ABC Classification & Treatment: The New Collected Classification of Tooth Structure Loss, Management and Treatment

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Abstract

As there are many old classifications for differently separated items of tooth structure loss, so dental clinicians can be faced with difficult diagnostic and treatment decisions with respect to the non-diseased and diseased tooth loss.

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

Comparing and accumulating data from different studies is extremely difficult due to the differences in the definitions and classifications used. With all these findings and observations we still have too many partial classifications, which could be confusable to clinicians or at least difficult to collect together in mind to be an aid for him during examination of the case.

For an aid to clinicians, these classifications and information about diagnosis should be collected in one classification. The aim of this paper is to collect and simplify the old classifications for general dental practitioners, and to collect the easy way for diagnosis and clinical decision of non-diseased and diseased tooth loss by focusing on those findings which do not and which do require treatment, and to identify other lesions where combined or alternative treatment is indicated. Also will give them the main idea how to manage and treat these cases.

Keywords: Diseased TSL; Tooth Structure Loss; Macrodontia; Mulberry molars; Dentinogenesis Imperfecta; Short Tooth Syndrome; Delayed maxillary incisors eruption

Introduction

Dental clinicians can be faced with difficult diagnostic and treatment decisions with respect to the non-diseased and diseased tooth loss, as there are many old classifications for differently separated items of tooth structure loss \cite{1}.

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 \cite{2}.

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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.

Diseased 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 [3].

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.

With all these findings and observations we still have too many partial classifications, which could be confusable to clinicians or at least difficult to collect together in mind to be an aid for him during examination of the case.

For an aid to clinicians, these classifications and information about diagnosis should be collected in one classification, with the way of management and treatment of each case [4-6].

Aim of the study
The aim of this paper is to collect and simplify the old classifications for general dental practitioners. Also, aim is to collect the easy way for diagnosis and clinical decision of non-diseased and diseased tooth loss by focusing on those findings which do not and which do require treatment, and to identify other lesions where combined or alternative treatment is indicated [7].

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

Part I: THE Non-Diseased TSL [T]

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 collected classification of non-diseased TSL (Trauma): (T)

<table>
<thead>
<tr>
<th>(TA) Crown Fracture</th>
<th>(TB) Crown Crack</th>
<th>(TC) Root Fracture</th>
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<tbody>
<tr>
<td>I: Incisal notch/ Occlusal chipping or facets (TAi):</td>
<td>I: Undefined Crack (Enamel only) (TBl):</td>
<td>I: Horizontal Fracture (usually anterior teeth) (TCi):</td>
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<td>a. Enamel only (TAia)</td>
<td>a. Short distance: (less than ½ of the tooth enamel) (TBlia)</td>
<td>a. Cervical (TCia)</td>
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<td>b. Enamel &amp; Dentin (TAib)</td>
<td>i. Incisal/occlusal (TBliai)</td>
<td>b. Middle (TCib)</td>
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<td>c. Enamel, Dentin &amp; involving the pulp (TAic)</td>
<td>ii. Cervical (TBliaii)</td>
<td>c. Apical (TCic)</td>
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</tbody>
</table>

II: Incisal/occlusal fracture involving one or both proximal sides (TAII): | II: Defined Crack (Enamel & Dentin) (TBII): | II: Oblique Fracture (anterior & posterior) (TCII): |
| a. Enamel only (TAIIa) | a. Short distance: (less than ½ of the tooth) (TBIIa) | a. Cervical (TCIIa) |
| b. Enamel & Dentin (TAIIb) | i. Incisal/Occlusal (TBIIai) | b. Middle (TCIIb) |
| c. Enamel, Dentin & involving the pulp (TAIIc) | ii. Cervical (TBIIaii) | c. Apical (TCIIc) |
| ![Image](image10.png) | ![Image](image11.png) | ![Image](image12.png) |
| ![Image](image13.png) | ![Image](image14.png) | ![Image](image15.png) |
| ![Image](image16.png) | ![Image](image17.png) | ![Image](image18.png) |

