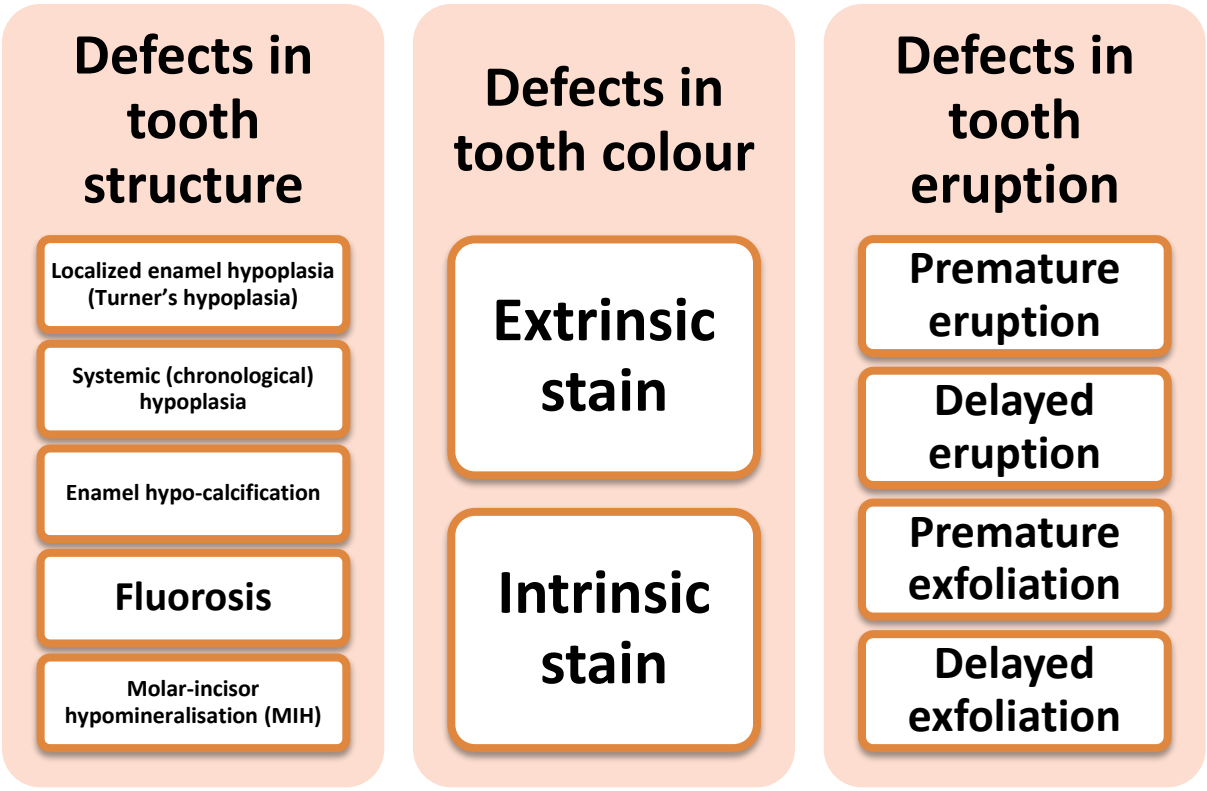
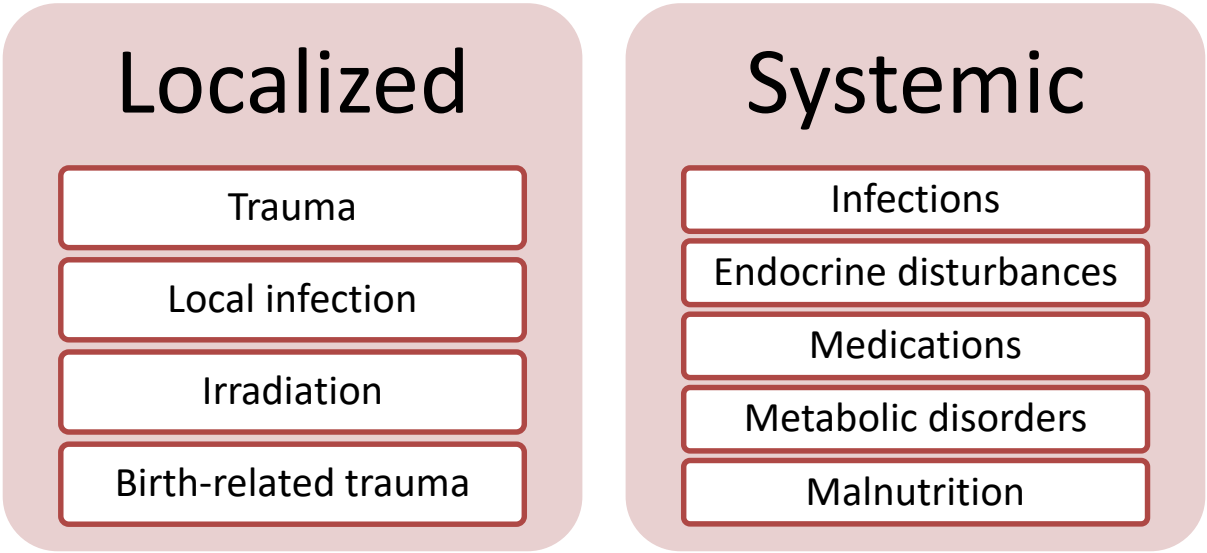
"Environmental Dental Anomalies"

