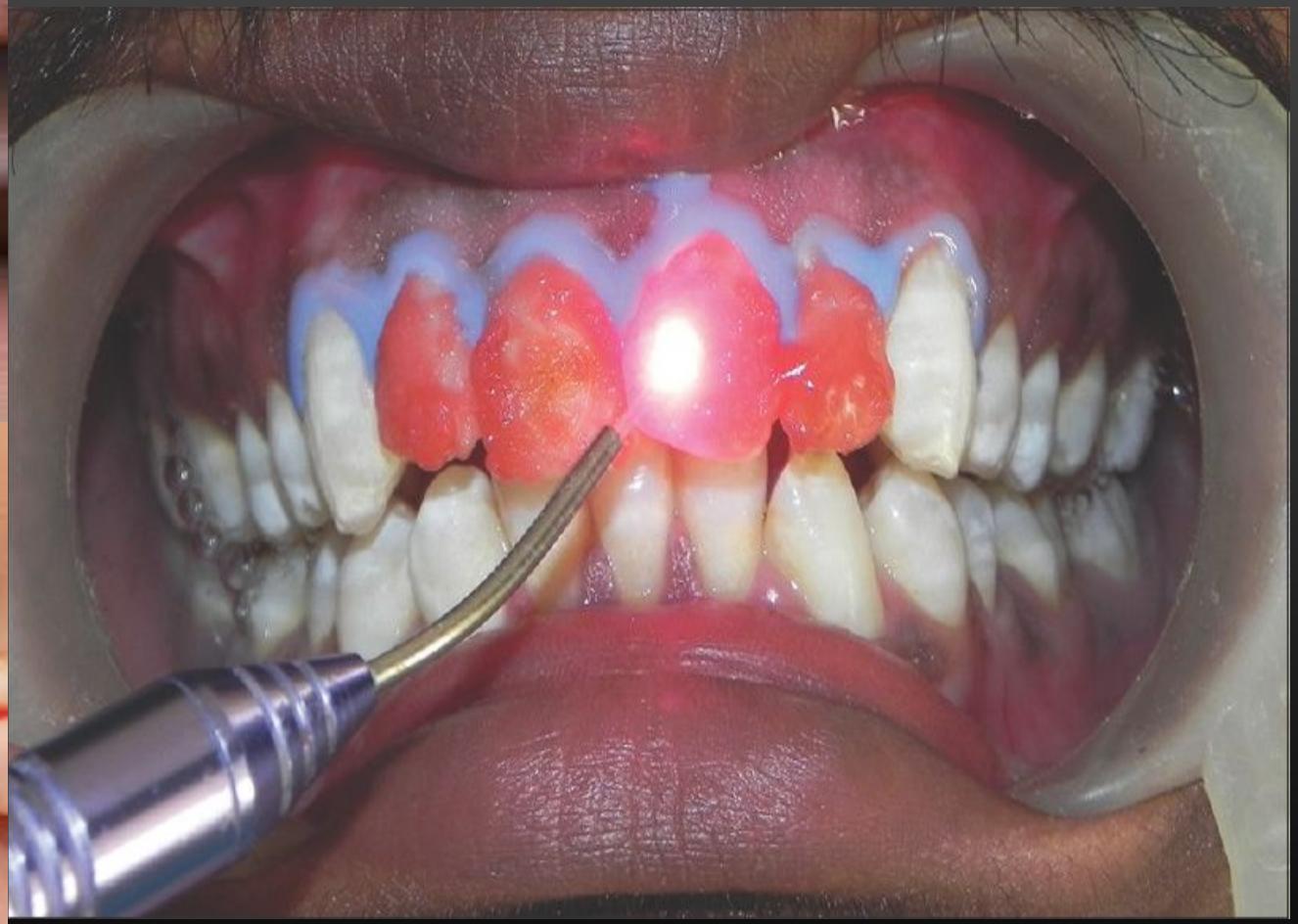
1. Fluoride in drinking water
2. Fluoride-containing dental products – dentifrices, mouth washes & fluoride supplements

Treatment of Dental Fluorosis

- Dental fluorosis is of cosmetic concern
- There is varying degrees of negative psychosocial effects

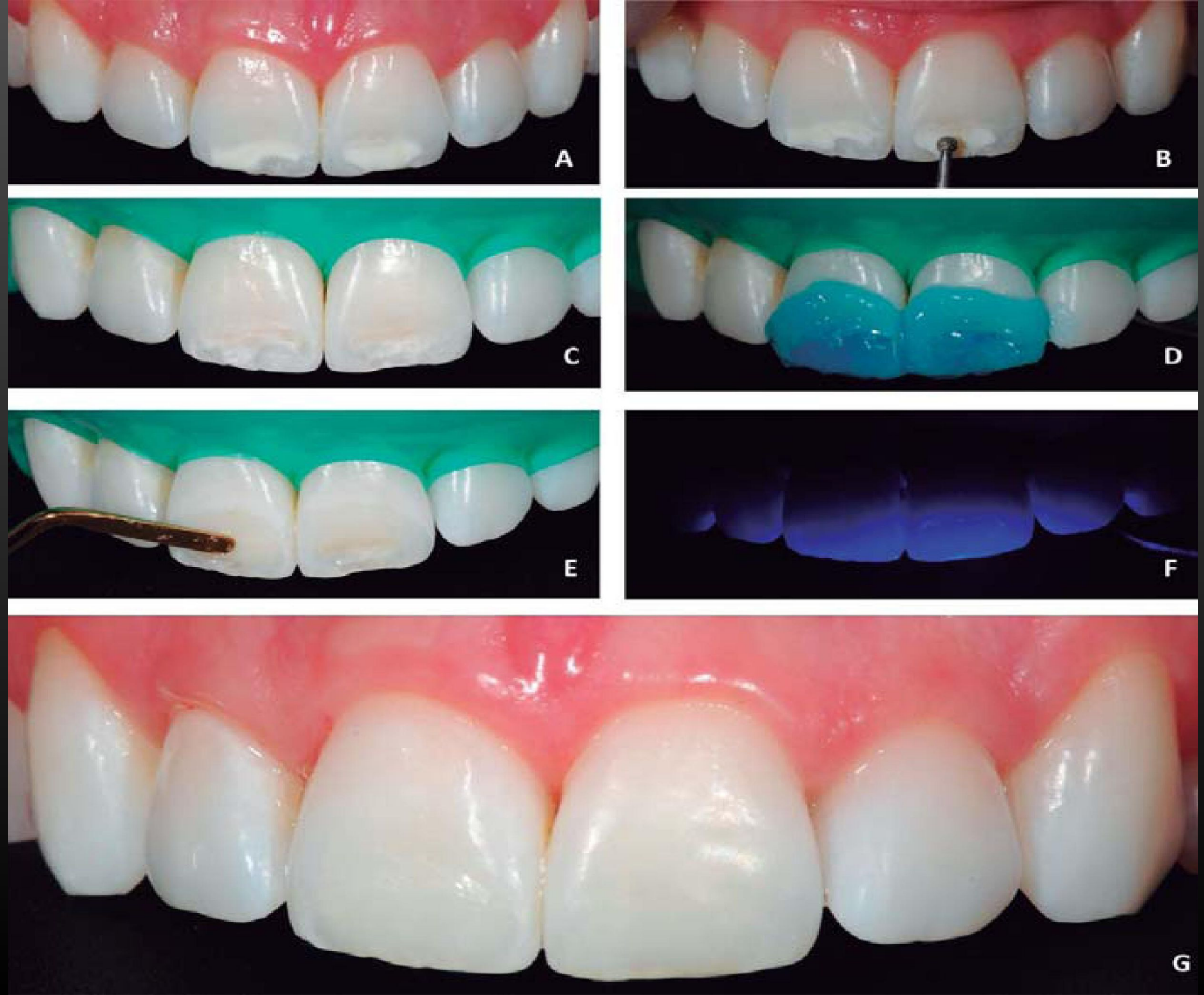
Treatment options:

- ❑ Mild cases: Tooth bleaching
- ❑ Moderate cases: Micro-abrasion (outer affected layer of enamel is abraded in an acidic environment)
- ❑ Severe cases: Composite fillings, Micro-abrasion, Veneers, Crowns



Bleaching

Vital Bleaching	Home Bleaching
1. Informed consent and case history 2. Rubber dam 3. First, in-office bleaching utilizes a high concentration of tooth-whitening agents (25 4. –40% hydrogen peroxide). 5. Light for around one hour in the dental office	1. Whitening agent (10–20% carbamide peroxide, which equals 3.5–6.5% hydrogen peroxide). 2. It is recommended that the 10% carbamide peroxide be used 8 h per day, and the 15–20% carbamide peroxide 3–4 h per day. 3. The bleaching gel is applied to the teeth through a custom-fabricated mouth guard worn at night for at least 2 weeks.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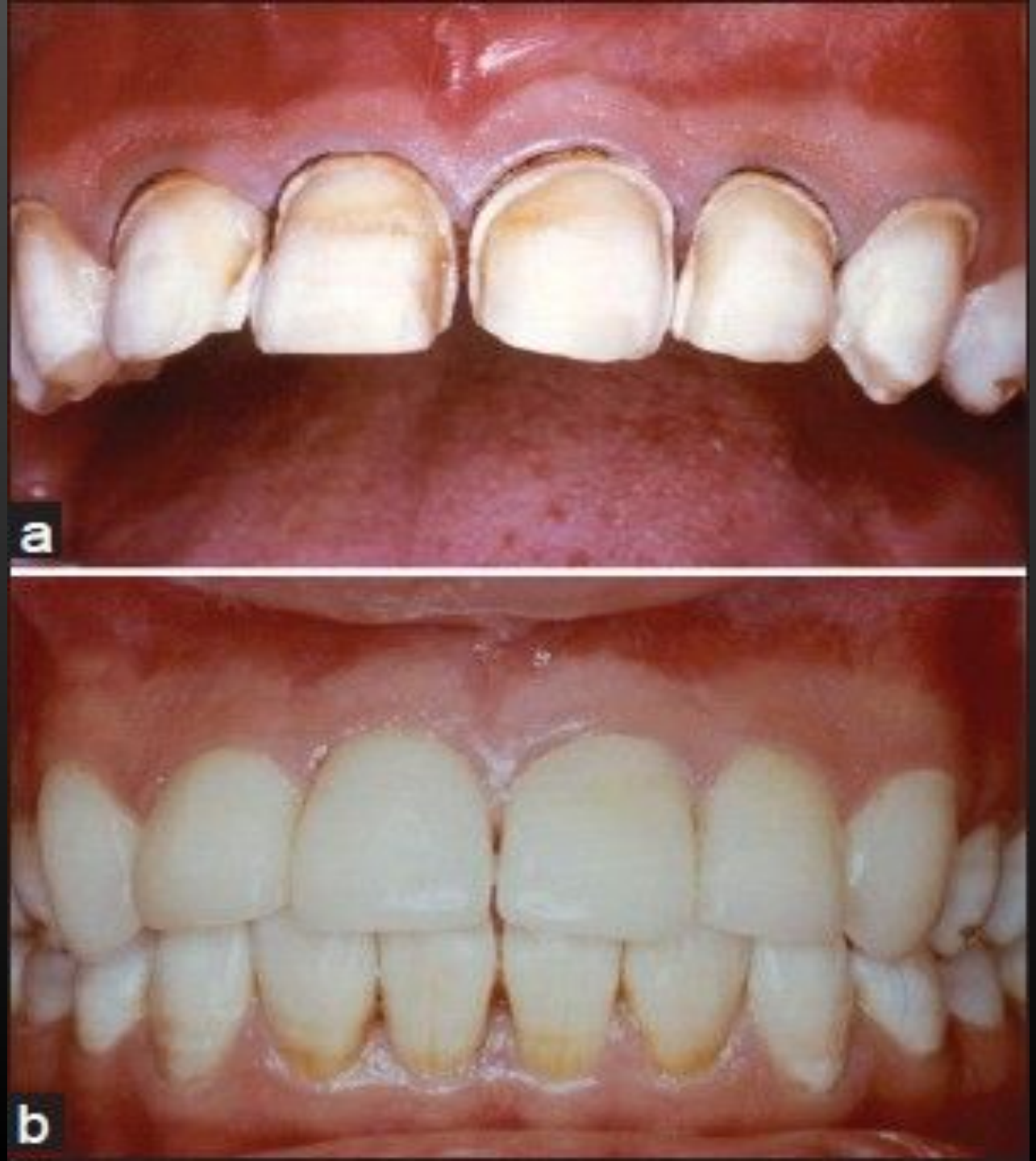
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Hypoplasia due to Idiopathic Factors

- Even with careful histories, majority of cases are of unknown origin
- Ameloblasts are sensitive type of cells & easily damaged & in those cases in which etiology cannot be determined, causative agent may have been some illness or systemic disturbance so mild that it made no impression on patient & was not remembered
- Even relatively severe cases of enamel hypoplasia arise with no pertinent past medical history to account for their occurrence

DEVELOPMENTAL DISTURBANCES IN THE STRUCTURE OF DENTINE

1. Dentinogenesis imperfecta
2. Dentin Dysplasia
3. Regional odontodysplasia
4. Dentin hypocalcification

- Dentinogenesis is the formation of dentin, which starts before amelogenesis
- Dentin is formed by odontoblast cells
- Dentinogenesis takes place in two phases:
    I. Formation of organic collagen matrix
    II. Deposition of hydroxyappatite crystals

DENTINOGENESIS IMPERFECTA

- Is an inherited disorder of dentin formation
- Autosomal dominant condition
- Affects deciduous & permanent teeth

- SHIELDS CLASSIFICATION

  TYPE – I, TYPE – II,TYPE- III

- REVISED CLASSIFICATION

  - DENTINOGENESIS IMPERFECTA 1
  - DENTINOGENESIS IMPERFECTA 2

- WITKOP CLASSIFICATION

  - DENTINOGENESIS IMPERFECTA
  - HEREDITARY OPALASCENT DENTIN
  - BRANDYWINE ISOLATE

SHIELDS CLASSIFICATION

- TYPE I: Occurs in patients affected with osteogenesis imperfecta
- TYPE II : Is not associated with osteogenesis imperfecta
- TYPE III: “Brandywine type” rare condition, seen in racial isolate of Maryland, exhibits multiple pulp exposures and periapical lesions in deciduous dentition

REVISED CLASSIFICATION

- DENTINOGENESIS IMPERFECTA 1 - Without osteogenesis imperfecta Corresponds to type II of shields classification
- DENTINOGENESIS IMPERFECTA 2 - Corresponds to type III of shields classification

THERE IS NO SUBSTITUTE IN THE PRESENT CLASSIFICATION FOR THE CATEGORY DESIGNATED AS TYPE I IN THE SHIELDS CLASSIFICATION

SYNONYMS

- OPALESCENT DENTIN
- DENTINOGENESIS IMPERFECTA WITHOUT OSTEOGENESIS IMPERFECTA
- OPALESCENT TEETH WITHOUT OSTEOGENESIS IMPERFECTA
- SHIELDS TYPE II CAPDEPONT TEETH

- MUTATION IN THE DENTIN SIALO PHOSPHO PROTEIN (DSPP) gene (chromosome 4 – gene map locus 4q21.3) encoding DENTIN PHOSPHOPROTEIN (DPP) & DENTIN SIALOPROTEIN (DSP)
- Clearly distinct from Osteogenesis Imperfecta (Type IV B) with opalescent teeth & affects only the teeth
- No increased frequency of bone fracture is seen
- Frequency: 1 in 6000-8000

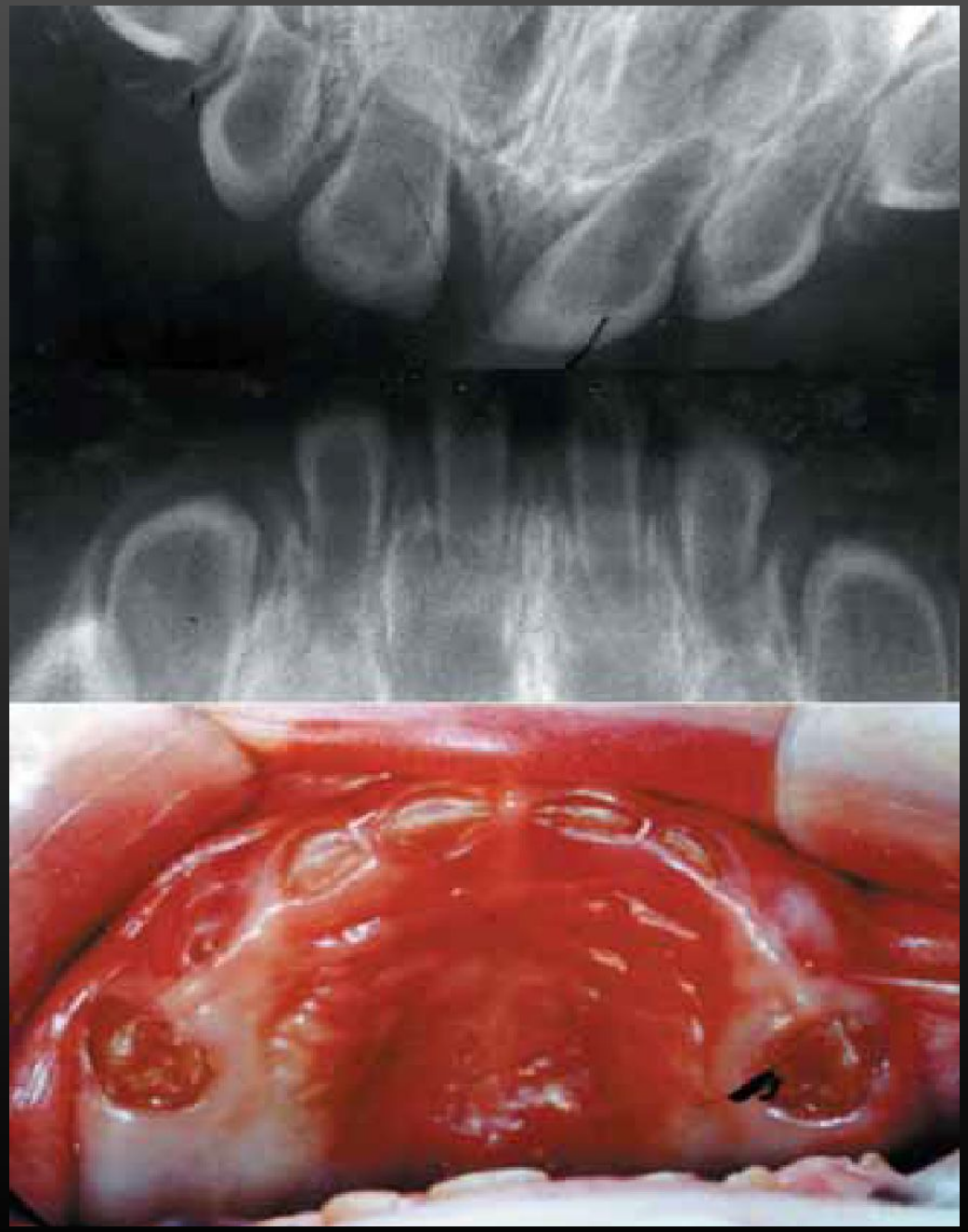
SHIELDS TYPE III, BRANDYWINE TYPE DENTINOGENESIS IMPERFECTA

- Some researchers say it is a separate mutation from DGI 1
- Shield et al 1973 stated that markedly enlarged pulp chambers & pulp exposures occurs in deciduous teeth do not occur in DGI 1
- Witkop 1975 suggested both are same
- Recent studies suggests both are result of mutation in two tightly linked genes
- MacDougall et al 1999 stated DGI 2 differ from DGI 1 by the presence of multiple pulp exposures, normal non mineralized pulp chambers & general appearance of shell teeth

