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Hand Book

Hand Book of Tooth Structure Loss (TSL): New Classification, Examination, Diagnosis, Management and Treatment

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Abstract and Aim of this Classification

There are many cases of tooth structure loss which could be tabulated under two main categories either non-diseased or diseased, whatever for long time researches always put classification only for part and leave other, so there are many old classifications for differently separated items of tooth structure loss.

We decided to collect these events in a new classification to be complete and easy for practitioners to identify the non-diseased cases and diseased cases of tooth structure loss. So, simplify for them the diagnosis and clinical management of tooth loss by focusing on those lesions which do not and those which do require treatment, and to identify other lesions where combined or alternative treatment is indicated.

Emphasis will be placed on preventive measures to control the progress and the treatment plan which is possible in each particular clinical situation.

This paper could be used by any practitioner around the world as hand book for identification of the case, diagnose, and do the ideal treatment for it.

Keywords: *TSL; Non-Diseased; Diseased*

Part I: THE Non-Diseased TSL [T]

Introduction

Trauma, by different ways and severities, is almost responsible for cases of non-diseased tooth structure loss.

The extent of injury is influenced by the severity of the traumatic event, the presence or absence of protective gear and the direction of force against the teeth and supporting structures. Traumatic dental injuries can cause serious aesthetic, functional, and psychological consequences.

Time is one of the most critical factors determining clinical outcome; all dental injuries should be considered as true emergencies.

An acute dental trauma may imply impact to the hard dental tissues and damage to the pulp and periodontium, including the surrounding alveolar bone.

Comparing and accumulating data from different studies is extremely difficult due to the differences in the definitions and classifications used.

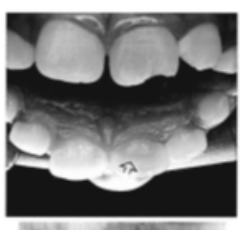
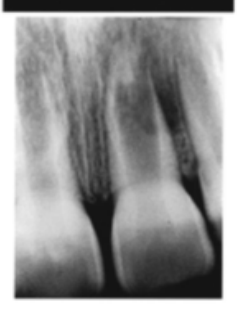
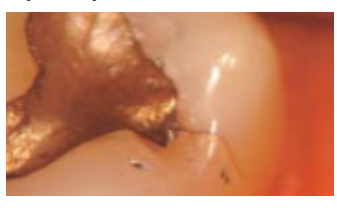
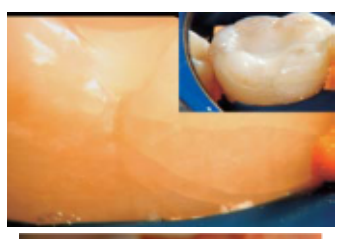
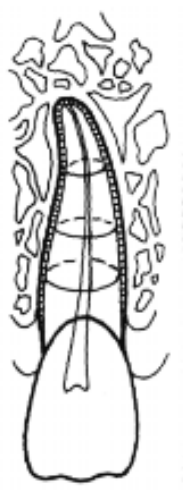
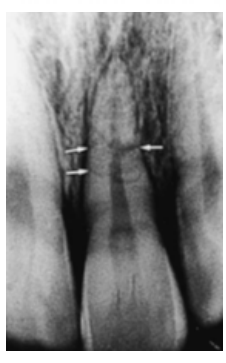
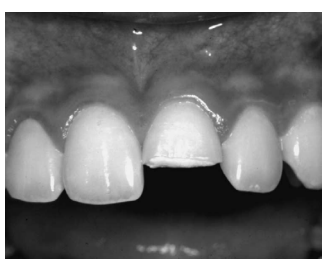
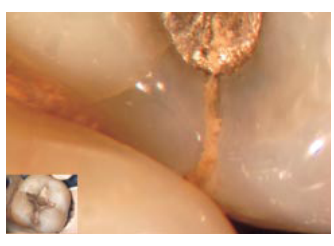
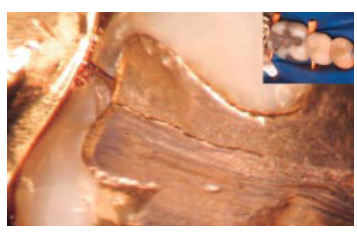
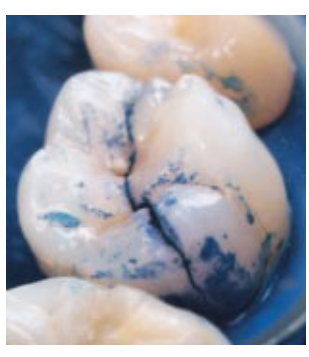
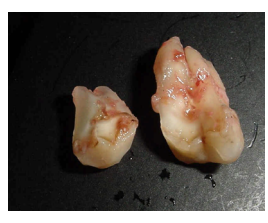
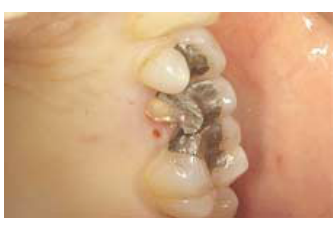
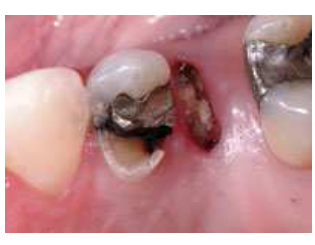
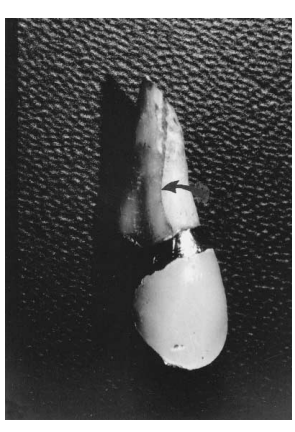
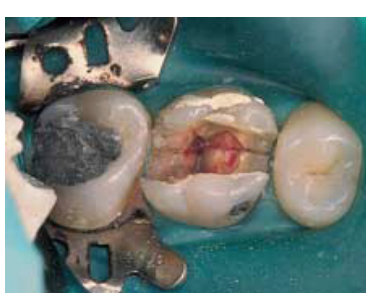
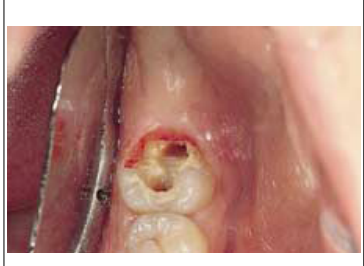
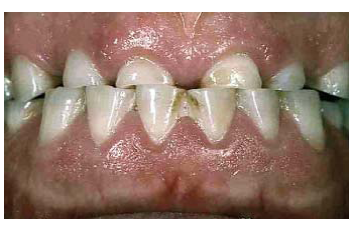
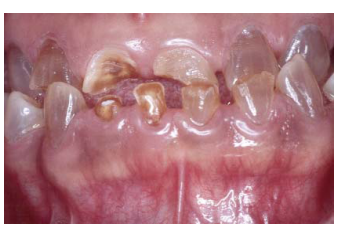
Etiological Factors

Trauma, by different ways and severities, is almost responsible for cases of non-diseased tooth structure loss.

Trauma responsible for non-diseased tooth structure loss could be classified as follows:

1) Habitual Trauma (Th): caused by bad habits of biting of hard structures for long times.
2) Friction Trauma (Tf): caused by wrong use of dental brushes.
3) Occlusal Trauma (To): caused by bad occlusal and biting forces due to uncontrolled reasons.
4) Accidental Trauma (Ta): caused by different impacts of traumatic injuries and could be: a. Mild b. Moderate c. Severe