They are acquired malformations of teeth that result from environmental disturbances to the tooth germ and can cause defects in tooth structure, colour or eruption.



➢ Causative factors for environmental dental defects:



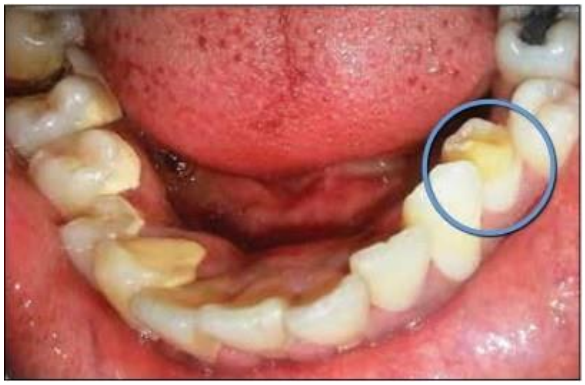
➢ Defects in tooth structure:

Enamel defects may be caused by genetic or environmental factors acting alone or in combination. Enamel usually develops in two phases, first an organic matrix deposition and second mineralization. Where less enamel matrix than normal is produced, the resulting enamel will be thinner (hypoplasia). If there is a defect in mineralization of the enamel matrix proteins, the result will be poorly mineralized enamel (hypomineralization—sometimes subdivided into hypocalcification for more severe defects and hypomaturation for milder changes). In many cases there will be a combination of both hypoplasia and hypomineralization, although sometimes the defect will be perceived clinically as predominantly one or the other. When enamel hypoplasia is seen the enamel may be either uniformly thin or grooved or pitted. In hypomineralization, the enamel will typically be discoloured, usually a yellow-brown colour, weak and prone to breakdown. This is particularly so where the defect is more severe (hypocalcification), whereas in a less severe presentation (hypomaturation) the enamel may be almost normal but appear mottled or even only slightly opaque rather than translucent.

1- Localized enamel hypoplasia (Turner's hypoplasia):

One or more permanent tooth-germs may be affected by infection from an overlying primary predecessor. Such teeth are termed Turner teeth and typically have areas of enamel hypoplasia and/or enamel hypomineralization. The mandibular premolars are most commonly affected **(Fig. 8-1).**

A traumatic blow to an anterior primary tooth that causes its apical displacement can interfere with matrix formation or calcification of the underlying permanent tooth. The trauma or subsequent periapical infection frequently produces localized enamel defects on the labial surface of the permanent incisor **(Fig. 8-2)**.