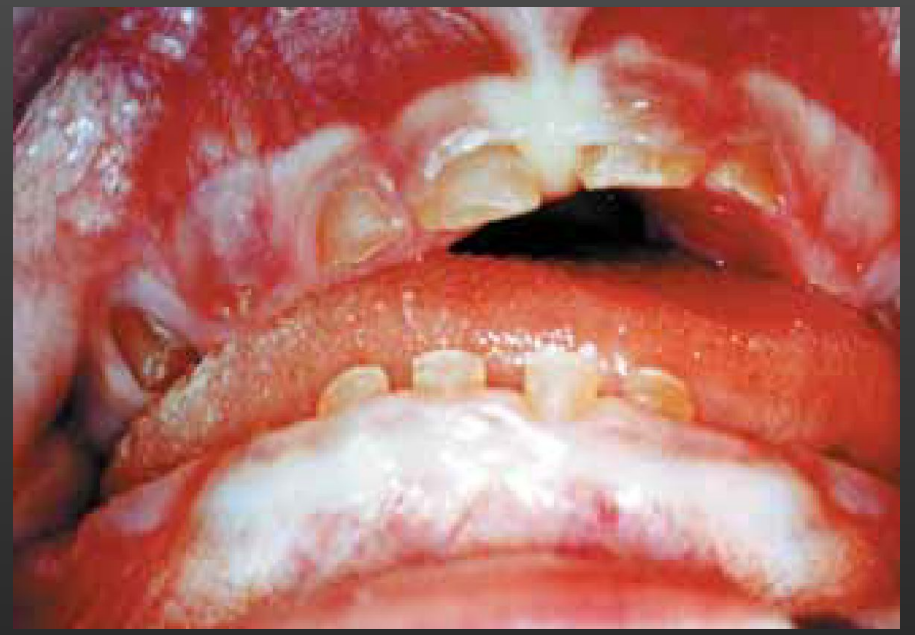
- In both dentitions tooth colour – may vary from normal to amber, grey or purple to bluish translucent discolouration
- Excessive attrition & tooth wear
- Abscess formation
- Tooth mobility
- Generally early loss of teeth
- The tooth enamel may have sheared off leaving dentine exposed; in such cases the exposed dentine often has a hard glassy appearance due to sclerosis. For this reason, patients rarely complain of sensitivity

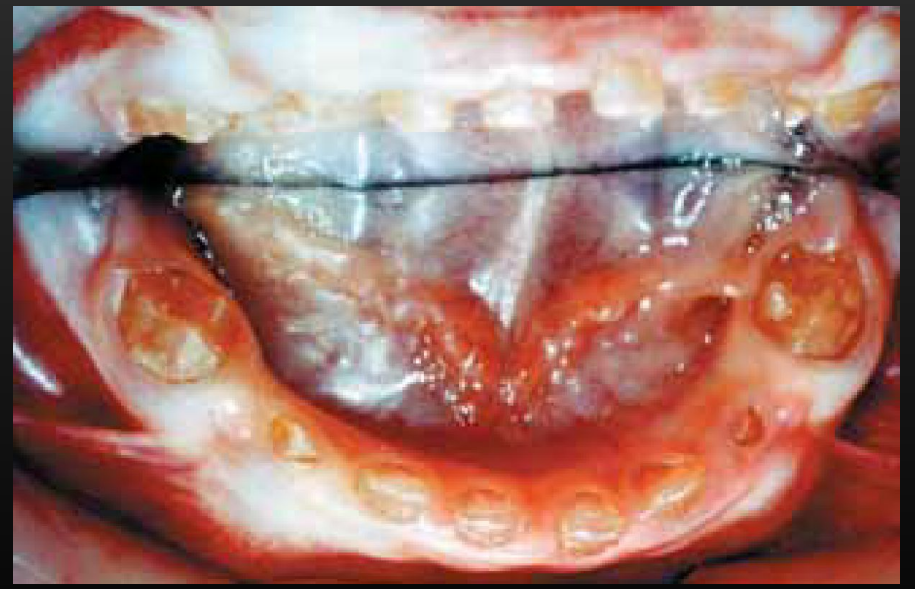
CLINICAL FEATURES

- Affects males & females equally
- Teeth are blue-gray or amber-brown & opalescent
- Few days after eruption teeth may achieve a normal color, following which they become translucent
- Finally become gray or brown with bluish reflection from enamel



- Enamel may split readily from dentin when subjected to occlusal stress
- Severe attrition of teeth
- Obliterated pulp chamber & root canals



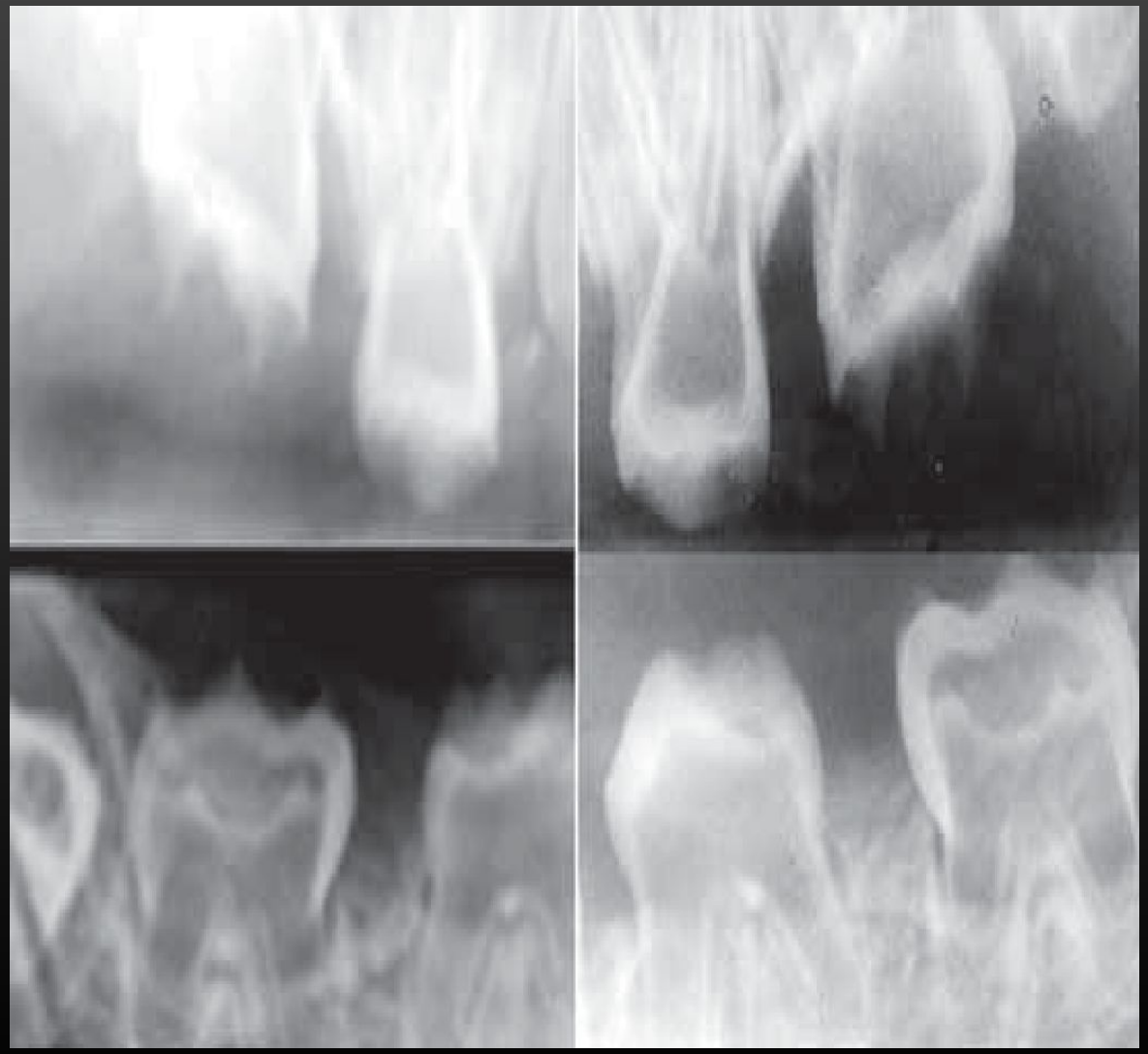


DIFFERENTIAL DIAGNOSIS OF DGI /OTHER CONDITIONS WITH SIMILAR FEATURES

- Exposure of underlying dentine-Hypocalcified forms of amelogenesis imperfecta
- Intrinsic discolouration
    - Congenital erythropoietic porphyria
    - Rhesus incompatibility
    - Tetracycline drug usage
- Mobility leading to early tooth loss - hypophosphatasia, immunological deficiencies e.g. severe congenital neutropenia (Kostmann's disease), cyclic neutropenia, Chediak-Hegashi syndrome, neutropenias, histiocytosis X, Papillon- Lefevre syndrome & leucocyte adhesion deficiency syndrome
- Vitamin D-dependent rickets & vitaminD-resistant rickets

RADIOGRAPHIC FEATURES

- Roots - thin & spiked
- Obliteration of coronal & radicular pulp chamber depending on age
- Cementum, alveolar bone & PDL appears normal
- Type 2 large pulp chambers with thin shell of dentin & enamel “shell teeth”



HISTOLOGIC FEATURES

- Mantle dentin (narrow zone of dentin below enamel) – normal
- Remaining dentin - severely dysplastic with vast areas of amorphous matrix with globular or interglobular foci of mineralization
- Reduced number of dentinal tubules/Large area of atubular dentin
- Tubules - distorted, irregular in shape, widely spaced ,larger in size
- Absence of odontoblastic processes & presence of degenerating cellular debris instead

PHYSICAL & CHEMICAL FEATURES

- Pulp chamber & root canal obliterated by abnormal dentin deposition
- DEJ - smooth or flattened instead of scalloped (responsible for early chipping of enamel)
- Increased water content (60 % than normal)
- Decreased mineral content
- Density, x-ray absorption & hardness are low
- Micro hardness near to cementum

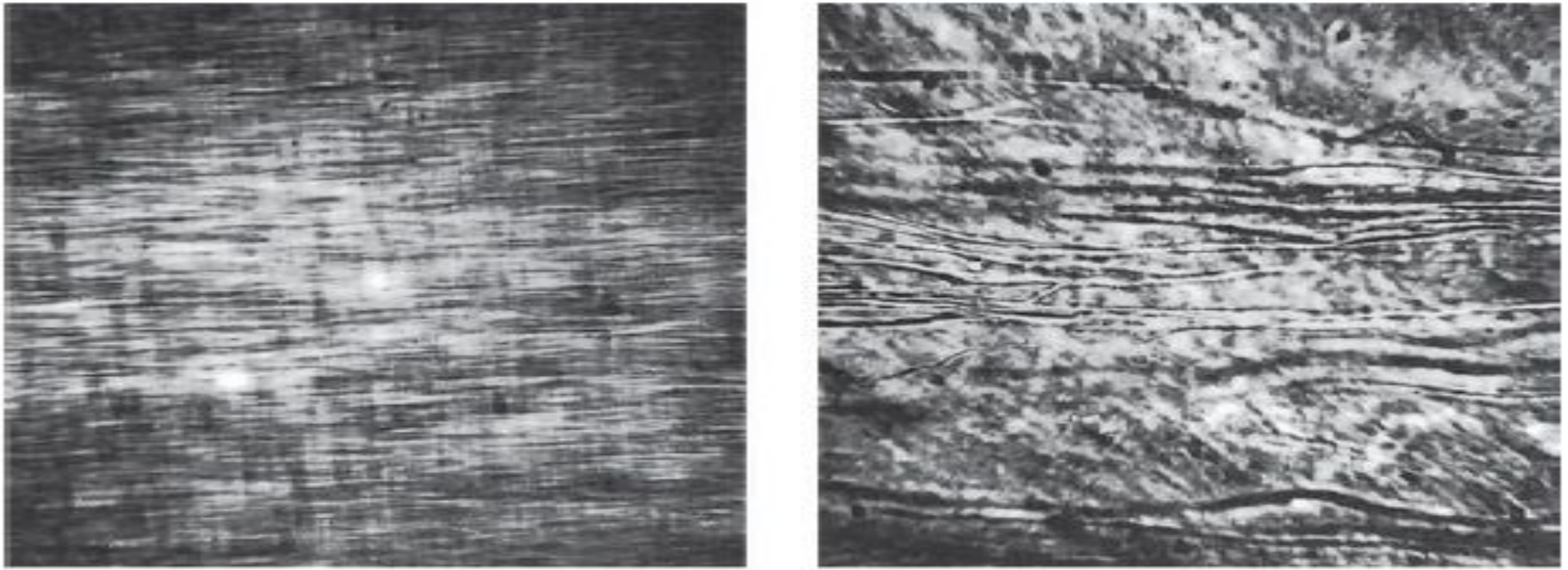


Figure 1-57
Dentinogenesis imperfecta.
(A) Normal dentin showing regular dentinal tubules. **(B)** large irregular dentinal tubules in dentinogenesis imperfecta. Both photomicrographs taken at same magnification.

AIMS OF TREATMENT/MANAGEMENT

- The aims of treatment are to remove sources of infection, restore function, aesthetics & protect posterior teeth from wear & maintain occlusal vertical dimension
- Treatment varies according to age of patient, severity of problem & presenting complaint

TREATMENT

- AIMED AT PREVENTING LOSS OF ENAMEL AND DENTIN THROUGH ATTRITION Mild –moderate cases (no enamel loss or rapid wear of teeth)

 Routine restorative techniques Eg: amalgam, composite

 Bonding of veneers for esthetics as they mask opalescence of anterior teeth

  Bleaching not used but can lighten the color

- Primary teeth:
  - Stainless steel in posteriors
  - Stainless steel with open face anterior teeth
  - Permanent composite for teeth
  - Porcelain fused metal crowns

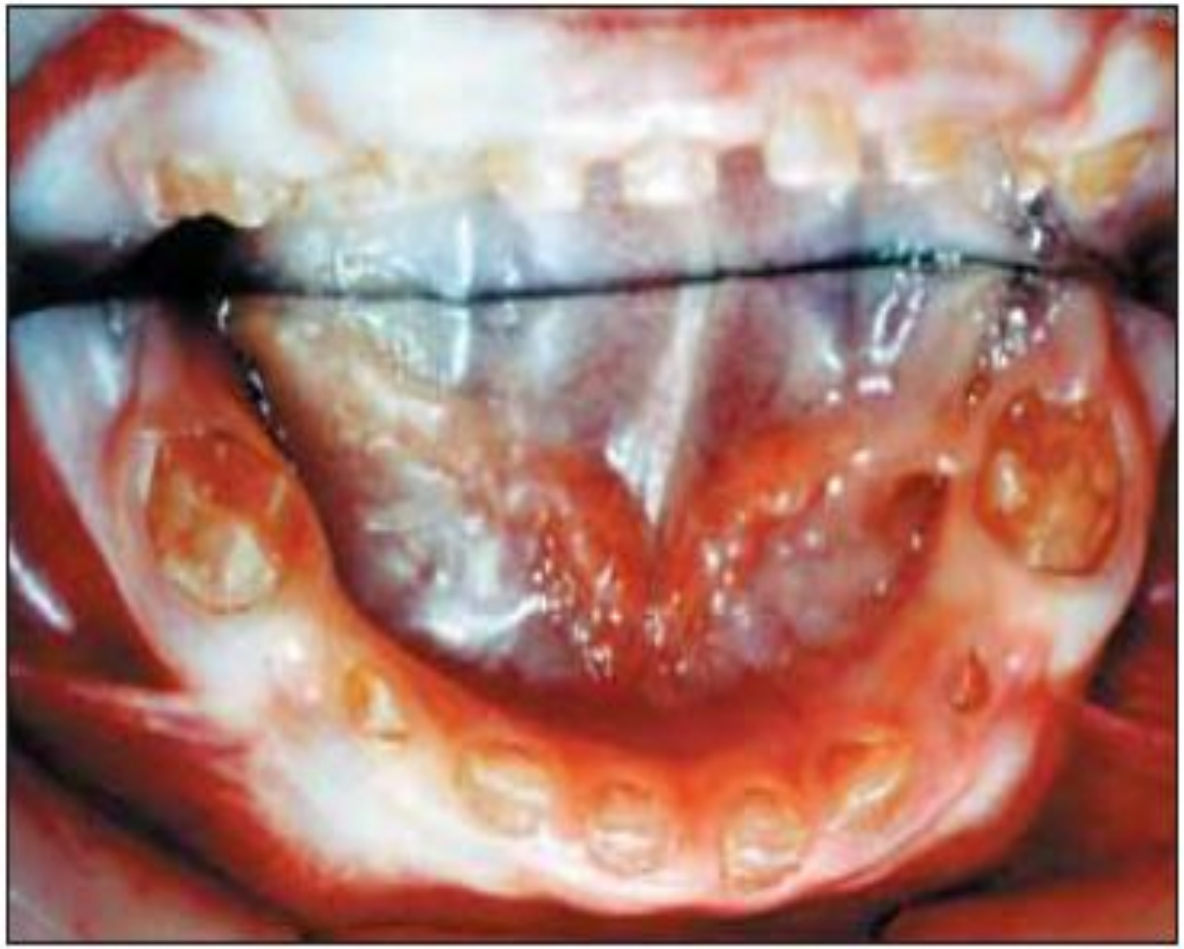


Fig 3. Preoperative clinical view at age 20 months, showing the brown-colored teeth, severe attrition, thin bucolingual dimensions of the incisors.

