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Non-carious cervical lesions (NCCL)

Abstract

The cervical junction constitutes the anatomic limit between the crown and the root.[1] Cervical lesions display two major variations: wedge-shaped and saucer-shaped lesions. Other lesion aspects have also been reported: shallow, concave, notched and irregular lesions. The cervical junction includes three different structures: enamel, dentin, and cementum, each displaying specific properties. Cervical lesions result from attrition, erosion, abrasion, or abfraction. There is a consensus on the etiology of three first types of lesions, however what is leading to an abfraction is a matter of discussion, with diverging etiological factors that remains controversial. Either it could be a stress-induced lesion related to the distribution of von Mises forces that potentiate cervical wear, or it is related to the physicochemical differences between enamel, cementum, and dentin, including their respective density and resistance to abrasives (brushing, toothpastes, acidic beverages and foods). Restorations are using mostly glass ionomer cements or resin-based composites. Failures of cervical adhesive restorations are numerous. The selection of appropriate treatment protocols implies substantial changes in the habits of the patient (tooth brushing, acidic beverages, and food intake), and the removal of excess material and final polishing.

Keywords: Cementum-dentin junction, enamel-cementum junction, wedge-shaped, saucer-shaped lesions, attrition, erosion, abrasion, abfraction, glass ionomer cements, resin-based cements

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Introduction

Carious lesions are prevalent lesions in the oral cavity. To summarize, 3.6 billion people have dental caries and need to be supported by an accurate diagnostic and subsequently treated. In addition to carious decays, non-carious lesions related to aging and located in the cervical area need also to be treated. This is the aim of this article.[2]

I. Anatomy of the cervical junction.

Histological structures:

Enamel, dentin and cementum are the three different structures that are implicated in non-carious lesions located in the cervical junction.

Enamel is formed by rods and interrods, with thin aprismatic enamel layers located in the inner dentino-enamel junction and in the outer border (surface layer).

This junction includes :

I. Acellular afibrillar cementum (AAC) (found in coronal cementum, 1-15µm thick). It doesn't contain collagens. Its formation may be due to the activity of alkaline phosphatase (ALP). This layer consists in fine filamentous or granular material of moderate electron density.[3] No fibrills are present in the AAC.

II. An acellular extrinsic fiber cementum (AEFC), is present near the cervical portion. Its thickness vary from 50 µm to 200 µm.

The junction comprises four different types of tissues (Figure 1) with variable relationship

a) Cementum ended over enamel (percentage: 60-65%)

b) Edge-to-edge junction (account for 30%)[4]

c) Gap between enamel and cementum with exposed dentin. This type of junction constitutes about one third of the junctions. # 1/3 (17.7%). (5-10%). Diverging results are found between the various reports.

d) Enamel is over dentin (1,6-2%).

Cemento-enamel junction type

Pattern 1: Cementum ended over enamel (60-65%).

Pattern 2: Cementum and enamel are edge to edge (30%).

Pattern 3: In the gap between cementum and enamel, the dentin is exposed (5-10%).

Pattern 4**:** Enamel ending is located over cementum (1,6%- 2%).[5]

Variations have been reported between publications of different authors**.**

Cervical enamel

It is the thinner layers of cervical enamel (buccal and lingual). Cervical enamel is subjected to Von Mises stress distribution (Figure 1). This is also the location of enamel tufts. These later are suggested to be formed by a 13-17 kd molecule, named sheathlin / amelin / ameloblastin), but it do not contain amelogenin. Keratin may be related to cell fragments of ameloblasts [the so-called Tomes'processes].[6,7] In addition to the tuft proteins, enamel lamellae / tuftelin are found.[8,9] Enamel prisms are directed horizontally and at the surface ended by a thin aprismatic enamel layer. Incremental lines have a daily circadian production about 4µm (3.5µm thick, and Von Ebner lines (4x 4µm, named incremental lines of Andresen or von Ebner lines. They are separated approximatively by a 16-20µm interval, or 10-14µm/day). In the root, Incremental lines are deposited accordiing to theoretical replacement. Dental longitudinal ssections revealed also Owen's contour lines formation, occurring between 13 and 26 µm/per day during the second half of root formation (Figure 2-4).

