Controversies in Periodontics

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Medical and Research Publications

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Medical and Research Publications

First printing, 2022.
ISBN: 978-81-955135-2-9

Published by

Medical and Research Publications,

124SpencerRd,Stoke-on-TrentST42BE,

United Kingdom.

www.medicalandresearch.com

Email: info@medicalandresearch.com

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Introduction

A controversy is a state of prolonged public dispute or debate, usually concerning a matter of conflicting opinion or point of view. The word was coined from the Latin word controversia, as a composite of controversus – "turned in an opposite direction". In dentistry, the reason of controversy on a particular topic may be related to inadequate knowledge of the etiological factors, technical difficulties, and patient related factors affecting the treatment outcomes or some other unknown factors. **[**1**]**

A hallmark of periodontology is the continual dialogue and debate among clinicians, research scientists and those who bridge both fields in translational research. For the periodontal specialist, as well as for other dental practitioners, this dialogue and debate starts from their early training, with a critical evaluation of both the classical and the contemporary literature. The dialogue and debate continues in study clubs and in regional, national and international meetings and workshops. Throughout the 20th century, and now in the 21st century, concepts, controversies and consensus regarding the full range of issues and disciplines in periodontology have been discussed, dissected and analyzed using the latest biological discoveries, and more recently using the emerging formal guidelines of evidence-based science. The subject of periodontology considers a broad range of issues that represent contemporary controversies in the discipline. The current challenge is to disentangle the controversies existing in various subjects like classification of periodontal diseases, pathogenesis of periodontal diseases including the role of occlusion, periodontal-endodontic controversy, lasers in periodontics and many more. **[**2**]**

This book attempts to highlight and analyse the background of some of the more significant issues that have been debated in the field of periodontology in recent years. Certainly, including all topics currently under debate would require a volume that would be several magnitudes larger than what could be presented in a single monograph. Therefore, a more limited list of issues that are of particular concern, not only to the periodontal specialist but also to the general dentist, hygienist and other dental practitioners, was selected.

This book also serves to show that - In a controversy, one often ends up where one started.

Classification of Periodontal Diseases

Any attempt to group the entire constellation of periodontal diseases into an orderly and widely accepted classification system is fraught with difficulty, and inevitably considerable controversy. No matter how carefully the classification is developed, and how much thought and time are invested in the process, choices need to be made between equally unsatisfactory alternatives. Many people appear to believe that classification systems are rigid and fixed entities that should not be changed. In fact, classification systems should be viewed as dynamic works-in-progress that need to be periodically modified based on current thinking and new knowledge.

Despite this dilemma, in the past hundred years, experts have periodically assembled to develop a new classification system for periodontal diseases, or to refine an existing one.

Dominant paradigms in the historical development of classification systems

The development and evolution of classification systems for periodontal diseases have been largely influenced by paradigms that reflect the understanding of the nature of periodontal diseases during a given historical period into three dominant paradigms primarily based on The clinical features of the diseases - 1870–1920,

The concepts of classical pathology - 1920–1970,

The infectious etiology of the diseases -1970 onwards. **[**3, 4, 5**]**

Classification systems in the modern era represent a blend of all three paradigms since there is a certain amount of validity to some of the earliest thoughts about the nature of periodontal diseases. Only those ideas that are believed to be clearly outmoded or incorrect have been discarded.

Clinical characteristics paradigm (1870 – 1920)

At the time, very little was known about the etiology and pathogenesis of periodontal diseases. Accordingly, the diseases were classified almost entirely on the basis of their clinical characteristics supplemented by unsubstantiated theories about their cause i.e whether they were caused by local or systemic factors.6 In the late 1800s and early 1900s clinicians used case descriptions and their personal interpretation of what they saw clinically as the primary basis for classifying periodontal diseases. Formal papers on the classification of periodontal diseases were rare in the late 1800s and early 1900s. Typical publications on the subject usually represented the opinion of a single person who almost always based the classification on clinical observations and theoretical explanations of causation. A good example is a paper published by C.G. Davis in 1879 who believed that there were three distinct forms of destructive periodontal disease: **[**7**]**

• Gingival recession with minimal or no inflammation.

• Periodontal destruction secondary to ‘lime deposits’.

• ‘Riggs’ Disease’ the hallmark of which was, ‘... loss of alveolus without loss of gum.’

Similarly, in 1886 G.V. Black published his thoughts on the classification of periodontal diseases based on their clinical characteristics and his understanding of their cause into five separate groups. **[**8**]**

• Constitutional gingivitis

• A painful form of gingivitis.

• Simple gingivitis

• Calcic inflammation of the peridental membrane.

• Phagedenic pericementitis

The point of these historical examples is to emphasize that little or no scientific evidence was used to support the opinions of the clinicians of the time. It is not surprising then, that no generally accepted terminology or classification system for periodontal diseases was adopted during this era. As a result, in the latter part of the 19th century periodontitis went Classifying periodontal diseases under numerous names including: 'pyorrhea alveolaris'. **[**9**]** 'Riggs' disease'. **[**10**]** 'calcic inflammation of the peridental membrane'. **[**11**]** 'phagedenic pericementitis'. **[**12**]** and 'chronic suppurative pericementitis'. **[**12**]** During this period, the dominant term used for destructive periodontal disease was pyorrhea alveolaris.

Classical pathology paradigm (1920–1970)

As the field of periodontology began to mature scientifically in the first half of the 20th century, many clinical scholars in both Europe and North America began to develop, and argue about, nomenclature and classification systems for periodontal diseases.

What emerged from this debate was the concept that there were at least two forms of destructive periodontal disease - Inflammatory and Non inflammatory ('degenerative' or 'dystrophic'). Gottlieb, in particular, had a significant influence on the field when he postulated that certain forms of destructive periodontal disease were due to degenerative changes in the periodontium. **[**13, 14**]** He believed that he had discovered histological evidence of an impairment in the continuous deposition of cementum (i.e. 'cementopathia'). The impact of Gottlieb's work on classification systems was profound since it suggested that some periodontal diseases were degenerative. Gottlieb's concept of cementopathia was so readily accepted, and for such a long time, although there was never any convincing evidence that the hypothesis was right. From then on, almost all classification systems used from approximately 1920–1970 included disease categories labeled as 'dystrophic', 'atrophic', or 'degenerative'.

Inflammation

I. Gingivitis (little or no pocket formation; can include ulcerative form – Vincent's)
   A. Local (calculus, food impaction, irritating restorations, drug action, etc.)
   B. Systemic
      • pregnancy
      • diabetes
      • other endocrine dysfunctions
      • tuberculosis
      • syphilis
      • nutritional disturbances
      • drug action
      • allergy
      • hereditary
      • idiopathic, etc.

II. Periodontitis
   A. Simplex (secondary to gingivitis) – bone loss, pockets, abscesses can form: cases have calculus.
   B. Complex (secondary to periodontosis) – etiologic factors similar to

Degeneration

I. Periodontosis (as a rule attacks young girls and older men; often caries immunity)
   A. Systemic disturbances
      1. diabetes
      2. endocrine dysfunctions
      3. blood dyscrasias
      4. nutritional disturbances
      5. nervous disorders
      6. infectious diseases (acute & chronic)
   B. Hereditary
   C. Idiopathic

Atrophy

I. Periodontal Atrophy (Recession, no inflammation, no pockets; osteoporosis)
   A. Local trauma (e.g., from toothbrush)
   B. Presenile
   C. Senile
   D. Disuse
   E. Following inflammation
   F. Idiopathic

Hypertrophy

I. Gingival Hypertrophy
   A. Chronic irritation (see inflammation)
   B. Drug action (e.g., Dilantin sodium)
   C. Idiopathic (e.g., gingivoma, elephantiasis, fibromatosis)

Traumatism

I. Periodontal Traumatism
   A. Occlusal trauma

Classification of Periodontal Diseases Following the ‘‘Classical Pathology’’ Paradigm (Orban 1942) [15]

Classification systems of the period were dominated by the ‘Classical Pathology’ paradigm which is based on the ‘principles of general pathology’ as articulated by Orban et al.: **[**16**]**

‘Periodontal diseases follow the same pattern as do diseases of other organs. There are minor differences which have to be recognized and labelled properly. The basic pathologic tissue changes, however, are the same as those of other organs.’ ‘... According to principles of general pathology, there are three major tissue reactions: inflammatory; dystrophic; neoplastic. Neoplastic changes are not in the therapeutic realm of periodontics. ‘Environmental factors, however, dictate the inclusion of a third and different category of pathologic reaction in Periodontology ...’ ‘... pathologic reactions ... produced by occlusal trauma’.

Conclusion that some forms of periodontal diseases were caused by non-inflammatory or degenerative process was primarily based on over-interpretation of histopathological studies. There was no scientific basis for retaining the concept that there were non- inflammatory or degenerative forms of destructive periodontal diseases and no convincing evidence that Gottlieb’s hypothesis (degenerative nature) was right.

At the 1966 World Workshop in Periodontics serious questions were raised about the existence of ‘periodontosis’ as a distinct disease entity. **[**17**]** Many in attendance at that meeting recommended that the term be discarded. Information summarized at the next World Workshop, held in 1977, meeting supported the conclusion that ‘periodontosis’ was actually an infection and ‘juvenile periodontitis’ should become the preferred term for this group of diseases. Indeed, around 1970 a different paradigm (i.e. the ‘Infection/ Host Response Paradigm’) had begun to dominate thoughts about the nature of periodontal diseases. **[**18**]**

Infection/host response paradigm (1970 onwards)

Soon after the 1876 publication of Robert Koch **[**19**]** in which he provided experimental proof of the germ theory of disease, some dentists began to think that periodontal diseases might be caused by bacteria. W.D. Miller, in particular, was an early proponent of the infectious nature of

periodontal diseases. Miller also recognized that certain systemic conditions (e.g. diabetes, pregnancy) could modify the course of the disease. **[**20**]**

Despite an extensive amount of work very little headway was made in establishing bacterial infections as the foundation upon which periodontal diseases should be classified. In addition, microbiological studies revealed that the periodontal microflora was exceedingly complex and no clear group of microorganisms could be causally linked to the diseases.

It was not until the classical 'experimental gingivitis' studies published by Harald Löe and his colleagues from 1965 to 1968 that the Infection/Host Response Paradigm began to move in the direction of becoming the dominant paradigm. **[**21,22**]** The next major discovery in periodontal microbiology was the preliminary demonstration in 1976–1977 of microbial specificity at sites with periodontosis. **[**23**]** This finding, coupled with the demonstration in 1977– 1979 that neutrophils from patients with juvenile periodontitis (periodontosis) had defective chemotactic and phagocytic activities, marked the beginning of the dominance of the Infection/Host Response paradigm. Indeed, what followed was over two decades of hard work that firmly established that juvenile periodontitis, the new name for periodontosis, was an infection.

The next major landmark in the classification of periodontal diseases emerged from the 1989 World Workshop in Clinical Periodontics where a new classification of periodontitis based on the Infection/ Host Response paradigm was suggested **[**24**]** (Fig. 2) :

I. Adult Periodontitis

II. Early Onset Periodontitis

A. Prepubertal Periodontitis

1. Generalized

2. Localized

B. Juvenile Periodontitis

1. Generalized

2. Localized

C. Rapidly Progressive Periodontitis

III. Periodontitis Associated with Systemic Disease

IV. Necrotizing Ulcerative Periodontitis

V. Refractory Periodontitis

The classification was a refinement of one that had been proposed by Page & Schroeder in 1982 and a similar one that had been adopted by the AAP in 1986. Five types of destructive periodontal disease were listed. **[**25**]**

• This classification, although soundly based in the Infection/ Host Response paradigm, depended heavily on the age of the affected patients and the rates of progression.

• Acknowledgment to some forms of periodontitis that could be significantly modified by host factors (i.e. the category of 'Periodontitis Associated with Systemic Disease') and still other forms that did not appear to respond well to conventional therapy (i.e. the 'Refractory Periodontitis' category), were also given.