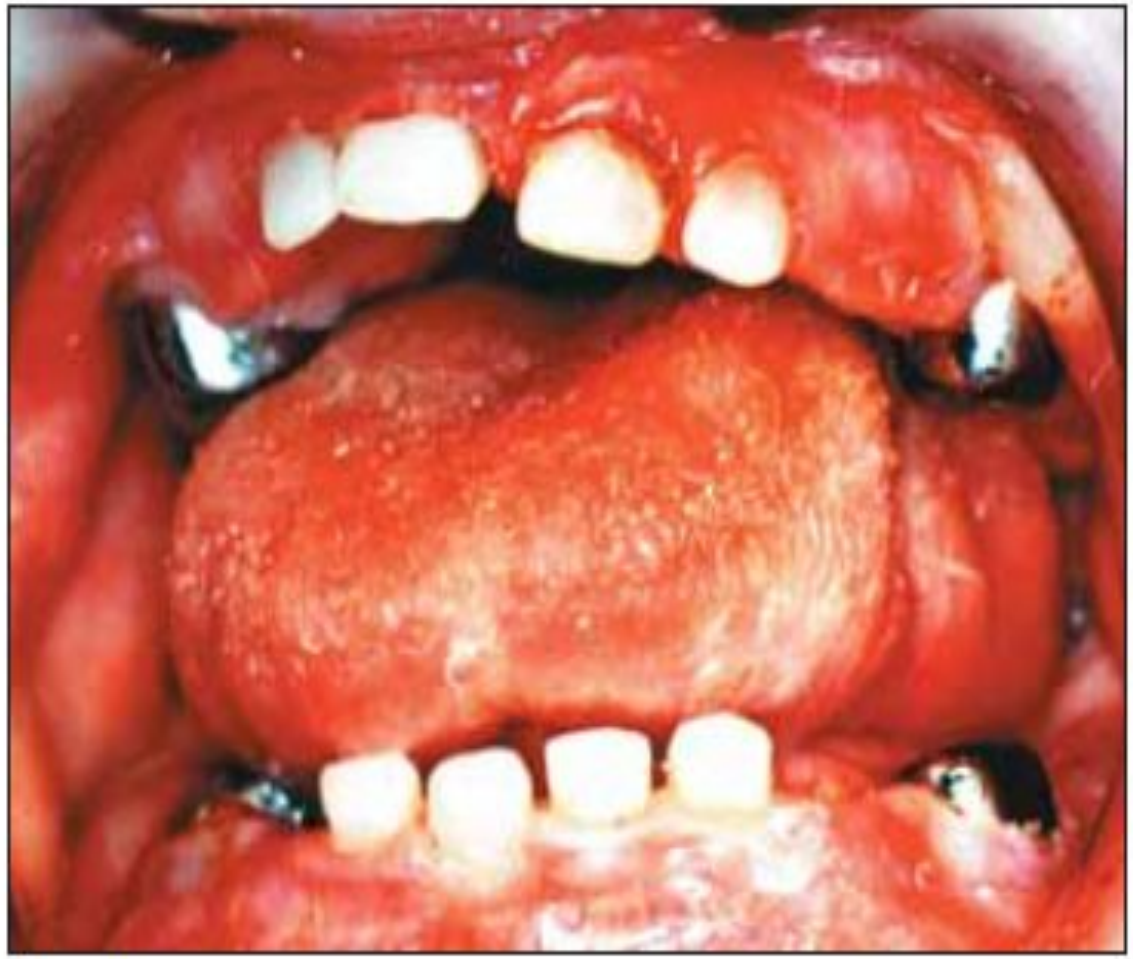


Fig 5. Postoperative clinical view at age 20 months, just after completing treatment under general anesthesia.

- Severe cases: (significant enamel loss and rapid wear)
- The emphasis should be on minimal tooth preparation until the child reaches adulthood. At this point, if clinically indicated, a **full mouth rehabilitation** may be considered with **full crowns & partial dentures/over dentures**
- Teeth with short thin roots & marked cervical constrictions however are often unfavourable for crowns . Obliteration of pulp chambers & root canals in teeth that develop abscesses makes endodontic therapy difficult if not impossible

DENTINE DYSPLASIA (DD)

- A rare disturbance of dentin formation characterized by normal enamel but atypical dentin formation with abnormal pulpal morphology
- Incidence of DD type I is 1 in 100,000 (US)
- Etiology: Dentin dysplasia, both type I and type II, appears to be a hereditary disease, transmitted as an autosomal dominant characteristics

CLASSIFICATION OF DENTINE DYSPLASIA (DD)

- Shields et al
  - Type I (dentin dysplasia)
  - Type II (anomalous dysplasia of dentin)
- Witkop
  - **Radicular dentin dysplasia (type I)** (More common) &
  - **Coronal dentin dysplasia (type II)**

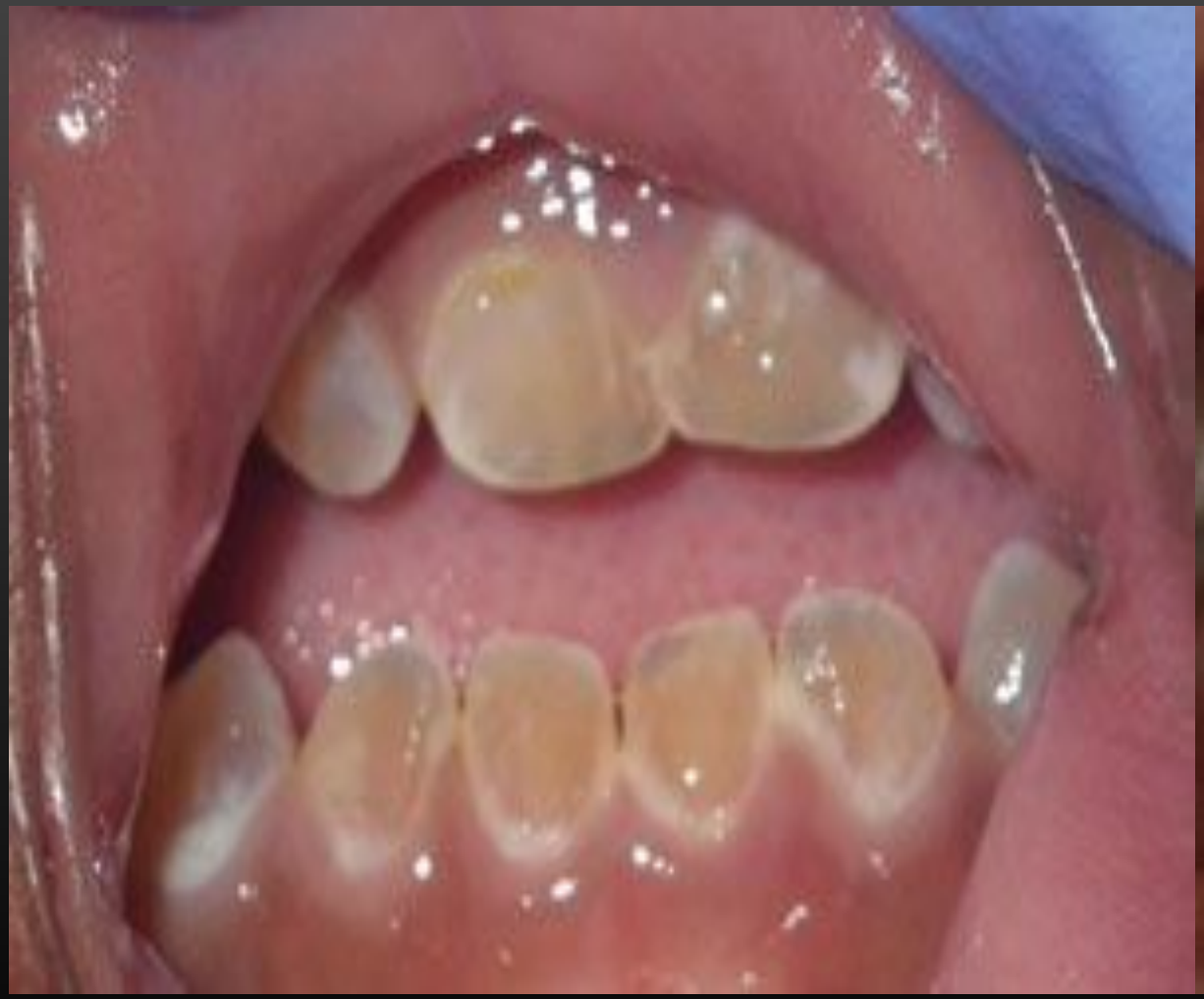
- First description of the disease - **Ballschmiede** (1920) ‘rootless teeth’
- The first concise description of disease was published in 1939 by **Rushton**, who was also first to designate it as ‘dentin dysplasia.’

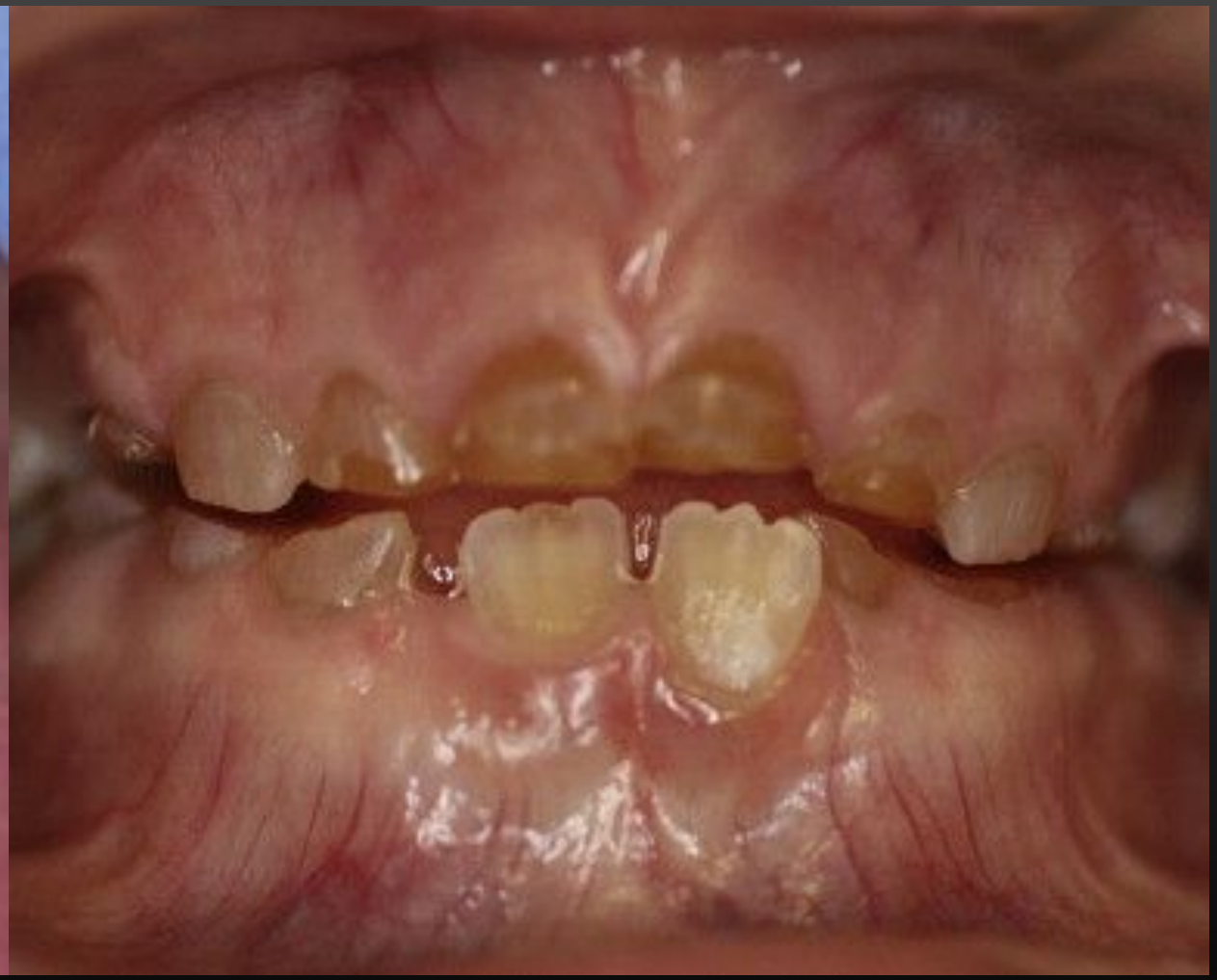
DD Type I

- Both dentitions affected
- Teeth appear clinically normal but occasional slight amber translucency
- Normal/Delayed Eruption pattern of teeth
- Extreme mobility & premature exfoliation, even after minor trauma (Short roots of teeth)

DD Type II

- Both dentitions affected
- Deciduous teeth appear yellow, brown, or bluish-gray opalescent appearance as seen in dentinogenesis imperfecta
- Permanent dentition clinically normal



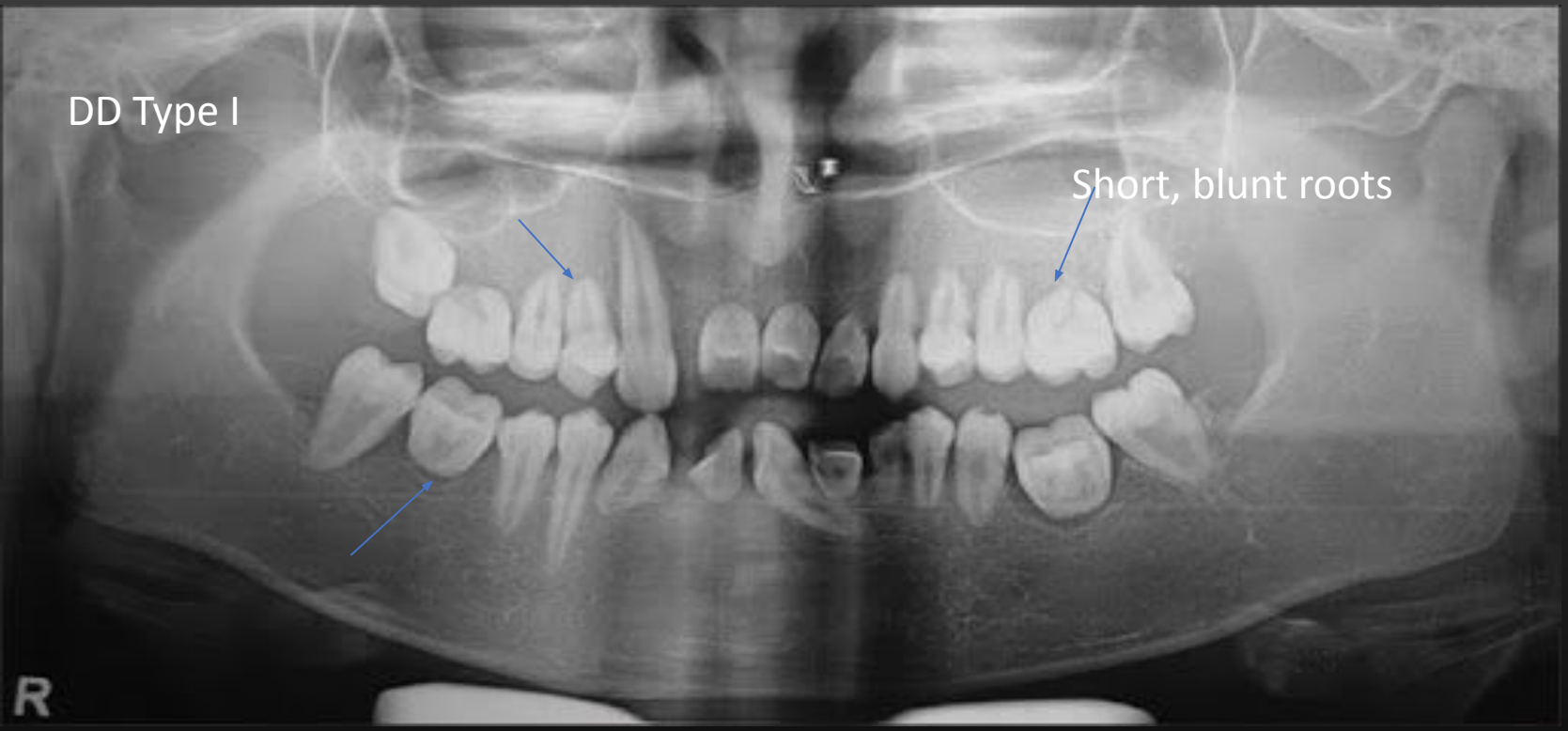


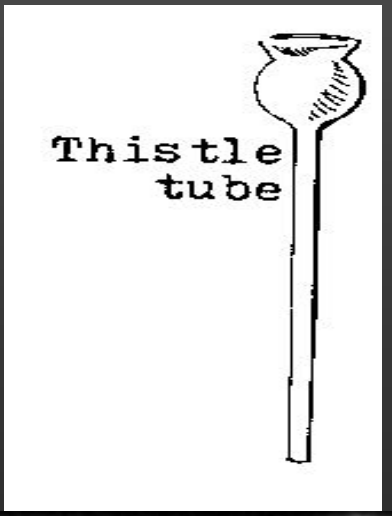
DD Type I (Radicular)