The formation of enamel is due to the tissue AAC is related to nonspecific alkaline phosphatase (TNAP) and osteopontin, a molecule taking origin in the blood circulation (blood serum). They are both acting as a major source of the AAC matrix. Osteopontin is a molecule predominantly identified in the AAC, as a non-collagenous protein (Figure 3).[10]

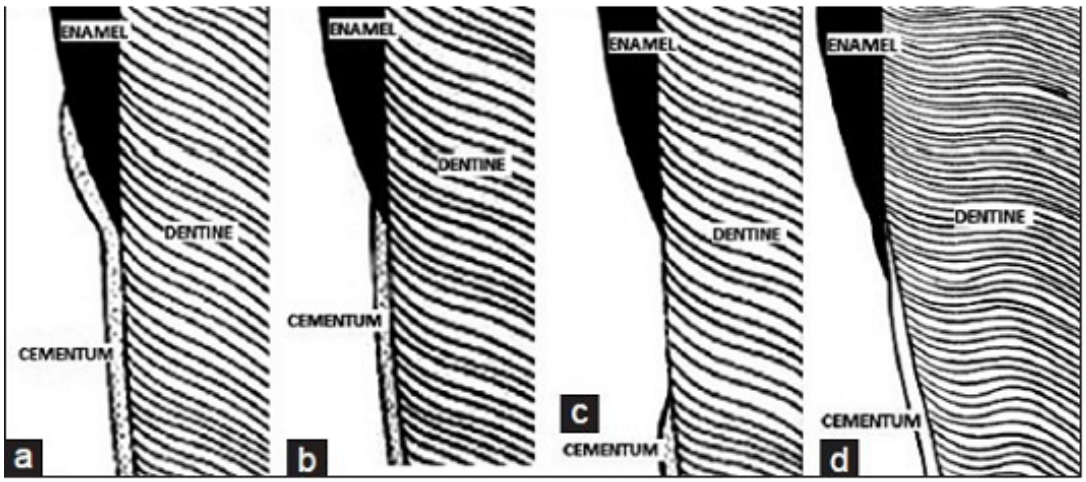

Figure 1

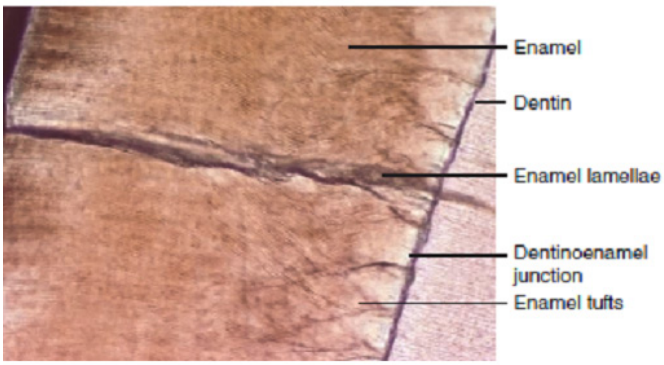

Figure 2

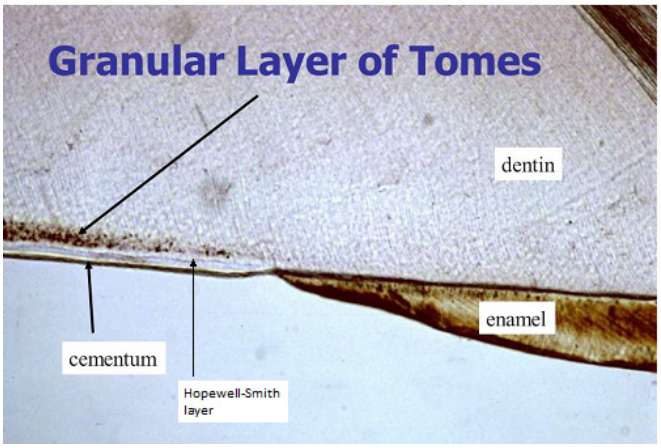

Figure 3

Cervical dentin:

The external (superficial) layer cover the outer part of dentin. The inner circumpulpal dentin forms the bulk of the dentin layer. Characterized as the continuation of the coronal mantle dentin, these outer layers are present only in the root part of the teeth. These outer layers, of the tooth, are formed by:

a) A thin atubular afibrillar layer: over the translucent Hopewell Smith layer, which is deprived of tubules. There are fewer tubules than in subjascent layers. It is a hyaline thin layer deposited over

b) The thicker granular layer of Tomes (Figure 5&6)

c) Characterized by the inclusion of NCP in the non-mineralized interglobular spaces.