The new classification: T

(TA) Crown Fracture	(TB) Crown Cracks	(TC) Root Fracture
I: Incisal notch/ Occlusal chipping or facets (TAI): a. Enamel only (TAIa) b. Enamel & Dentin (TAIb) c. Enamel, Dentin & involving the pulp (TAIc)  	I: Undefined Crack (Enamel only) (TBI): a. Short distance: (less than ½ of the tooth enamel) (TBIa) i. Incisal/occlusal (TBIai) ii. Cervical (TBIaii)  b. Long distance: (more than ½ of the tooth enamel) (TBib)   i. Incisal/occlusal (TBIbi) ii. Cervical (TBIbii)	I: Horizontal Fracture (usually anterior teeth) (TCI): a. Cervical (TCIa) b. Middle (TCIb) c. Apical (TCIc) Mainly caused by direct trauma  
II: Incisal/occlusal fracture involving one or both proximal sides (TAII): a. Enamel only (TAIIa) b. Enamel & Dentin (TAIIb) c. Enamel, Dentin & involving the pulp (TAIIc) 	II: Defined Crack (Enamel & Dentin) (TBII): a. Short distance: (less than ½ of the tooth) (TBIIa) i. Incisal/Occlusal (TBIIai) ii. Cervical (TBIIaii)   b. Long distance: (more than ½ of the tooth) (TBIIb) i. Incisal/occlusal (TBIIbi) ii. Cervical (TBIIbii)  	II: Oblique Fracture (anterior & posterior) (TCII): a. Cervical (TCIIa) b. Middle (TCIIb) c. Apical (TCIIc) Caused by high oblique biting forces on mostly Periodontally affected tooth 
III: Extensive Crown Fracture (TAIII): a. Less than ½ of the crown (TAIIIa)  b. More than ½ of the crown but above the CEJ (TAIIIb)  c. All the crown and extend to the root (TAIIIc) 	III: Fractured-Crack (Enamel, Dentin & involving Pulp) (TBIII): a. Mild (TBIIIa): incomplete fracture of the crown & not involving periodontal apparatus b. Moderate (TBIIIb): incomplete fracture of the crown & involving the periodontal apparatus c. Sever (TBIIIc): complete fracture (detachable) of the tooth crown with pulpal & periodontal involvement, no root involvement d. Complicated (TBIIId): complete fracture (detachable) involving crown & part of the root (E., D., C., pulp & periodontal apparatus) e. Complex (TBIIIe): fracture involving crown, part of the root & alveolar bone  	III: Vertical Fracture (TCIII) (usually in posterior teeth): May be caused by teeth clenching or being struck with the jaw closed. Most commonly happened to molars during RCT. 
IV: Occlusal/Cervical tooth loss (TAIV): (due to normal attrition, normal abrasion or abfraction) a. Enamel only (TAIVa) b. Enamel & Dentin (TAIVb) c. Enamel, Dentin & involving the pulp (TAIVc)  		

Tooth parts affected by each event are as follows:

T.S.L. class T	Enamel	Dentin	Pulp	Cementum	Periodontal apparatus	Alveolar Bone
TAIa	*	-	-	-	-	-
TAIb	*	*	-	-	-	-
TAIc	*	*	*	-	-	-
TAIIa	*	-	-	-	-	-
TAIIb	*	*	-	-	-	-
TAIIc	*	*	*	-	-	-
TAIIIa	*	*	*	-	-	-
TAIIIb	*	*	*	-	-	-
TAIIIc	*	*	*	*	-	-
TAIVa	*	-	-	-	-	-
TAIVb	*	*	-	-	-	-
TAIVc	*	*	*	-	-	-
TBIai	*	-	-	-	-	-
TBIaii	*	-	-	-	-	-
TBIbi	*	-	-	-	-	-
TBIbii	*	-	-	-	-	-
TBIIai	*	*	-	-	-	-
TBIIaii	*	*	-	-	-	-
TBIIbi	*	*	-	-	-	-
TBIIbii	*	*	-	-	-	-
TBIIIa	*	*	*	-	-	-
TBIIIb	*	*	*	-	*	-
TBIIIc	*	*	*	-	*	-
TBIIId	*	*	*	*	*	-
TBIIIe	*	*	*	*	*	*
TCIa	-	*	*	*	*	-
TCIb	-	*	*	*	*	*
TCIc	-	*	*	*	*	*
TCIIa	-	*	*	*	*	-
TCIIb	-	*	*	*	*	-
TCIIc	-	*	*	*	*	-
TCIII	-	*	*	*	*	-

Examination and Diagnosis of Traumatic TSL

An initial comprehensive examination is performed, including a thorough medical and dental history and an orofacial and dental clinical examination. Radiographs and other special tests may then be carried out. Such tests may include fracture finder, pulpal sensibility testing, illumination testing, etc. Questions regarding lifestyle, medications, stress, brushing habits, etc. can help in aiding diagnosis. From clinically observed features and habits and careful collation of all this information determination of the risk factors is helpful to minimize long term damage of teeth.

Diagnosis involves all findings to identify the factor(s) contributing to tooth structure loss. This is to preserve the remaining dentition and to improve the long term prognosis of any restorative treatment completed.

Treatment plan

Treatment plan will vary according to the class of loss, from simple filling to extraction and construction of fixed prosthesis or even removable one.

Suggested treatment of every case will be as follows:

Classes	Treatment Plan
TAIa	Simple filling (composite or amalgam).
TAIb	Simple filling (composite or amalgam).
TAIc	RCT, then filling (composite or amalgam). If tooth weakened crowning is must.
TAIIa	Filling for destructed sides (composite or amalgam)
TAIIb	Filling for destructed sides (composite or amalgam)
TAIIc	RCT, then filling (composite or amalgam), followed by crowning.
TAIIIa	RCT, then filling (composite or amalgam), followed by crowning.
TAIIIb	RCT, post and core will be used for reconstruction of tooth and then crowning.
TAIIIc	This will depend on the extension of root fracture: A. Fracture above or at alveolar bone level: RCT, post and core will be used for reconstruction of tooth, followed by crown lengthening and then crowning. B. Fracture extends far beyond the alveolar bone level: Extraction followed by either implant or fixed prosthesis.
TAIVa	Filling (composite or amalgam)
TAIVb	Filling (composite or amalgam)
TAIVc	RCT, post and core will be used for reconstruction of tooth and then crowning.
TBIai	To be left under observation or sealed with resins.
TBIaii	To be left under observation or sealed with resins.
TBIbi	To be sealed by resins.
TBIbii	To be sealed by resins.
TBIIai	Restored by filling.
TBIIaii	Restored by filling.
TBIIbi	Restored by filling.
TBIIbii	Restored by filling.
TBIIIa	Restored by filling.
TBIIIb	Restored by filling, periodontal intervention (treatment or crown lengthening).
TBIIIc	RCT, post and core build up, followed by periodontal intervention, and then crowning.
TBIIId	This will depend on the extension of root fracture: A. Fracture above or at alveolar bone level: RCT, post and core will be used for reconstruction of tooth, followed by crown lengthening and then crowning. B. Fracture extends far beyond the alveolar bone level: Extraction followed by either implant or fixed prosthesis.
TBIIIe	RCT as emergency treatment, tooth fixation to stabilize the bone, after that treatment will be completed as in case of BIIId. Extraction followed by implant and may need bone augmentation in case of extensive fracture and deep alveolar bone fracture.
TCIa	a. If the fracture line is above the alveolar bone crest the fractured part with the crown are removed, RCT performed, then post and core done, after that crown lengthening to get 2mm ferrule is a must, followed by crowning of the tooth. b. If the fracture line is far beyond the alveolar bone crest and there is no mobility and the two parts are intact RCT and fixation of the tooth will be the solution with follow up period to insure that there is no root resorption happened at the fracture site, and if happened surgical intervention using root-end filling at the site of resorption will be the solution. c. If the fracture line is far beyond the alveolar bone crest and there is mobility and the two parts are separated and moved away, extraction will be the solution.
TCIb	a. If there is no mobility and the two parts are intact RCT and fixation of the tooth will be the solution with follow up period to insure that there is no root resorption happened at the fracture site, and if happened surgical intervention using root-end filling at the site of resorption will be the solution. b. If there is mobility and the two parts are separated and moved away, extraction will be the solution.
TCIc	a. If there is no mobility and the two parts are intact RCT and fixation of the tooth will be the solution with follow up period to insure that there is no root resorption happened at the fracture site, and if happened surgical intervention to remove the apical part, and then retro-grade filling will be the solution. b. If there is mobility and the two parts are separated and moved away, surgical removal of the apical part and retro-grade filling after RCT will be the must.
TCIIa	a. If there is no mobility and the two parts are intact RCT and fixation of the tooth will be the solution with follow up period to insure that there is no root resorption happened at the fracture site, and if happened surgical intervention using root-end filling at the site of resorption will be the solution. b. If there is mobility and the two parts are separated and moved away, extraction will be the solution.
TCIIb	a. If there is no mobility and the two parts are intact RCT and fixation of the tooth will be the solution with follow up period to insure that there is no root resorption happened at the fracture site, and if happened surgical intervention using root-end filling at the site of resorption will be the solution. b. If there is mobility and the two parts are separated and moved away, extraction will be the solution.
TCIIc	a. If there is no mobility and the two parts are intact RCT and fixation of the tooth will be the solution with follow up period to insure that there is no root resorption happened at the fracture site, and if happened surgical intervention to remove the apical part, and then retro-grade filling will be the solution. b. If there is mobility and the two parts are separated and moved away, surgical removal of the apical part and retro-grade filling after RCT will be the must.
TCIII	In this case almost the vertical fracture passes through the long area of the root which will complex the treatment and prognosis, so extraction could be mandatory.