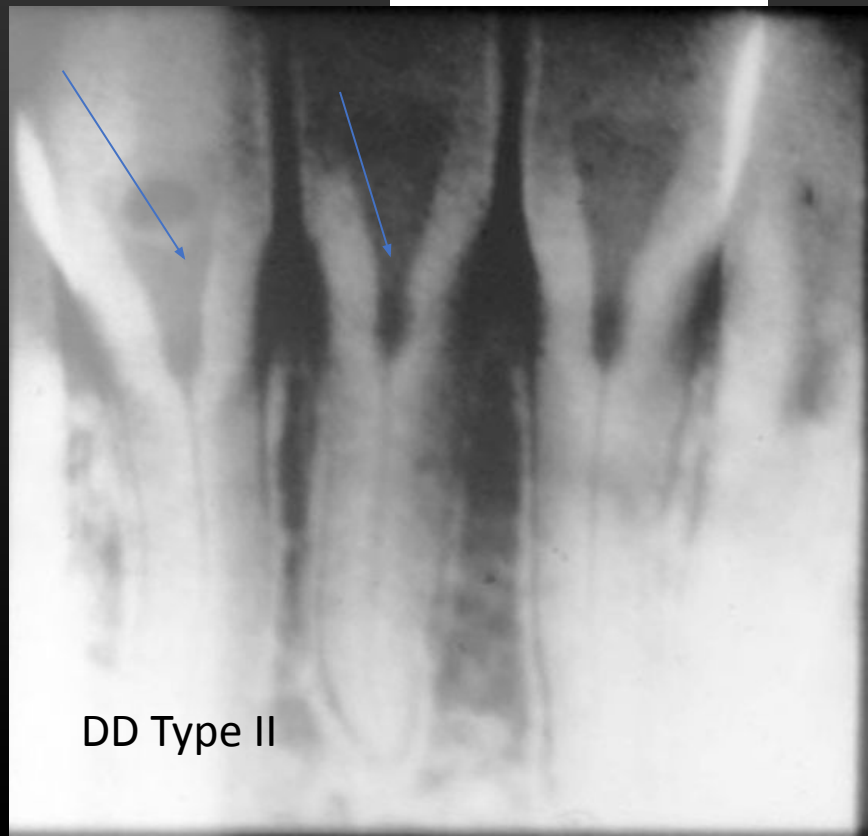
- Short, blunt, conical, or similarly malformed roots of teeth
- Deciduous teeth: pulp chambers, root canals completely obliterated
- Permanent dentition - crescent-shaped pulpal remnant may still be seen in pulp chamber, pre-eruptively
- Periapical radiolucencies (granulomas, cysts or abscesses) involving apparently otherwise intact teeth

DD Type II (Coronal)

- Pulp chambers of deciduous teeth become obliterated
- Permanent teeth - abnormally large pulp chamber in coronal portion of tooth, ‘thistle-tube’ in shape
- Radiopaque foci resembling pulp stones
- Periapical radiolucencies do not occur unless for an obvious reason


DD Type I
Short, blunt roots
R


Thistle
tube


DD Type II

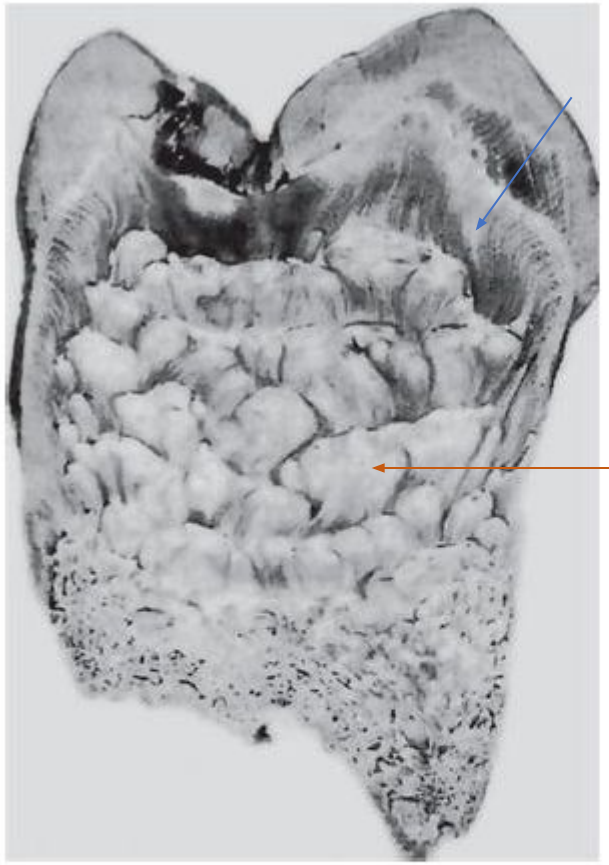
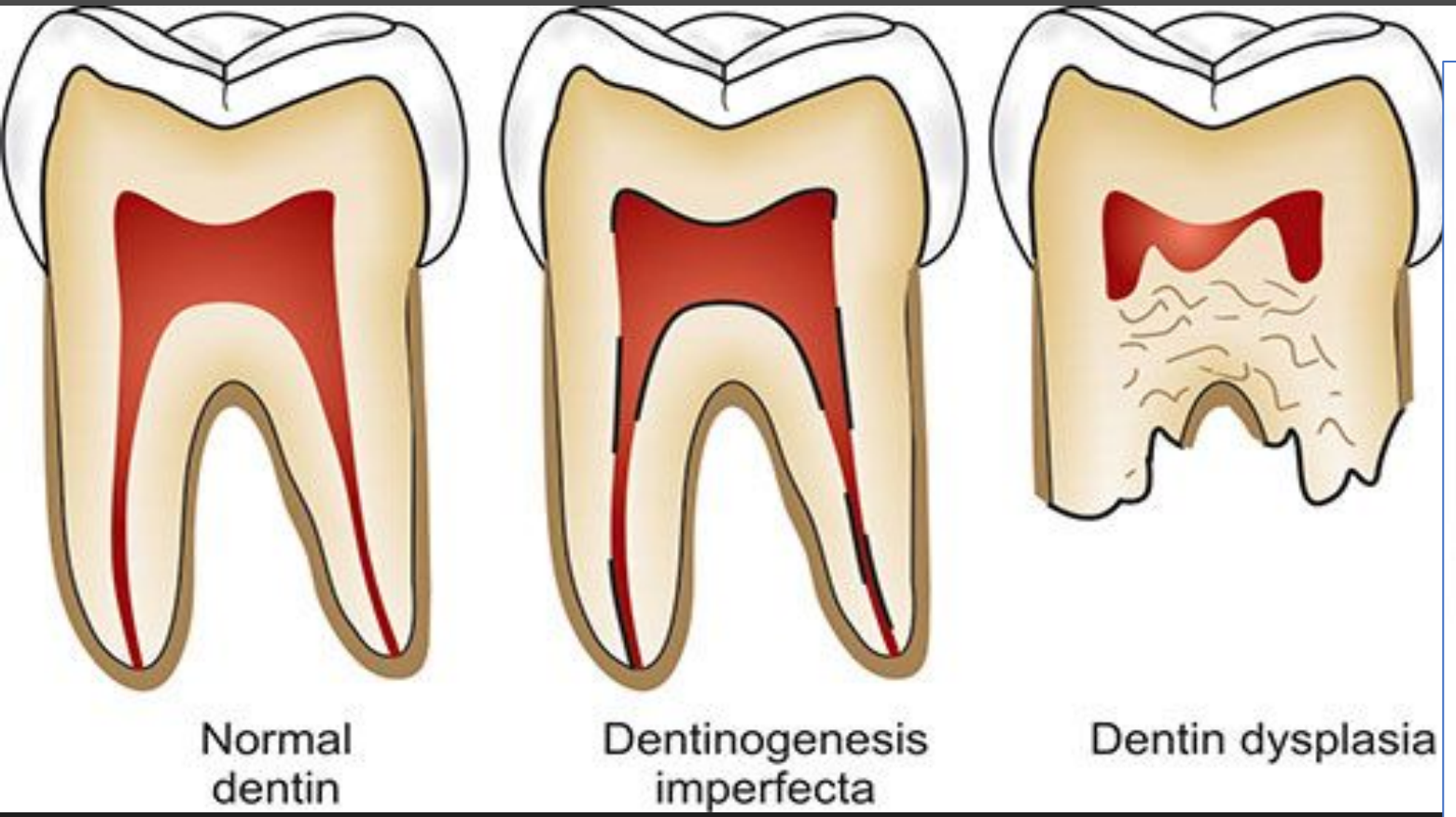


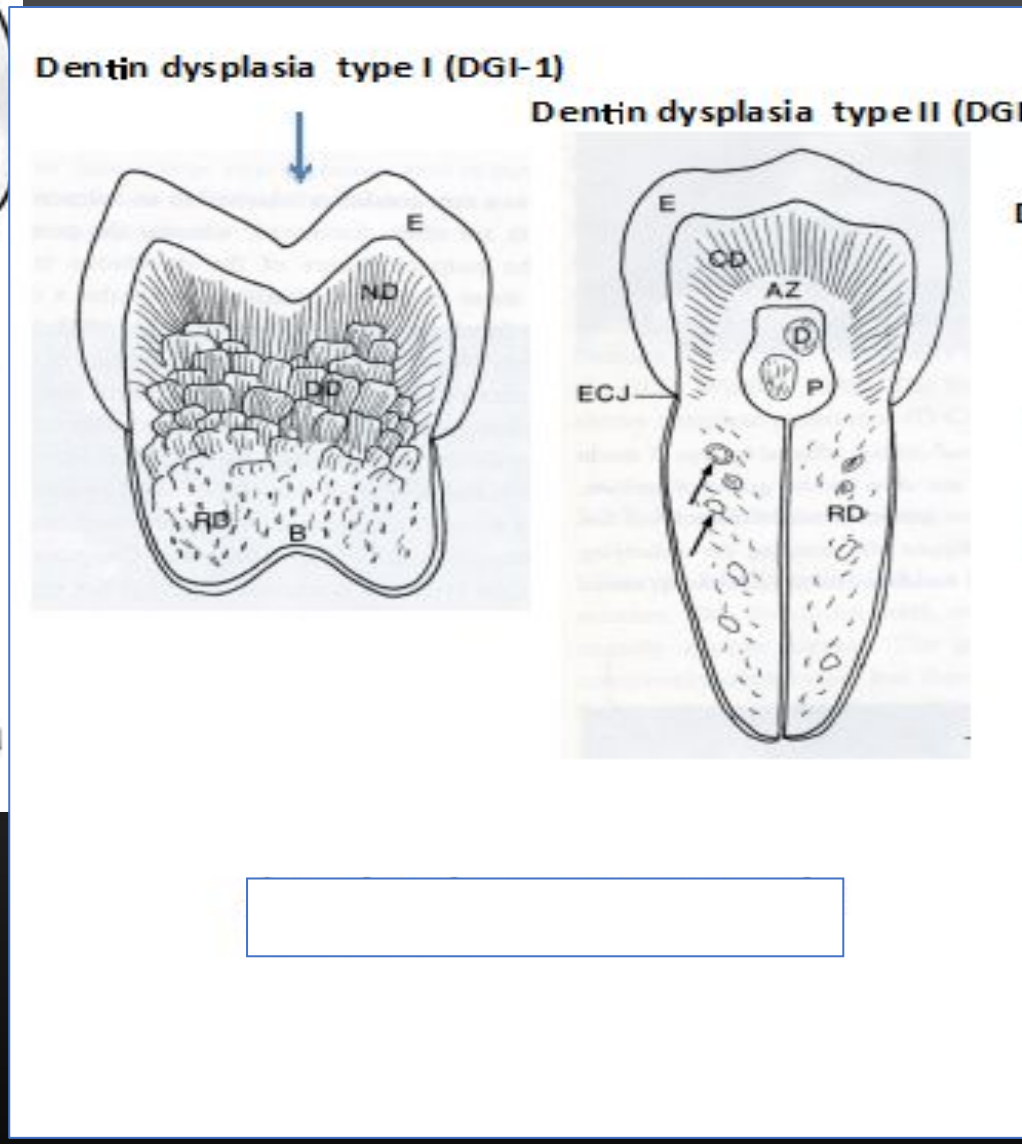
Figure 1-59
Dentin dysplasia, type I (radicular).
The atypicality of root dentin and dentin filling the pulp chamber is seen in this ground section of tooth *(Courtesy of Dr Richard K Wesley and George Wysocki. From Oral Surg,*

- Dentin itself is histologically normal but is simply disoriented
- Portion of coronal dentin is usually normal
- Apical to this, calcified tubular dentin, osteodentin & fused denticles
- Characteristic appearance described as ***'lava flowing around boulders'***
- Electron microscopic studies 'cascades of dentin' (sauk & his coworkers)

DD TYPE II HISTOLOGICAL FEATURES

- Deciduous teeth exhibit amorphous & atubular dentin in radicular portion, while coronal dentin is relatively normal
- Permanent teeth - normal coronal dentin, but pulp has multiple pulp stones or denticles


Normal dentin
Dentinogenesis imperfecta
Dentin dysplasia


Dentin dysplasia type I (DGI-1)
Dentin dysplasia type II (DGI
E
ND
DP
RD
B
E
CD
AZ
D
P
ECJ
RD

TREATMENT & PROGNOSIS of Dentine Dysplasia (DD)

- Management of increased mobility of teeth, it's exfoliation
- Periapical lesions – Extraction of teeth
- Rehabilitation - Dentures

REGIONAL ODONTODYSPLASIA - synonyms

- Odontodysplasia
- Odontogenic dysplasia
- Odontogenesis imperfecta
- Ghost teeth

- An unusual dental anomaly in which one or several teeth in a localized area are affected in an unusual manner
- Frequently involved teeth:
    - Maxillary anteriors (I, C)
    - Mandibular anteriors (I, C)
    - Both deciduous & permanent teeth

ETIOLOGY OF GHOST TEETH

- Unknown
- Possibility of somatic mutation
- Latent virus in Odontogenic epithelium getting activated during development?
- Local vascular defects (Walton & Co)

CLINICAL FEATURES

- Delay or a total failure in eruption
- Very irregular in shape/appearance
- Evidence of defective mineralization.

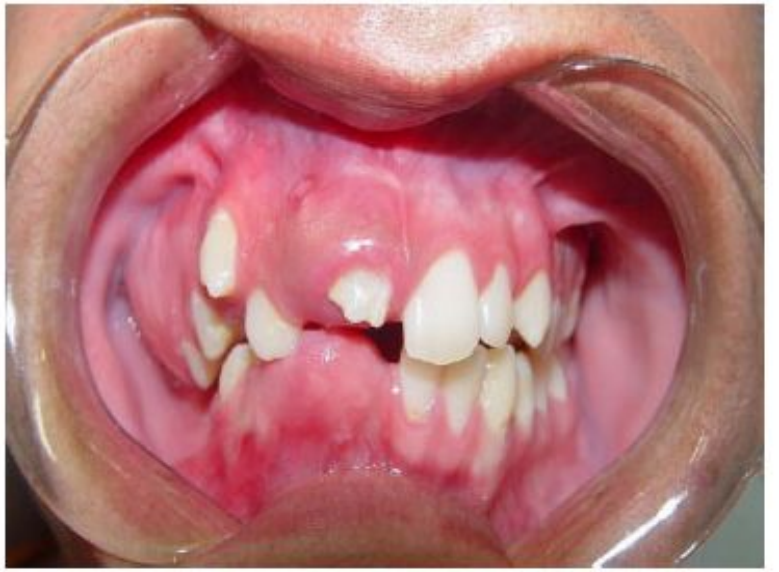


Fig. 1. Intraoral view of the patient with regional odontodysplasia.

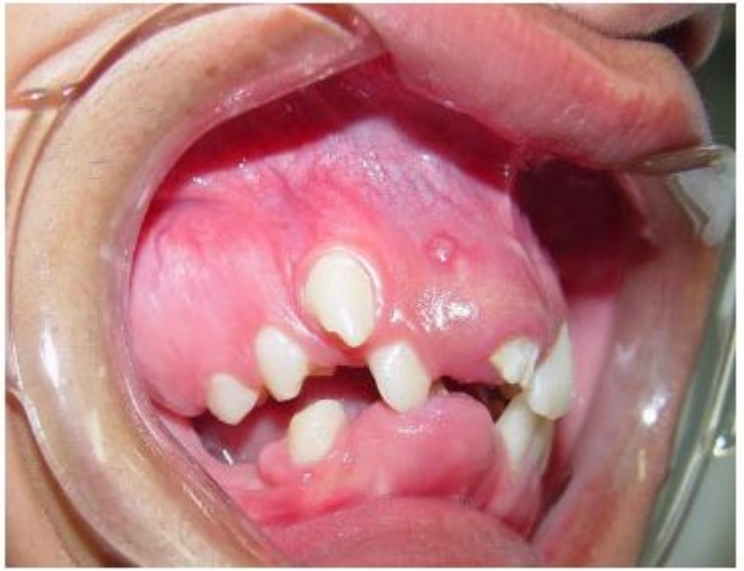


Fig. 2. Intraoral view of the right quadrant of the patient with regional odontodysplasia.