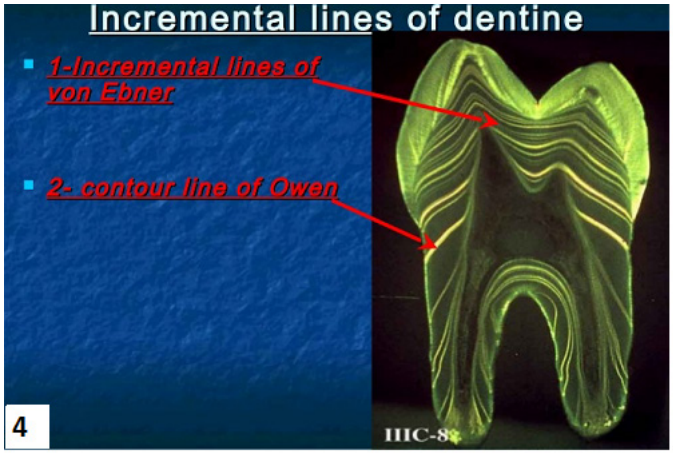

Figure 4

In addition, contour lines of Owen are found exclusively in the crown.

In the root, the Hopewell-Smith layer (0.5-0.8 □m thick)[11] and the granular layer of Tomes correspond to the mantle dentin located exclusively in the crown (Figure 5).[11]

Enamel makes a union with dentin. The layer of Hopewell-Smith seals the dentinal tubules and therefore contribute to decrease for its part sensitivity to cold, sweet and acids beverages, and horizontal abrading due to tooth brushing (Figure 2-5).

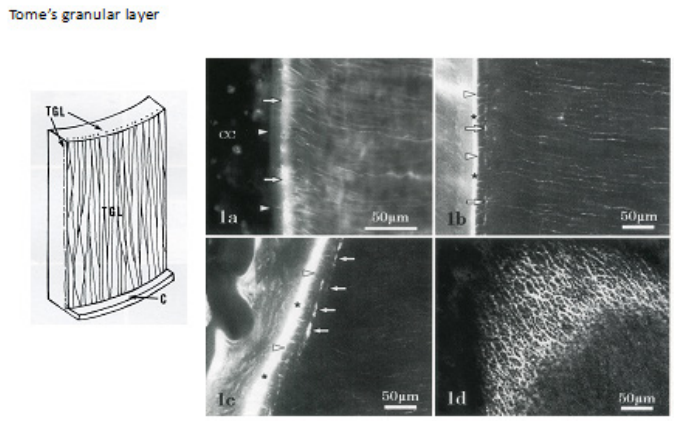

Figure 5

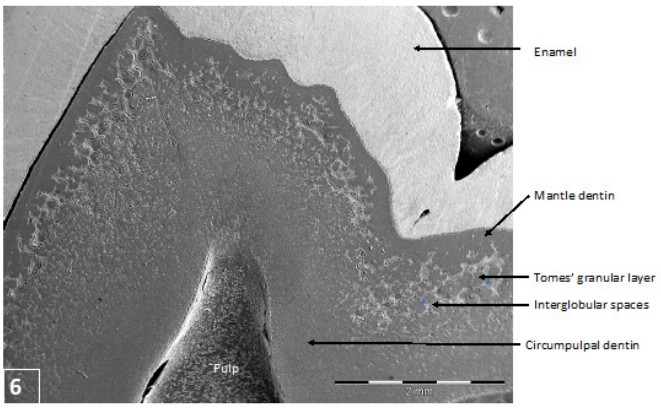

Figure 6

Tubule density in human deciduous (HD) was 18,243+/- 3845 , and in human permanent (HP) (18,781+/-5855), versus 24,162+/-5338 (human deciduous), and 21,343+/-7290 (human permanent).

The mean diameter for human deciduous, was 2.55 micrometers+/-0.16/ 2.82 micrometers +/- 0.28; and for HP, 2.65 micrometers +/-0.19/2.90 micrometers +/-0.22.[12]

Again variations are noted between the different reports, due to the methods which were used and variations between the populations examined.