Part II: THE Diseased TSL [D]

Introduction

Dental clinicians can be faced with difficult diagnostic and treatment decisions with respect to the diseased tooth loss.

Tooth loss in the primary and permanent dentition has been extensively studied and the complex processes involved in the removal of the organic and inorganic components of tooth structure by clastic cells continue to evolve through basic research.

Knowledge gained from experimental studies and observations of histo-pathological material has provided a sound basis for the diagnosis and treatment of many diseased tooth loss processes.

The new classification with etiologic factors: D

(DA) Congenital & Syndromes	(DB) Systemic, Cellular & Environmental disorders	(DC) Bacterial Invasion
I: Ectodermal Dysplasia: Hypodontia, Oligodontia & Microdontia (peg lateral incisor) [DAI]: • Mutation of Pax 9 gene which maps to chromosome #4. • The best known of the missing teeth syndromes is X-Linked hypohidrotic Ectodermal dysplasia.	**I: Erosion** [DBI]: * Dental erosion is defined as the progressive, irreversible loss of hard dental tissues due to a chemical process not involving bacteria. * It is a condition of growing concern in the dental profession as it causes irreversible damage to the dentition in all ages of the population. * It is often difficult to compare the outcomes of different epidemiological studies on dental erosion due to the use of different examination standards. a- Dietary Erosion[DBIa]: Acidic foods & drinks b- Environmental Erosion[DBIb]: Airborne proteolitic enzymes & high levels of hydrochloric acid c- Medications & Xerostomia [DBIc]: can be another cause of possible erosive conditions. d- Regurgitation Erosion[DBId]: Involuntary or voluntary * Involuntary regurgitation: or gaestroesophageal reflux can occur due to hiatus hernia or as a consequence of pregnancy or chronic alcoholism. * Voluntary regurgitation: is usually associated with an underlying psychological problem. Eating disorders commonly associated are anorexia nervosa and bulimia nervosa. The effect of acid regurgitation in bulimic patients often exhibits perimolysis - erosive lesions localized to the palatal aspect of maxillary teeth	**Caries** [DC]: Tooth decay is caused by certain types of acid-producing bacteria which cause damage in the presence of fermentable carbohydrates such as sucrose, fructose, and glucose. The resulting acidic levels in the mouth affect teeth because a tooth's special mineral content causes it to be sensitive to low pH. a- Pits & Fissures [DCa]: For all types of pits and fissures, the deep enfolding of enamel makes oral hygiene along these surfaces difficult, allowing dental caries to be common in these areas 1- Occlusal [DCa1] 2- Buccal & Lingual [DCa2] 3- Combined [DCa3] b- Smooth surfaces [DCb]: 1- Interproximal [DCb1] 2- Cervical [DCb2] 3- Incisal & Cuspal [DCb3] 4- Buccal & Lingual [DCb4] 5- Root (exposed) [DCb5] However, as caries can be a progressive disease, it is desirable to be able to define the size and extent of the lesion. It is possible then to define five separate sizes as the lesion progresses: Size **0** [DC--S0]: the earliest lesion that can be identified as the initial stage of demineralization. Size **1** [DC--S1]: minimal surface cavitation with involvement of dentine just beyond treatment by remineralization alone. Size **2** [DC--S2]: moderate involvement of dentine. Following cavity preparation remaining enamel is sound, well supported by dentine and not likely to fail under normal occlusal load. Size **3** [DC--S3]: the lesion is enlarged beyond moderate. Remaining tooth structure is weakened to the extent that cusps or incisal edges are split, or are likely to fail if left exposed to occlusal load. Size **4** [DC--S4]: extensive caries or bulk loss of tooth structure e.g. loss of a complete cusp or incisal edge, has already occurred.
II: Macrodontia & Dense in Dente [DAII]: a- Macrodontia is a molarization of bicuspid leading to its uneruption. [DAIIa] b- Dense in dente is a tooth within a tooth by invagination of the cingulum which resulted in enamel being reflected into the tooth. [DAIIb]	**II: Abnormal Attrition &** **Abrasion [DBII]:** a- Dietary Attrition [DBIIa]: This type of tooth wear can be significant in patients with "primitive diets e.g.the aboriginal population - high quantity of dietary abrasives b- Restorative Attrition [DBIIb]: the use of porcelain can accelerate tooth wear, especially if this porcelain is unglazed and rough/unpolished c- Drug Attrition [DBIIc]: can be another cause of bruxism and has an effect on attrition d- Attrition due to loss of posterior support [DBIId]: It has been suggested that there is an increase in force per unit area in the remaining dentition, thereby causing an increase in tooth wear. **e-** Habitual Abrasion [DBIIe]: Rubbing of pipes, hairclips, musical instrument mouthpieces, excessive tooth picking, etc, could cause this. The most common cause is incorrect or over-vigorous tooth brushing	
III: Screw driver incisors & Mulberry molars [DAIII]: Seen in congenital syphilis and caused by direct invasion of tooth germs by Treponema organisms.	**III: Cellular changes (inflammatory) [DBIII]:** The response of the dento-alveolar apparatus to infection is characterized by inflammation which may result in cellular changes leading to tooth resorption. Osteoclasts (dentinoclasts?) arising in the dental pulp inexorably resorb dentin and enamel a) Internal resorption [DBIIIa] 1- Apical [DBIIIa1]: more common in teeth with various inflammatory periapical pathosis. 2- Intraradicular [DBIIIa2]: fully contained within an otherwise intact root. Common finding is a large accessory canal communicating from the periodontal ligament to the resorbed area; this may have allowed the passage of a collateral blood supply which probably played an important role in the development of the internal resorptive process. **b)** External resorption [DBIIIb]: A prerequisite for external inflammatory root resorption is damage to the normally protective cementum which then initiates surface resorption exposing the underlying dentine to the passage of bacteria or their metabolites from the root canal to the external root surface. Although the cause may be idiopathic, in some cases the cause is apparent (keratocyst, tumor, and ossifying fibroma). A normal inflammatory response ensues including the activation of clastic cells which results in resorption of both tooth and bone. As the inflammatory response is chronic in nature it is generally asymptomatic unless the infection becomes acute, in which case the tooth will become tender to touch and there may be development of an overlying swelling. **c)** Internal-External resorption [DBIIIc]: Where resorption has extended from an internal inflammatory resorption to involve the external surface a communicating lesion is created. d) Invasive cervical resorption [DBIIId]: **1**- Invasive coronal [DBIIId1] 2- Invasive radicular [DBIIId2] 3- Hyper-plastic invasive resorption [DBIIId3] : [proliferative fibro-vascular or fibro-osseous disorder in which micro-organisms become secondary invaders]. There is a potential predisposing factor. All types have a clinical classification classes: Class 1: a small invasive resorptive lesion near the cervical area with shallow penetration into dentin. [DBIIId3C1] Class 2: A well defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but show little or no extension into the radicular dentin. [DBIIId3C2] Class 3: A deeper invasion of dentin by resorbing tissue, not only involving the coronal dentin but also extending into the coronal third of the root. [DBIIId3C3] Class 4: A large invasive resorptive process that has extended beyond the coronal third of the root. [DBIIId3C4]	
IV: Dentinogenesis Imperfecta (DI) [DAIV]: * Gene maps to chromosome #4. * It encodes a protein called dentin sialophosfoprotein which constitutes about 50% of the noncollagenous component of dentin matrix. * Enamel is easily broken leading to exposure of dentin that undergoes accelerated attrition. Type **a-** With Osteogenesis Imperfecta [DAIVa] Type **b-** Without Osteogenesis Imperfecta [DAIVb] Type **c-** Shell teeth with little dentine (rare) [DAIVc] • Types a & b radiographically, the teeth appear solid, but lacking pulp space. • Type c (more rare) paradoxically characterized by too little rather than too much dentin resulting in shell teeth. • It is a different mutation in the same gene.	IV: Environmental Effects on Tooth Structure Development [DBIV]: a- Enamel Hypoplasia: associated with exanthematous fevers [DBIVa]: * If occur during the first two years of life; horizontal rows of pits or diminished enamel on anterior teeth and first molars; enamel loss is bilateral. * Similar pattern in cuspids, bicuspids, and second molars when the inciting event occurs at age 4-5. b- Turner's Hypoplasia: [DBIVb] * Secondary to periapical inflammatory disease of the overlying deciduous tooth. * Enamel defects vary from focal areas of white, yellow or brown to extensive hypoplasia involving the entire crown. * Most frequently affects permanent bicuspids. c- Antineoplastic Therapy Hypoplasia [DBIVc]: * Degree and severity related to age, form of therapy (chemotherapy/radiotherapy) and dose. * Radiotherapy effects more severe than chemotherapy alone but sometimes used together. * Defects include radicular hypoplasia, enamel hypoplasia and discolorations.	
V: Amelogenesis Imperfecta(AI) [DAV]: • Rare as 1:14,000 • At least 14 phenotypes have been identified and autosomal dominant, recessive & X-Linked inheritance have been reported. • The matrix of enamel is comprised mainly of a protein called amelogenin. • The gene for this protein is on the short arm of the X chromosome (Xp22.1). • Autosomal dominant AI has been traced to a gene on chromosome #4 near the site as the gene for DI and dental dysplasia. Type **a-** Hypo-plastic type: Inadequate formation of enamel matrix, both pitting and smooth types exist. Enamel may be reduced in quantity but is of normal hardness. Tooth loss occurs in this type by attrition of thin enamel surface. [DAVa] Type **b-** Hypo-calcification type: A defect not in the quantity of enamel but in the quality of the enamel which is poorly mineralized, soft & chips or wears easily. [DAVb]		
VI: Short Tooth Syndrome [DAVI]: **a-** Altered eruption [DAVIa]: In altered eruption, one finds the gingival margin located excessively incisal to the CEJ, covering a portion of the anatomic crown with soft tissue **1**- Active: is reflective of subcategory B, where the osseous crest is at the CEJ **2**- Passive: is indicative of subcategory A, where the osseous crest is *apical* to the CEJ (physiologic normal) **b-** Compensatory eruption: Excessive incisal attrition [DAVIb]: Tooth structure loss is physiologic and occurs as a natural consequence of aging and wear. However, if excessive generalized tooth loss affecting occlusal surfaces of the teeth has occurred, then it is highly likely that a reduction in occlusal face height VDO or an increase in freeway space (FWS) has occurred. This diagnosis may be convoluted by forward posturing of the mandible. **c-** Delayed maxillary incisors eruption: Excessive eruption of mandibular incisors [DAVIc]: It is not uncommon that the primary maxillary incisors (A's and B's) can be lost at any early age prior to the final development and eruption of the permanent teeth. The delayed eruption of the maxillary anterior dentition frequently allows the mandibular incisors to over erupt thereby creating an unfavorable esthetic tooth proportion of the anterior teeth. The resultant occlusion tends to be unfavorable as well since a Class III maxillomandibular relationship frequently results as the centric occlusion scheme as a consequence of inadequate interocclusal space. The lack of length of the maxillary incisors give the false pretense that there has been a loss or decrease of vertical dimension of occlusion. **d-** Vertical maxillary excess [DAVId]: This gummy smile frequently results from a skeletal dysplasia, specifically the hyperplastic growth of the maxillary skeletal base.		