RADIOGRAPHIC FEATURES

- Radiographs are uniquely characteristic
  - The enamel layer often is not evident
  - Both enamel & dentin appear very thin
  - Pulp chamber is exceedingly large
  - Marked reduction in radiodensity so that teeth assume a **'ghost'** appearance

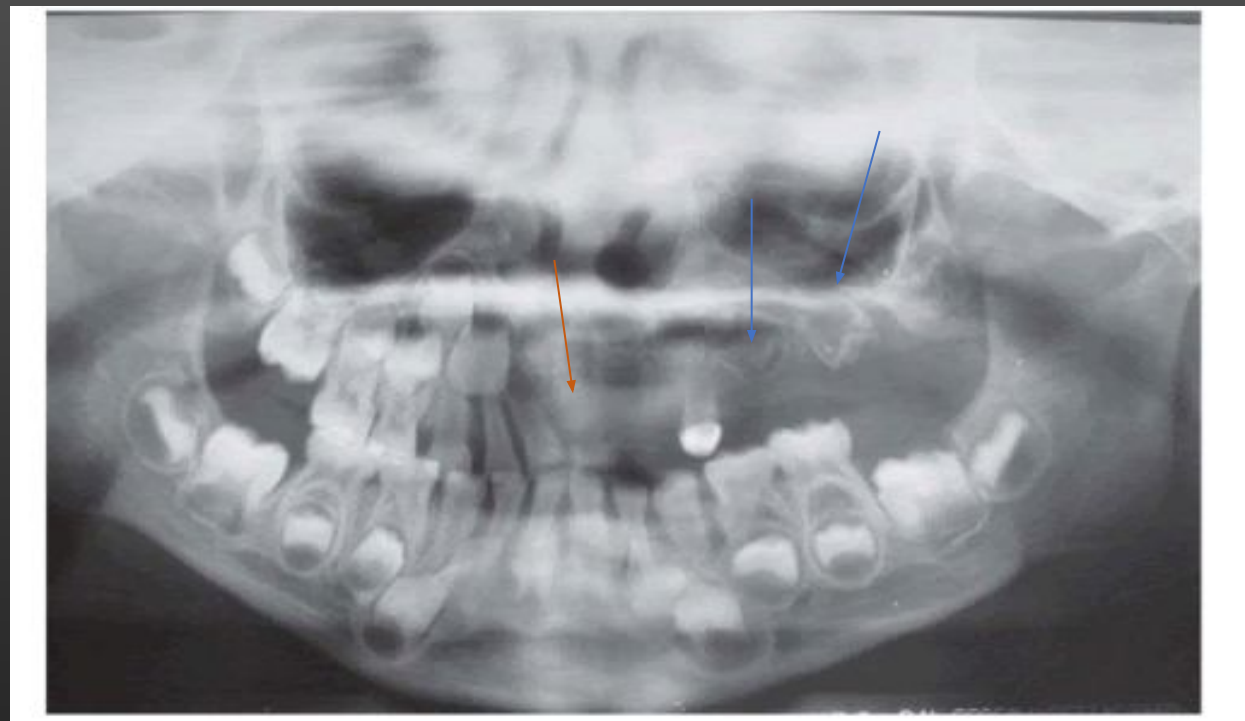
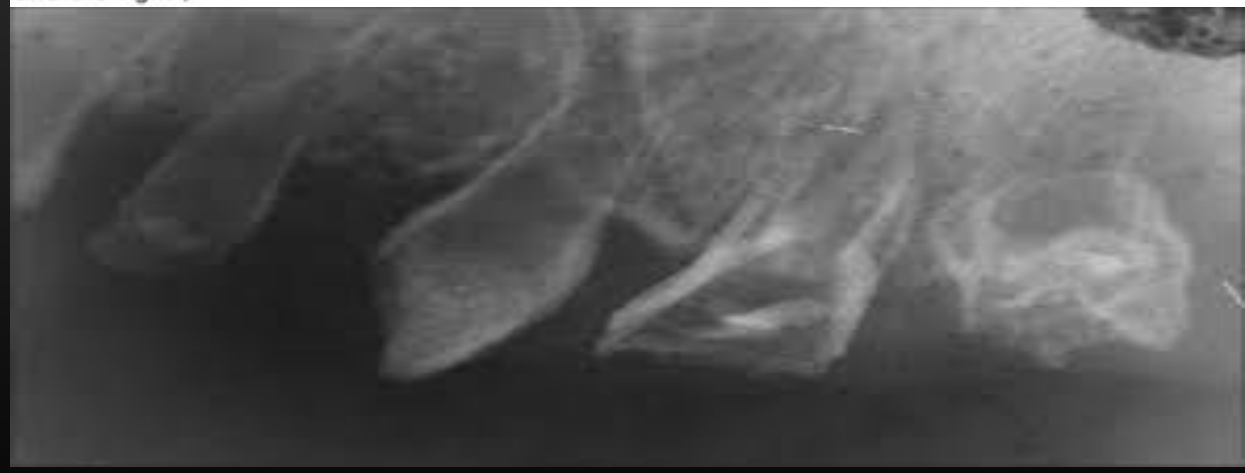
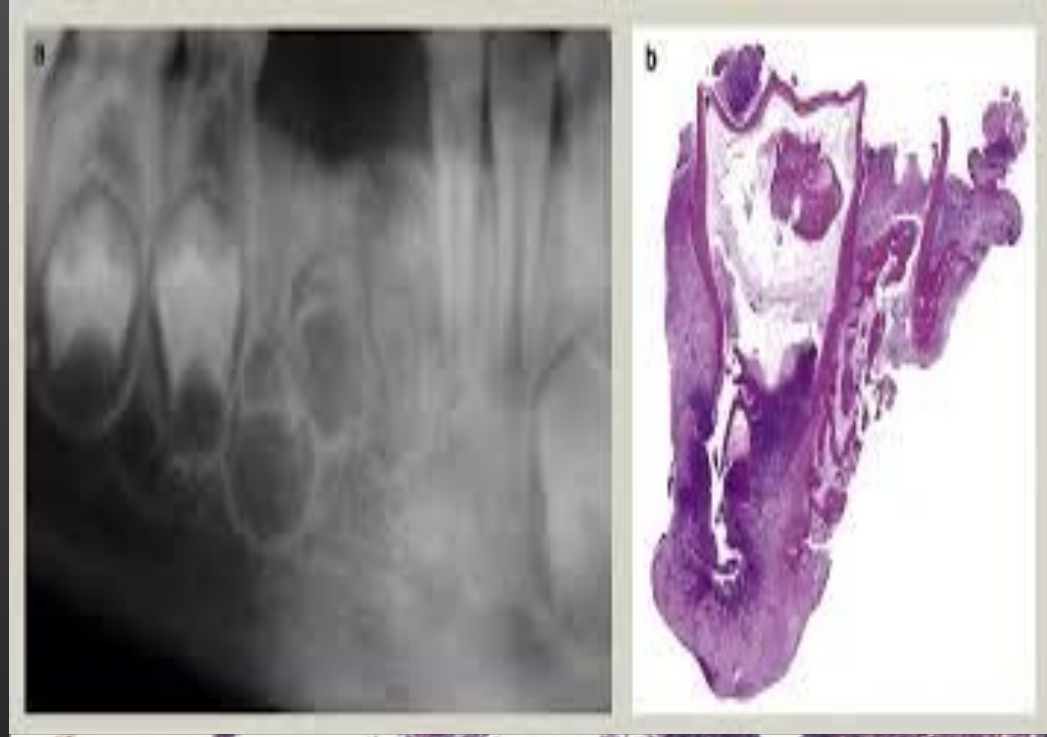


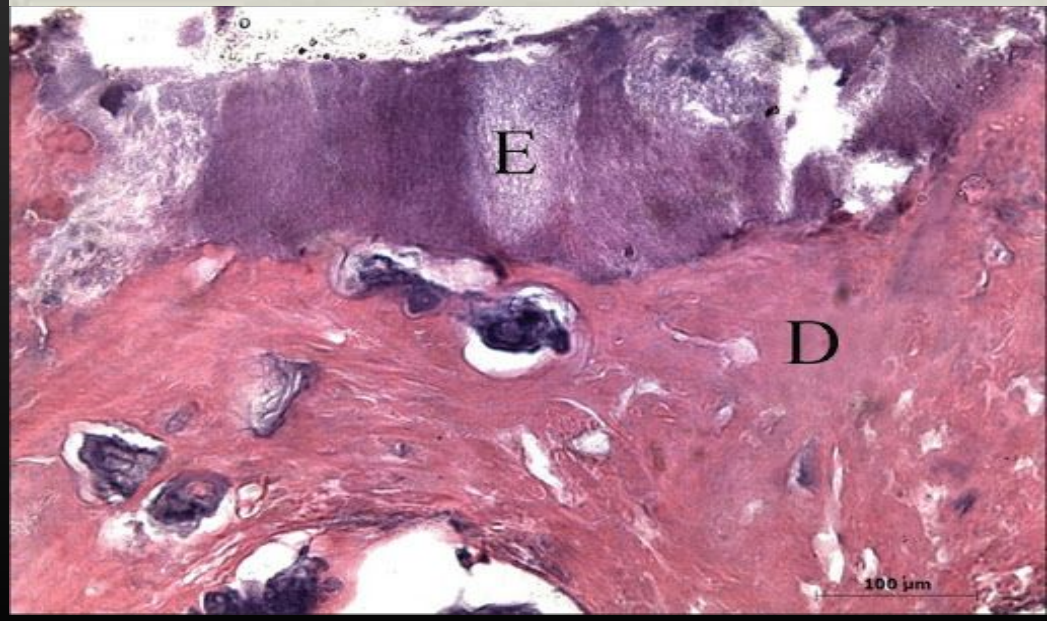
FIGURE 7- Panoramic radiograph showing the teeth with "ghostlike" appearance in maxillary left quadrant and the right permanent central incisor



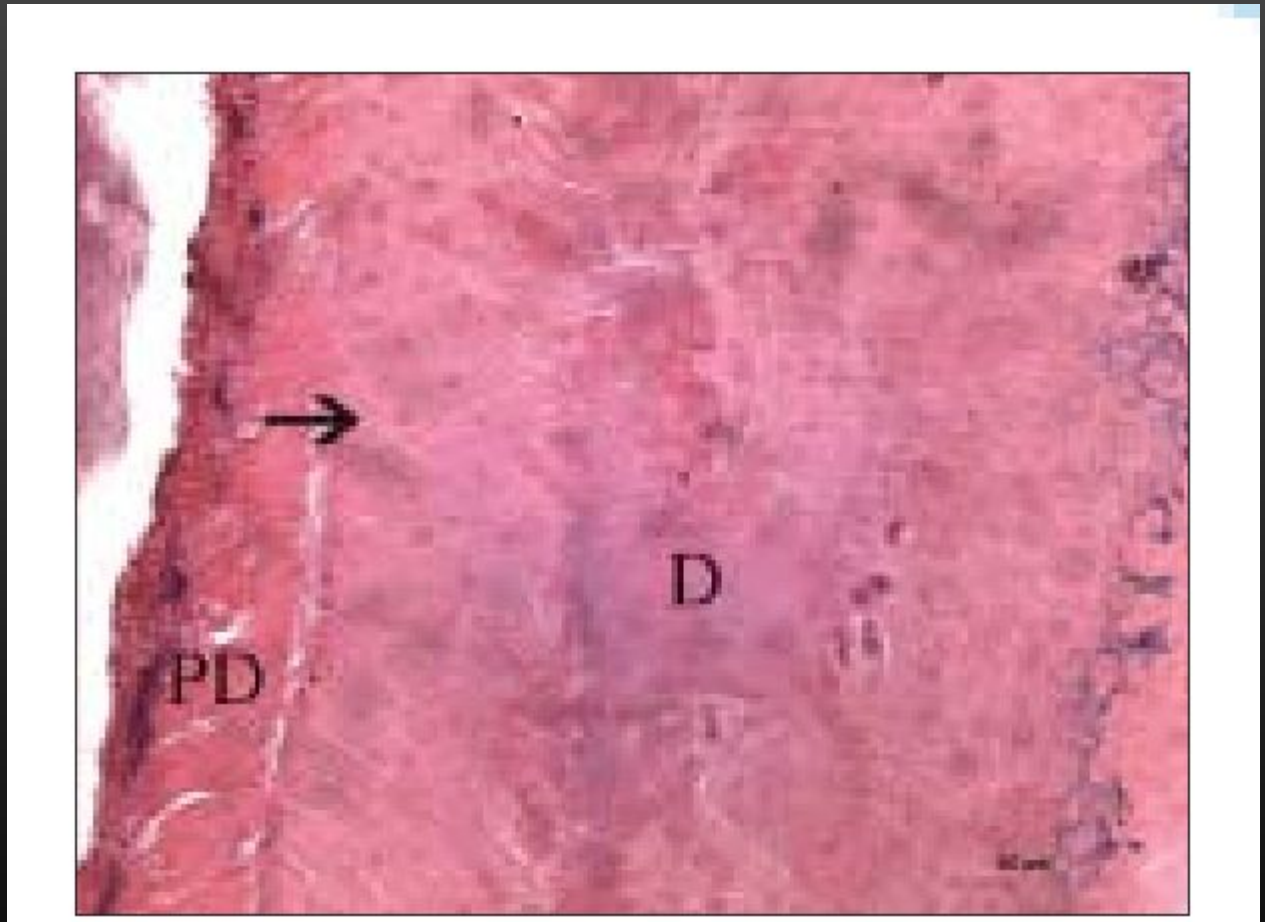
Histopathology

- Marked reduction in amount of dentin
- Widening of predentin layer
- Presence of large areas of interglobular dentin
- Irregular tubular pattern of dentin
- Reduced enamel epithelium around nonerupted teeth shows many irregular calcified bodies





WIDENED PREDENTINE



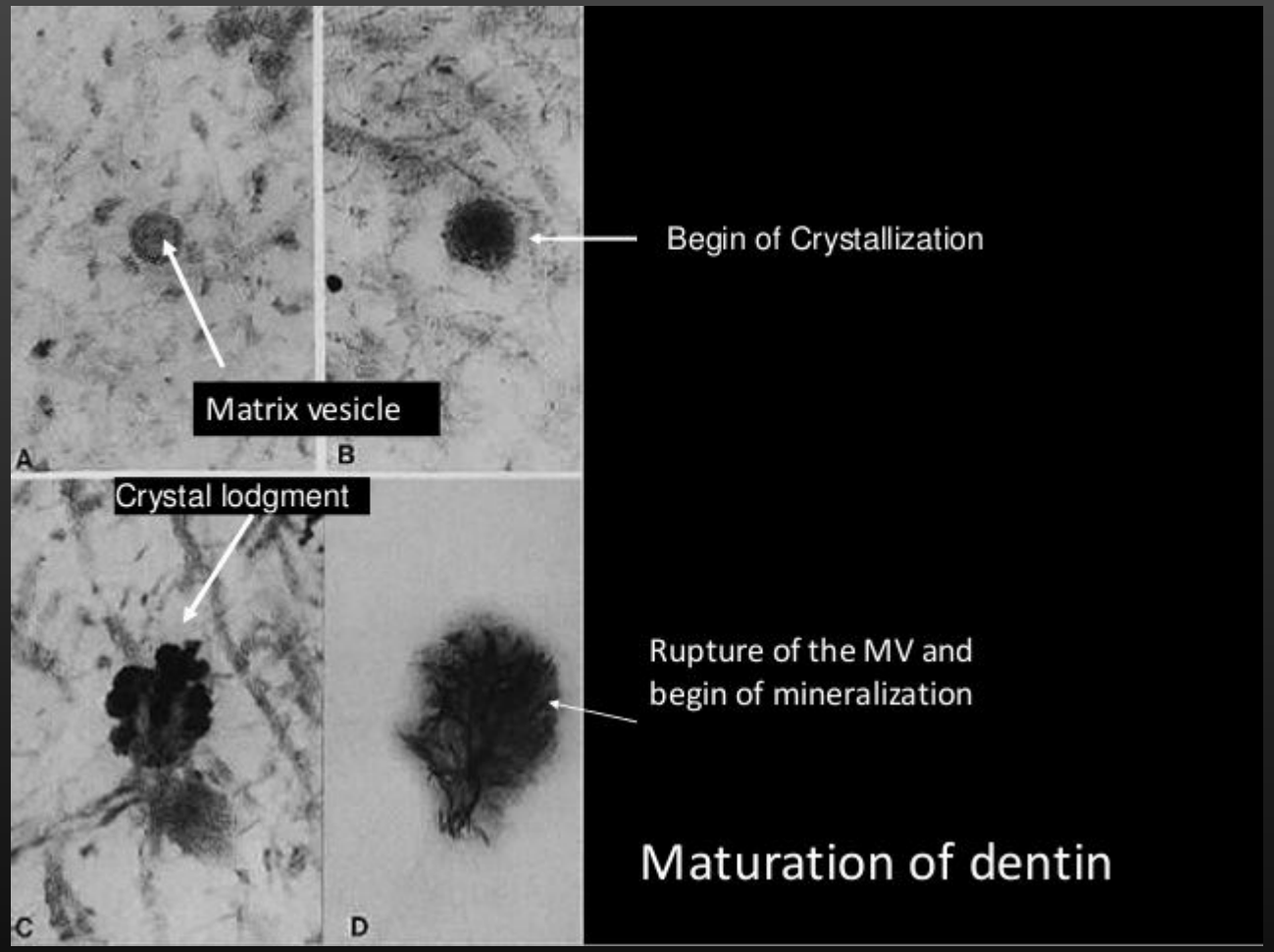
gure 5: Portion of radicular dentin in which a thick band