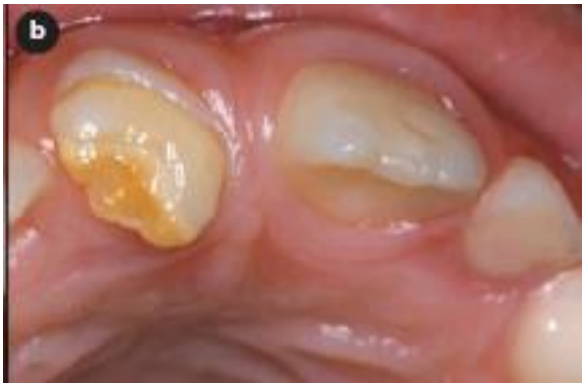


Figure 8-1 **Figure 8-2**

2- Chronological enamel hypoplasia:

It is a type of enamel hypoplasia that results from a deficiency state or a systemic condition. It is usually evident and follow a definite pattern on all the teeth that were undergoing matrix formation and calcification at the time of the insult.

The maternal and foetal conditions that might result in chronological enamel hypoplasia include endocrine disturbances (hypoparathyroidism), infections (rubella), drugs (thalidomide), nutritional deficiencies (particularly in vitamins A, C, & D as well as calcium & phosphorus), and haematological and metabolic disorders (rhesus incompatibility).

A knowledge of the timing of commencement of formation of the teeth will aid in understanding the timing of such insult. When there is a systemic upset or marked physiological changes occur during pregnancy, corresponding enamel defects may be seen in the primary dentition **(Fig. 8-3)**. Illness in the neonatal period may affect the tips of the first permanent molars as these commence development at around birth **(Fig. 8-4)**. Enamel defects in permanent teeth may also arise as a result of acute or chronic childhood illnesses.

Syphilis produces classic patterns of hypoplastic dysmorphic permanent teeth. The tapered and notched incisal edges of anterior teeth with screwdriver shapes are called Hutchinson's incisors **(Fig. 8-5)**, and the crenated occlusal patterns of posterior teeth known as mulberry molars **(Fig. 8-6)** are classic clinical findings for prenatal syphilis infection.

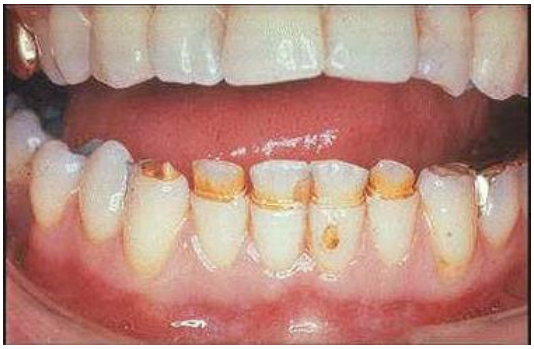
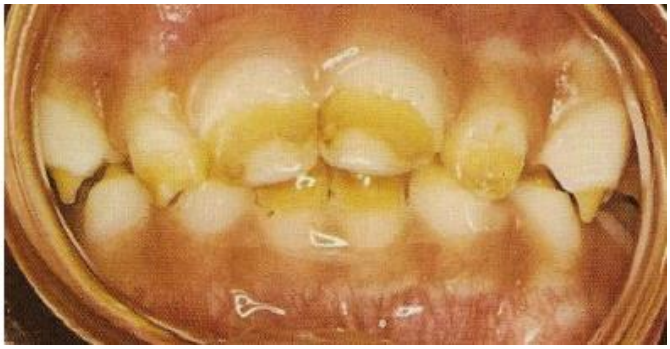