Examination and Diagnosis of Diseased TSL

An initial comprehensive examination is performed, including a thorough medical and dental history and an orofacial and dental clinical examination. Radiographs and other special tests may then be carried out. Such tests may include saliva tests, fracture finder, pulpal sensibility testing, illumination testing, etc.

Questions regarding lifestyle, medications, stress, brushing habits, etc. can help in aiding diagnosis. Saliva testing may be appropriate; a food diary may also be required. From clinically observed features and habits and careful collation of all this information determination of the risk factors is helpful to minimize long term damage of teeth.

Diagnosis involves all findings to identify the factor(s) contributing to tooth structure loss. This is to preserve the remaining dentition and to improve the long term prognosis of any restorative treatment completed. Diagnosis needs to also be made as to whether the wear is physiological or pathologic? If wear has produced an unsatisfactory appearance, sensitivity, reduction in facial height and vertical dimension of occlusion then tooth wear is considered pathologic and this may constitute the need for treatment. A period of monitoring may be required to decide on appropriate management.

This monitoring may be carried out by:

- Photographic records;
- Measurements of teeth;
- Study model comparison;
- Tooth wear index;
- Impression of splint and comparison of changes over 3 months;
- Indices

Ectodermal Dysplasia: Hypodontia, Oligodontia and Microdontia [DAI].

Hypodontia: having less than 6 congenitally missing teeth. (partial anodontia).

Oligodontia: having 6 or more congenitally missing teeth.

Diagnosis is done by clinical examination and panoramic x-ray for the patient, to differentiate if the case is kind of missing teeth or the teeth are impacted due to other reasons.

An example of a cone-shaped lateral incisor, a peg lateral, is a form of 'Microdontia'. This may be inherited as a dominant trait. Diffuse microdontia occurs in some hereditary disorders and sometimes associated with hypodontia. Increased in Down's, pituitary dwarfism and a few other syndromes. If both parents have "peg laterals", the homozygous child will have total anodontia of succedaneous teeth.

Examples are shown in figures.

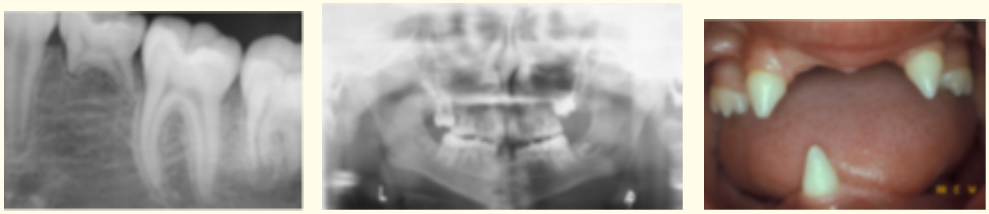
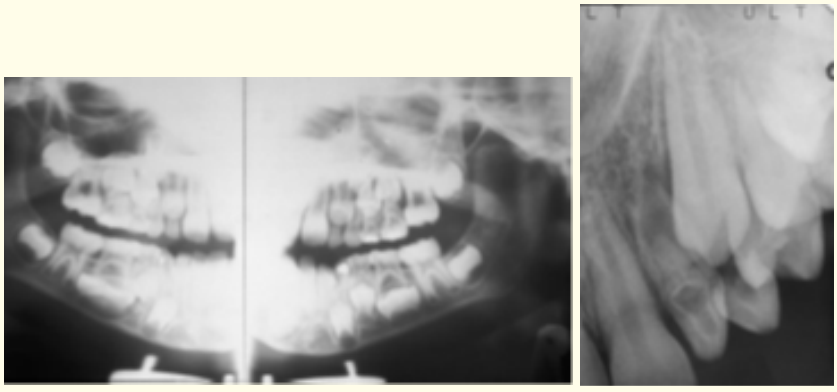


Figure 1

Macrodontia and Dense in Dente [DAII]

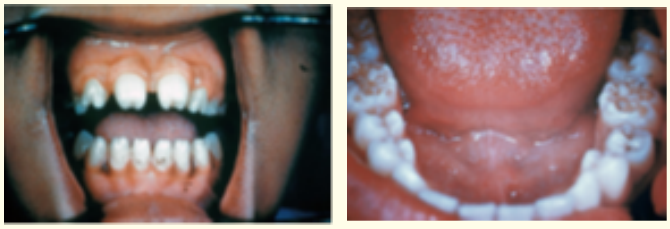
Diagnosis is carried out through clinical and x-ray examinations. Examples are shown in figures.



Macrodontia *Dense in Dente (Figure 2)*

Screw driver incisors and Mulberry molars [DAIII]

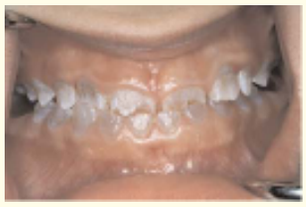
Diagnosis is done by clinical examination, and the appearance of the incisors and molars are shown in figures.