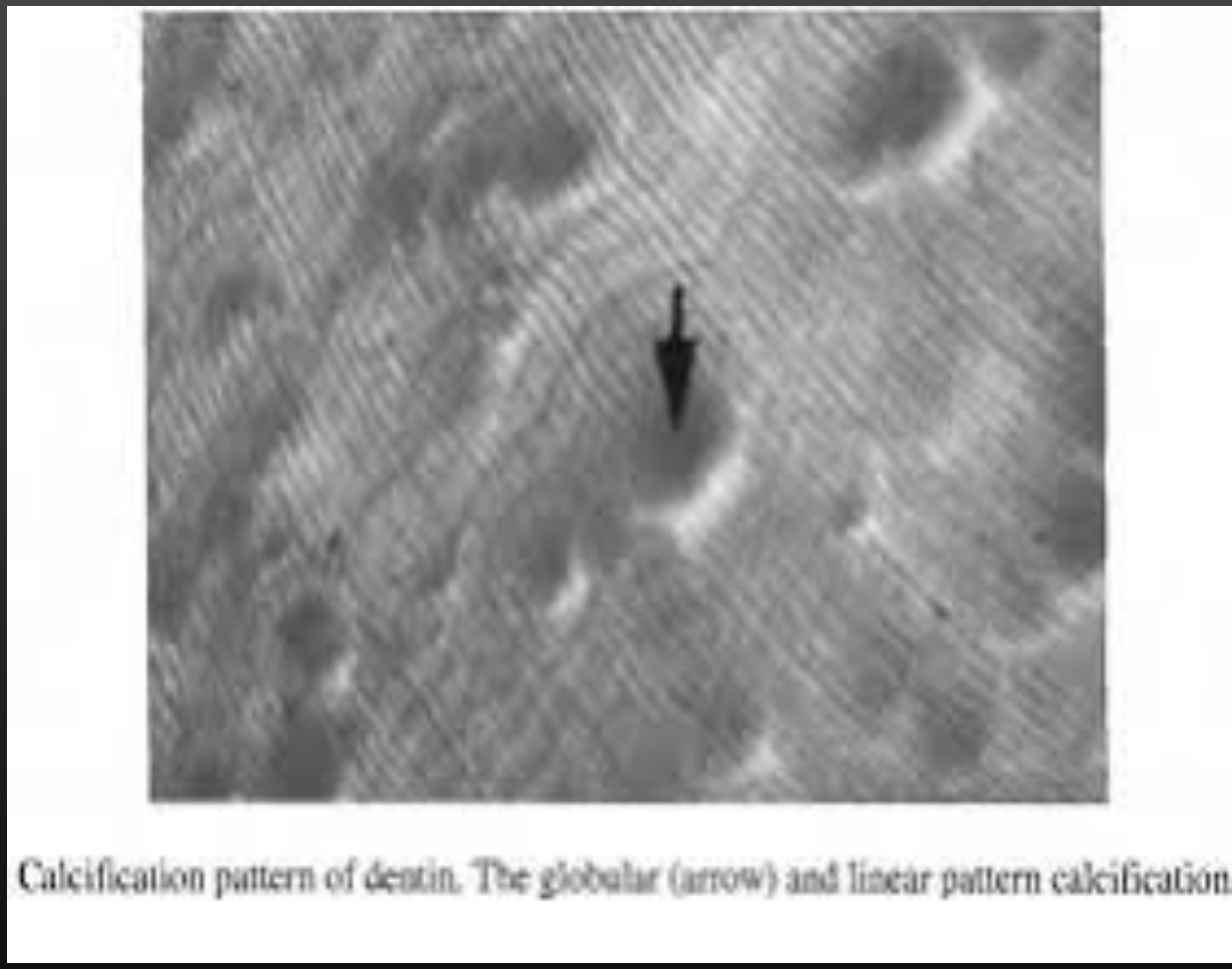
TREATMENT

- Cosmetic reasons - extraction with restoration by a prosthetic appliance

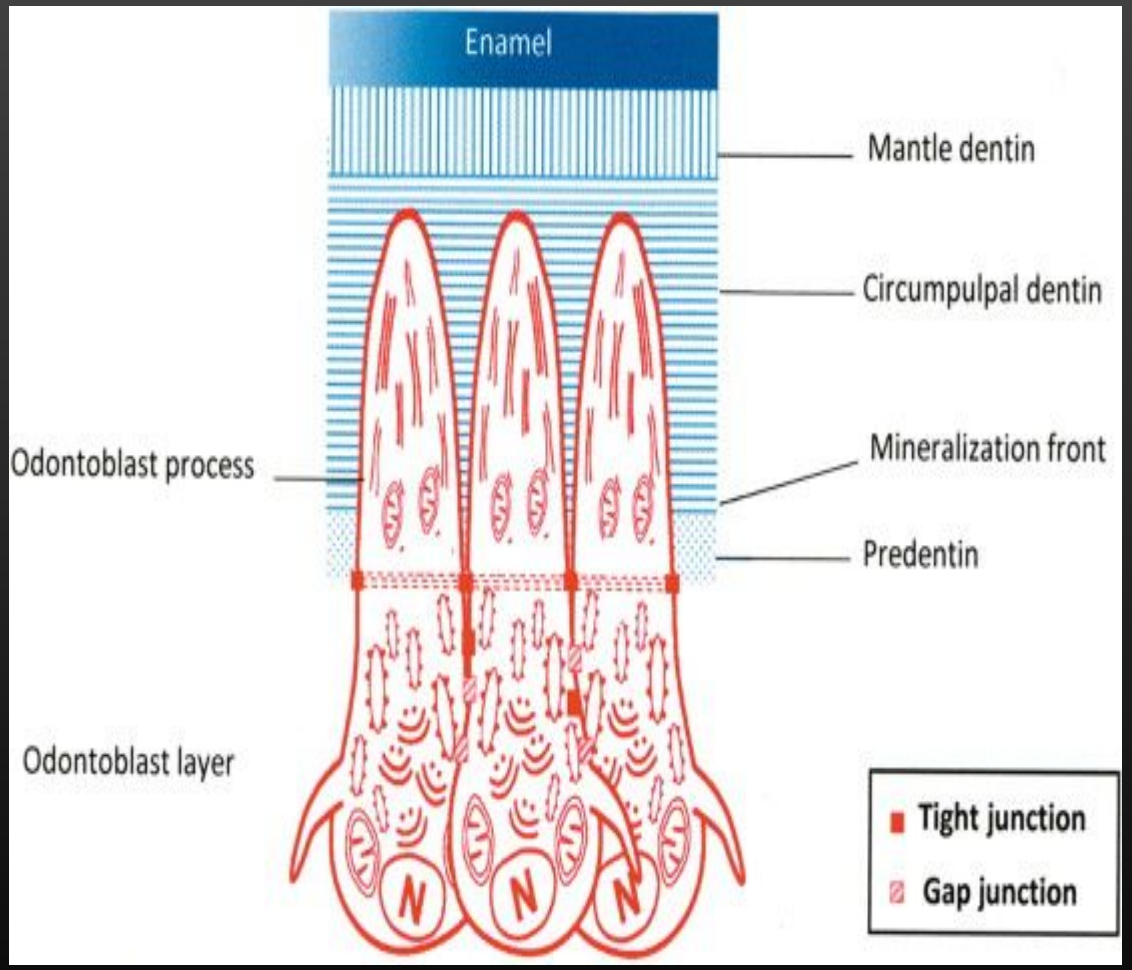
DENTIN HYPOCALCIFICATION - causes

- Similar to those of environmental enamel hypocalcification & enamel hypoplasia
- Any factor which interferes with normal calcification, such as parathyroid deficiency or rickets, could produce hypocalcification
- Hypocalcified dentin - softer





Calcification pattern of dentin. The globular (arrow) and linear pattern calcification

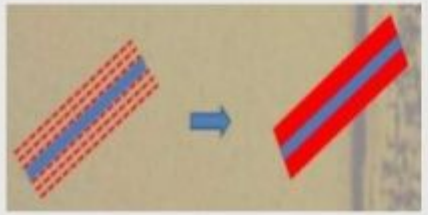


PATTERN OF MINERALIZATION

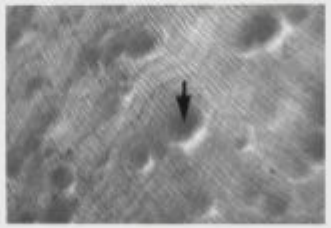
LINEAR

- SLOW RATE
- UNIFORM

(CIRCUMPULPAL DENTIN)