Figure 8-3



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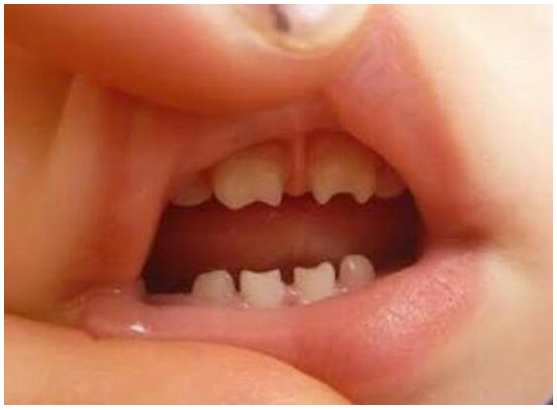

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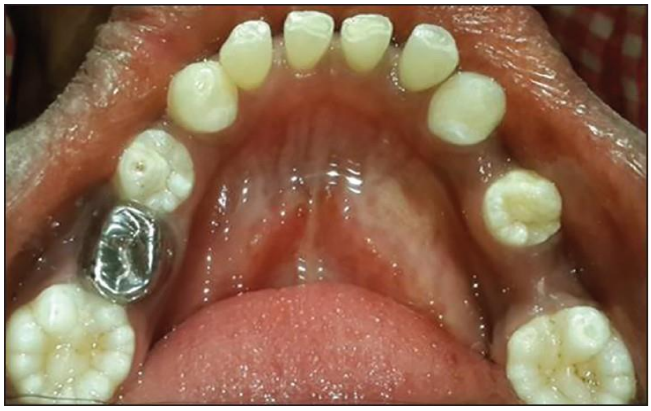


Figure 8-6

3- Enamel hypo-calcification:

Enamel Hypocalcification defects can be directly related to faults in the mineralization of the organic matrix in enamel formation **(Fig. 8-7)**. The same factors that cause enamel hypoplasia also cause hypocalcification. The majority of localized hypocalcific defects, as in the case of Turner hypoplasia, are subsequent to localized infection and trauma. Excess exposure to citric acid resulting from habitual sucking on citrus fruits can produce generalized erosive hypocalcified lesions that mimic the hypocalcification type of amelogenesis imperfecta.

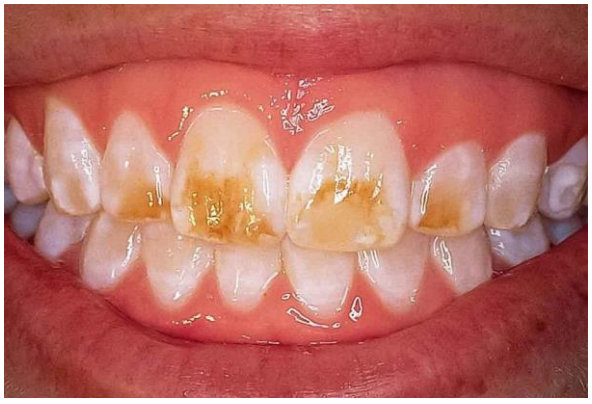


Figure 8-7

4- Dental fluorosis:

Excess ingestion of systemic fluoride can produce generalized enamel defects. Dental fluorosis can be manifested as a defect in the calcification of the teeth in milder forms, with significant pigmentation and ameloblastic impairment in the more severe forms. Fluorosis occurs when the concentration of ingested fluoride is above 1.8 ppm/day. There is a 90% chance of some degree of dental fluorosis when the amount of ingested fluoride is greater than 6 ppm, although the severity of morphologic defects cannot be predicted from specific quantities of ingested fluoride.

In its mildest form, fluorosis appears as an opacity of the enamel. The condition is dose dependent, with increasing intake of fluoride being associated with more marked opacity, areas of discolouration of the enamel, and pitting or more extensive hypoplastic defects **(Fig. 8-8)**. Local fluorotic lesions may respond very well to the micro-abrasion technique.