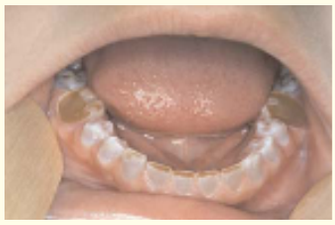


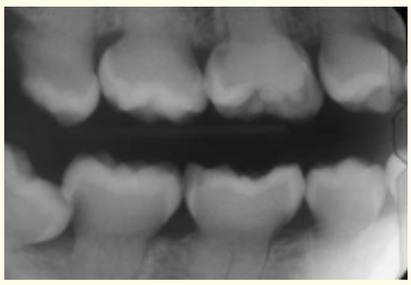
Screw driver incisors *Mulberry molars (Figure 3)*

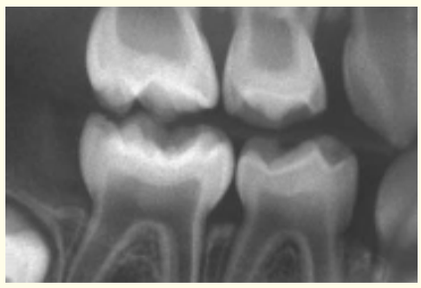
Dentinogenesis Imperfecta (DI) [DAIV]

This is an autosomal dominant condition affecting both deciduous and permanent teeth. Affected teeth are gray to yellow-brown and have broad crowns with constriction of the cervical area resulting in a "tulip" shape. Radiographically, the teeth appear solid, lacking pulp chambers and root canals. Enamel is easily broken leading to exposure of dentin that undergoes accelerated attrition (types IandII). DI type III is even rarer and paradoxically characterized by too little rather than too much dentin resulting in "shell teeth". Type III DI may be and allelic variant of type II DI.





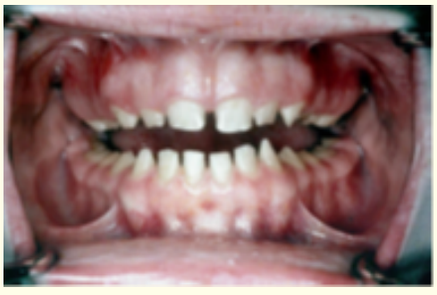


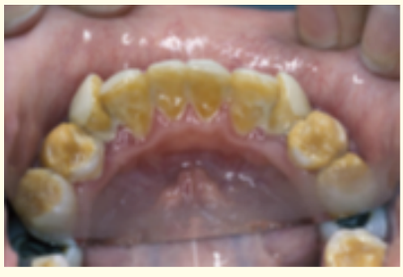


DI types I & II *DI type III (Figure 4)*

Amelogenesis Imperfecta (AI) [DAV]

The appearance depends on the type of AI, varying from the mild hypomature 'snow-capped' enamel to the more severe hereditary hypoplasia with thin, hard enamel which has a yellow-brown appearance. Dental problems, which depend on the severity of the condition, include sensitive teeth and poor appearance due to tooth loss and staining. If tooth tissue loss is severe there is vertical loss resulting in reduced masticatory function and poor appearance. Pulpal involvement may occur in severe cases, and because the roughness makes cleaning more difficult, gingivitis and periodontitis may develop. Examples are shown in figures.



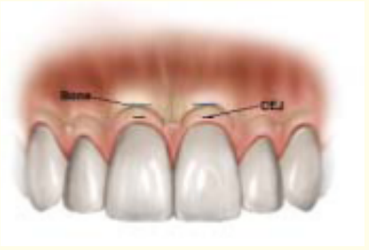


Hypoplastic amelogenesis imperfecta *Hypocalcified amelogenesis imperfecta (Figure 5)*

Short Tooth Syndrome [DAVI]

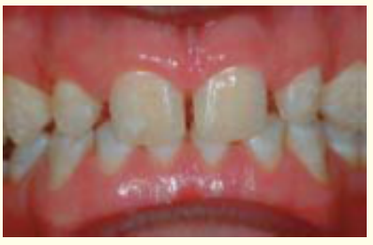
From clinical and x-ray examination, findings will differentiate between the different types of short tooth syndrome as follows:

1) Altered eruption [DAVIa]:
    a. Altered Active Eruption:
        i. Gingival margin located incisal to the CEJ.
        ii. Osseous crest failed to resorb to a level 2 mm apical to the CEJ.
    b. Altered Passive Eruption:
        i. Gingival margin located incisal to the CEJ.
        ii. Osseous crest located normally at a level 2 mm apical to the CEJ.
2) Compensatory eruption: Excessive incisal attrition [DAVIb]:
        i. Reduction in the facial height and increased free way space (generalized).
        ii. VDO unaffected and free way space constant (localized).
3) Delayed maxillary incisors eruption: Excessive eruption of mandibular incisors [DAVIc]:
        i. Class III maxillo-mandibular relationship.
        ii. Over-erupted mandibular incisors.
        iii. Short maxillary incisors.
4) d- Vertical maxillary excess [DAVId]:
    a. Teeth positioned farther away from skeletal base.
    b. Excessive gingival display.

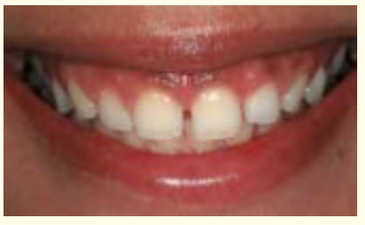


Diagrammatic representation of the normal physiologic position of the: Osseous-crest, free gingival margin, and clinical crown exposure relative to the CEJ. (Figure 6)

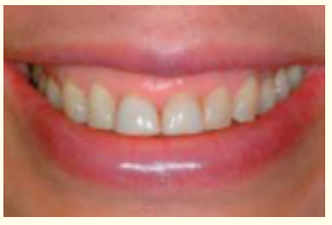
Figures showing different types of short tooth syndrome



Active Altered

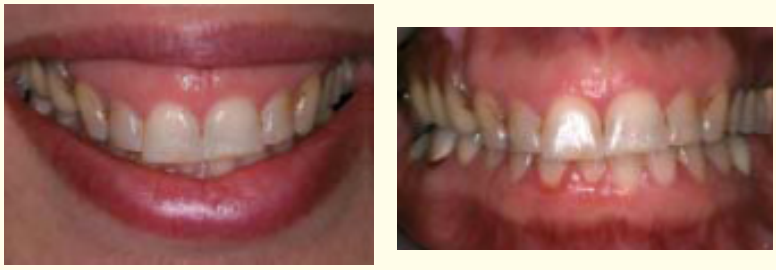


Passive Altered



Compensatory eruption (Figure 7)

Diagrammatic representation of delayed eruption



Vertical maxillary Excess (Figure 8)

Erosion [DBI]

At the present time, dentists commonly estimate tooth wear by comparing sequential study casts taken over long periods of time. This method satisfies most clinical needs in deciding whether the patient requires restoration and prevention counseling. However, more accurate methods are useful if the dentists would like to detect tooth erosion earlier and begin preventive treatment immediately, such as ultrasound, profilometry, and quantitative light-induced fluorescence have been suggested as potential tools for diagnosing dental erosion more accurately. An important consideration for the diagnosis of dental erosion is whether expensive technological devices are truly necessary for diagnosis and whether they are worth the cost from the perspective of the patients and the dentists. If a trained clinician can detect the loss of tooth structure in a pattern suggestive of acid wear, then this should be enough to know that the cause of the erosion must be identified and eliminated and the damage must be repaired. Therefore, better development of standard indices for visual diagnosis may be a more practical and cost efficient goal. Dental erosion is a multi-factorial condition with both extrinsic and intrinsic causes, consisting mainly of erosive acids. The most common extrinsic acids that can lead to erosion are dietary acids, such as fruit, fruit juices, carbonated drinks, and sports drinks. Behavioral factors can influence the impact of these dietary acids on the dentition. For instance, excessive consumption of acidic food or beverages, or unusual eating and drinking habits such as sipping an acidic drink over a long period of time, will increase the acid challenge to the teeth. The erosive effects of acids are exacerbated by decreased salivary gland function. Saliva is a significant factor in the prevention of dental erosion since it helps to directly neutralize and clear acids, as well as forming a protective coat over the teeth and promoting remineralization.

Other extrinsic causes can contribute to erosion including oral hygiene products and medications with a low pH, such as toothpastes, fluoride rinses, and vitamin C tablets. Environmental acids are also potential risk factors. People who work in battery factories are exposed to acid fumes and professional wine tasters sip low pH beverages for long periods of time; thus, these professions have been suggested to be high risk.

Intrinsic causes of dental erosion are gastric acids that are regurgitated into the mouth.

This is seen in patients with gastro-esophageal reflux disease (GERD) or with chronic excessive vomiting such as patients with anorexia, bulimia, alcoholism or gastrointestinal disorders.