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### ABC Classification & Treatment: The New Collected Classification of Tooth Structure Loss, Management and Treatment

**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)

**b. Long distance: (more than ½ of the tooth) (TBIIib)**
- i. Incisal/occlusal (TBIIibi)
- ii. Cervical (TBIIibii)

### 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.
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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)

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<th>Enamel</th>
<th>Dentin</th>
<th>Pulp</th>
<th>Cementum</th>
<th>Periodontal apparatus</th>
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Tooth parts affected by each event are as follows:

**Part II: THE Diseased TSL [D]**

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<th>(DA)</th>
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| **I:Ectodermal Dysplasia**  
[DA]: Hypodontia, Oligodontia & Microdontia (peg lateral incisor): 1. Mutation of Pax 9 gene which maps to chromosome #4. 2. The best known of the missing teeth syndromes is X-Linked hypohidrotic Ectodermal dysplasia.  
**Hypodontia:** having less than 6 congenitally missing teeth. (partial anodontia)  
**Oligodontia:** having 6 or more congenitally missing teeth.  
**Microdontia** (peg lateral incisor): | **Erosion** [DB]:  
* 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[DBa]: Acidic foods & drinks  
  b- Environmental Erosion[DBb]: Airborne proteolitic enzymes & high levels of hydrochloric acid  
  c- Medications & Xerostomia [DBc]: can be another cause of possible erosive conditions.  
  d- Regurgitation Erosion[DBd]: Involuntary or voluntary  
* Involuntary regurgitation: or gastroesophageal 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.  
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<th>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. A photo-micrograph using transmitted light showing the earliest signs of a caries lesion at the base of an occlusal fissure.</th>
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<tr>
<td>II: Macrodontia &amp; Dens in Dente [DAII]: a- Macrodontia is a molarization of bicuspid leading to its uneruption. [DAIIa] b- Dens in dente is a tooth within a tooth by invagination of the cingulum which resulted in enamel being reflected into the tooth. [DAIIb]</td>
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<tr>
<td>II: Abnormal Attrition &amp; 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 [DBIIId]: 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.</td>
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</table>
**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 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).

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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 [DBIIlc]: 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 sialophosphoprotein 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]**

### IV: Environmental Effects on Tooth Structure Development [DBIV]:
- 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.
3. Types a & b radiographically, the teeth appear solid, but lacking pulp space.
Type c- Shell teeth with little dentine (rare) [DAIVc]
4. Type c (more rare) paradoxically characterized by too little rather than too much dentin resulting in shell teeth.
5. It is a different mutation in the same gene.

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]:
6. Rare as 1:14,000
7. At least 14 phenotypes have been identified and autosomal dominant, recessive & X-Linked inheritance have been reported.
8. The matrix of enamel is comprised mainly of a protein called amelogenin.
9. The gene for this protein is on the short arm of the X chromosome (Xp22.1).
10. 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

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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 maxillo-mandibular 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.
## Summary of TSL Classification