However, the 1989 classification was criticized shortly after it was published -

• Considerable 'heterogeneity' existed within the Refractory Periodontitis category.

• Different forms of periodontitis proposed in the classification shared many microbiologic and host response features, which suggested extensive overlap and heterogeneity among the categories.

• Did not include gingivitis or gingival disease category.

• Periodontitis categories had non-validated age-dependent criteria.

• Extreme cross-over in rates of progression of the different categories of periodontitis.

• Refractory periodontitis & Pre pubertal periodontitis were a heterogeneous categories.

With these transparent limitations of the proposed classification, the classification lost some of its clinical utility.

A different classification system was proposed by Ranney. He suggested elimination of the 'Refractory Periodontitis' category since it was a heterogeneous group and it was impossible to standardize the treatment that necessarily would have to be given prior to making the diagnosis. In addition, he recommended elimination of the 'Periodontitis Associated with Systemic Disease' category since the, '... expression of all forms of periodontitis can be modified by some systemic diseases or abnormalities, it is probably better to consider them in that specific context, rather than treating them as a unique category.' **[**26**]**

The first European Workshop 1993 accepted the fact that there is insufficient knowledge to separate truly different diseases (disease heterogeneity) from differences in the presentation / severity of the same disease (phenotypic variation) and stated that the existing classifications are unsatisfactory. **[**27**]**

The 1993 classification proposed a simple classification distinguishing between

1. Early onset periodontitis,

2. Adult periodontitis,

3. Necrotizing periodontitis.

However, there was a need to use additional secondary descriptors for defining the clinical situation. These include: Distribution within the dentition, rate of progression, response to treatment, relation to systemic diseases, microbiological, ethnic group characteristics, and other factors.

1999 Classification of Periodontal Diseases and Conditions

Problems, inconsistencies, and deficiencies associated with the 1989 classification led many clinicians and investigators to call for a revision of the currently used system. This resulted in a 1999 international workshop on the classification of periodontal diseases. **[**28**]**

What emerged was a classification that was even more firmly based on the Infection/Host Response paradigm, but without some of the inherent problems of the 1989 classification.

A quick comparison of the 1989 and the 1999 classifications could lead to the misconception that all that was done was to arbitrarily change the names of ‘Adult Periodontitis’ to ‘Chronic Periodontitis’ and ‘Juvenile Periodontitis’ to ‘Aggressive Periodontitis.’ These changes were specifically made to eliminate the non validated age- dependent designations.

Other important changes to be noted in the classification

• A badly needed gingivitis or gingival disease category was added.

• The heterogeneous disease categories of pre pubertal, refractory and rapidly progressive periodontitis were eliminated as distinct or stand-alone entities.

• The ‘refractory’ designation remains in the new classification, but not as a single entity. Conceptually, all forms of periodontitis can be unresponsive to treatment.

• Furthermore, the troublesome criteria of age and rate of progression were removed as a basis for classifying different forms of periodontitis. It was suggested that the classification be based on extent and severity of the disease, age, and rate of progression.

• There was the addition of new categories like abscess, endodontic-periodontic lesion, development and acquired deformity which were not explained in the previous classifications. **[**29**]**

But this classification also falls short to answer many issues. The term “chronic” and “aggressive” periodontitis arose the need for the rate of progression of the disease and how much severe disease could be categorized as “aggressive periodontitis.” Primary criteria given by Lang for aggressive periodontitis need a detailed systemic investigation, familial history and rate of progression, which are difficult to access in clinical practices. Further, the studies related to the rate of disease progression seem to be insufficient. There are many questions, which remain unanswered like why Diabetes mellitus has not been given place for the systemic condition modifying the periodontal disease. There is no description about environmental factors affecting the periodontitis and gingivitis, when there are so many proven studies available for them, e.g., smoking. Moreover, the entities like “color of gingiva” and “gingival enlargements” are included in periodontal disease category. There is no description about what degree of increase in size of gingiva could be designated as gingival enlargement. In case of multiple diagnoses, as is the case most of the times, which diagnosis is to be put first? Further there are situations, where the etiology of a disease is unknown; in those conditions the relation between diagnosis and the treatment plan creates a state of perplexity. **[**29**]**

Although, AAP 1999 classification is the most widely accepted classification and used for most of research and academic purposes, after the review of the previous classifications, it can be concluded that none of the classification comes near to the idealistic classification.

A new classification scheme for periodontal and peri-implant diseases and conditions - 2018

The workshop which was held in Chicago on November 9 to 11, 2017 was co-sponsored by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) and included expert participants from all over the world. An organizing committee from the AAP and EFP commissioned 19 review papers and four consensus reports covering relevant areas in periodontology and implant dentistry. The authors were charged with updating the 1999 classification of periodontal diseases and conditions1 and developing a similar scheme for peri-implant diseases and conditions. The workshop addressed unresolved issues with the previous classification. **[**30**]**

In this workshop, it was agreed that bleeding on probing should be the primary parameter to set thresholds for gingivitis. **[**31, 32**]** The workshop also characterized periodontal health and gingival inflammation in a reduced periodontium after completion of successful treatment of a patient with periodontitis. This distinction was made to emphasize the need for a more comprehensive maintenance and surveillance of the successfully treated patient with periodontitis.

The workshop agreed that, consistent with current knowledge on pathophysiology, three forms of periodontitis can be identified: necrotizing periodontitis, periodontitis as a manifestation of systemic disease, and the forms of the disease previously recognized as “chronic” or “aggressive”, now grouped under a single category, “periodontitis”.

In revising the classification, the workshop agreed on a classification framework for periodontitis further characterized based on a multidimensional staging and grading system that could be adapted over time as new evidence emerges. **[**33**]**

Staging is largely dependent upon the severity of disease at presentation as well as on the complexity of disease management, while grading provides supplemental information about biological features of the disease, including a history based analysis of the rate of disease progression, assessment of the risk for further progression, anticipated poor outcomes of treatment, and assessment of the risk that the disease or its treatment may negatively affect the general health of the patient. Staging involves four categories (stages 1 through 4) and is

determined after considering several variables including clinical attachment loss, amount and percentage of bone loss, probing depth, presence and extent of angular bony defects and furcation involvement, tooth mobility, and tooth loss due to periodontitis. Grading includes three levels (grade A – low risk, grade B – moderate risk, grade C – high risk for progression) and encompasses, in addition to aspects related to periodontitis progression, general health status, and other exposures such as smoking or level of metabolic control in diabetes. Thus, grading allows the clinician to incorporate individual patient factors into the diagnosis, which are crucial to comprehensive case management.

The new classification of periodontal diseases and conditions also includes systemic diseases and conditions that affect the periodontal supporting tissues including rare systemic disorders, such as Papillon Lefèvre Syndrome. **[**34**]**

A few other notable terminological/conceptual changes noted in the classification can be listed:

• The consensus report presents a new classification of gingival recession that combines clinical parameters including the gingival phenotype as well as characteristics of the exposed root surface. In the consensus report the term periodontal biotype was replaced by periodontal phenotype.

• Traumatic occlusal force, replacing the term excessive occlusal force, is the force that exceeds the adaptive capacity of the periodontium and/or the teeth.

• The section on prostheses-related factors was expanded in the new classification.

The term biologic width was replaced by supracrestal attached tissues.

Periodontal Health and Gingivitis: Consensus Report
Chapple, Mealey, et al. 2018
Active link to consensus report

Gingival Diseases: Case Definitions and Diagnostic Considerations
Trombelli, Tatakis, et al. 2018
Active link to case definitions

PERIODONTAL HEALTH, GINGIVAL DISEASES/CONDITIONS

1. **Periodontal health and gingival health**
   Lang & Bartold 2018 link
   a. Clinical gingival health on an intact periodontium
   b. Clinical gingival health on a reduced periodontium
      i. Stable periodontitis patient
      ii. Non-periodontitis patient

2. **Gingivitis – dental biofilm-induced**
   Murakami et al. 2018 link
   a. Associated with dental biofilm alone
   b. Mediated by systemic or local risk factors
   c. Drug-influenced gingival enlargement

3. **Gingival diseases – non-dental biofilm induced**
   Holmstrup et al. 2018 link
   a. Genetic/developmental disorders
   b. Specific infections
   c. Inflammatory and immune conditions
   d. Reactive processes
   e. Neoplasms
   f. Endocrine, nutritional & metabolic diseases
   g. Traumatic lesions
   h. Gingival pigmentation

Periodontitis Consensus Report
Papapanou, Sanz et al. 2018
Active link to consensus report

Staging and Grading of Periodontitis: Framework and Proposal of a New Classification and Case Definition
Tonetti, Greenwell, Kornman 2018
Active link to case definitions

FORMS OF PERIODONTITIS

1. **Necrotizing Periodontal Diseases**
   Herrera et al. 2018 link
   a. Necrotizing Gingivitis
   b. Necrotizing Periodontitis
   c. Necrotizing Stomatitis

2. **Periodontitis as Manifestation of Systemic Diseases**
   Jepsen, Caton et al. 2018 Consensus Rept link Albandar et al. 2018 link
   Classification of these conditions should be based on the primary systemic disease according to the International Statistical Classification of Diseases and Related Health Problems (ICD) codes

3. **Periodontitis**
   Fine et al. 2018 link Needleman et al. 2018 link Billings et al. 2018 link
   a. **Stages:** Based on Severity[1] and Complexity of Management[2]
      Stage I: Initial Periodontitis
      Stage II: Moderate Periodontitis
      Stage III: Severe Periodontitis with potential for additional tooth loss
      Stage IV: Severe Periodontitis with potential for loss of the dentition
   b. Extent and distribution[3]: localized; generalized; molar-incisor distribution
   c. **Grades:** Evidence or risk of rapid progression[4], anticipated treatment response[5]
      i. Grade A: Slow rate of progression
      ii. Grade B: Moderate rate of progression
      iii. Grade C: Rapid rate of progression

[1] Severity: Interdental clinical attachment level (CAL) at site with greatest loss; Radiographic bone loss & tooth loss
[2] Complexity of management: Probing depths, pattern of bone loss, furcation lesions, number of remaining teeth, tooth mobility, ridge defects, masticatory dysfunction
[3] Add to Stage as descriptor: localized <30% teeth, generalized ≥ 30% teeth
[4] Risk of progression: direct evidence by PA radiographs or CAL loss, or indirect (bone loss/age ratio)
[5] Anticipated treatment response: case phenotype, smoking, hyperglycemia

Periodontal Manifestations of Systemic Diseases and Developmental and Acquired Conditions: Consensus Report
Jepsen, Caton et al. 2018
Active link to consensus report

PERIODONTAL MANIFESTATIONS OF SYSTEMIC DISEASES AND DEVELOPMENTAL AND ACQUIRED CONDITIONS

1. **Systemic diseases or conditions affecting the periodontal supporting tissues**

   Albandar et al. 2018 link

2. **Other Periodontal Conditions**

   Papapanou, Sanz et al. 2018 link Herrera et al. 2018 link

   a. Periodontal Abscesses
   b. Endodontic-Periodontal Lesions

3. **Mucogingival deformities and conditions around teeth**

   Cortellini & Bissada 2018 link

   a. Gingival phenotype
   b. Gingival/soft tissue recession
   c. Lack of gingiva
   d. Decreased vestibular depth
   e. Aberrant frenum/muscle position
   f. Gingival excess
   g. Abnormal color
   h. Condition of the exposed root surface

4. **Traumatic occlusal forces**

   Fan & Caton 2018 link

   a. Primary occlusal trauma
   b. Secondary occlusal trauma
   c. Orthodontic forces

5. **Prostheses and tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis**

   Ercoli & Caton 2018 link

   a. Localized tooth-related factors
   b. Localized dental prostheses-related factors

Peri-implant Diseases and Conditions Consensus Report
Berglundh, Armitage et al. 2018
Active link to consensus report

PERI-IMPLANT DISEASES AND CONDITIONS

1. **Peri-implant health**

   Araujo & Lindhe 2018 link

2. **Peri-implant mucositis**

   Heitz-Mayfield & Salvi 2018 link

3. **Peri-implantitis**

   Schwarz et al. 2018 link

4. **Peri-implant soft and hard tissue deficiencies**

   Hammerle & Tarnow 2018 link

Renvert et al. 2018 Case Definitions link

This new classification for peri-implant health, peri-implant mucositis and peri- implantitis was developed by the workshop was direly needed. An effort was made to review all aspects of peri-implant health, diseases, and relevant aspects of implant site conditions and deformities to achieve a consensus for this classification that could be accepted worldwide. Notable consensus that was derived here was that bleeding on probing helps to differentiate between healthy and inflamed peri-implant mucosa, whereas bone loss helps to differentiate between peri-implant mucositis and peri- implantitis.