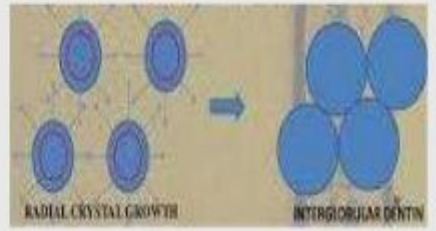
MIXED



GLOBULAR

- RAPID
- NON UNIFORM

MANTLE DENTIN



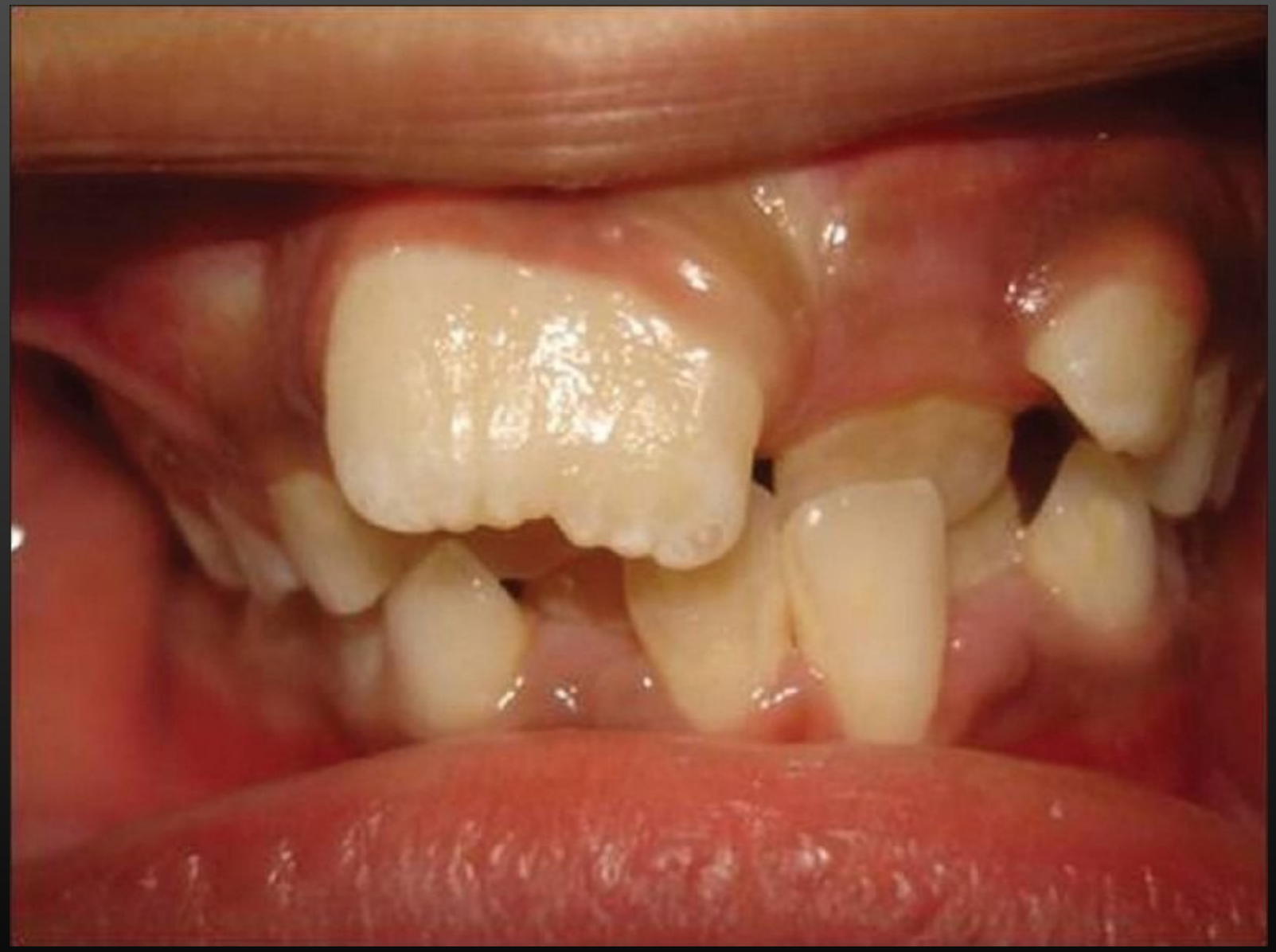
Thank you

SUMMARY

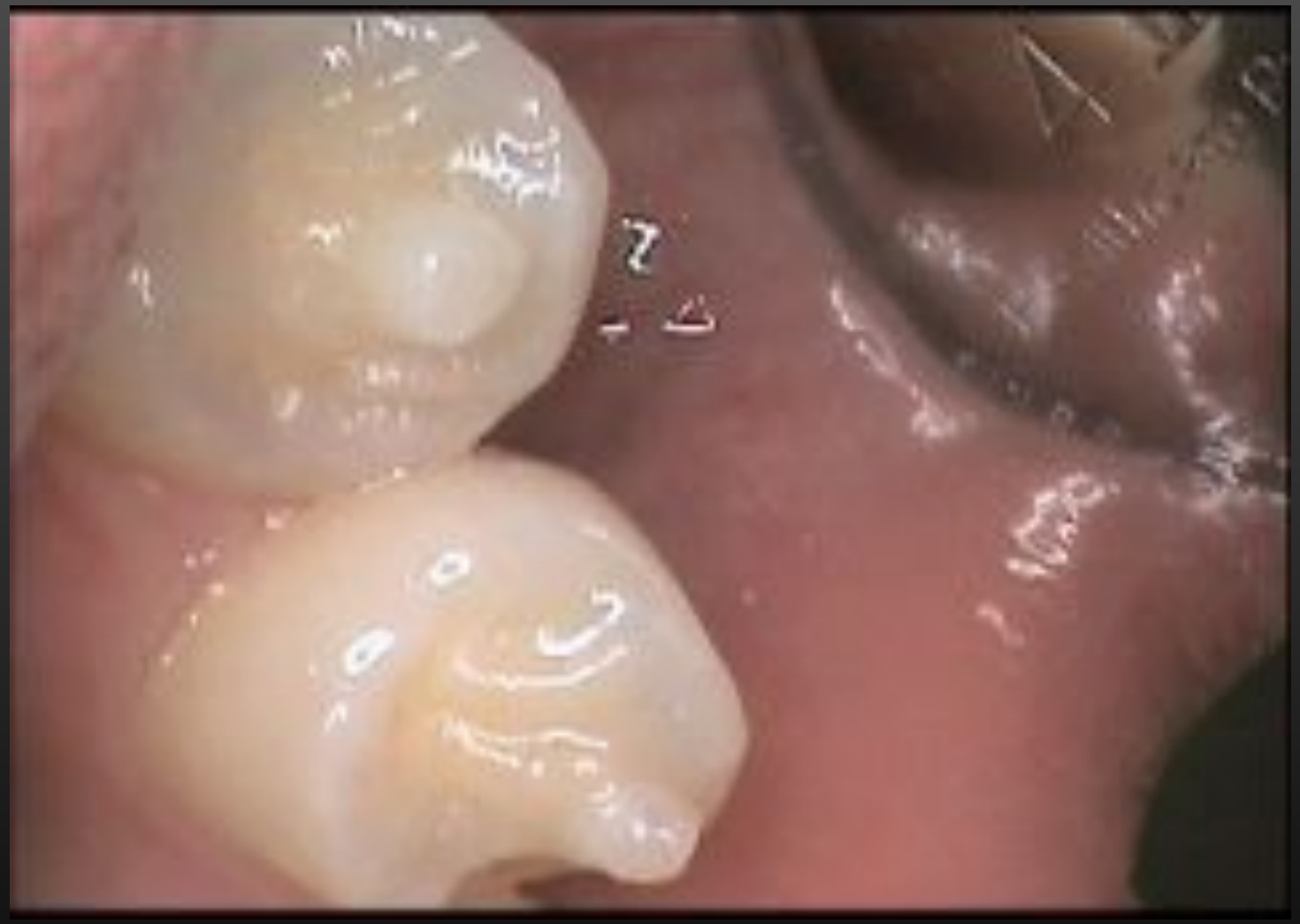
DEVELOPMENTAL DISTURBANCES OF TEETH in

1. Size
2. Shape
3. Number
4. Structure
5. Eruption

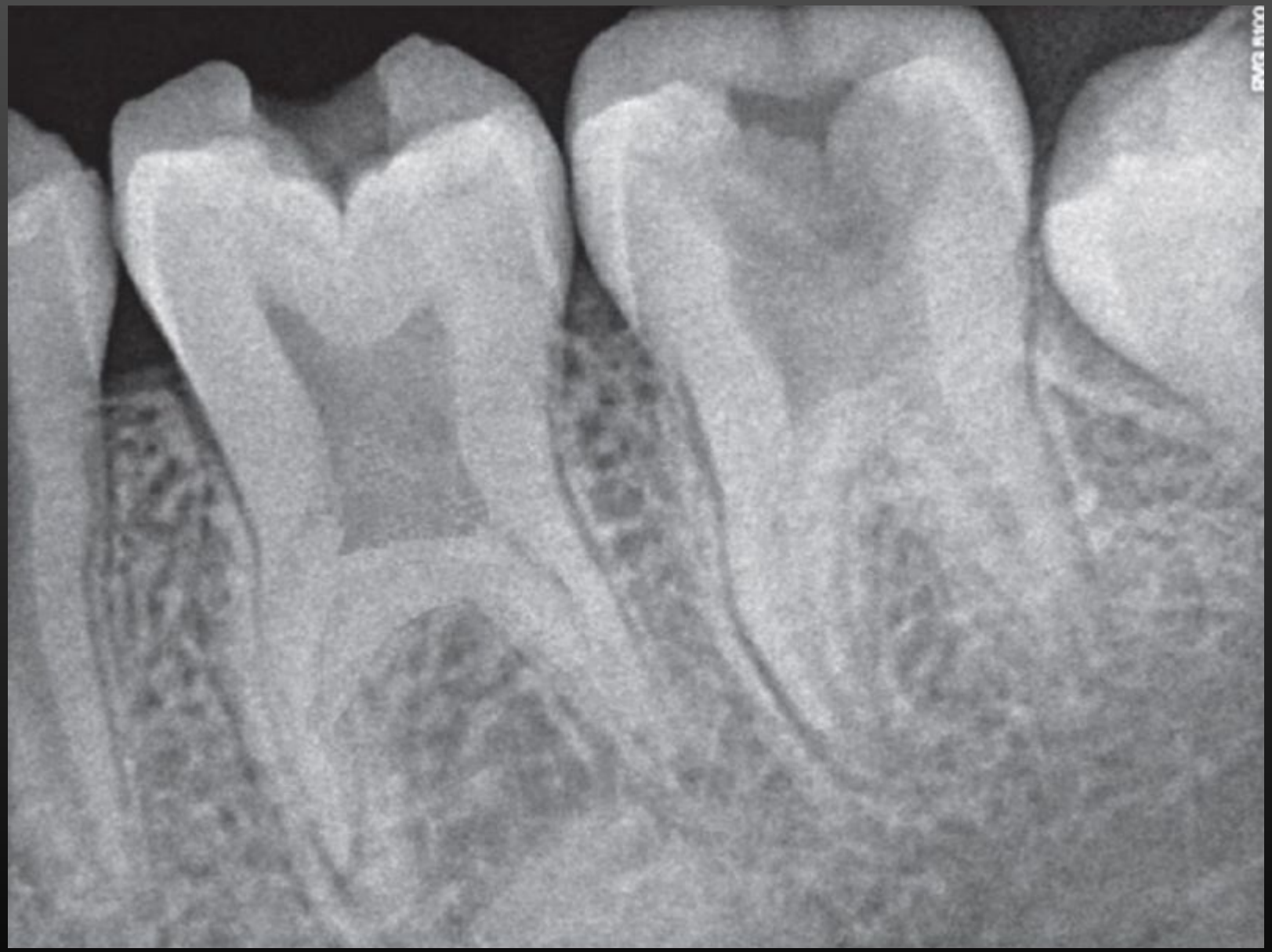

TEST
ctrl



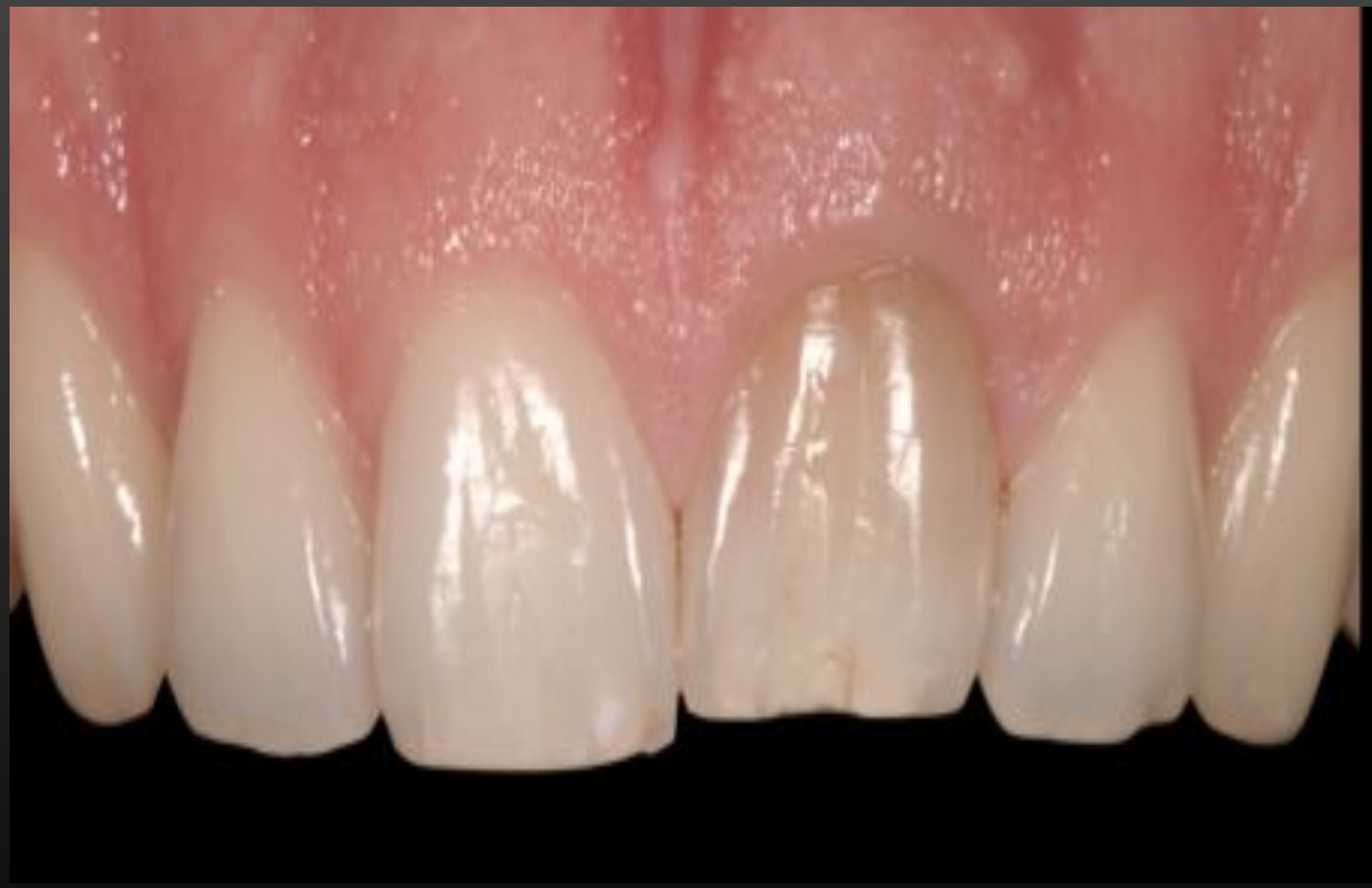
Hint: This condition seen in both dentitions but more common in deciduous

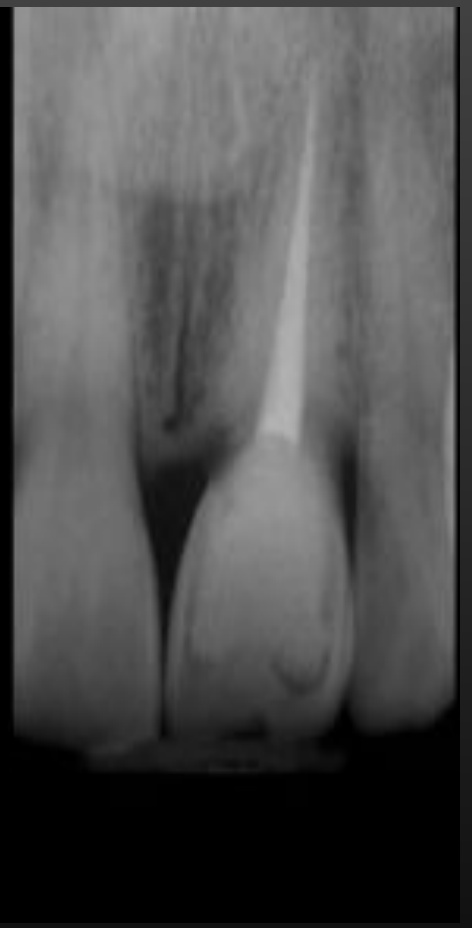


Hint:
developmental anomaly seen only in persons of Mongoloid ancestry

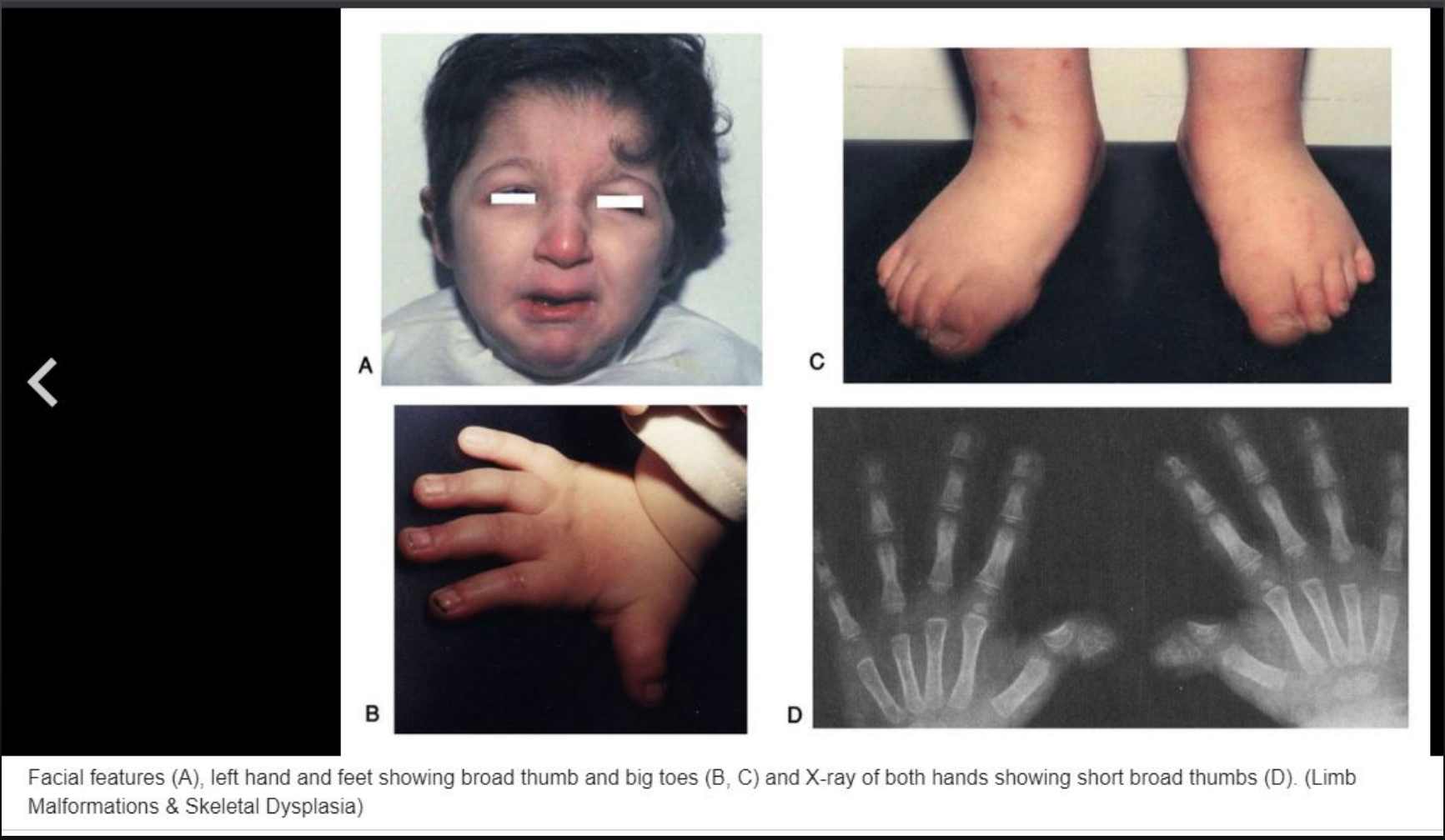


Hint: Many patients with Klinefelter syndrome(Extra X in males) exhibit this feature.
Also seen in Type IV Amelogenesis Imperfecta





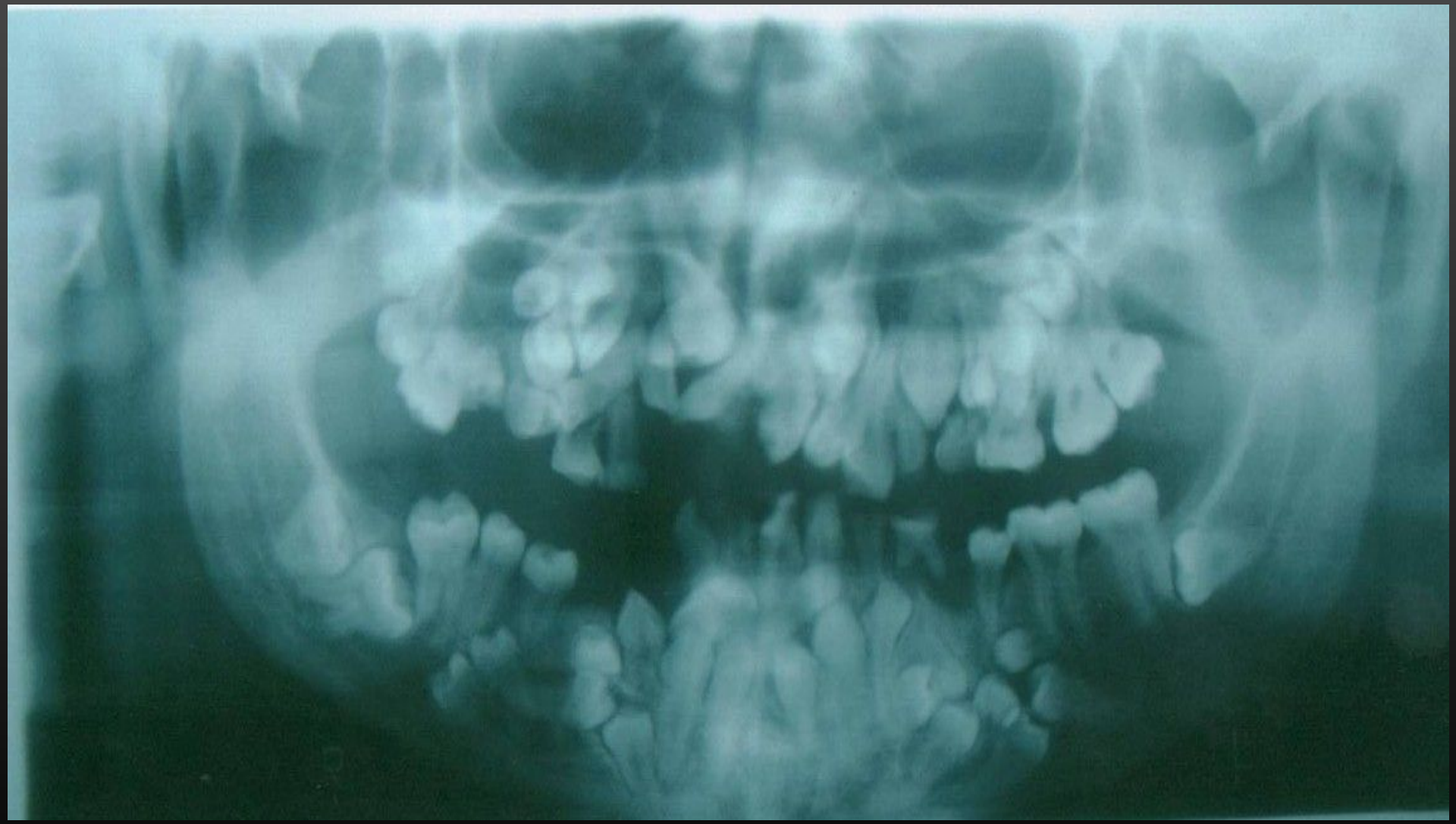
Hint: Characteristic solid (Metallic) sound on percussion while clinical examination



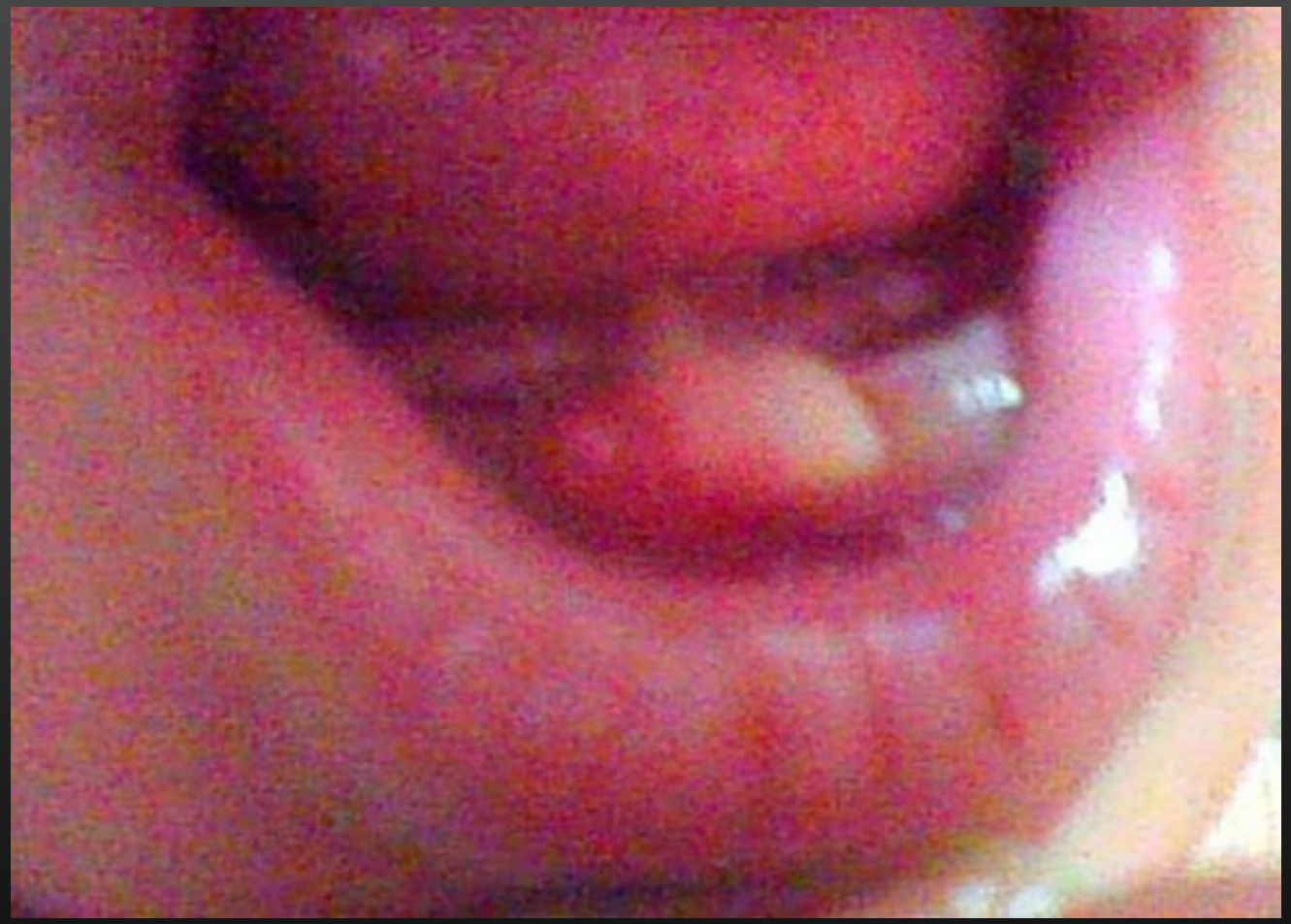
Facial features (A), left hand and feet showing broad thumb and big toes (B, C) and X-ray of both hands showing short broad thumbs (D). (Limb Malformations & Skeletal Dysplasia)

Typical features of this syndrome include:

Broad thumbs, broad first toes,
Mental disability
Small height, low bone growth, small head
Cryptorchidism in males
Unusual facies involving the eyes, nose, palate
+
An anomaly of tooth shape resembling an Eagle's talon



Hint:
Multiple Supernumerary Impacted teeth, Failure of eruption of permanent dentition, Missing clavicles, open skull sutures, large fontanelles, Frontal bossing, prognathic mandible (maxilla-micrognathia)

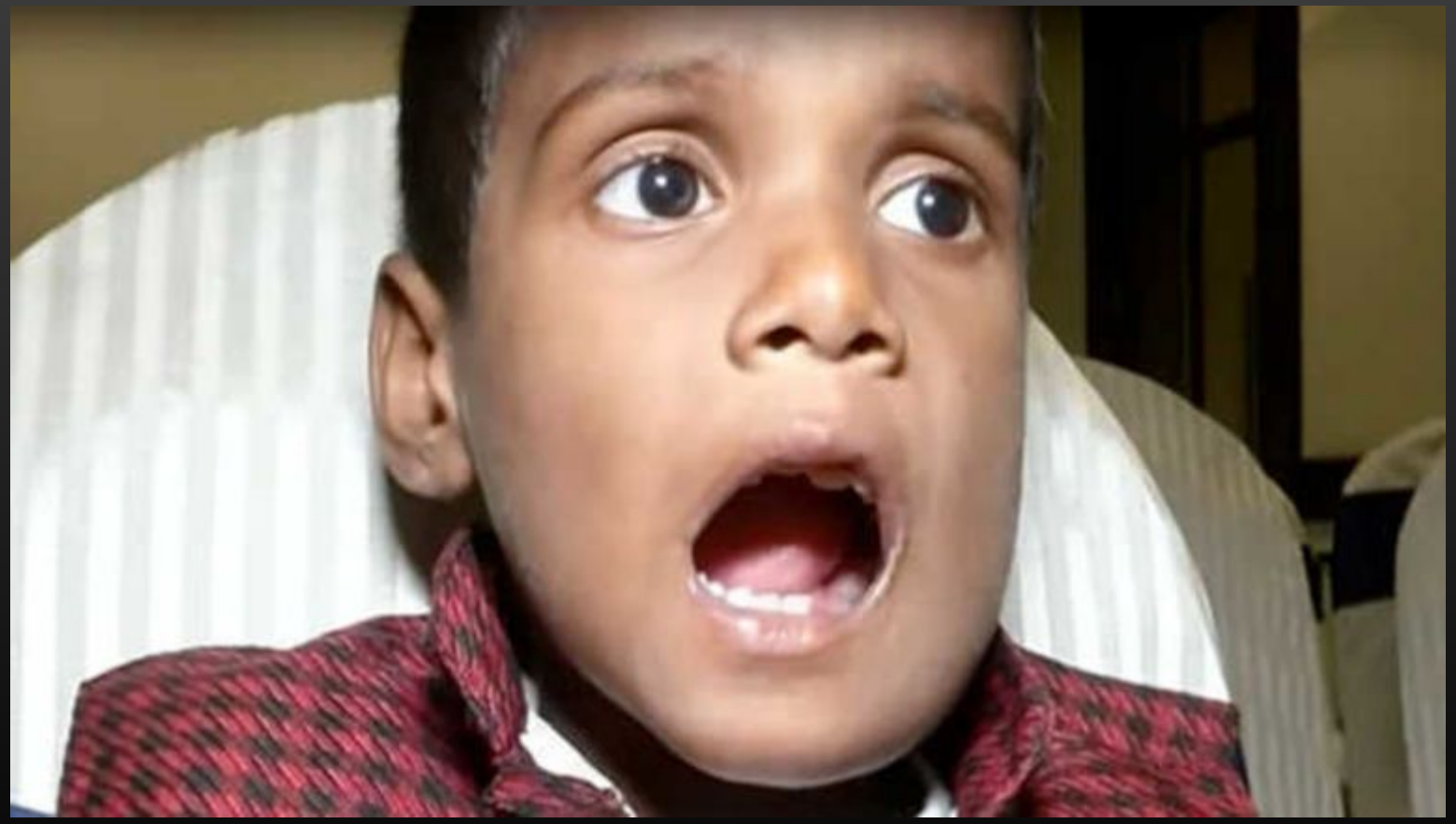


Hint: Pic of 2 day old baby. Shakespeare contributed his thoughts on this anomaly in “King Henry the Sixth” when he refers to Richard the Third in his quotation, “teeth hadst thou in thy head when thou wast born to riguity thou camest to bite the word.”

