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.

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

Etiological Factors

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:

I: Incisal notch/Occlusal chipping or facets (TAI):
   a. Enamel only (TAIa)
   b. Enamel & Dentin (TAIb)
   c. Enamel, Dentin & involving the pulp (TAIc)

II: 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)

III: 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)

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)

V: Vertical Fracture (TCV) (usually in posterior teeth):
   a. Cervical (TCVa)
   b. Middle (TCVb)
   c. Apical (TCVc)

May be caused by teeth clenching or being struck with the jaw closed. Most commonly happened to molars during RCT.
Tooth parts affected by each event are as follows:

<table>
<thead>
<tr>
<th>T.S.L. class T</th>
<th>Enamel</th>
<th>Dentin</th>
<th>Pulp</th>
<th>Cementum</th>
<th>Periodontal apparatus</th>
<th>Alveolar Bone</th>
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<tbody>
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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:

<table>
<thead>
<tr>
<th>Classes</th>
<th>Treatment Plan</th>
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</thead>
<tbody>
<tr>
<td>TAIIa</td>
<td>Simple filling (composite or amalgam)</td>
</tr>
<tr>
<td>TAIIb</td>
<td>Filling for destructed sides (composite or amalgam)</td>
</tr>
<tr>
<td>TAIIc</td>
<td>RCT, post and core will be used for reconstruction of tooth and then crowning.</td>
</tr>
<tr>
<td>TAIa</td>
<td>Simple filling (composite or amalgam)</td>
</tr>
<tr>
<td>TAIb</td>
<td>Fillings (composite or amalgam)</td>
</tr>
<tr>
<td>TAIc</td>
<td>To be sealed by resins.</td>
</tr>
<tr>
<td>TAIai</td>
<td>To be left under observation or sealed with resins.</td>
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<tr>
<td>TAIaii</td>
<td>To be sealed by resins.</td>
</tr>
<tr>
<td>TBIIa</td>
<td>Simple filling (composite or amalgam).</td>
</tr>
<tr>
<td>TBIIb</td>
<td>RCT, post and core will be used for reconstruction of tooth, followed by crown lengthening and then crowning.</td>
</tr>
<tr>
<td>TBIIc</td>
<td>RCT, then filling (composite or amalgam), followed by crowning.</td>
</tr>
<tr>
<td>TBIIi</td>
<td>RCT, then filling (composite or amalgam), followed by crowning.</td>
</tr>
<tr>
<td>TBIIai</td>
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</tr>
<tr>
<td>TBIIaii</td>
<td>RCT, post core will be used for reconstruction of tooth, followed by crown lengthening and then crowning.</td>
</tr>
<tr>
<td>TBIIbi</td>
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</tr>
<tr>
<td>TBIIIa</td>
<td>Restored by filling.</td>
</tr>
<tr>
<td>TBIIIb</td>
<td>Restored by filling.</td>
</tr>
<tr>
<td>TBIIIc</td>
<td>Restored by filling, periodontal intervention (treatment or crown lengthening).</td>
</tr>
<tr>
<td>TBIVa</td>
<td>Filling (composite or amalgam)</td>
</tr>
<tr>
<td>TBIVb</td>
<td>RCT, post and core will be used for reconstruction of tooth and then crowning.</td>
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</tr>
</tbody>
</table>

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 diag-

The new classification with etiologic factors: D

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Citation:

Treponema organisms.

Seen in congenital syphilis and caused by direct invasion of tooth germs by

b- Dense in dente is a tooth within a tooth by invagination of the cingulum which

a- Macrodontia is a molarization of bicuspid leading to its uneruption. [DAIIa]

II:

1 the CEJ, covering a portion of the anatomic crown with soft tissue

[DAVI]:

I

poorly mineralized, soft & chips or wears easily. [DAVb]

A defect not in the quantity of enamel but in the quality of the enamel which is

b - Hypo-calcification type: Enamel may be reduced in quantity but is of normal hardness. Tooth loss occurs

- Hypo-plastic type: a

- Shell teeth with little dentine (rare) [DAIVc]

- Abrasion: uncontrolled attrition of tooth structure by the physical action of opposing tooth surfaces which

- Erosion: the chemical loss of enamel or dentin by acids from diet, juices, beverages, or regurgitation.

- Acid erosion: the chemical loss of tooth structure by both acid and mechanical factors: 2

- Effects of fluoride may vary from tooth to tooth, and within a tooth from cervix to incisal edge.

- The reduction of tooth structure may be caused by direct or indirect trauma of various etiologies.

- Abrasion: mechanical wear of tooth structure by repeated frictional action.

- Erosion: chemical loss of tooth structure by acids.

- Hypocalcification:

- Hypoplasia: loss of enamel from imperfect mineralization.

- Tetracycline staining: permanent yellow or green discoloration of tooth structure from systemic ingestion of tetracycline.

- Dyes and stains: the use of stains, dyes and chemical agents to improve esthetics.

- Acidulated phosphate fluoride (APF) varnish: a material containing high concentrations of fluoride in a hydrocolloid vehicle.

- Silver-based filling materials: filling materials based on silver-tin or silver-mercury.

- Glass ionomer cements: filling materials which contain glass or glass-flour in a water-based vehicle.

- Bonding materials: materials that bond to tooth structure.

- Composite resins: a tooth-colored filling material which is a mixture of inorganic fillers and a thermosensitive polymer matrix.

- Amalgam: a filling material composed of various percentages of silver, tin, copper, antimony, and mercury in a lead-tin alloy.

- Composite resins, amalgams, and bond systems are extensively used in modern dentistry.

- Composite resins: tooth-colored filling materials that bond to tooth structure.

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- Vertical maxillary excess [DAVId]:

- At least 2 mm of the upper incisors are labially positioned.

- Degree and severity related to age, form of therapy (chemotherapy/radiotherapy) and dose.

- Class 4: A large invasive resorptive process that has extended beyond the coronal third of the root. [DBIIId3C4]

- All types have a clinical classification classes: Class 1: a small invasive resorptive lesion near the cervical area with shallow penetration into dentin. [DBIIId3C1]

- d) Invasive cervical resorption [DBIIId]:

- resorption [DBIIIc]: Where resorption has extended from an internal inflammatory resorption to involve the external surface a communicating lesion is created.

- a) Internal resorption [DBIIIa]

- 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

- b) External resorption [DBIIIb]: The result of a non-inflammatory process and often associated with use of a tooth as an implant.

- a) Internal resorption [DBIIIa]

- The response of the dento-alveolar apparatus to infection is characterized by inflammation which may result in cellular changes leading to tooth resorption. Osteoclasts (denti

- d) Invasive cervical resorption [DBIIId]:

- resorption [DBIIId]: [proliferative fibro-vascular or fibro-osseous disorder in which micro-organisms become secondary invaders]. There is a potential predisposing factor.

- Microorganisms present in the root canal can pass through the periodontal ligament and into the external root surface. Although the cause may be idiopathic, in some cases the cause is apparent (keratocyst, dento-alveolar abscess).

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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.

Macrodontia and Dense in Dente [DAII]
Diagnosis is carried out through clinical and x-ray examinations. Examples are shown in figures.

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.

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 I and II). 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 an allelic variant of type II DI.

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.

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:

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.

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).

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.

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, bicuspid, and second molars (when the inciting event occurs at age 4 - 5).
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.

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

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 severe cases with severe 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 [DBIIIId]**

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.

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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**


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