Takeaways from the new classification -

• New classification is based on ICD (International Classification of Diseases).

• The new classification is broader than the previous classification and will help the clinicians in better diagnosis and treatment of periodontal diseases.

• It was earlier difficult to differentiate between chronic and aggressive periodontitis. So, now staging and grading method is applied which clearly defines the disease.

• The new system for grading introduces biomarkers for better understanding of progression of disease and more treatment options.

• Staging depends not only on the severity but also on the complexity of disease management. Four types of staging are mentioned.

• Periodontal biotype is replaced by the term periodontal phenotype.

• A traumatic occlusal force has replaced the term excessive occlusal forces.

• The new classification has included clinical procedures used in the fabrication of indirect restorations.

• Peri-implant diseases and conditions are added in new classification. **[**35**]**

The practical applicability, relevance and utilization of the current classification is yet to be gauged. The plethora of debates around classification systems has led us to conclude that every aspect of periodontology is rewritable as science and logic needs to govern.

Conclusion

In the past 130 years classification systems for periodontal diseases have evolved based on the understanding of the nature of these diseases at the time the classifications were proposed. One consistent feature of the development of classification systems is the guaranteed controversy surrounding any suggested revisions to the previously accepted system of nomenclature. Revisions to existing systems have been largely influenced by three dominant paradigms. Before a classification firmly based on the etiological and pathogenic characteristics of periodontal infections can be devised, numerous fundamental breakthroughs will have to occur in our understanding of host– microbial interactions and the environmental factors that affect them. Until this happens, all classification systems will continue to create a dilemma in that choices will need to be made between equally unsatisfactory alternatives.

Case Definition of Periodontal Disease

A fundamental prerequisite for any epidemiologic study is an accurate definition of the disease under investigation. However, the threshold values that determine disease varies among studies, which in turn affects diagnosis and potentially gives rise to controversy. At present, it is difficult to accurately assess epidemiologic data on periodontal disease because of the wide variety of indices and measurements used. The American academy of Periodontology (AAP) reported numerous classification systems in the last 25 years. This is one of the reason why the prevalence of gingivitis and periodontal disease can range widely, depending on which reference levels are considered to be the normal versus diseased. **[**36**]**

Periodontitis is a long-lasting inflammatory disease affecting the soft and hard tissues around the teeth and it is common worldwide. This disease is related to common and preventable biological risk factors (e.g., high blood pressure, high blood cholesterol, diabetes, genetic factors, and obesity) and behavioral risk factors (e.g., an unhealthy diet, physical inactivity, and tobacco use). Based on published studies, the severity and prevalence of the disease vary significantly among populations. **[**37**]**

Case definitions and criteria that are used to diagnose this disease are not yet consistent worldwide. This can affect the accuracy of any comparison made between studies.

Manau et al. found that a different case definition can change the statistical significance and effect size when he sought association between periodontitis and prematurity or low birth weight. Thus, little is known about the common case definition of chronic periodontitis in epidemiological literature or the most common risk factors/predictors associated with it. **[**38**]**

Objectives of a periodontitis case definition system

A case definition system should facilitate the identification, treatment and prevention of periodontitis in individual patients. Given current knowledge, a periodontitis case definition system should include three components:

1. Identification of a patient as a periodontitis case,

2. Identification of the specific form of periodontitis,

3. Description of the clinical presentation and other elements that affect clinical management, prognosis, and potentially broader influences on both oral and systemic health. **[**39**]**

In early epidemiologic studies, the two major periodontal diseases, gingivitis and periodontitis, were combined and considered to be a continuum. National United States surveys were conducted in 1960 to 1962, 1971 to 1974, 1981, 1985 to 1986, 1988 to 1994, and 1999 to 2000. The case definitions and protocols used in the six national surveys reflect a continuing evolution and improvement over time. **[**40-46**]**

Case Definitions and Prevalences Used in United States National Surveys

Study	Age (years)	Case Definition	Prevalence (%)
HES 1960 to 1962	18 to 79	≥1 tooth with pocket	25
HES 1971 to 1974	18 to 74	≥1 tooth with pocket	25.5
1981 National Survey	≥19	Periodontitis: ≥1 site, PD ≥4 mm	36
		Moderate: ≥1 site, PD 4 to 6 mm	28
		Severe: ≥1 site, PD ≥6 mm	8
NIDR 1985 to 1986	18 to 64	≥1 site with CAL ≥3 mm	43
		≥1 site with PD 4 to 6 mm	13.4
		≥1 site with CAL 5 mm	12.8
		≥1 site with PD ≥7 mm	0.6
NHANES III	30 to 90	Periodontitis: ≥1 tooth with CAL ≥3 mm + PD ≥4 mm (same site)	35
		Mild: ≥1 tooth with PD ≥3 mm or ≥1 Class I furcation	21.8
		Moderate: ≥1 tooth with PD ≥5 mm or ≥2 teeth with PD ≥4 mm or ≥1 Class I furcation + PD 3 mm	9.5
		Severe: ≥2 sites with PD ≥5 mm or ≥4 sites with PD ≥4 mm or ≥1 tooth with Class II furcation	3.2
		≥3 mm CAL	53.1 (19.6% of teeth)
		≥3 mm PD	63.9 (19.6% of teeth)

Case Definitions and Prevalence Used in Other Clinical Studies

Study	Case Name	Case Definition
Machtei et al.[13]	Established periodontitis	≥2 teeth with CAL ≥6 mm + ≥1 site with PD ≥5 mm
Moore et al.[68]	Severe generalized periodontitis	≥8 teeth with CAL ≥5 mm, PD ≥6 mm
Burmeister et al.[8]	Severe generalized periodontitis	≥8 teeth with CAL ≥5 mm (≥3 teeth not first molars)
Beck et al.[64]	Severe destructive periodontitis	≥4 sites with CAL ≥5 mm; ≥1 same sites PD ≥4 mm
Tomar and Asma[69]	Periodontitis	≥1 site with CAL ≥4 mm, PD ≥4 mm

Many studies have been conducted using different diagnostic classifications regarding periodontitis. The most common classification that used a single criterion was CPI/CPITN (Community Periodontal Index/Community Periodontal Index of Treatment Needs), which used PD ≥ 3.5mm as a cut point (13.4%). This classification consumes less time and is easy to apply in large samples of people. **[**47**]** International uniformity is the most important advantage, but it does not record irreversible changes such as recession or loss of periodontal attachment.

Another common definition which used two criteria was the CDC/AAP working group, published in 2007, for moderate and severe. **[**37**]**

Clinical Case Definitions Proposed by the CDC Working Group for Use in Population-Based Surveillance of Periodontitis*

	Clinical Definition		
Disease Category	CAL		PD
Severe periodontitis	≥2 interproximal sites with CAL ≥6 mm (not on same tooth)	and	≥1 interproximal site with PD ≥5 mm
Moderate periodontitis	≥2 interproximal sites with CAL ≥4 mm (not on same tooth)	or	≥2 interproximal sites with PD ≥5 mm (not on same tooth)
No or mild periodontitis	Neither "moderate" nor "severe" periodontitis		

* Third molars excluded.

In a systematic review of 351 studies on case definitions of chronic periodontitis, Natto et al (2018) found that overall

• 121 (34.5%) articles used both probing depth (PD) and clinical attachment loss (CAL) combined

• PD only (110 studies, 31.3%),

• CAL only (54 studies, 15.4%), or

• Radiograph only (19 studies, 5.4%). **[**36**]**

Several other criteria have been used and were summarized in a table.

Method	Number of studies(%) N=351
Single criteria	
CAL	54(15.4)
Radiograph	19(5.4)
PD	110(31.3)
ICD	1(0.3)
Combined criteria	
PD+CAL	121(34.5)
Radiograph + PD	5(1.4)
CAL + furcation	1(0.3)
CAL + radiograph	4(1.1)
PD+CAL+BOP	21(6.0)
PD+BOP	7(2.0)
Edema +BOP +PD+ recession +mobility	1(0.3)
PD+ CAL+ Radiograph	4(1.1)
PD+ Furcation	1(0.3)
PD+ Radiograph +BOP	2(0.6)

CAL: clinical attachment level, PD: probing depth, and BOP: bleeding on probing.

• A minimum of two sites was the most common diagnostic criterion used.

• Bitewing was the most common method used in radiographic studies (11 studies, 3.1%),

• In the combination diagnostic criteria, PD ≥ 4mm and CAL ≥ 3mm were the most common with 34 studies (9.7%), followed by PD ≥ 5 mm, CAL ≥ 4mm (26 studies, 7.4%).

The recent 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP), introduced new parameters based on certain articles. Several clinical examination methods, threshold values, and criteria of chronic periodontitis were used in this research, including measurement of probing depth (PD), clinical attachment loss (CAL), bleeding on probing (BOP), and alveolar bone loss with or without radiographs. **[**39**]**

Definition of a patient as a periodontitis case

In the context of the 2017 World Workshop, it was suggested that a single definition be adopted. A patient is a periodontitis case in the context of clinical care if:

1. Interdental CAL is detectable at ≥2 non-adjacent teeth, or

2. Buccal or oral CAL ≥3 mm with pocketing >3 mm is detectable at ≥2 teeth and the observed CAL cannot be ascribed to non-periodontal causes such as:

• Gingival recession of traumatic origin;

• Dental caries extending in the cervical area of the tooth;

• The presence of CAL on the distal aspect of a second molar and associated with malposition or extraction of a third molar;

• An endodontic lesion draining through the marginal periodontium

• The occurrence of a vertical root fracture.

Identification of the form of periodontitis

Based on pathophysiology, three clearly different forms of periodontitis have been identified:

1. Necrotizing periodontitis

2. Periodontitis as a direct manifestation of systemic diseases

3. Periodontitis

Additional elements

Since the 1999 International Classification Workshop, it has become apparent that additional information beyond the specific form of periodontitis and the severity and extent of periodontal breakdown is necessary to more specifically characterize the impact of past disease on an individual patient's dentition and on treatment approaches needed to manage the case.

The following additional factors were proposed before diagnosing a case of periodontitis:

• Severity of the disease

• Complexity of management

• Extent of the disease

• Rate of progression

• Risk factors associated with the disease

• Interrelationship with general health

A staging and grading system for classification of disease was also introduced.39 Goals of staging a periodontitis patient:

• Classify severity and extent of an individual based on currently measurable extent of destroyed and damaged tissue attributable to periodontitis.

• Assess complexity.

• Assess specific factors that may determine complexity of controlling current disease and managing long-term function and esthetics of the patient's dentition.

Goals of grading a periodontitis patient:

• Estimate future risk of periodontitis progression and responsiveness to standard therapeutic principles, to guide intensity of therapy and monitoring.

• Estimate potential health impact of periodontitis on systemic disease and the reverse, to guide systemic monitoring and co-therapy.