The best diagnostic tools for erosion are the full medical and habitual history of the patient with thorough clinical examination. Some cases of erosive lesions are shown in figures:

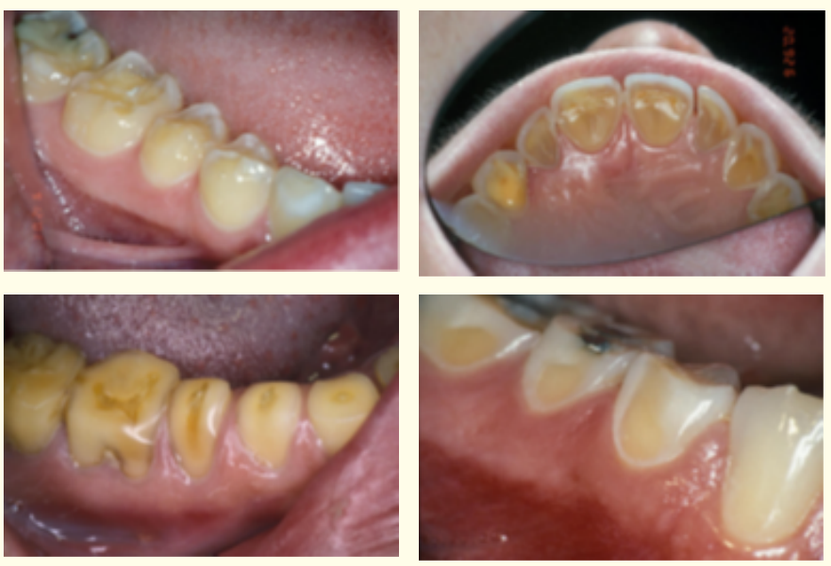
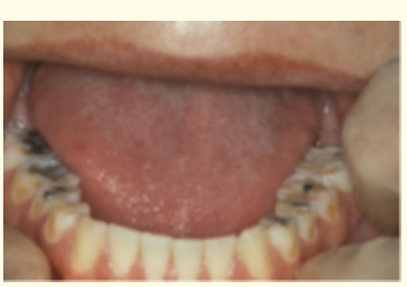


Figure 9

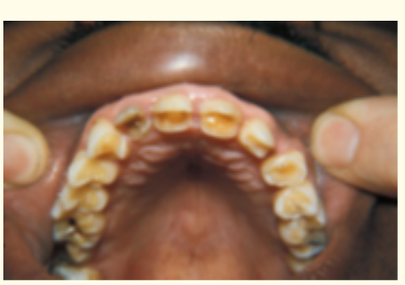
Abnormal Attrition and Abrasion [DBII]

Wear beyond normal caused by mechanical forces. This sounds like pathologic attrition but the difference is the pattern of wear. Attrition is ordinarily confined to the occlusal and incisal surfaces. Abrasion is ordinarily used when the loss is on a non-occluding surface. Case is shown in figures.

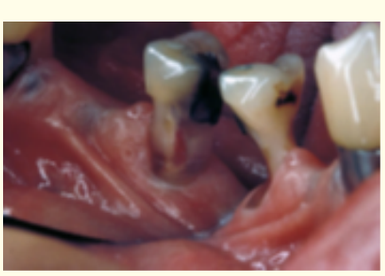
As the following figures will show, it is not always easy to distinguish between attrition, erosion and abrasion, they may coexist. Figures of a 21-year old man are shown, who had occlusal wear that had flattened the occlusal surfaces and loss of enamel on the buccal surfaces that cannot be explained by occlusion. Is the occlusal wear just an example of advanced attrition and the buccal lesions caused by erosion or abfraction? We could not identify a reason for erosion and he denied nocturnal bruxing or coarse diet that could account for the wear. Figure shows advanced wear on the occlusal and incisal surfaces presumably due to end to end occlusion coupled with erosion. We could not identify an erosive agent but there is an almost identical picture in your text that identifies it as erosion. Data collected from the patient about dietary and personal habits may be helpful to some extent in the diagnosis of the case.

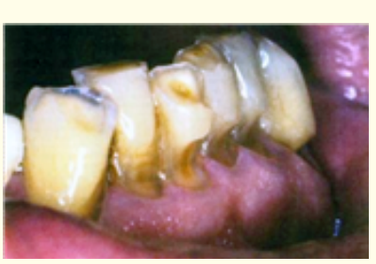


pathologic attrition on occlusal surface

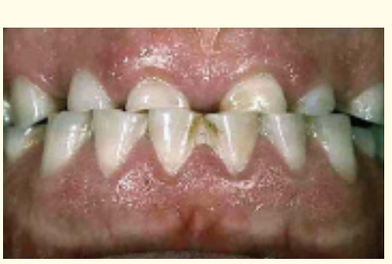


pathologic attrition or erosion or both?





Abrasion



Severe tooth attrition (Figure 10)

Cellular changes (inflammatory) [DBIII]

Past history, clinical examination and radiographic examination all are needed to diagnose the case of resorption which sometimes mimic caries or escape undiagnosed. Dental clinicians can be faced with difficult diagnostic and treatment decisions with respect to tooth resorption. When a patient presents with tooth resorption, the following basic questions must be addressed in arriving at a diagnosis and treatment plan:

(1) What type of resorption is present?

(2) Is the resorption external (Periodontally derived), internal (pulpally derived) or communicating?

(3) Will the resorptive process be self-limiting or transient and not require management other than careful monitoring of healing processes?

(4) If the resorptive process is progressive will there be a favorable response to treatment and, if so, what is the appropriate therapy?

(5) If treated what are the short and long-term prognoses?

(6) When is extraction and prosthetic therapy indicated?

One or many teeth may be involved and the cause is a total mystery. (*Skeletal bone has a counterpart in which a bone or adjacent bones mysteriously disappear, so-called vanishing bone disease or Gorham's syndrome).

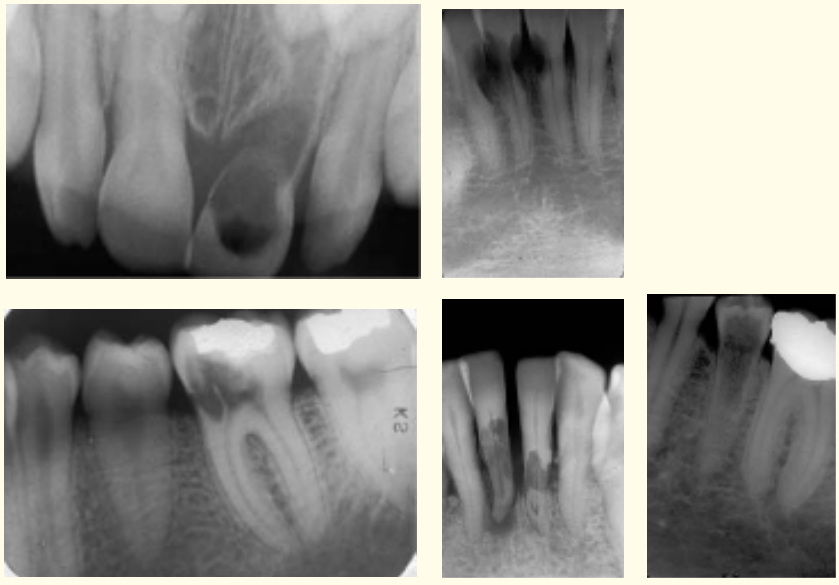


Figure 11

Radiographically, apical internal resorption may be difficult to diagnose when the resorption is of the lower grade, while intraradicular internal resorption can be recognized as round or oval-shaped radiolucencies contained within the tooth root.

Radiographically external inflammatory root resorption can be recognized by bowl-like radiolucencies in both the tooth root and the adjacent bone.

Communicating internal-external inflammatory resorption can be recognized radiographically by radiolucency within the tooth structure extending to the exterior surface and the surrounding bone.

The clinical presentation of invasive cervical resorption varies considerably depending on the extent of the resorptive process. The condition is usually painless and while a pink discoloration of the crown indicates the resorptive process, some teeth give no visual signs and diagnosis is usually the result of a routine radiological examination. Multiple resorptions can occur, particularly when there has been a history of orthodontic treatment and a full mouth radiographic examination should follow the identification of any tooth showing evidence of invasive cervical resorption. The radiographic appearance generally shows an irregular mottled or 'moth-eaten' image in the main lesion area.