Figure 8-8

5- Molar-incisor hypomineralization:

In recent years, reports have been published of children with mineralization defects of the first permanent molars and, sometimes, the permanent incisors **(Fig. 8-9)**. This has been referred to as molar incisor hypomineralization or hypoplasia and also as ‘cheese molars’ because of the friable nature of the enamel of the molar tooth enamel. Affected 6s have hypomineralized defects of enamel, varying from discoloration to severe enamel dysplasia with post-eruptive breakdown, sensitivity and secondary caries. Defects may affect anything from one to all 6s. The defects in the incisors, which are usually less severe and most likely to show demarcated isolated mottling, will likewise be irregularly distribute and are less prone to enamel breakdown than 6s. Childhood infections, specific antibiotics, and repeated fevers have all been suggested as causes. It has been suggested that a genetic predisposition combined with an environmental insult might produce these changes, but this has yet to be substantiated.

Treatment options include intracoronal restoration, stainless steel crowns, or extraction (E Extraction of poor quality first permanent molars) or partial composite veneering for incisors.

Figure 8-9

➢ Defects in tooth colour:

1- Extrinsic stain:

It is caused by extrinsic agents and can be removed by prophylaxis. Green **(Fig. 8-10)**, black **(Fig. 8-11)**, orange, or brown stains are seen, and may be formed by chromogenic bacteria or be dietary in origin. Chlorhexidine mouthwash causes a brown stain by combining with dietary tannin. Where the staining is associated with poor oral hygiene, demineralization and roughening of the underlying enamel may make removal difficult.

Treatment involve the use of a mixture of pumice powder and toothpaste or an abrasive prophylaxis paste together with a bristle brush. Also, oral hygiene instructions must be given to prevent recurrence.

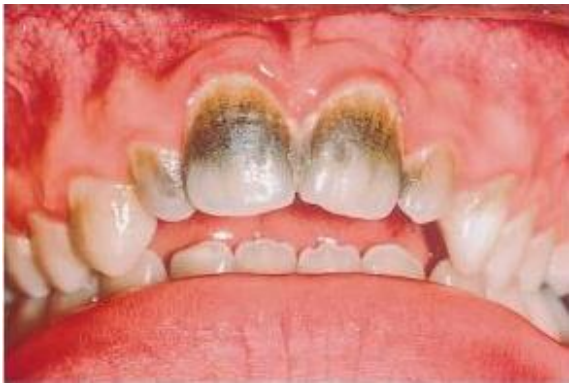


Figure 8-10

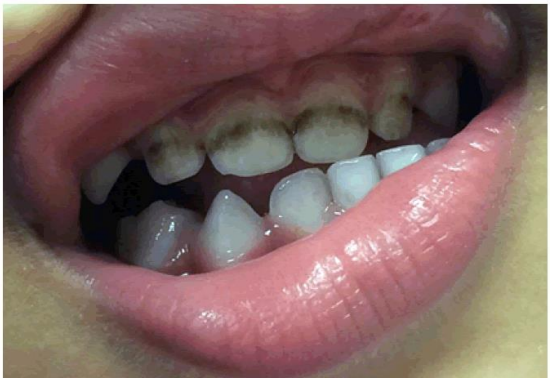


Figure 8-11

2- Intrinsic stain:

It can be caused by:

- Changes in the structure or thickness of the dental hard tissues, e.g. enamel opacities **(Fig. 8-12)**.
- Incorporation of pigments during tooth formation, e.g. tetracycline staining (blue/brown) **(Fig. 8-13)**, Hyperbilirubinemia (green) **(Fig. 8-14)**
- Diffusion of pigment into hard tissues after formation, e.g. pulp necrosis products (grey), root canal medicaments (grey) **(Fig. 8-15)**.

One of the treatment approaches include the use of micro-abrasion technique with phosphoric acid (slow but potentially safe technique) **(Fig. 8-16)**. The method include etching the enamel surface with 30–50% orthophosphoric acid

for 1‑2 min, wash, then use pumice and water slurry with rubber prophy cup for 1 min (take care not to overheat the tooth). Wash. Repeat etch and pumice stage ×2, washing between. Dry tooth and apply topical fluoride solution (avoid pigmented varnishes). May be repeated up to ×2, but leave at least 6 weeks before each repeat to check for improvement.