Periodontitis stage		Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third (<15%)	Coronal third (15% to 33%)	Extending to mid-third of root and beyond	Extending to mid-third of root and beyond
	Tooth loss	No tooth loss due to periodontitis		Tooth loss due to periodontitis of ≤4 teeth	Tooth loss due to periodontitis of ≥5 teeth
Complexity	Local	Maximum probing depth ≤4 mm Mostly horizontal bone loss	Maximum probing depth ≤5 mm Mostly horizontal bone loss	In addition to stage II complexity: Probing depth ≥6 mm Vertical bone loss ≥3 mm Furcation involvement Class II or III	In addition to stage III complexity: Need for complex rehabilitation due to: Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defect Bite collapse, drifting, flaring

Periodontitis grade			Grade A: Slow rate of progression	Grade B: Moderate rate of progression	Grade C: Rapid rate of progression
Primary criteria	Direct evidence of progression	Longitudinal data (radiographic bone loss or CAL)	Evidence of no loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss/age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g., molar/incisor pattern; lack of expected response to standard bacterial control therapies)
Grade modifiers	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
		Diabetes	Normoglycemic/ no diagnosis of diabetes	HbA1c <7.0% in patients with diabetes	HbA1c ≥7.0% in patients with diabetes
Risk of systemic impact of periodontitis[a]	Inflammatory burden	High sensitivity CRP (hsCRP)	<1 mg/L	1 to 3 mg/L	>3 mg/L
Biomarkers	Indicators of CAL/bone loss	Saliva, gingival crevicular fluid, serum	?	?	?

However, it is too early to assess the actual effect of this consensus and its acceptance globally.

A case definition system needs to be a dynamic process that will require revisions over time in much the same way the Tumor, Node, Metastasis (TNM) staging system for cancer has been shaped over many decades.

It needs to be:

1. Simple enough to be clinically applicable but not simplistic: additional knowledge has distinguished dimensions of periodontitis, such as complexity of managing the case to provide the best level of care.

2. Standardized to be able to support effective communication among all stakeholders.

3. Accessible to a wide range of people in training and understood by members of the oral health care team around the world.

Conclusion

The case definition for a disease is the key factor for any specialty. It is different from diagnosis because case definitions must be more quantitative, specific, and accurately measurable and relatively few in number. There are different definitions for chronic periodontitis in the literature, which can affect estimates of prevalence, incidence, and treatment strategies. It is also clear that variation in threshold values—for CAL, PD, radiograph, or any combination at a given site—leads to different diagnosis of chronic periodontitis at that site. In addition, the number of involved sites are also equally important. Clear definitions of the disease and associated threshold values and criteria should be established worldwide to ensure accurate results in future studies.

Role of Genetics in Periodontal Disease

Periodontal diseases are a heterogeneous group of diseases that affect hundreds of millions around the world. Periodontal disease may be regarded as a range of different diseases for which certain individuals are at relatively high risk. There is now significant clinical and scientific evidence apart from microbial and other environmental factors, genetic factors are important determinants of periodontitis susceptibility and progression by influencing inflammatory and immune responses in general, and periodontitis experience specifically. **[**48**]**

Periodontal diseases have many of the characteristics of complex diseases that make genetic studies difficult. These characteristics include difficulty in measuring and classifying disease phenotypes, the temporal nature of disease and the complex interaction of host, genetic, microbial and other environmental factors. However, the advent of current research tools makes it possible to begin to identify and partition specific elements of susceptibility and to incorporate these into periodontal disease models. **[**49**]**

Human Genes

• Each chromosome contains a single, very long duplex of DNA.

• DNA consists of chemically linked sequences of nucleotides which constitute the building blocks of DNA.

• Nucleotide always contains a nitrogenous base Adenine (A), Guanine (G), cytosine

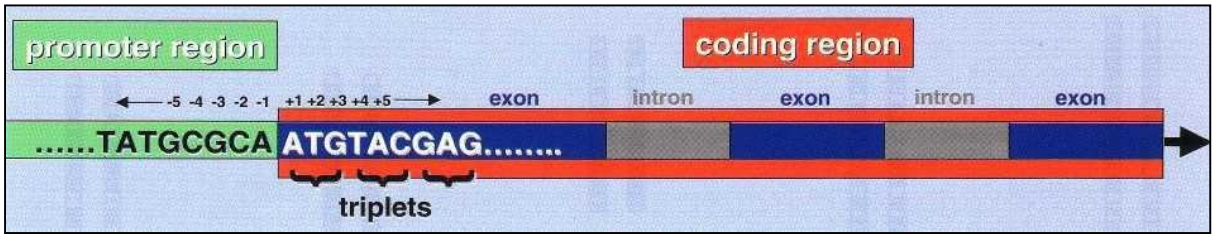
(C) and Thymine (T).

• In chromosome, base pairing is complementary, i.e., its always A-T & G-C.

• The haploid human gene i.e., 22 chromosomes and one sex chromosome consists of 3.3 x109 nucleotides i.e., 3.3x109 base pairs (bp).

• DNA contains the genetic code and given specific sequence of nucleotides encodes for the sequence of amino acids that constitutes the corresponding protein.

• The genetic code is read in groups of three nucleotides and each trinucleotide sequence i.e., Triplet is a Codon. **[**50**]**



Gene structure consisting of coding region - that codes for a sequence of amino acids to form a protein; and non coding region - essential for the regulation of the transcription of the coding region.

Geneticists refer to the different forms of a gene as allelic variants or alleles. Allelic variants of a gene differ in their nucleotide sequences.

• Gene polymorphism is the occurrence in a population of two or more alleles at a locus in frequencies greater than can be maintained by mutation. Polymorphisms are genetic differences that provide variation within species. A polymorphism is "Silent" when a nucleotide change in a codon does not alter the protein synthesis.

- Gene polymorphisms may be a result of gene mutation OR insertion/ deletion.
- The variation at the site harboring such changes has recently been termed a "single nucleotide polymorphism" (SNP). **[**51**]**
- Restriction Fragment length polymorphism (RFLP) Digestion of a piece of DNA containing the relevant site with an appropriate restriction enzyme could then distinguish alleles or variants based on the resulting fragment sizes via electrophoresis. **[**52**]**
- Repeated base patterns can consist of several hundreds of base pairs (size 1Kb-30Kb in length), known as "variable number of tandem repeats" (VNTRs)
- Mutation is an alteration in the genomic sequence compared to a reference state. When a nucleotide change is very rare, and not present in many individuals, it is often called Mutation, whereas genetic polymorphisms are usually considered normal variants

in the population. An alteration that changes only a single base pair is called a point mutation.

The contribution of an allelic variant to a disease can vary from being deterministic to having only a minor effect on the etiology. The manner and extent to which genetic factors play a role in disease have important implications for identifying the genetic basis of etiology and for utilizing this information for diagnosis and treatment.

Many disease-associated genetic polymorphisms are common in the population and can be present at allele frequencies of >20%, with some disease-associated alleles reported in

>50% of\ populations studied.

Currently, major classes of genetic diseases include

• Single Gene Disorders

• Chromosomal abnormalities

• Multifactorial Traits

Single Gene Disorders

These include the otherwise called Mendelian or Monogenic disorders i.e., conditions that are produced by the effects of one of the gene or a gene pair. They are usually transmitted in simple patterns as originally described by Gregor Mendel. It can be Autosomal dominant, Autosomal recessive or X-linked.

Chromosomal abnormalities

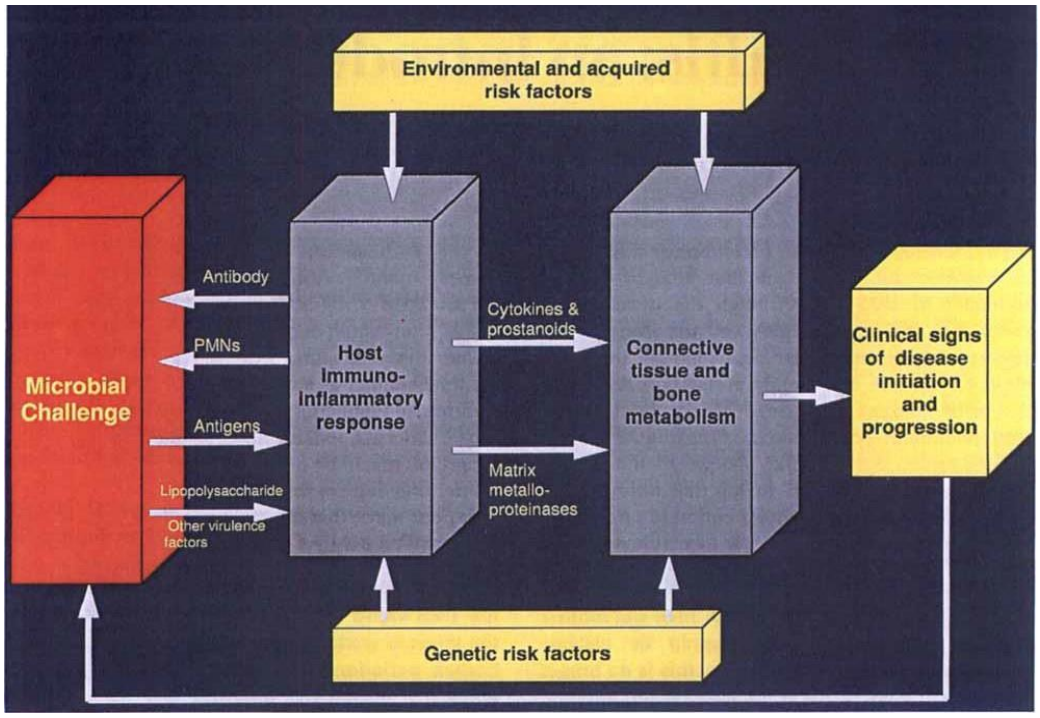
This group of diseases occurs due to deviations from normal chromosome number or structure. Major example includes Down syndrome, Turner syndrome etc.

Multifactorial Traits

This group is the largest and is due to combined effects of multiple genetic and non- genetic influences.

Periodontitis is considered to be a complex disease. The pathophysiology of complex diseases is characterized by various biological pathways, leading to similar clinical phenomena.

Periodontitis is not a single homogeneous disease but rather consists of a family of closely related diseases each of which may vary somewhat in etiology, natural history, and response to therapy. Nevertheless, a common underlying chain of events in the pathogenesis is shared by all forms of the disease. This common chain of events is influenced by other factors including genetic and other risk factors that may differ from one form of the disease to another. **[**53**]**



Pathogenesis of human periodontitis

A study from 1966 is one of the earliest studies from which it could be deduced that certain individuals are more at risk for periodontitis than others (Trott & Cross 1966). In this study the principal reasons for tooth extractions in over 1800 subjects were investigated. The figures

showed that in each age category the percentage of teeth lost due to periodontal disease is always higher than the percentages of patients who lost teeth due to periodontal disease. This means that relatively many teeth are lost in relatively few patients. **[**55**]**

The concept of high risk for the development of periodontitis was further confirmed in longitudinal studies investigating the natural history of periodontal disease. (Hirschfeld & Wasserman 1978; McFall 1982). **[**55**]**

Löe et al. (1986), in a Sri Lankan population without dental care and absence of oral hygiene, identified three subpopulations:

~ a group with no progression (11%),

~ a group with moderate progression (81%), and

~ a group with rapid progression of periodontal breakdown (8%).

In a recent study the initiation and progression of periodontal breakdown was studied in a population deprived from regular dental care in a remote village on Western Java (Van der Velden et al. 2006). The authors found that 20% of the subjects developed severe breakdown whereas the remaining population developed minor to moderate breakdown. **[**55**]**

These variations must have been attributable to either unrecognized environmental factors or to individual differences in susceptibility to disease which may be defined in terms of genetic variation. Because of complex etiology and pathogenesis of periodontitis, variations in any number or combinations of genes that control the development of periodontal tissues or the competency of the cellular and humoral immune systems could affect an individual's risk for disease. The phenomenon that a relatively small proportion of the population is at risk for developing severe forms of periodontitis may suggest that not everybody is equally susceptible to periodontitis. The existence of high-risk groups cannot be explained by the microbiology alone. There are, however, other factors that may play a major role in the development of periodontitis, i.e. the inflammatory and immune response both locally and systemically.

Geneticists use a variety of techniques to demonstrate the genetic basis of disease. Some methods are general, whereas others permit precise identification of genetic variants that cause or contribute to disease.