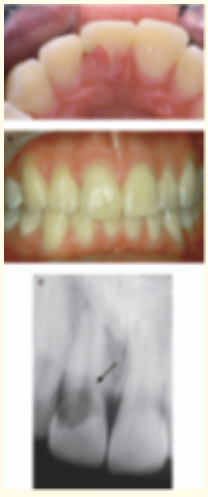


Figure 12

Environmental Effects on Tooth Structure Development [DBIV]

Enamel Hypoplasia: (due to systemic influences, such as exanthematous fevers): From clinical examination and the past medical history, the practitioner findings will be horizontal rows of pits or diminished enamel on anterior teeth and first molars; enamel loss is bilateral (if the fever occurred at the first 2 years of life), or the affected part is cuspids, bicuspids, and second molars (when the inciting event occurs at age 4 - 5).

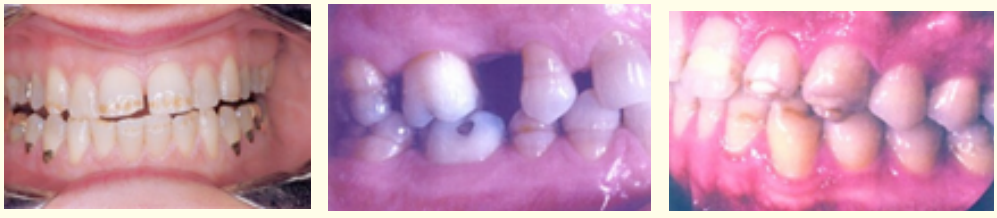


Figure 13

Turner's Hypoplasia: From clinical examination, x-ray of the affected part and the medical history of past deciduous tooth injury, findings will be enamel defects which vary from focal areas of white, yellow or brown to extensive hypoplasia involving the entire crown. Most frequently affects permanent bicuspids. Severe trauma early in tooth development can cause disorganization of the bud resembling a complex odontoma. Severe trauma later on can lead to partial or total arrest of root formation.

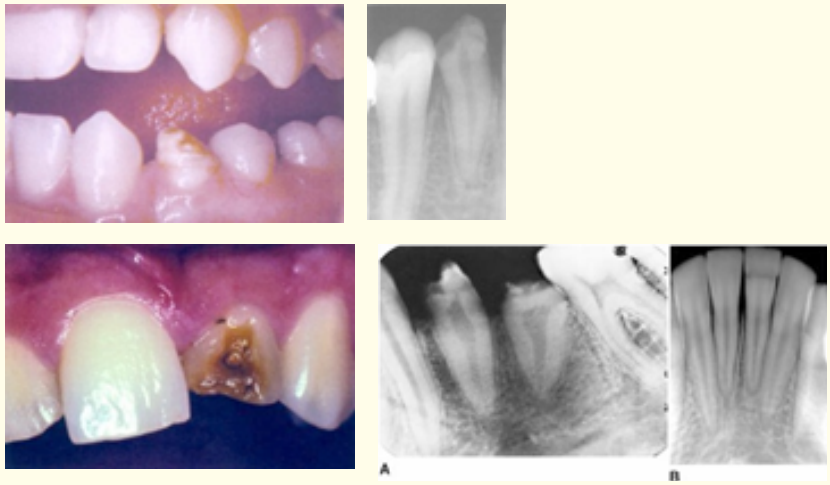


Figure 14

Antineoplastic Therapy Hypoplasia: From past medical history and by clinical and radiographic examination symptoms may include hypodontia, microdontia, radicular hypoplasia, enamel hypoplasia and discolorations.

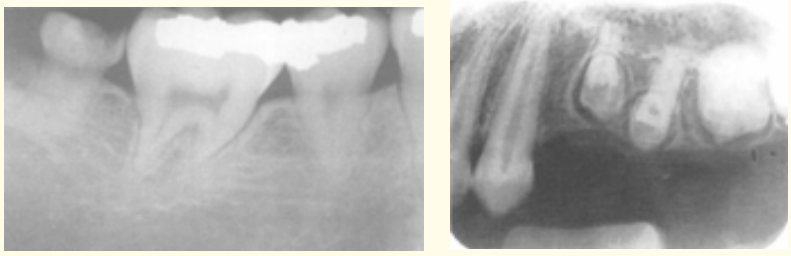
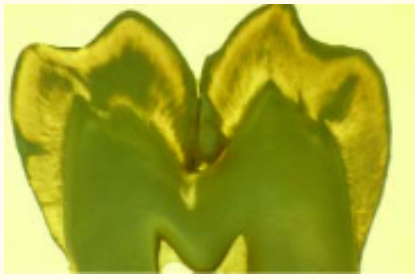


Figure 15

Caries [DC]

Primary diagnosis involves inspection of all visible tooth surfaces using a good light source (clinical examination). Dental radiographs may show dental caries before it is otherwise visible, particularly in the case of caries on interproximal (between the teeth) surfaces.

Large dental caries are often apparent to the naked eye, but smaller lesions can be difficult to identify. Unextensive dental caries was formerly found by searching for soft areas of tooth structure with a dental explorer. A common technique used for the diagnosis of early (uncavitated) caries is the use of air blown across the suspect surface, which removes moisture, changing the optical properties of the unmineralized enamel. This produces a white 'halo' effect detectable to the naked eye. Fiber-optic transillumination, lasers and disclosing dyes have been recommended for use as an adjunct when diagnosing smaller carious lesions in pits and fissures of teeth.



A photo-micrograph using transmitted light showing: The earliest signs of a caries lesion at the base of an occlusal fissure. (Figure 16)

Management and Treatment

DA) Congenital and Syndromes

Ectodermal Dysplasia: Hypodontia, Oligodontia and Microdontia [DAI]:

Hypodontia: often no treatment required for individual missing teeth as there is no space available; prosthetic replacement for multiple missing teeth.

Oligodontia: prosthetic replacement for multiple missing teeth either by fixed appliance for short distance or removable appliance for long distance.

Microdontia: Crowning of the affected tooth for esthetic reason is a must.

Macrodontia and Dense in Dente [DAII]

Macrodontia: In case with lose deciduous molar and enlarged bicuspid, extraction of the deciduous and surgical removal of the impacted tooth is a must before prosthetic replacement starts.

Dense in Dente: Restoring the defect caused by tooth invagination, using suitable restorative material must be done, even without carious lesion to avoid food stagnation in the defect which will lead to caries.

Screw driver incisors and Mulberry molars [DAIII]

Screw driver incisors: For esthetic reasons all the affected teeth must be crowned to give the normal appearance and to close the spacing between teeth.

Mulberry molars: If the occlusal defect is minimal, the treatment will be repairing these defects by fillings. If the defect occupies the whole occlusal surface, with cuspal destruction, fixed appliance will be a must.

Dentinogenesis Imperfecta (DI) [DAIV]

Mild cases with minimal attrition need to be restored for esthetic veneers, while sever cases with sever attrition need complete mouth rehabilitation, by fixed appliances, as the VDO is already lost and to prevent the food accumulation from causing caries and gingival problems.

Amelogenesis Imperfecta (AI) [DAV]

Treatment using fixed appliances must be done, starting from veneers through full coverage prosthetics, for esthetic and preventive measures.

Short Tooth Syndrome [DAVI]

(a) Altered eruption [DAVIa]:

1) Active: Treatment includes periodontal surgery with ostectomy.
2) Passive: Treatment depends on the amount of the attached gingiva and the position of the alveolar crest relative to the CEJ.
    * Gingivectomy
    * Flap surgery with or without ostectomy
    * Apical positioning of the flap

(b) Compensatory eruption: Excessive incisal attrition [DAVIb]:

* Increase VDO by restorations (generalized)
* Crown lengthening for some teeth (localized)

(c) Delayed maxillary incisors eruption [DAVIc]:

* Selective incisal reduction followed by crown lengthening, or orthodontic intrusion of the mandibular incisors.
* Orthodontic extrusion of the maxillary incisors, or prosthetic solution as fixed restorations.

(d) Vertical maxillary excess [DAVId]:

Treatment depends on the severity of the gingival display

- Orthodontics
- Periodontics
- Elective RCT
- Restorative therapy
- Orthognathic surgery

DB) Systemic, Cellular and Environmental disorders

Erosion [DBI]

The management of dental erosion consists of two essential components, prevention and therapy. The ultimate treatment for dental erosion is to prevent this irreversible damage from occurring in the first place. It is essential for dental professionals to have a strong understanding of the risk factors for erosion so that they can inform and educate their patients of how to avoid erosive damage. If dentists or hygienists perform a dietary analysis with patients who have a high risk of dental erosion or who are showing early signs, causative factors could be identified and the patients could be instructed as to how to modify their behaviors in order to preserve their dentition. Surprisingly, no studies were found regarding elimination of risk factors as a means of managing dental erosion.