Reparative dentin

In tertiary dentin (also named reactionary or reparative dentin) sclerotic tubules are occluded by mineral reprecipitations (from the cristallo-chemical family of hydroxyapatite but bearing other crystallographic forms (wheddellite, calcite, brushite, whitlockite and octocalcium phosphate, amorphous calcium phosphate). Beneath the cervical lesion, a calcio-traumatic line differentiates the secondary dentin (tubular) from the osteodentin and/or atubular dentin (Figure 6&7). The number and diameter of dentin tubules is shown in Table 1.

Pulp Immunocompetent cells in healthy human dental pulp[13]: $CD45^{+}$ 0.94% ± 0.65%

With a sub-population $CD16^{+}CD14^{+}$, granulocyte/ neutrophils are representing the major subpopulation in $CD45^{+}$, CD14+ monocytes. Minor subpopulations include natural killer cells and B lymphocytes. Pulp immuno-competent cells: including innate immune responses of the dental pulp that abolish the insult, cellular-mediated immunity and specific humoral immunity. Dendritic cells, are located at the periphery of the pulp, other cells are located more centrally with a macrophage-like appearance. The two groups of cells have the capacity to induce T-cell proliferation.[14,15] Dendritic cells activate T-lymphocytes classified into Th1 and Th2 cells. B-lymphocytes are rarely encountered in normal pulp. Class II molecule-expressing pulpal DCs are of primary importance. Histiocytes /macrophages and other immune cells are essential for the induction of antigen-specific reactions in the dental pulp.

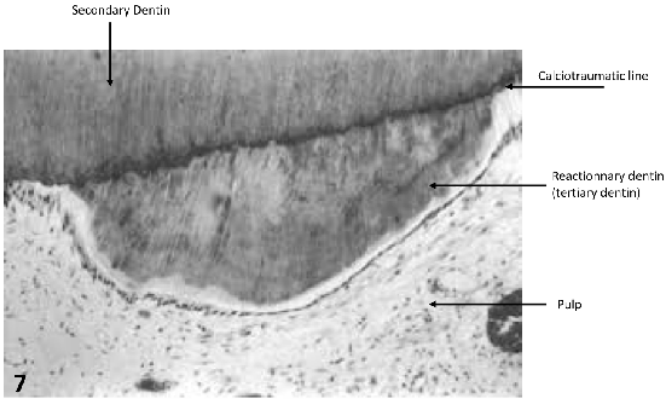

Figure 7

Figure 8

Table 1

Number of tubules (1000/ mm2)		Diameter in micrometers wedge-shaped lesions
Floor of the lesion	18 ±8.2	0.5 mm
Between the floor and pulp	44 ±9	1.6 mm
Primary dentin near the pulp	51±9.8	2.3mm

In addition to fibroblast-like cells [or pulpoblasts, according to Baume (1980)],[16] and pulp stem cells, inflammatory cells are found (pulpal dendritic cells, histiocytes/macrophages, T-lymphocytes). These cells are members of the class I and class II major histocompatibility complex (MHC). Macrophages implicated in the immune reaction are involed in the phagocytosis of apoptotic

bodies and bacteria. Macrophages are located at the pulp periphery at perivascular location. These cells are present mostly in the sub-odontoblastic zone. Lymphocytes, and mast cells are located near the vessels implicated in blood vascularization. Differences were detected between the coronal pulp and the root. Terminal capillaries are organized as loops in the coronal pulp (implying areas between 100 and 150 □m) , whereas in the root, they form a fishernet-like arrangement. Endothelial capillaries are continuous. They eliminate extracellular exudates, and control the vascularization and lymph vascularization, avoiding inflammatory aggregates, and keeping sound in the pulp facing the dental wear as NCCL despite the loss of tissue in the cervical area (Figure 7). In reaction to cervical erosion, the volume occupied by the pulp is gradually reduced. Calcosphrites, pulp stones and diffuse mineralization are produced within the pulp (Figure 8).

1- Non-carious cervical lesions (NCCL) are characterized by a loss of hard dental tissue located at the cementum-enamel junction.