- Familial aggregation - Familial aggregation of a trait or disease can suggest genetic etiology.
- Twin studies - The influences of genetics in disease and the relative contribution of genes and environment to a trait can be examined through the phenomenon of twins, in particular monozygous twins, who arise from one fertilized egg.
- Segregation analysis - Geneticists can study the pattern of disease transmission in families using a method called segregation analysis.
- Linkage analysis - is a technique used to localize the gene for a trait to a specific chromosomal location. Linkage is often used as a first step to determine the approximate location of a gene of interest, permitting subsequent studies to identify the mutation responsible for a disease trait.
- Association studies – These studies are done when multiple, perhaps many, genes act with environmental factors to contribute to disease liability or if a disease gene is neither necessary nor sufficient to cause a disease. **[**56**]**

Genetic studies in periodontics

It is clear that periodontitis severely affects a high-risk group representing around 10- 15% of the population, in whom the disease quickly progresses from chronic gingivitis to destructive periodontitis (Jenkins and Kinane, 1989). **[**55**]**

The results of twin studies and clinical observations regarding the aggregation of cases of aggressive forms of periodontitis in families, along with the presence of genetic diseases or syndromes in which periodontitis is a major clinical characteristic, implicate genes in the etiology of periodontitis. Many twin studies have concluded a significant association between periodontal disease and genetic susceptibility. **[**55**]**

Corey et al. (1993) studied 4908 twin pairs and found that 9% of subjects, consisting of 116 identical and 233 nonidentical twin pairs, reported a history of periodontitis. The concordance rate, or level of similarity in disease experience, ranged from 0.23 to 0.38 for monozygous twins, and was much lower (0.08–0.16) for dizygous twins. They concluded that heritable factors are important in the reported periodontitis experience. Michalowicz et al. (1991) studied dizygous twins reared apart (dizygous-A) and reared together (dizygous- T) and monozygous twins reared

apart (monozygous-A) and reared together (monozygous-T). The mean probing depth and clinical attachment level scores were found to vary less for monozygous- T than for dizygous-T twin pairs, further supporting the role of genetics in this disease. Michalowicz et al. (1991) investigated alveolar bone height and showed significant variations related to genotype. The twin groups had similar smoking histories and oral hygiene practices. It was concluded that genetics plays a role in susceptibility to periodontal disease. In a subsequent study of 117 adult twin pairs, Michalowicz and coworkers (2000) estimated genetic and environmental variances and heritability for gingivitis and chronic periodontitis. Monozygous twins were found to be more similar than dizygous twins for all clinical measures. Statistically significant genetic variance was found for both the severity and the extent of disease. **[**55**]** However, it seems clear that most forms of periodontitis with postpubertal onset are not inherited as Mendelian diseases, that is, they are probably not caused by major genes. Rather, many polymorphic genes with relatively small but significant associations with disease risk may interact to contribute to overall risk.

These results confirm previous studies and indicate that approximately half of the variance for chronic periodontitis is attributable to genetic variance and the basis for the heritability of periodontitis appears to be biological and not behavioural.

Segregation analyses can evaluate the relative support for different models to identify the one that most closely represents the clinical data. Various forms of aggressive periodontitis (e.g., prepubertal and juvenile periodontitis) have been observed in the same family and found to occur sequentially in the same individual. These findings suggest that there are common genetic risk factors for the subforms of AP disease. Melnick et al (1976) proposed X-linked inheritence because of the preponderence of female probands and affected family members. **[**55**]**

Saxen L et al (1980, 1984): An autosomal recessive mode of inheritance was clearly favored in Finnish populations where parents of probands were not consistently unaffected.

Marazita and coworkers (1994) ~ studied more than 100 North American families segregating aggressive forms of periodontitis. Their results supported an autosomal dominant transmission.

They concluded that autosomal dominant inheritance with approximately 70% penetrance occurred for both Blacks and non- Blacks. Beaty et al (1987) recognized that the narrow age range

in which AP diseases can reliably be diagnosed increases the chance that an incorrect model of inheritance is favored over the true one. **[**55**]**

Linkage studies have been performed on families with localized aggressive periodontitis. Boughman et al. (1986) identified an autosomal dominant form of localized aggressive periodontitis in an extended family from Southern Maryland. In this family, an autosomal dominant form of AP disease was found to cosegregate with dentinogenesis imperfecta-

III. The putative AP disease gene was localized to the long arm of chromosome 4(4q11- 13) near the gene for dentinogenesis imperfecta-III. They demonstrated a relatively close linkage with the suspected locus for aggressive periodontitis.

Hart et al. (1993) evaluated support for linkage to this region of chromosome 4 in a different population of families (14 African American and 4 Caucasian). Findings supported genetic locus heterogeneity of aggressive periodontitis, as they excluded a chromosome 4 major gene locus for aggressive periodontitis in the families they studied. These findings support genetic heterogeneity, with at least one gene locus responsible for aggressive periodontitis located on chromosome 4. Li and coworkers (2004) reported evidence of a gene responsible for localized aggressive periodontitis located on chromosome 1q25. Combinations of genes, whether organized along a segment of a chromosome or not, may also be associated with risk in a manner not reflected by the risk imparted by individual genes. It is important to recognize that probably only a few of the genetic variants that can contribute to increasing or decreasing risk for periodontitis have been identified, and once they are, their interrelationships and modes of interaction with each other and the environment will have to be assessed.(10)

Polymorphism	Gene
IL-1A (+ 4845) and IL-1B (+ 3954)	IL-1 gene
TNF-α-308 allele 1	TNF-α gene
TNF-β NcoI, ET-1 gene, and ACE gene insertion/ deletion polymorphism	lymphotoxin alpha (TNF-β), ET-1 and ACE genes
FcγRIIIb-NA2 allotype	Fc receptor polymorphism
NAT2	*N*-acetyltransferase polymorphism

Table - Genes associated with chronic periodontitis

Polymorphism	Gene	Disease association
IL-1A (+4845) and IL-1B (+3954)	IL-1 gene	early onset periodontitis
IL-4 promotor and intron polymorphisms	IL-4 gene	early onset periodontitis
FcγRIIIb-NA2 allele (and possibly FcγRIIIa-158F)	Fc receptor gene polymorphisms	early onset periodontitis/ generalized early onset periodontitis
Gc locus chrom 4q	unknown	early onset periodontitis/ localized juvenile periodontitis
fMLP receptor	*N*-formyl peptide receptor polymorphisms	early onset periodontitis/ localized juvenile periodontitis
VDR gene	vitamin D receptor polymorphism	early onset periodontitis/ localized juvenile periodontitis

Table - Genes that have been found to have association with aggressive periodontitis (10)

Syndromic forms of periodontitis

Severe periodontitis presents as part of the clinical manifestations of a number of monogenic syndromes and the gene mutation and biochemical defect is known for many of these conditions. A commonality of these conditions is that they are inherited as simple Mendelian traits due to genetic alterations of a single gene locus. **[**57**]**

Genetic defect	Disease	Phenotype
Collagen folding defect	Ehlers–Danlos syndrome type 8	early onset periodontitis/ localized juvenile periodontitis
CTSC gene on chromosome 11q14-q21	Papillon–Lefèvre syndrome, Haim–Monk syndrome	prepubertal periodontitis
Multiple possible mutations in alkaline phosphatase gene	hypophosphatasia, alkaline phosphatase deficiency	prepubertal periodontitis
LAD1 (integrin), LAD2 (selectin) gene defect	leukocyte adhesion deficiency	prepubertal periodontitis
OCRL1 gene, X-chromosome	Lowe syndrome	prepubertal periodontitis (atypical finding)

The significance of these conditions is that they clearly demonstrate that a genetic mutation at a single locus can impart susceptibility to periodontitis. Additionally, these conditions illustrate that this genetic susceptibility may segregate by different transmission patterns. Because altered proteins function in different structural and immune pathways, genetic modulation of a variety of different genes can affect a variety of different physiological and cellular pathways.

Cytokine gene polymorphisms

Rationale for studying cytokine gene polymorphisms -

• To enhance the understanding of the etiology and pathology of human disease.

• To identify potential markers of susceptibility, severity, and clinical outcome.

• To identify potential markers for responders vs. non-responders in therapeutic trials.

• To identify targets for therapeutic intervention.

• To identify novel strategies to prevent disease or to improve existing preventions such as vaccines.

In a recent review by Heidari et al (2019) that summarized some functional biomarkers that are associated with CP susceptibility. There is some evidence that SNPs in the IL- 1α, IL-1β, IL-6, IL-10, TNF-α, IFN-γ and may be associated with CP susceptibility. **[**59**]**

Cytokine	No. of studies that show association of the gene with periodontal disease	No. of studies that show no association of the gene with periodontal disease
IL 1alpha	15	20
IL 1beta	25	14
IL 6	10	7
IL 10	9	16
IFN GAMMA	2	5
TNF ALPHA	7	15
TGF beta	6	7

Thus, not all studies have demonstrated the relationship of these genotypes with either disease severity or prognostic ability.

Vitamin D receptor (VDR) gene polymorphisms:

Till now the actual role of the VDR SNPs in CP susceptibility have not been clarified completely. it has been identified that VDR SNPs, such as Taq1, Bsm1, Fok1, and Apa1 were associated with CP(12). In some studies, the Taq1N-allele has been related to CP susceptibility. There was not an association between VDR Bsm1 SNP and CP **[**160– 162**]**.Given that the VDR gene can affect both immune functions and bone metabolism, therefore, VDR SNPs, specially the VDR Taq1 may be risk factors for CP susceptibility. Further studies should be undertaken to confirm the current preliminary data.

HLA gene polymorphisms

Human leukocyte antigens (HLA) are involved in genetically predetermined humoral immune response via recognition of foreign antigens. ‘‘Classical’’ MHC Class I molecules (HLA-A, -B, and -C) are expressed on most nucleated cells. MHC Class II molecules (HLA -DP, -DQ, -DR) are expressed on cells that immunosurvey host cells including B and T cells, macrophages and accessory cells for the presence of foreign peptides. MHC molecules play a central role in immune responses to protein antigens and in autoimmunity. The MHC genes are the most polymorphic genes present in the genome of every species analyzed.

The most clinically relevant genes are the DRA, DRB1, DQA1, DQB1, and DPA1, DPB1 genes encoding the DR, DQ, and DP heterodimers, respectively. Takashiba S et al 1999

~ suggested that patients with the HLA-DRB1*1501-DQB1*0602 genotype may have an accelerated T cell response to P. gingivalis and an increased susceptibility to EOP in Japanese patients.

Studies have shown that HLA A9 and B15 is associated with the generalized form, but not the localized form, of early-onset periodontal diseases (Shapira et al., 1994). Also, HLA-A10 shows a significantly increased incidence in the resistant population (Amer et al., 1988) and significant association has been found between JP and HLA-DR2 and HLA-A33 (Cogen et al., 1986). **[**55**]**

Clinical implications of studies of genetic polymorphisms

The studies showing polymorphisms associated with periodontitis illustrate a number of important points with respect to the clinical interpretation of this type of information.

• The associations between particular genes and disease may only (thus far) be apparent in certain populations and not in others. Thus, genetic tests based upon these genes may not apply to all patients.

• The associations between groups of interacting genes and disease may be stronger than those between individual genes and disease. Therefore, as more genetic risk factors are found, genetic tests for disease risk will continually evolve and merit scrutiny and evaluation.

• The associations between disease and genes may be indirect, that is, genetic factors may be associated with environmental risk factors for periodontitis (e.g. smoking) and thereby influence disease only in those patients with the relevant biological exposure. **[**57**]**

These points could be of great importance when interpreting the results of genetic testing or evaluating clinical approaches based upon genetic tests.

Conclusion

The determination of all environmental and genetic factors that can influence disease and the development of assessment protocols to determine an individual patient's risk profile should allow optimal determination of risk and individualized approaches to both prevention and therapy. In addition, life-long screening modalities for both genetic and environmental factors should limit the effects of disease and enhance the effectiveness of therapies. Although current dental practice does not commonly integrate current (and incomplete) knowledge of genetic risk factors into patient care, there will ultimately be a revised approach to patient care that will incorporate genetic information on a regular basis.