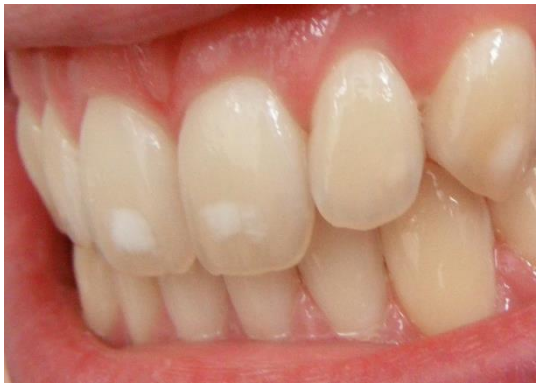
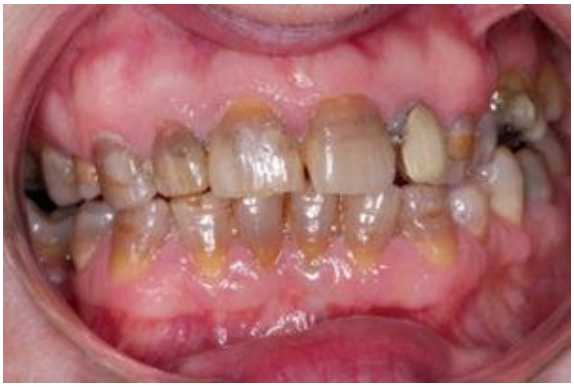


Figure 8-12



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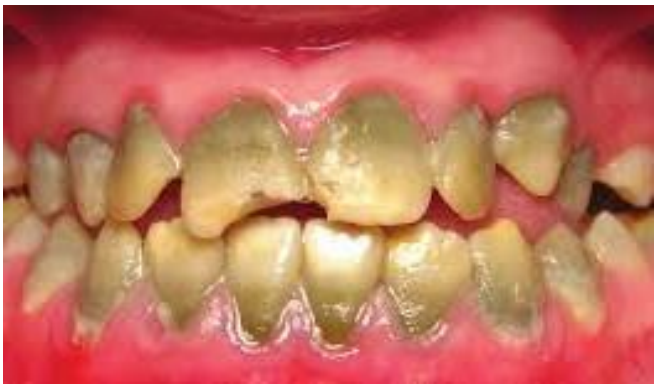

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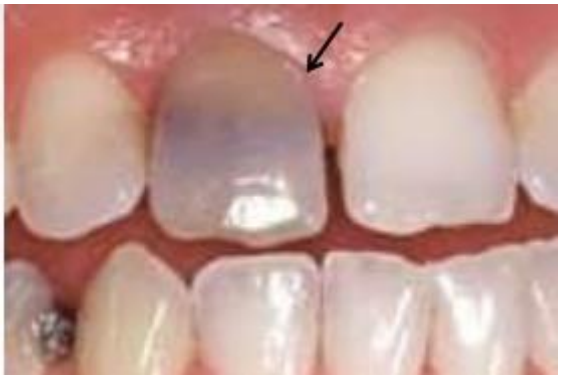

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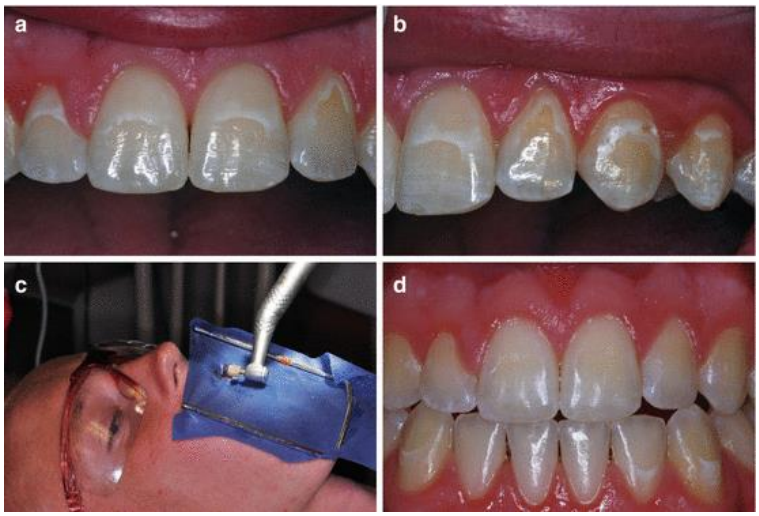