The other important aspect of managing dental erosion is the restorative treatment of the condition. There are many unfavorable consequences to dental erosion, including sensitivity and compromised esthetics. Restoration of lesions can be effective in resolving many of the problems, but without eliminating the cause of the erosion, the destructive process will continue. Many studies suggest that glass ionomer is the most superior restorative material for these types of lesions. GI restorations undergo less stress and gap formation due to less polymerization shrinkage and thermal expansion/contraction. Use of a GI liner is thought to reduce micro-leakages by imparting some flexibility to the restoration.

Abnormal Attrition and Abrasion [DBII]

Causes of tooth surface loss must be understood to adopt appropriate preventive measures. Abrasive effects of aggressive tooth brushing can be reduced with education, but can be difficult to change especially with in-built memory. Patients must be informed of correct technique and to use a soft brush. Preference for abrasive dentifrice may need to be changed to a low abrasive one. Other abrasive habits can also be changed like pipe smoking, aggressive use of inter-dental sticks, etc.

Bruxism and attrition may be prevented with the use of occlusal splints and stress management. Occlusal adjustment and addition with restorations may also be required.

Monitoring of all preventive measures needs to be performed even if no restorative treatment is performed as to the effectiveness of the program to ensure long-term success and maintenance for patients suffering from tooth surface loss.

Replacement of lost posterior teeth and avoidance of edge-to-edge occlusion, is the only solution with cases of attrition due to loss of posterior support.

Cellular Changes (inflammatory) [DBIII]

Internal inflammatory resorption [DBIIIa]

1) **Apical [DBIIIa1]:** Endodontic treatment to level of resorption. Long-term calcium hydroxide dressing before placement of root filling.
2) **Intra-radicular [DBIIIa2]:** Endodontic treatment and root canal filling (hot GP technique, Obtura etc).

External inflammatory resorption [DBIIIb]

Endodontic treatment and intra-canal medication with either Ledermix paste followed by long-term calcium hydroxide or calcium hydroxide alone will be done first. Fill root canals when resorption is controlled.

Prevention following replantation of mature tooth; pulp extirpation and Ledermix paste dressing is done as soon as possible.

Communicating internal external resorption [DBIIIc]

Endodontic treatment must be done to resorptive defect. Induce calcification by use of calcium hydroxide alone or following careful topical application of 90% trichloroacetic acid. ProRoot MTA may also be used.

Invasive cervical resorption [DBIIId]

Class 1, 2: Treatment could be topical application of 90% trichloroacetic acid, curettage, and glass ionomer cement restoration.
Class 3: Treatment could require topical application of 90% trichloroacetic acid to resorptive tissue, curettage, elective pulpectomy and canal preparation to gain access to deeper and encircling infiltrative channels. Ledermix paste intra-canal dressing, followed by root filling and final glass ionomer cement restoration. Adjunctive orthodontic extrusion is necessary. Alternative therapy is periodontal flap reflection, curettage, TCA application to the defect, endodontic therapy and restoration.
Class 4: Leave untreated and monitor or extract and implant.

Environmental Effects on Tooth Structure Development [DBIV]

Enamel Hypoplasia [DBIVa]: Treatment depends on the severity of the case. Mild cases could be restored by esthetic fillings as reinforced GI or composite resin restorations, while severe cases could be restored by either veneers or complete coverage restorations depending on the age of the patient.

Turner's Hypoplasia [DBIVb]: Treatment depends on the severity of the tooth structure lost. The mild and moderate tooth structure loss requires restorative treatment with restoration and may be coverage of the tooth, while the severe cases require RCT first then post and core build up, after which full coverage restorations are must.

Antineoplastic Therapy Hypoplasia [DBIVc]: Treatment depends on the symptoms of the case. Cases with hypodontia will be treated as cases of hypodontia, cases with microdontia will be treated as cases of microdontia, cases with hypoplasia will be treated as hypoplastic cases.

Caries [DC]: Many of the old limitations are no longer applied, and it is now appropriate to think again about the problems presented by a carious lesion. Without in any way denigrating the achievements due to Black's concepts and work, the following thoughts are offered and a new approach to the definition of cavity design is outlined. The proposed classification is designed for the identification of lesions from the very earliest stage of demineralization and to define their increasing complexity as the lesion extends. It is expected to provide benefits for both the profession and their patients.

However, as caries can be a progressive disease, it is desirable to be able to define the size and extent of the lesion at the time of identification and, therefore, the potential complexity of the restorative procedures required for treatment.

Size 0: This needs to be recorded but will be treated by eliminating the cause and should therefore not require further treatment.

Size 1: Some form of restoration is required to restore the tooth surface affected and prevent more food accumulation at the site.

Size 2: Removal of the carious lesion by conservative cavity preparation, as the remaining tooth structure is sufficiently strong to support the restoration in place against loads.

Size 3: The cavity needs to be further enlarged so that the restoration can be designed to provide support to the remaining tooth structure.

Size 4: This more extensive carious lesion needs larger cavity preparation filled with a restoration having high fracture resistance, high flexure strength as well as high abrasion resistance. Some cases need a complete coverage restoration to withstand the occlusal forces [1-22].

Bibliography

1. Addy M and Shellis RP. "Interaction between attrition, abrasion and erosion in tooth wear". *Monographs in Oral Science* 20 (2006): 17-31.

2. "Advanced Oral Pathology PowerPoint Seminar: Abnormalities of The Teeth". Oral Pathology Department, Faculty of Oral and Dental Medicine, Cairo University (2009).

3. Bakland LK. "Endodontic Considerations in Dental Trauma". Bakland Endodontics, Chapter 15: 796-843.

4. Barbour ME and Rees GD. "The role of erosion, abrasion and attrition in tooth wear". *Journal of Clinical Dentistry* 17.4 (2006): 88-93.

5. Bastone EB., *et al*. "Epidemiology of Dental Trauma: A review of the literature". *Australian Dental Journal* 45.1 (2000): 2-9.

6. Chu SJ., *et al*. "Short Tooth Syndrome: Diagnosis, Etiology and Treatment Management". *Journal of the California Dental Association* 32.2 (2004): 143-152.

7. Ck Ho C. "Non-Carious Tooth Surface Loss: A look at the causes, diagnosis and prevention of wear". *Australasian Dental Practice* (2007): 184-190.

8. Clark DJ., *et al*. "Definitive Diagnosis of Early Enamel and Dentinal Cracks". *Journal of Esthetic and Restorative Dentistry* 15.1 (2003): 391- 401.

9. Clarck DJ. "The Epidemic of Cracked and Fracturing Teeth". *Dentistry Today* 26.5 (2007): 90-95.

10. Dunlap C. "Abnormalities of Teeth". Mosgoogle Center (2004).

11. Ellis RG and Davey KW. "The Classification and Treatment of Injuries to the Teeth of Children (5th Edition)". Year Book Medical Publisher Inc. Chicago (1970).

12. Ellis SGS., *et al*. "Predisposing and Contributing Factors for Complete and Incomplete Tooth Fractures". *Dental Update* 26.4 (1999): 150-158.

13. Ellis SGS., *et al*. "Influence of Patient Age on the Nature of Tooth Fracture". *Journal of Prosthetic Dentistry* 82.2 (1999): 226-230.

14. Ellis SGS. "Incomplete Tooth Fracture – Proposal for New Definition". *British Dental Journal* 190.8 (2001): 424-428.

15. Fatimani K., *et al*. "Diagnosis, Risk Factors and Management of Dental Erosion: An Evidence Based Report". Faculty of Dentistry, University of Toronto (2009).

16. Fuss Z., *et al*. "Root resorption – Diagnosis, classification and treatment choices based on stimulation factors". *Dental Traumatology* 19.4 (2003): 175-182.

17. Heithersay GS. "Invasive cervical resorption". *Endodontic Topics* 7.1 (2004): 73-92.

18. Hunt KH. "Abfraction Lesions: A Bioesthetic Approach to Improve Function and Appearance". Continuing Education GlaxoSmithKline Inside Dentistry (2006): 22-27.

19. Mount GJ and Hume WR. "Classification and Cavity Preparation for Caries Lesions". Preservation and Restoration of Tooth Structure; Chapter 14: 243-249.

20. Salvi V. Cracked Tooth Syndrome.

21. Tai Ho EH. "Amelogenesis Imperfecta: A case report". *Hong Kong Dental Journal* 3 (2006): 123-127.

22. Westergaard J., *et al*. "Occupational exposure to airborne proteolitic enzymes and lifestyle risk factors for dental erosion – a cross-sectional study". *Occupational Medicine* 51.3 (2001): 189-197.

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