They appear as a wedge, or depressions (dome or cup). But they may also appear as shallow, concave, notched or irregular (Figure 9-11). They have been classified as erosion, abrasion, or as abfraction.[17] They are also named "Dental Wear", also identified under the acronymic appellation of NCCL. The different interactions between attrition, abrasion and erosion in tooth wear are summarized in the figures 9-11[18,19]

a) Abrasion (is produced by interaction between teeth) tooth (attrition-abfraction), lip piercings (erosion & abrasion)

b) Attrition is caused by chewing or grinding

c) Erosion (resulting from dissolution of hard tissue by acidic substances). Progressive loss of tooth substance by chemical or acid effects (no bacteria are involved in NCCL). The composition of enamel and dentin as volume percent of total tissues contributes to NCCL (Table 2).[20]

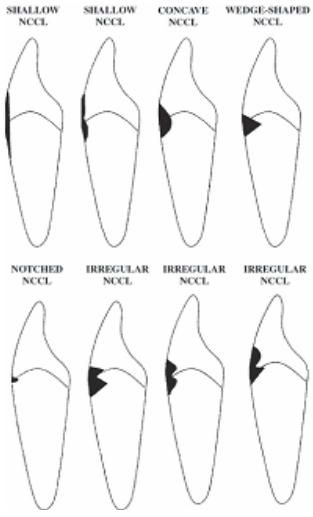

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Figure 9

Chemical erosion occurs either by the hydrogen ion, or by anions which can bind or complex calcium. Inorganic acids, such as hydrochloric acid dissociates in water to hydrogen and chloride ions. It dissolves the mineral surface.

However, we are mostly concerned by weak acids, such as citric and acetic acid. The citric acid directly attacks the crystal surface.

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Figure 10

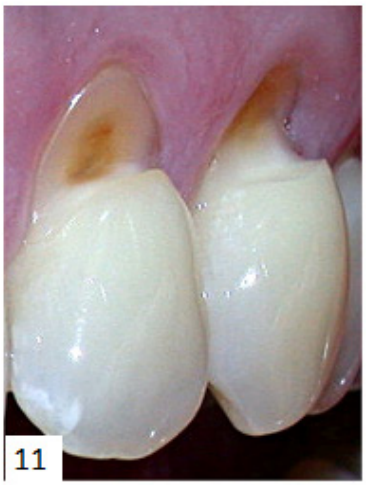

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Figure 11

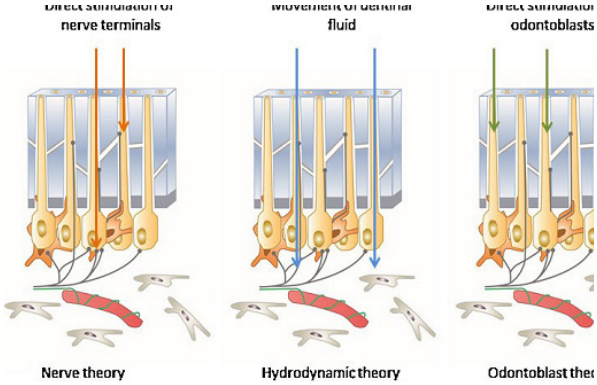

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Figure 12

Abfraction potentiate wear. The term was introduced by Grippo.[21] There are 3 types: C-shaped lesions, V-shaped lesions, and mixed. They have been identified as "Stress-Induced lesions". Microstructural loss of tooth substance in area of stress concentration. Stress analysis on occlusal forces in canine teeth focus on a strong relationship with NCCL.[22] The concentration of tensile stresses seems to be linked to cervical lesion, inducing lesions appearing as a wedge-shaped cervical lesion. However, a re-evaluation of the relationship between occlusal loading and abfraction lesion do not conclude on the validity of theory of abfraction as the primarily factor in the formation of the lesion.[23]

Table 2

Component	Enamel % by vol.	Dentin % per volume
Carbonated hydroxyapatite	85	47
Water	12	20
Protein and lipid	3	33

Table 3

Morphological NCCL category	Synonymous terms
Shallow	Dished-out, saucer-shaped
Concave	Cupped, c-shaped
Wedge-shaped	V-shaped
Notched	Shallow grooves, grooved, gingival notching, deep notches

A number of research have proposed that tensile and compressive stresses play a primary role in causing enamel and dentin fracture creating wedge-shaped cervical lesion, while abrasion and erosion play a secondary role.