Stress and Periodontal Disease

In 2016, major depressive disorder ranked in the top 10 of Years Lived with Disability (YLDs) in all 195 countries and territories except only 4 countries. Anxiety disorders also ranked in the top 10 of YLDs in more than half of the countries and territories. Stress, a term continually being redefined in the scientific study of disease and illness, is nevertheless a confirmed and important factor in the etiology and maintenance of many inflammatory diseases, including periodontal disease. **[**60**]**

The association of stress with periodontal disease is difficult to prove as there are many factors influencing the incidence and severity of periodontal disease, some of which are assumed and have not been identified. Nevertheless, studies indicate that psychosocial stress represents a risk indicator for periodontal disease and should be addressed before and during the treatment. **[**61**]**

Basic concepts and mechanisms

At the heart of the concept of stress is an attempt to understand how the body regulates itself to maintain smooth, adaptive and homeostatic functioning when confronted with disruptive endogenous or exogenous forces. **[**60**]**

It was Hans Selye, who was largely responsible for giving the term ‘stress’ its current saliency in relation to the contest between health and disease. Selye defined stress as a response state of the organism to forces acting simultaneously on the body which, if excessive – that is, straining the capacity of adaptive processes beyond their limits – led to diseases of adaptation and eventually to diseases of exhaustion and death. Selye defined forces that had the potential to challenge the adaptive capacity of the organism as ‘stressors’. **[**64**]**

Psychosocial stressors can generally be classified as: **[**62**]**

1. Major stressful life events

2. Minor daily stressors or “hassles.”

Stressors acting to produce changes in the body could be

1. Positive (e.g. exciting, pleasurable), leading to a response state ‘eustress’,

2. Negative, threatening homeostasis with pain, discomfort and physical pathology, leading to a negative response state as ‘distress’. **[**64**]**

Research has shown that psychosocial stress can modulate the immune system through the neural and endocrine systems in at least 4 different ways:

1. Through the autonomic nervous system pathways

2. Through the release of neuropeptides

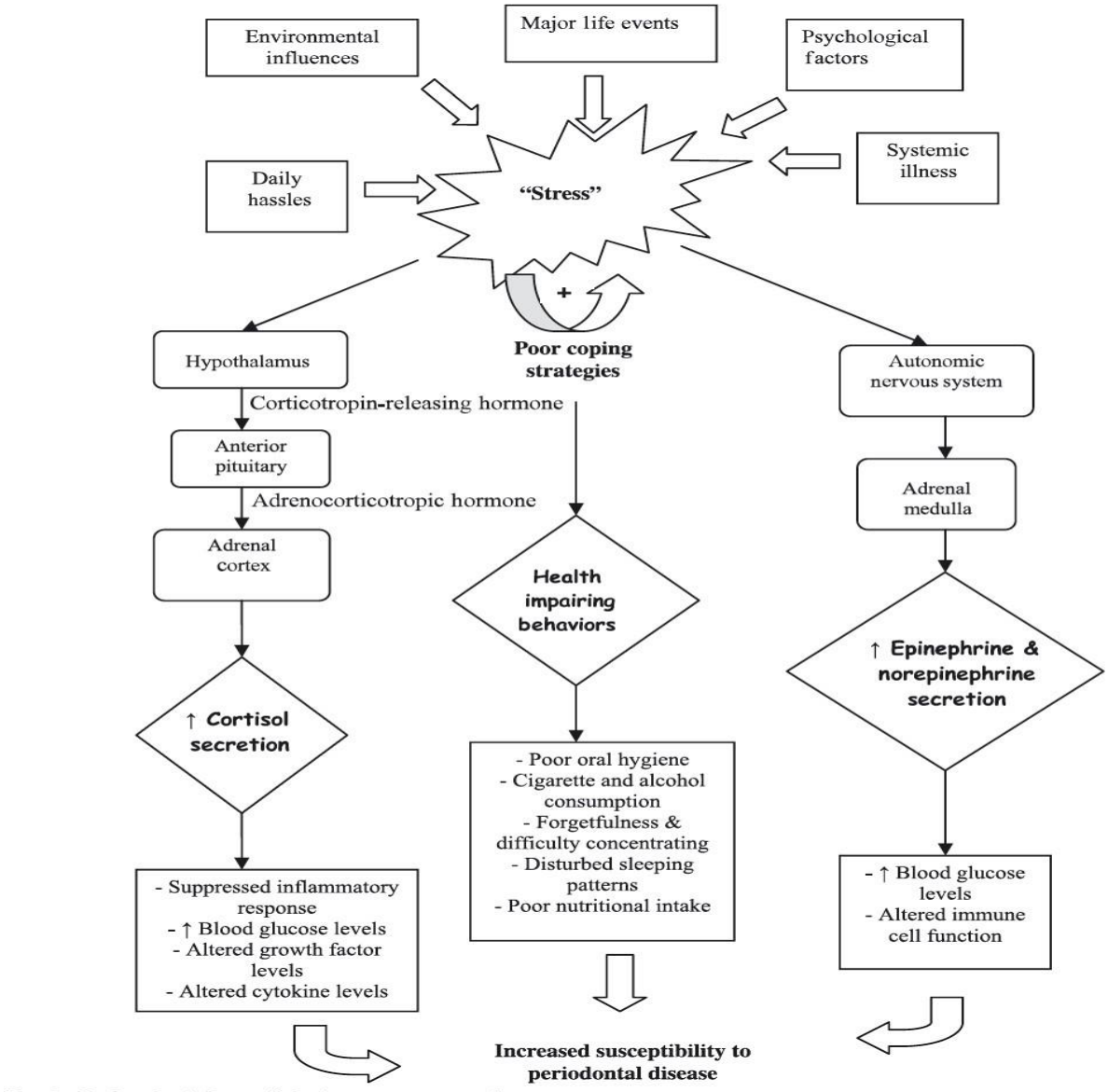
Stress can induce the release of neuropeptide from sensory nerve fibers (neurogenic inflammation), and the presence of neuropeptides has been implicated as a neurogenic promoter in various inflammatory processes modulating the activity of the immune system and the release of cytokine.

3. Through the release of hypothalamic, pituitary and adrenal hormones.

Stress induced response is transmitted to HPA axis and promotes the release of corticotropin releasing hormones from pituitary gland and glucocorticoid hormone from adrenal cortex-altering production of inflammatory cytokines (IL1,TNF a, PGE2).

4. The sympathetic nervous system

Exposure to stressor can induce sympathetic nervous system to release adrenaline and noradrenaline from adrenal medulla and exerts immunosuppressive effects which can indirectly promote periodontal tissue breakdown. **[**60, 62**]**



Pathophysiology of stress response

When an infection occurs it is the inflammatory response that allows marshalling of immune system elements at specific sites. Early events in the inflammatory reaction to infection are typically clinically undetectable. As the infectious process becomes more chronic, clinically evident inflammation occurs, generating high levels of cytokines and other mediators of inflammation associated with activation of the stress system. If the inflammatory reaction is

both prolonged enough and profound enough, systemic illness manifestations can also become clinically evident. [65]

However, accumulating evidence over several decades makes it clear that corticotropin releasing hormone, catecholamines and other elements of the stress system may in fact influence the immune system in both directions, whether at resting (baseline) levels or at elevated levels associated with stress. Locally, stress may then exert a pro- or anti- inflammatory effect on tissues that may be influenced by such factors as the specific organ involved or the presence or absence of particular immune cell receptor subtypes. It seems fair to conclude that dysregulation of the stress system is involved in a number of major health problems, but it would be difficult to distinguish between cause and effect since the system is, to a large extent, nonspecific and responds in similar ways to a wide variety of endogenously and exogenously arising stressors. [65]

Stress and periodontal disease

Models of stress and periodontal disease

Several reviews have sought to synthesize current concepts underlying stress phenomenology into evidence-based models linking stress with periodontal disease. Some of these models include elaboration of stressors from both the physical and psychosocial domains that may serve as risk factors for periodontal disease. [66, 67]

Model 1

Mental stress response triggering the HPA axis with immunosuppressive effects. According to MODEL-1, psychosocial stress can activate the central nervous system.

The hypothalamus releases CRH which, among other things, stimulates release of ACTH from the pituitary, which in turn results in production of cortisol by the adrenal cortex. Glucocorticosteroids, including cortisol, then depress immunity including secretory IgA, IgG, and neutrophil functions, all of which may be important in protection against infection by periodontal organisms.

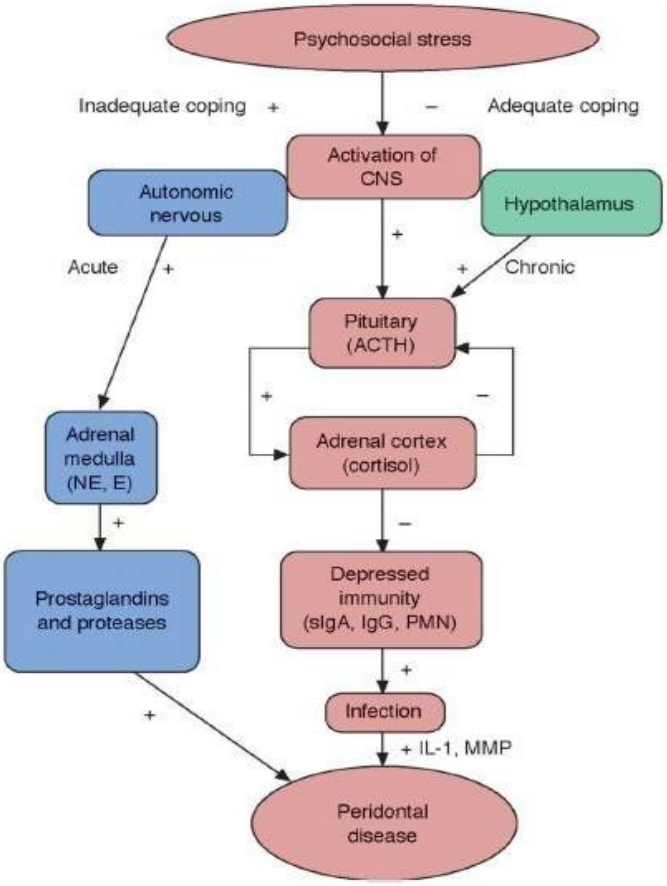


Figure : MODEL 1 - Mental stress response triggering the HPA axis with immunosuppressive effects

Model 2

In the second model of the role of psychosocial stress on periodontal disease, it is hypothesized that the main effects of stress occur through behavioural changes which affect at risk health behaviors such as smoking, poor oral hygiene, and poor compliance with dental care.