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<thead>
<tr>
<th>Non-diseased Tooth Loss (Trauma) (T)</th>
<th>Diseased Tooth Loss (D)</th>
<th>DC: Bacterial Invasion (Caries)</th>
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<tbody>
<tr>
<td><strong>TA</strong></td>
<td><strong>TB: Crown Crack</strong></td>
<td><strong>TC: Root Fracture</strong></td>
</tr>
<tr>
<td>I</td>
<td>Incisal notch/ Occlusal chipping or facets (TAI):&lt;br&gt; a. Enamel only (TAIa)&lt;br&gt; b. Enamel &amp; Dentin (TAIb)&lt;br&gt; c. Enamel, Dentin &amp; involving the pulp (TAIc)</td>
<td>Horizontal Fracture (usually anterior teeth) (TCI):&lt;br&gt; a. Cervical (TCIa)&lt;br&gt; b. Middle (TCIb)&lt;br&gt; c. Apical (TCIc)&lt;br&gt; Mainly caused by direct trauma</td>
</tr>
<tr>
<td>II</td>
<td>Incisal/occlusal fracture involving one or both proximal sides (TAIi):&lt;br&gt; a. Enamel only (TAIia)&lt;br&gt; b. Enamel &amp; Dentin (TAIib)&lt;br&gt; c. Enamel, Dentin &amp; involving the pulp (TAIic)</td>
<td>Defined Crack (Enamel &amp; Dentin) (TBII):&lt;br&gt; a. Short distance: (less than ½ of the tooth) (TBIIa)&lt;br&gt; i. Incisal/Occlusal (TBIIai)&lt;br&gt; ii. Cervical (TBIIaii)</td>
</tr>
</tbody>
</table>

### Citation:
### ABC Classification & Treatment: The New Collected Classification of Tooth Structure Loss, Management and Treatment

| III | Extensive Crown Fracture (TIIIa): a. Less than ½ of the crown (TIIia) b. More than ½ of the crown but above the CEJ (TIIib) c. All the crown and extend to the root (TIIlc) | Fractured-Crack (Enamel, Dentin & involving Pulp) (TBIi): a. Mild (TBIi-a): incomplete fracture of the crown & not involving periodontal apparatus b. Moderate (TBIib): incomplete fracture of the crown & involving the periodontal apparatus c. Sever (TBIic): complete fracture (detachable) of the tooth crown with pulpal & periodontal involvement, no root involvement d. Complicated (TBIId): complete fracture (detachable) involving crown & part of the root (E., D., C., pulp & periodontal apparatus) e. Complex (TBIIe): fracture involving crown, part of the root & alveolar bone | 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. | Screw driver incisors & Mulberry molars (DAIII): Seen in congenital syphilis and caused by direct invasion of tooth germs by Treponema organisms | 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. a) Internal resorption [DBIIia] 1- Apical [DBIIia1] 2- Intraradicular [DBIIia2] 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 d- Invase 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] 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] | 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. |

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**Citation:** Abdelfattah AH., et al. "ABC Classification & Treatment: The New Collected Classification of Tooth Structure Loss, Management and Treatment". *EC Dental Science* 4.1 (2016): 673-696.
| 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) | Dentinogenesis Imperfecta (DI) [DAIV]: * Gene maps to chromosome #4. * It encodes a protein called dentin sialoprophosphoprotein 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] Types a&b radiographically, the teeth appear solid, but lacking pulp space. Type c- Shell teeth with little dentine [rare] [DAIVc] Type c paradoxically characterized by too little rather than too much dentin resulting in shell teeth. It is a different mutation in the same gene. | 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 cusps, 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]: Type a- Hypoplastic 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- Hypocalcification 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] | | |

**Citation:** Abdellattah AH., et al. "ABC Classification & Treatment: The New Collected Classification of Tooth Structure Loss, Management and Treatment". *EC Dental Science* 4.1 (2016): 673-696.
VI  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)  

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short Tooth Syndrome [DAVI]:</td>
<td></td>
</tr>
<tr>
<td>a. Altered eruption [DAVIa]:</td>
<td>In altered eruption, one finds the gingival margin located excessively incisal to the CEJ, covering a portion of the anatomic crown with soft tissue</td>
</tr>
<tr>
<td>b. Compensatory eruption: Excessive incisal attrition [DAVIb]:</td>
<td>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.</td>
</tr>
<tr>
<td>c. Delayed maxillary incisors eruption: Excessive eruption of mandibular incisors [DAVIc]:</td>
<td>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.</td>
</tr>
<tr>
<td>d. Vertical maxillary excess [DAVID]:</td>
<td>This gummy smile frequently results from a skeletal dysplasia, specifically the hyperplastic growth of the maxillary skeletal base.</td>
</tr>
</tbody>
</table>

Examination & Diagnosis of 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 [8].