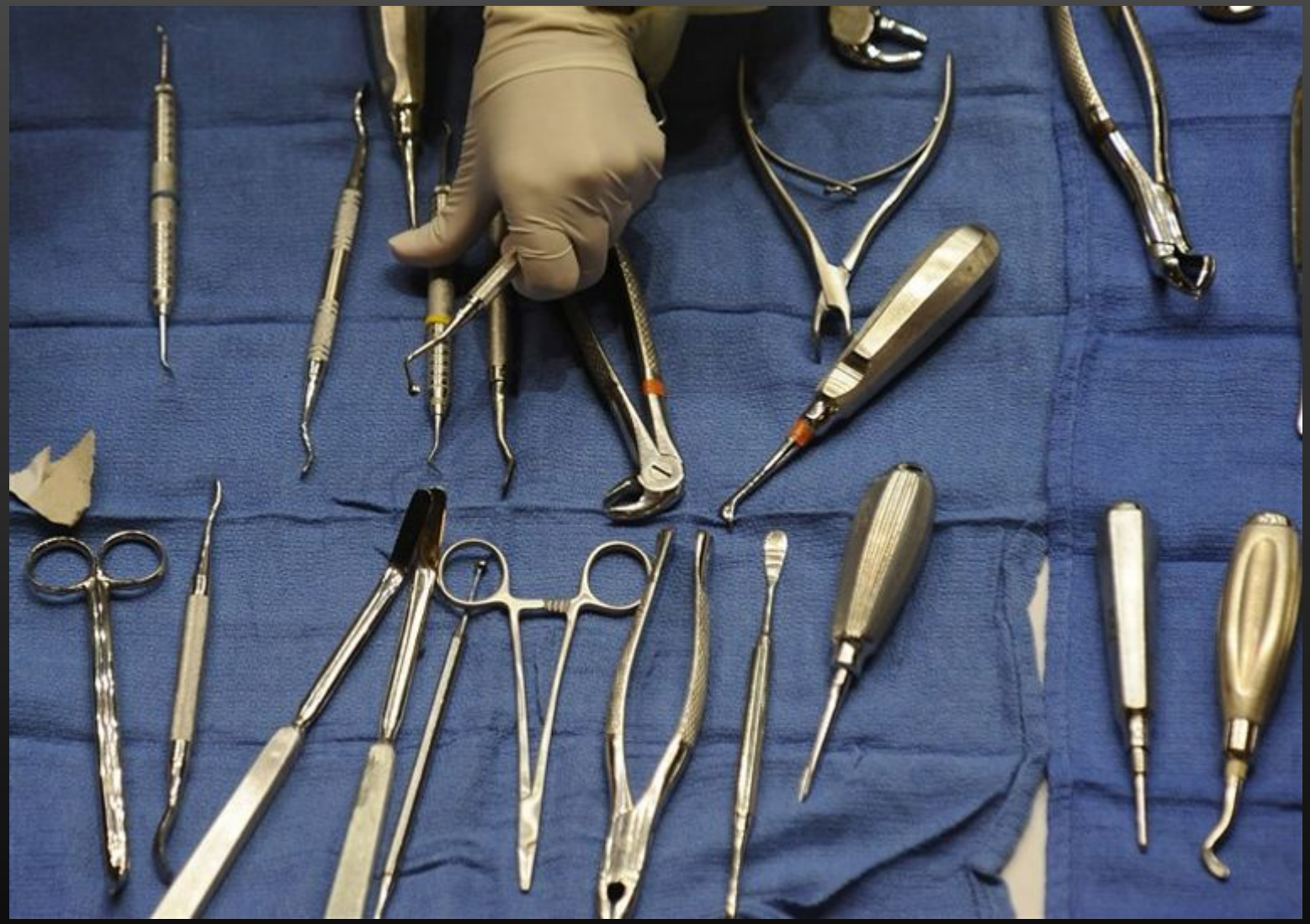
In England, the belief was that this condition would guarantee the conquest of the world.

More common in females than males, frequently bilateral, commonly seen in mandibular anterior region

Boy Complains Of Tooth Pain And Stuns The Dentist After Opening His Mouth



Ravindranath had a toothache. The 7-year-old told his parents, who gave his mouth a quick once over and concluded their boy needed to see a dentist. Was it growing pains? Surely a boy his age was too young to be suffering from any lapse in oral hygiene.

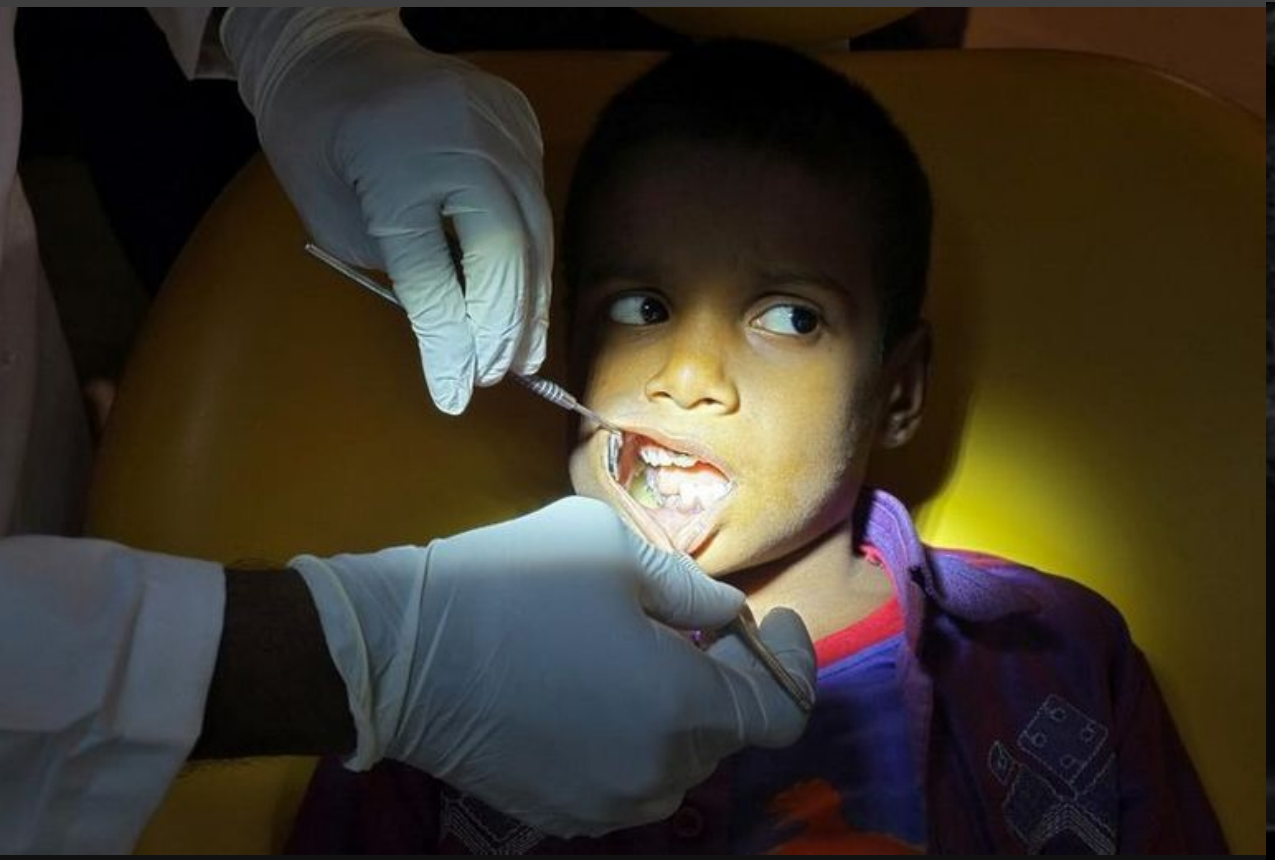


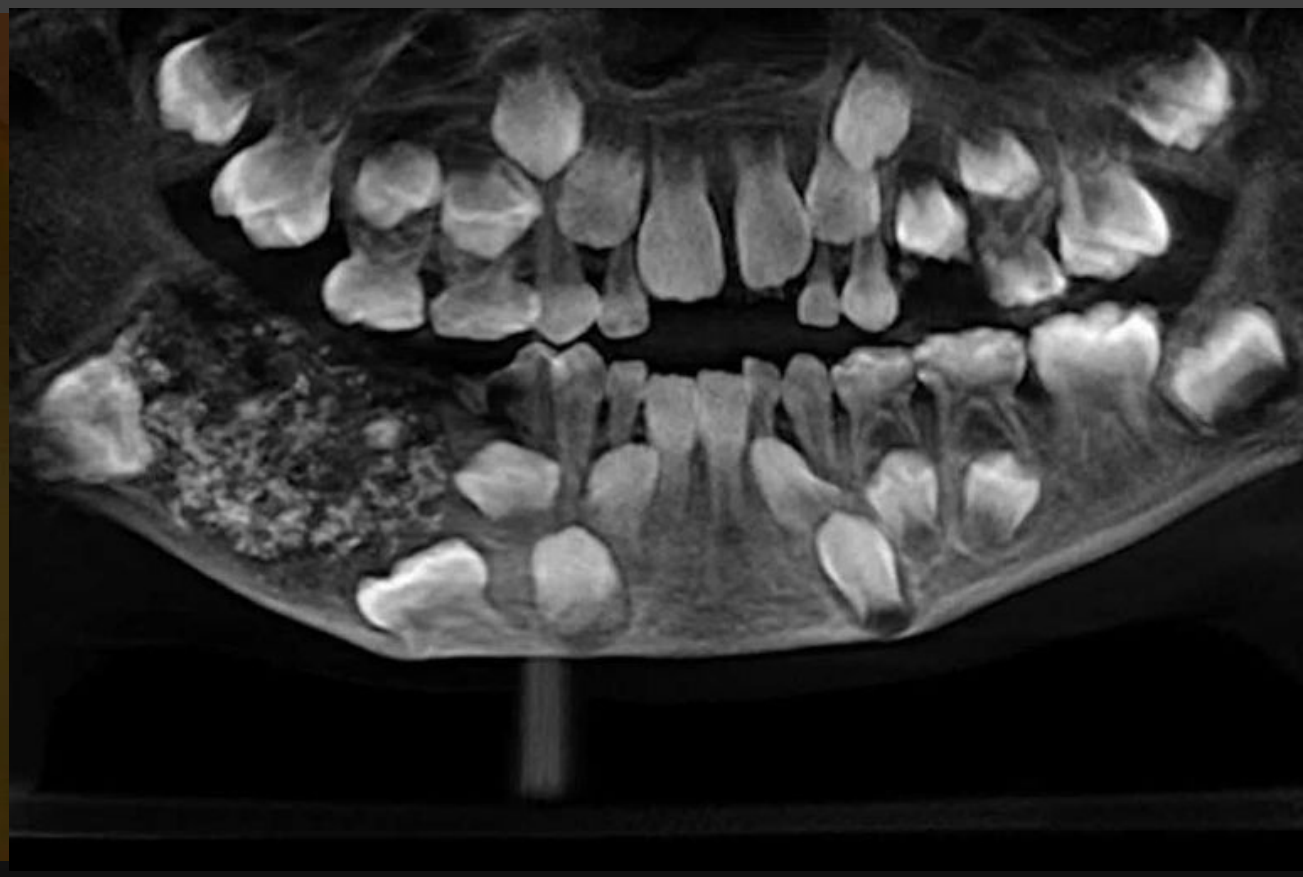
This wasn’t the first time they responded to their son’s complaints of oral discomfort. Back when Ravindranath was three, his father, S Prabudoss, noticed his son’s right cheek was slightly swollen.

Off they went to the dentist, but the boy was too young to sit still and cooperate for any examinations. Both parents agreed they’d rather not force the issue, so they opted to wait. The swelling eventually decreased...

...until five years later, when Ravindranath's right cheek swelled up again. This time, his parents stepped into action. Hoping to knock out whatever was causing him pain, they brought their son to a facility that came highly recommended

From the moment the dentist examined his mouth, they knew the boy was in serious pain. The medical staff immediately ordered extensive tests, x-rays, and CT scans to get to the root of what was really wrong with his right lower jaw





Once the results came back, the dentists' jaws dropped. Secretly tucked into Ravindranath's gums were many tiny little teeth. From the images, they couldn't determine how many extras he had, only that it was way more than normal.

The dentist explained to Ravindranath's parents the peculiarity of his condition. They felt it was medically necessary that the boy have surgery to remove his many spare teeth and to do it fast since their child was in pain.

Once they opened him up it was clear that they were dealing with a medical anomaly. “They looked like pearls in an oyster. Even the smallest piece had a crown, root, and an enamel coating like a tooth,” said one of the dentists involved in the surgery

One after another, they removed each tooth and dropped in into the nurse’s surgical tray. It seemed like a magic trick — the teeth just never stopped. When the number of teeth removed was well into the triple digits the operating room staff grew giddy with excitement over this groundbreaking medical event.

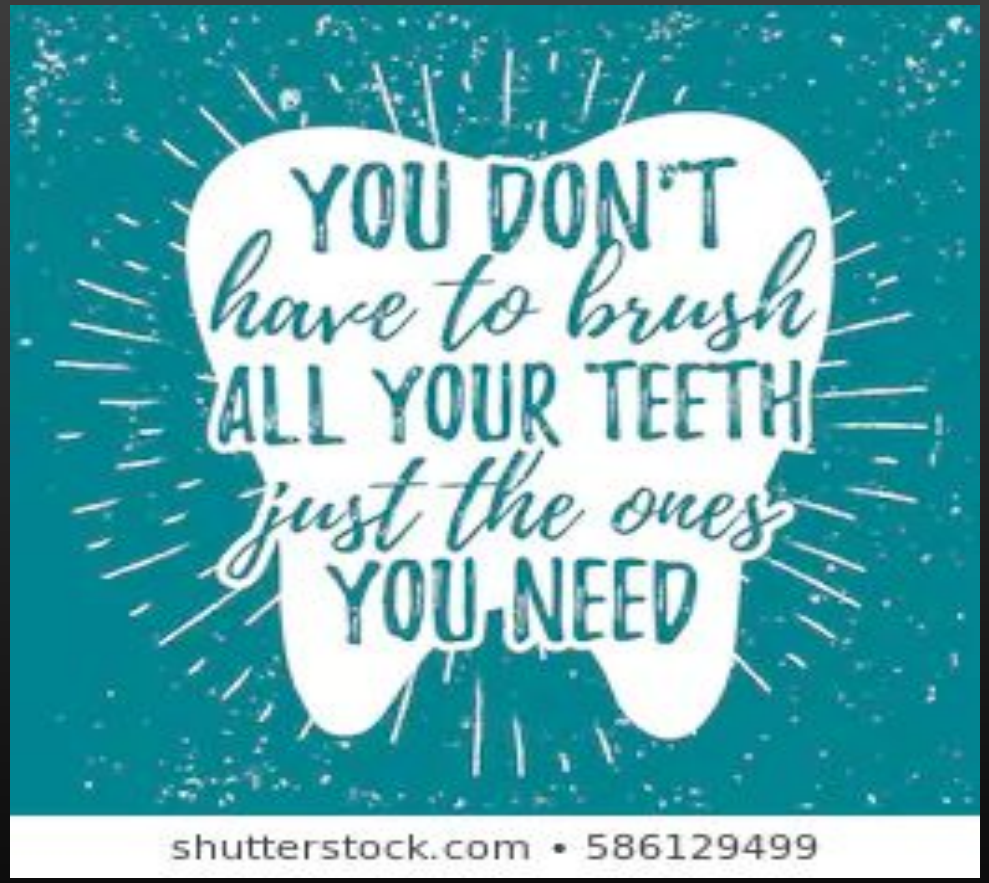
It amazed everyone that Ravindranath was carrying around a heavy burden in his regular-sized head, showing zero physical signs of his specialness. At the final count, the dentists removed a staggering 526 teeth from his jaws!

Post-surgery, he was left with his adult teeth still intact.

The removed teeth varied in size, the smallest at .1mm and the largest at 15mm

DIAGNOSIS ???


STRATUM GRINULOSUM
by @wittowoo
www.ihearthisto.com


YOU DON'T
have to brush
ALL YOUR TEETH
just the ones
YOU NEED
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