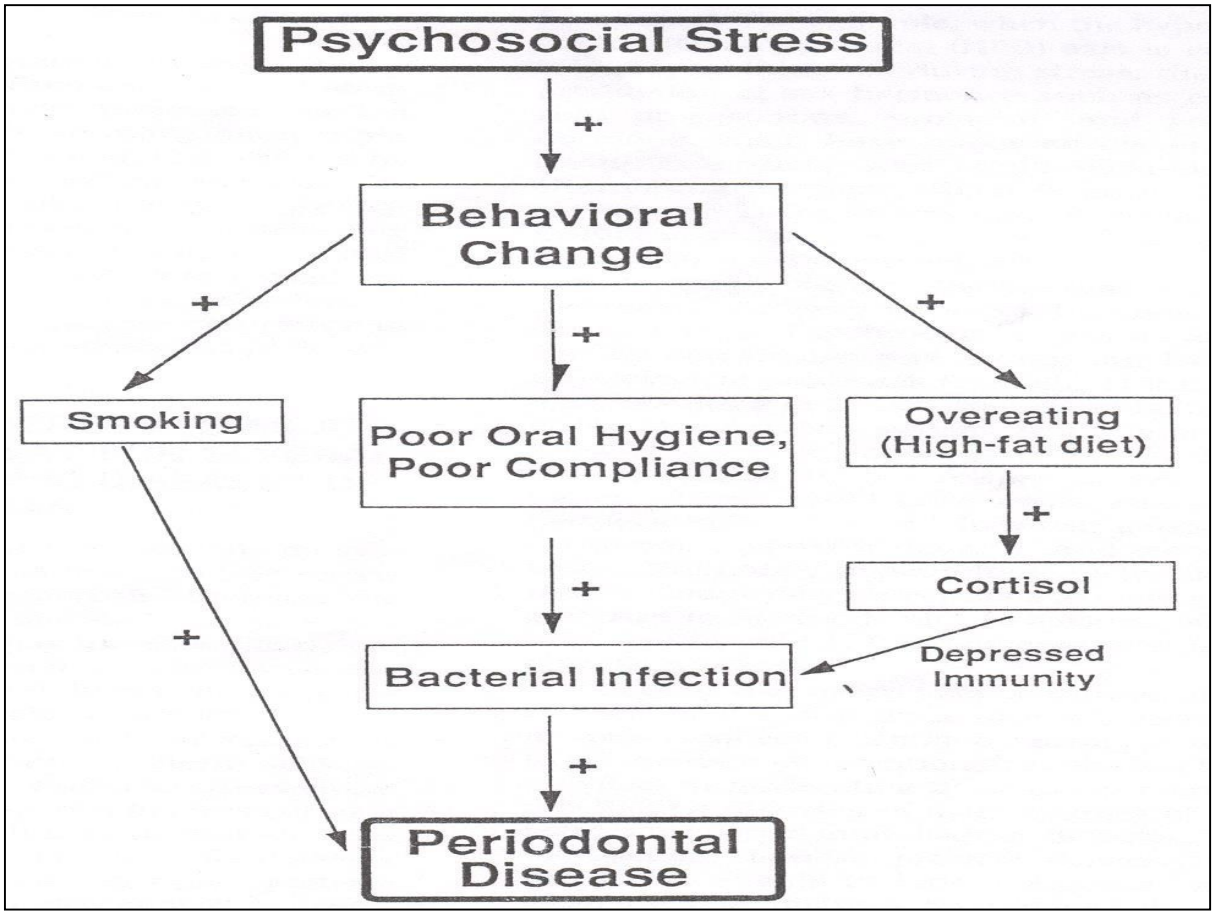


Figure : MODEL 2 - Effects of stress occur through behavioural changes

A number of mechanisms have been proposed, which could mediate the putative relationship between psychosocial conditions and inflammatory periodontal diseases.

Endocrine changes

It has been suspected that periodontal status is related to alterations in the concentration of adrenal corticoids and by altering the response of oral tissues to bacterial toxins and other hormones involved in the general adaptation. **[**60**]**

Studies have confirmed the fact that the concentration of cytokines (IL-6, IL-1 β etc.,) and cortisol in GCF is higher in persons showing signs of depression. **[**68-72**]**

Neglect of oral hygiene

It is obvious that proper oral hygiene is partially dependent on the mental health status of the patient. It has been reported that psychological disturbances can lead patients to neglect oral hygiene and that the resultant accumulation of plaque is detrimental to the periodontal tissue. Academic stress was reported as a risk factor for gingival inflammation with increasing crevicular interleukin-b levels and a diminution of quality of oral hygiene. **[**73-78**]**

Changes in dietary intake

Emotional conditions are thought to modify dietary intake, thus indirectly affecting periodontal status. Psychological factors affect the choice of foods, the physical consistency of the diet, and the quantities of food eaten. This can involve, for instance, the consumption of excessive quantities of refined carbohydrates and softer diets requiring less vigorous mastication and therefore predisposing to plaque accumulation at the approximal risk site. **[**62**]**

Smoking and other harmful habits

Circulating nicotine results in vasoconstriction, produced by the release of adrenaline and noradrenaline, which is supposed to result in a lack of nutrients for the periodontal tissue; suppression of secondary antibody responses and inhibition of oral neutrophil function. **[**63**]**

Alteration in salivary flow and its components

Manhold et al. tested the hypothesis that in long or continued emotions a constant constriction of the blood vessels would produce a lack of oxygen and nutrient materials for the periodontal tissue. They found a lower ability of the tissues of rats under stress to utilize oxygen.

Furthermore, smoking and stress have been implicated in reducing gingival blood flow which in turn, could increase the possibility of necrosis of tissue, with subsequent reduced resistance to plaque. **[**63**]**

It is assumed that both increase and decrease in salivary flow, induced by emotional disturbance, may affect the periodontium adversely. Emotional distress may also produce changes in saliva pH and chemical composition like IgA secretion. These relationships between salivary physiology and psychological status do not necessarily demonstrate causation of periodontal disease, but they show a pathway in which periodontal health is influenced by salivary changes.79

Lowered host resistance

As outline previously, stress and its biochemical mediators may modify the immune response to microbial challenge, which is an important defense against inflammatory periodontal disease.

Stress and systemic inflammatory diseases

It is well-established that cardiovascular disease, diabetes mellitus, preterm delivery, osteoporosis, rheumatoid arthritis, inflammatory bowel disease, systemic lupus erythematosus etc., are associated with stress either as a physiological response to stress or as a behavioral response. It may be that stress is a significant common risk factor for diabetes mellitus, cardiovascular disease, preterm delivery, and osteoporosis, as well as periodontal disease. The more severe bouts of all these conditions involve activation of the immune response and an associated increase in inflammation. **[**63**]**

Stress and wound healing

Patients with maladaptive coping strategies have more advanced disease and poor response to non-surgical treatment, whereas positive correlation was observed in reduction of dental plaque and gingival bleeding in patients having an active coping. **[**80**]**

Furthermore, the cellular immune response plays a vital role in wound healing. Not only does it protect the wound site from infection, it also prepares the wound for healing and regulates its repair. Cytokines such as IL-1, IL-8, and TNF are extremely important in recruiting phagocytic cells to clear away the damaged tissue and to regulate the rebuilding by fibroblasts and epithelial cells. A decrease in expression in any of these cytokines could theoretically impair wound healing. Stress could suppress certain aspects of the cellular immune response such as mitogen stimulation, antibody and cytokine production, and NK cell activity.

Since stress deregulates inflammatory and immune response, stress can alter the course of oral wound healing and affect the management of other oral diseases, e.g., periodontitis. **[**81**]**

Evidence for the role of stress in periodontal disease

Belting CM and Gupta P (1961) conducted a study and reported that psychiatric patients presented significantly higher periodontal scores than their controls when brushing frequency, calculus, bruxism and clenching were held constant. The authors suggested that the periodontal changes in the psychiatric patients were mediated through one or more processes related to anxiety, and under the control of the autonomic nervous system. **[**63**]**

In a review of psychosocial factors in inflammatory periodontal disease reported in 1995, Monteiro da Silva et al. distinguished between acute necrotizing ulcerative gingivitis and adult periodontitis, concluding that the evidence is strong for stress as a predisposing factor to acute necrotizing ulcerative gingivitis, while the evidence for psychosocial factors as etiological agents in periodontitis is not as substantive. **[**82**]**

In a series of laboratory studies using mice, Shapira et al. found that an ‘emotional’ stressor (isolation) and a physical stressor (cold), compared to control, had the effect of modifying the

inflammatory response following introduction of Porphyromonas gingivalis, through suppression of macrophages, increased secretion of nitric oxide and reduction of TNF-a. **[**83, 84**]**

On the contrary, in a study of gingivitis in 314 children aged 6–8years, levels of urinary catecholamines (epinephrine, norepinephrine and dopamine) were not related to gingivitis as recorded by a gingival bleeding index. **[**85**]**

In a prospective study, severe deterioration in gingival health from baseline levels was observed significantly more frequently in a cohort after they had undergone a period of academic examinations compared to a peer-control group not experiencing such academic testing.86 In a separate paper, Deinzer et al. report an experiment to assess the relationship between academic stress and gingival inflammation, examination students showed significantly higher levels of interleukin-1b at both the experimental gingivitis sites and the sites of good oral hygiene, indicating that stress may affect periodontal health through suppressed immune system activity, and that such a relationship would be more pronounced when oral hygiene was not maintained.87 Hypothesis generating studies concerning psychosocial variables including the case reports by Moulton et al. and De Marco. observed a presumed primary relationship between stress variables and periodontitis. **[**88, 89**]**

In a recent meta-analysis, Araujo et al in 2016 on association between depression and periodontitis the authors failed to affirm any association between depression and periodontitis. **[**90**]**

In another recent meta-analysis in 2018 on emotional disorders as risk factors for periodontitis, the authors suggest that the association between emotional disorder and chronic periodontitis was statistically significant. **[**63**]**

Conclusion

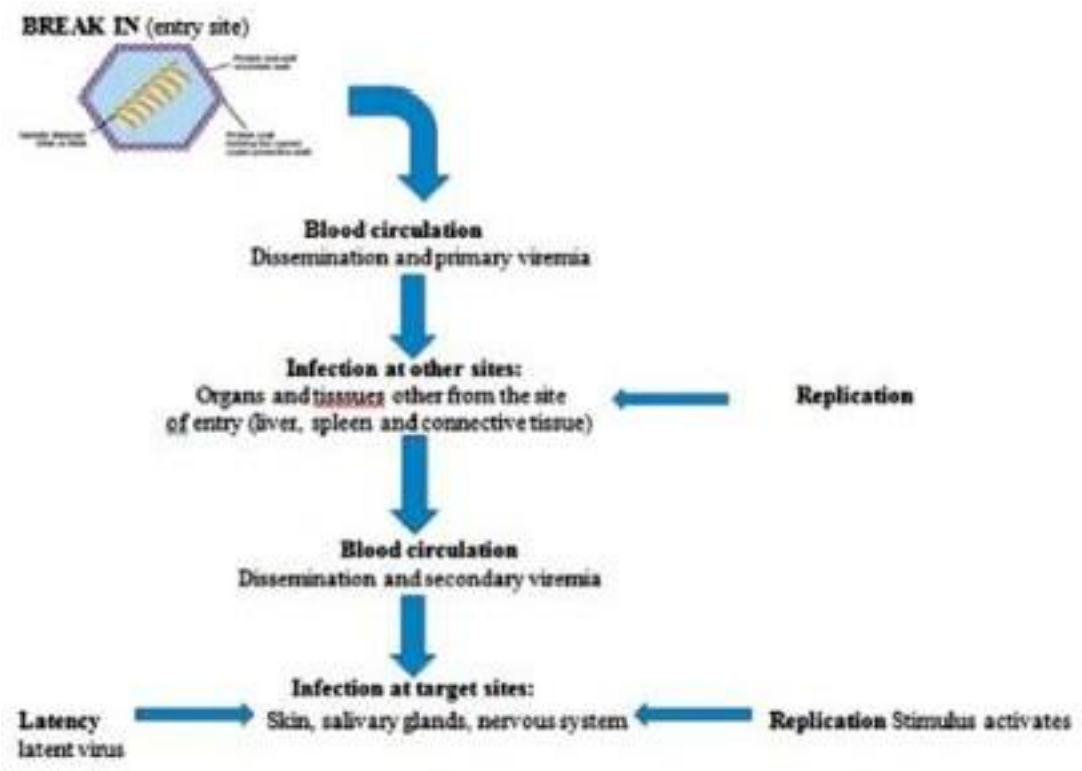
Most studies showed a positive relationship between stress/psychological factors and periodontal disease. But lack of adequate study models and difficulty in quantifying the amount and duration of stress, could lead to varying results. Furthermore, multiple variables affect the severity of periodontal disease and there is uncertainty about the individual's onset of the disease. Moreover, it is not possible to separate the effects of physical stress from emotional stress in these studies. Also, it is likely that systemic diseases associated with periodontal disease such as diabetes, cardiovascular disease etc., may share psychosocial stress as common risk factor. The available scientific evidence thus, does not definitively support a causal relationship between psychosocial factors and inflammatory periodontal diseases. The information, nevertheless, does indicate the possible influence of psychosocial factors in the etiology of inflammatory periodontal diseases.