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.

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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 [9].

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

Management & Treatment Plan of Tooth Structure Loss

Treatment plan of Traumatic TSL (Class T):

Suggested treatment of every case will be as follows:

<table>
<thead>
<tr>
<th>Classes</th>
<th>Treatment Plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>TA Ia</td>
<td>Simple filling (composite or amalgam).</td>
</tr>
<tr>
<td>TA Ib</td>
<td>Simple filling (composite or amalgam).</td>
</tr>
<tr>
<td>TA Ic</td>
<td>RCT, then filling (composite or amalgam). If tooth weakened crowning is must.</td>
</tr>
<tr>
<td>TA IIa</td>
<td>Filling for destructed sides (composite or amalgam)</td>
</tr>
<tr>
<td>TA IIb</td>
<td>Filling for destructed sides (composite or amalgam)</td>
</tr>
<tr>
<td>TA IIc</td>
<td>RCT, then filling (composite or amalgam), followed by crowning.</td>
</tr>
<tr>
<td>TA IIIa</td>
<td>RCT, then filling (composite or amalgam), followed by crowning.</td>
</tr>
<tr>
<td>TA IIIb</td>
<td>RCT, post and core will be used for reconstruction of tooth and then crowning.</td>
</tr>
</tbody>
</table>
| TA IIIc | 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. |
| TA IVa  | Filling (composite or amalgam) |
| TA IVb  | Filling (composite or amalgam) |
| TA IVc  | RCT, post and core will be used for reconstruction of tooth and then crowning. |
| TB IaI | To be left under observation or sealed with resins. |
| TB IaII | To be left under observation or sealed with resins. |
| TB IbIi | To be sealed by resins. |
| TB Ibii | To be sealed by resins. |
| TB IIaI | Restored by filling. |
| TB IIaII | Restored by filling. |
| TB IIbI | Restored by filling. |

Management & Treatment of Diseased TSL (Class D):

DA) Congenital & Syndromes:

Ectodermal Dysplasia: Hypodontia, Oligodontia & 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 & 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 [10].

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 & 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 [11].

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 [12].

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)
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(c) Delayed maxillary incisors eruption [DAVIc]

*Selective incisal reduction followed by crown lengthening, or orthodontic intrusion of the mandibular incisors [13].
*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

a. Orthodontics
b. Periodontics
c. Elective RCT
d. Restorative therapy
e. Orthognathic surgery

**DB) Systemic, Cellular & 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 [14].

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 [15].

**Abnormal Attrition & 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 [16].

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 [17].

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Cellular Changes (inflammatory) [DBIII]

a. Internal inflammatory resorption [DBIIIa]:
   A. Apical [DBIIIa1]: Endodontic treatment to level of resorption. Long-term calcium hydroxide dressing before placement of root filling
   B. Intra-radicular [DBIIIa2]: Endodontic treatment and root canal filling (hot GP technique, Obtura etc).

b. 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.

c. 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% trichloracetic acid. ProRoot MTA may also be used [18].

d. Invasive cervical resorption [DBIIIId]:
   Class 1, 2: Treatment could be topical application of 90% trichloracetic acid, curettage, and glass ionomer cement restoration.

Class 3: Treatment could require topical application of 90% trichloracetic 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 [19-21].

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]:

a. 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.

b. 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 [22].

c. 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, and cases with hypoplasia will be treated as hypoplastic cases [23].

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

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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 [24,25].

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 [25].

Conclusion
Every GP dentist must have a leaflet containing the collected classification and management with the treatment of each case of tooth structure lost. This will help him to get the right decision about the diagnosis and to which consultant he is going to refer the case. This collection facilitate to GP dentist the examination, diagnosis and treatment without any confusion.

Bibliography