It should be emphasized that noncarious cervical lesions (NCCL) may occur as part of a multifactorial event whose mechanism is not elucidated completely yet. Moreover, the association of occlusal loading factors and noncarious cervical lesions may not necessarily support a causal relationship. Therefore, there is little direct evidence supporting abfraction as the primary factor in causing non-carious cervical lesions.

Additional research is needed to assess more definitively the mechanism through which noncarious cervical lesions are initiated and propagated, as well as the potential role of abfraction in non-carious cervical lesions and restorations (Figure 11).

More recently, authors have introduced new terms describing tooth wear: “Biodental engineering factors” they 0have been defined as the effect of piezoelectricity at the cervical area, and “stress corrosion”.. They have been used to describe a multifactorial physiochemical degradation of the CEJ area. In addition, “dental compression syndrome” is tooth deformation related to malocclusion, parafunctional habits and temporo-mandibular joint disorders.[23] Multifactorial aetiologies were proposed by Osborne-Smith et al.[24] (Figure 12).

Corrosion: ***endogenous*** source of corrosion: vomitus pH 3.8 determine the site and extent of dental corrosion.

Categories in the morphological NCCL with synonymous terms are provided in the literature (Table 3).

Etiological factors involved in non-carious cervical lesions[25]: excessive consumption of acidic and carbonated beverages (71,42%); gastro-oesophagyal reflux disease (14,28%); incorrect technique of brushing (28,57%), vicious habits-nail biting (14,28%), daily consumption of sunflower seeds (9,52%), use of toothpicks as auxiliary hygiene (19,04%) and night teeth grinding (4,76%).

Intrinsic factors: parafunctions, vit C, aspirin tablets, powders, Ectasy, acid food and drinks. Gastrointestinal reflux (gastroesophageal reflux disea (or GERD), bulimia and anorexia, bruxism.

Extrinsic (exogenous) factors: acid food (erosion), reduced saliva flow, drugs that change the buffering power of saliva, dental floss, toothbrushes, poorly washed vegetables, immune system disease.

Flat or concave, irregular and wedge-shaped with pitting, striations, scratches, and enamel cracks.

When wear is limited to enamel: facets are observed. In an advanced stage, they become concave (dentin exposed- cupped). Gingival crevicular fluid contributes to the formation of cervical NCCL.

Static stress corrosion, cyclic (fatigue) stress corrosion (combined with 1% lactic acid, buffered to pH 4,5) (Figure 12).

Stress (micro fracture/abfraction)

Endogenous: parafunction /occlusion/ deglutition

Exogenous: mastication, Habbits, occupational behaviors Use of dental appliance

Corrosion (chemical degradation)

Endogenous plaque, gingival crevicular fluid, gastric juice

Exogenous: diet, occupational exposure, use of certain drugs or alcohol

Friction (wear) (Figure 8-11**)**

Hypersensitive non-carious cervical lesions[26]

Therapeutic options are the following: sealants, restoration treatment, potassium nitrate dentifrice.

Restoration[27]

Non Carious Tooth Defects (NCTDs) Abrasion, Abfraction or Erosion (AAE) are using mostly Resin Modified Glass Ionomers Cement (RMGIC) or Resin Based Composite (RBC). The restorations of non-carious cervical lesions are increasing in number. This is due to the growth of elderly population, less tooth loss, and increasing factors such as erosion, abrasion, corrosion (critical pH of less than 5.5), and abfraction. The decision is taken by the dental practitioner either to « ignore or restore ».[28]

Conclusion

Failures of cervical adhesive restorations are attributed to inadequate moisture control, adhesion to different substrate (enamel and dentin), with different composition, and cusp movements (Figure 12). Composite resins can be used. Either as microfilled resins or as a flowable resin.

Cavity cleaning involving etching the dentin with 37% phosphoric acid for 19 seconds, and 20 seconds on enamel It is suggested to start with two or three layers in the cervical zone (incremental insertion), and to finish with the enamel margin.

The selection of appropriate treatment protocols implies substantial changes in the habits (brushing, dietary) of the patient, the removal of excess material and final polishing of the restauration.[29–31]

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