Figure 8-16

➢ Defects in tooth eruption:

1- Premature eruption:

Premature eruption may be familial. There is a tendency to early eruption in children with a high birth weight. Excessively early eruption is seen in endocrine abnormalities (e.g. increase of thyroid and growth hormone).

Natal and neonatal teeth Natal (present at birth) and neonatal (erupts within 30 days of birth) teeth occur in about 1 in 2000–3000 births **(Fig. 8-17)**.

Management: Teeth are left if possible to allow normal root formation. Extraction may be necessary because of extreme mobility and airway danger, painful suckling or tongue ulceration.

2- Delayed eruption:

Delayed eruption of primary teeth tends to occur in preterm children or those of very low birth weights. It is also associated with a number of conditions such as Down syndrome, nutritional deficiency, hypothyroidism and hypopituitarism.

In the permanent dentition, localised causes are more frequent: ectopic crypt positions, supernumeraries, odontomes and impaction.

3- Premature exfoliation:

Apart from trauma (accidental or non-accidental), there are a number of rare conditions that may result in premature loss of primary teeth such as Hypophosphatasia.

Papillon-Lefèvre syndrome can also cause premature loss of primary and permanent teeth.

4- Delayed exfoliation:

Delay in normal exfoliation of primary teeth may be seen in association with:

- Double primary teeth
- Hypodontia affecting permanent successors
- Ectopic permanent successors

- Subsequent to trauma or severe periradicular infection of primary teeth
- Infraocclusion **(Fig. 8-18)** or ankylosed teeth **(Fig. 8-19)**

Figure 8-17

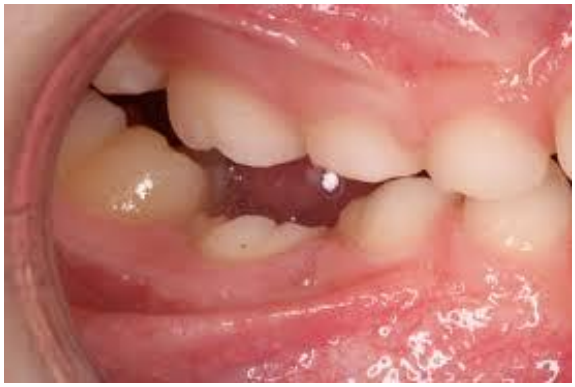

Figure 8-18

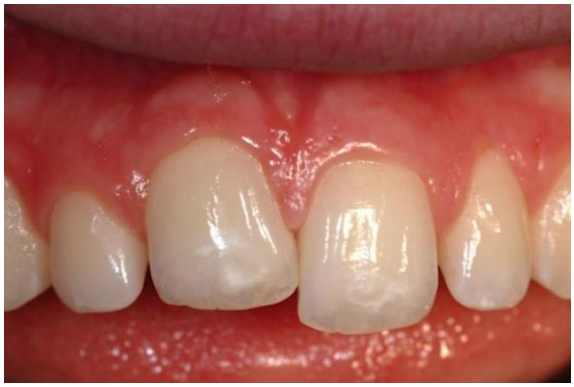

Figure 8-19

❖ References:

- Dean, JA. et al. 2015. *McDonals and Avery's Dentistry for the child and adolescent*. 10th edition. Missouri: Elsevier.
- Welbury, R. et al. 2012. Paediatric Dentistry. 4th edition. Oxford: Oxford University Press.