Role of Virus in Periodontal Disease

Although the critical role of bacteria in the development of periodontitis is universally recognized, bacteria alone seem unable to explain the site-specificity and other characteristic features like rapid attachment loss and bone destruction with minimal plaque and existences of disease activity and quiescence phases. It is not understood why, in hosts with comparable levels of risk factors, some periodontal infections result in loss of periodontal attachment and alveolar bone while other infections are limited to inflammation of the gingiva with little or no discernible clinical consequences. Also, many periodontitis patients do not show a remarkable level of classical risk factors. **[**91**]**

A plausible etiopathogenetic explanation for destructive periodontal disease includes interactions among viruses, specific bacteria and immune reactions. Various studies found association of virus in periodontitis sites. Nevertheless, despite a large body of compelling research data, definitive proof is still asking whether viruses (ex. Herpes virus) play a causal role in periodontitis development and do not occur merely as an epiphenomenon to the periodontal disease process. The pathogenic significance and the concept of viral infections of the diseased periodontium may guide in a new level of understanding of the importance of preventing and controlling periodontal diseases. **[**92**]** The uncertainty about the infectious and clinical events of periodontal breakdown has given rise to a number of hypotheses about the etiology of periodontitis. Some researchers proposed that specific infectious agents act as a key to periodontal breakdown while others emphasized the importance of host immune factors or genetic characteristics in the development of periodontitis. **[**93**]**

The role of viruses in periodontology still remains a grey area since the progress is slow even with advanced technologies in the past century. Conventionally viruses are a challenging task for detection and treatment as compared to bacteria. Hereby, more and more studies have paid attention to the relationship between herpes viruses and different types of periodontitis.



Viral infection steps : Entry, replication, dissemination and infection of target cells/organs

Viruses gain entry into the host through different routes which include:

(a) Inoculation via the skin and mucosa as in case of needle stick injury, or accidental abrasions

(b) Inhalation through the respiratory tract as in aerosol or droplet

(c) Ingestion via the gastrointestinal tract as in the oro –fecal route

(d) The genitourinary tract as in sexual activity.

Once, the virus gains entry to the host cell through direct local spread on epithelial and subepithelial surfaces, lymphatic spread, vascular spread, central nervous system and peripheral nerve spread, the viruses will interact with the host cell in two main ways namely permissive and non permissive mode.

• Permissive infection: In permissive infections, the synthesis of viral components, their assembly and release can lead to consequent death of the host cell.

• Non permissive infection: In non permissive infections, infection can result in cell transformation often with the integration of viral DNA into the host genome. Viral replication occurs within the cell but the cell remains alive. Examples for non permissive infections include hepatitis B viruses, herpes viruses and retroviruses infection. **[**94**]**

These viruses play a fundamental role in the pathogenesis of periodontal diseases by a number of mechanisms operating alone or in combinations namely:

• Direct cytopathic effect on inflammatory cells such as polymorphonuclear, leukocytes, lymphocytes, macrophages, and other cells such as fibroblasts, endothelial cells and even bone cells. Herpes virus-induced cytopathic effects will hamper tissue turnover and repair as the involved are the main constituents of inflamed periodontal tissue.

• Cytokines and chemokines release: Their release from inflammatory and noninflammatory host cells are mediated by viruses.

• Interference with the immune system of the host

• Promotion of bacteria colonization : The down regulations of the cells involved in the periodontal defense may lead to bacterial superinfection resulting in increased virulence of resident bacteria including Porphyromonas gingivalis, Prevotella intermedia, Prevotella nigrescens, Campylobacter rectus, Treponema denticola and Aggregatibacter actinomycetemcomitans. **[**95**]**

• Latent viruses remaining in the body can become reactivated by various immune compromising events, such as smoking, stress, inflammation, trauma, and immunosuppressive diseases, and participate in the pathogenesis of periodontitis. **[**96**]**

Viral infection contributes to the development of various forms of periodontal diseases including severe chronic periodontitis, localized and generalized aggressive periodontitis, HIV-associated periodontitis and acute NUG. The possible role of viruses in periodontal diseases is suggested by the recovery of a patient from a chronic and highly refractory periodontal condition upon antiviral

treatment. Moreover, these viruses are found significantly more frequently in samples taken from disease active pockets, and gingival crevicular fluid compared to healthy pockets. **[**97**]**

Many bacterial infections in humans occur as super infections of viral diseases. Bacterial activity, for example, bacterial enzymes or other inflammation-inducing products, on the other hand, can also activate periodontal herpes viruses, which are considered as the vicious circle concept. Due to this, the controversy remains whether viruses can be classified as primary causative organisms for periodontitis.

Herpes viruses

Herpes viruses is considered as the most common viruses in humans, infecting 80–90% of the global adult population. Eight members of the herpes viridae family are known to cause human disease. These include Ebstein Barr Virus , Human Cytomegalo Virus, Herpes Simplex Virus 1 and 2 (HSV-1, HSV-2), Varicella Zoster Virus, Human Herpes Virus HHV- 6, HHV-7, and HHV-8. **[**91**]**

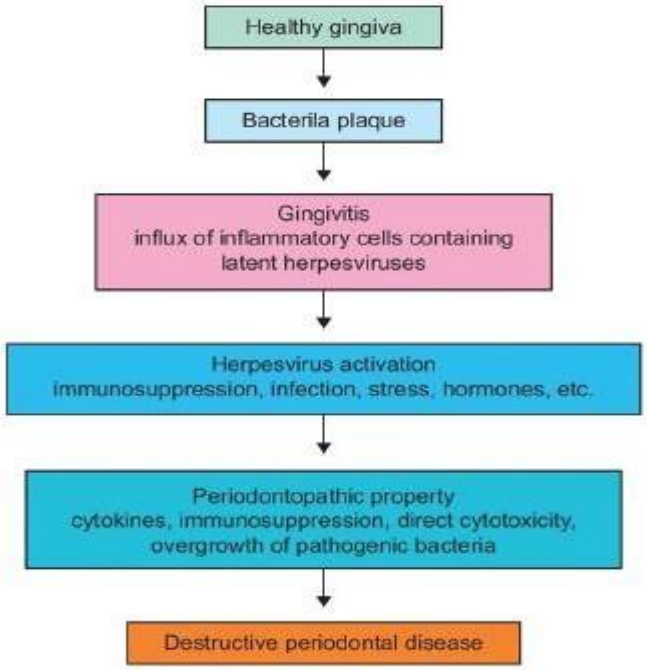
To survive, herpes viruses need to infiltrate macrophages, lymphocytes, or other host cells for replication, while minimizing antiviral inflammatory responses of the host. Herpes virus can cause periodontal disease as a direct result of virus infection and replication, or as a result of virally induced impairment of periodontal host defences with increased aggressiveness of resident bacterial pathogens.

Herpesvirus associated periodontal sites also tend to harbor elevated levels of periodontopathic bacteria, like Porphyromonas gingivalis, Tannerella forsythia, Dialister pneumosintes / Dialister invisus, Prevotella intermedia, Prevotella nigrescens, Treponema denticola, Campylobacter rectus, and Aggregatibacter actinomycetemcomitans. The coexistence of periodontal HCMV, EBV, and other viruses, along with periodontopathic bacteria, and local host immune responses should be considered as a precarious balance which has the potential to lead to periodontal destruction. **[**96, 97**]**

Presence of a gingival herpesvirus is further indicated by the presence of Immunoglobulin A antibodies against HCMV, EBV, and HSV in gingival crevicular fluid which is believed to

originate mainly from local plasma cell synthesis rather than from passive transudation from serum. **[**98**]**

After reaching a critical virus load, activated macrophages and lymphocytes may trigger a cytokine/chemokine storm of interleukin (IL)-1β, TNF-α, IL-6, prostaglandins, interferons, and other inflammatory mediators, some of which have the potential to stimulate bone resorption. In a vicious circle, the triggering of cytokine responses may stimulate latent herpes viruses, and that may lead to further aggravation of periodontal disease. It is proposed that herpes viruses rely on coinfection with periodontal bacteria to produce periodontitis and, inversely, periodontopathic bacteria may depend on viral presence for the initiation and progression of some types of periodontitis. **[**99, 100**]**



Herpes virus – bacterial model of periodontitis

Nibali et al. concluded that prevalence of herpes viruses in plaque sample of periodontitis subjects is not universal. Viruses have been detected in latent stages in various periodontal patients indicating their role as mere innocent bystander. **[**101**]** Saygun et al. concluded that periodontal pockets might act as a main source of viruses in the saliva of periodontitis patients where viruses grow owing to immunosuppression caused by bacteria. **[**97**]**

According to the studies conducted by Parra and Slots J in 1996, they found a significantly higher prevalence of human cytomegalovirus (HCMV) and Epstein–Barr virus (EBV) in sub gingival specimens from adult periodontitis patients as compared to periodontally healthy or gingivitis patients. **[**102**]**

In a systematic review (2016) Azahrani concluded that human herpes virus (HSV, CMV and EBV) levels are increased and are found to be associated with AgP and AP as compared to healthy individuals. **[**103**]**

Furthermore, in a meta analysis (2015) Zhu et al found significant associations between herpes virus (especially EBV and HCMV) and chronic periodontitis but concluded that a cause-effect relationship is yet to be established. **[**104**]**

Epstein–barr virus

EBV is generally transmitted by oral secretions or blood. The virus replicates in epithelial cells or B cells of oropharynx. Memory B cells are the main site where EBV viruses remain latent.105

In periodontitis, the presence of EBV is related to an elevated presence of periodontopathic bacteria like Porphyromonas gingivalis, Tannerella forsythia, Campylobacter spp. etc. Bacteria induced gingivitis is said to lead EBV-infected B lymphocytes to enter the periodontium. These cells are seen more prominently in progressive periodontal lesions.

EBV may induce proliferation of cytotoxic T lymphocytes whose main purpose is recognition and destruction of virally infected cells. However, various aspects of the periodontal immune response may be hampered secondarily by EBV. Together, these mechanisms probably contribute to the pathogenesis of periodontitis. **[**105**]**

Numerous studies have reported that herpes viruses, especially EBV and human cytomegalovirus have significant associations with increased risks of varieties of periodontitis, such as Chronic Periodontitis. **[**107-113**]** However, there are still some controversies in these findings. Some literatures indicated that a weak or even no relationship exists between herpes viruses and risks of periodontitis. **[**101, 114-116**]**

The results of a meta analysis conducted in 2015 have shown a significant association between EBV and periodontitis. EBV-detecting frequencies were associated with increased risks of CP. The meta-analysis also demonstrated that EBV was associated with the increased risk of AgP. **[**104**]** Dawson et al analyzed the correlation between EBV

and probing depth (PD), clinical attachment loss (AL), and bleeding on probing (BOP) and found that the relationship only exists between EBV and BOP. **[**117**]** Wu et al have found the similar conclusion. **[**118 **]**

Vincent et al found that EBV already exists in epithelial cells of periodontium (pECs) before the initiation of periodontitis, and the extent of EBV in pECs is increased with periodontitis severity. Similar results were described by Kato et al. **[**112**]** Analysis conducted according to ethnicity indicated that EBV was associated with periodontal diseases in Asian, European and Americans.

Human immunodeficiency virus (HIV)

Patients with HIV can manifest a number of oral lesions and conditions which are associated with a compromised immune response. A dentist may be the first professional to make a diagnosis of these common oral lesions. HIV is associated with periodontal conditions like: linear gingival erythema, necrotizing gingivitis, necrotizing periodontitis, oral candidiasis and chronic periodontitis. HIV positive patients with chronic periodontitis also tend to show a greater loss of attachment over time. **[**119 **]**

The primary target of HIV is the T-helper cell, which affects and impairs the immune function. It is this reduction in immune function that predisposes the individual to various opportunistic infections including periodontal diseases and may also facilitate herpes viruses reactivation or reinfection. In individuals suffering from AIDS, HIV-infected lymphocytes and monocytes are abundant in periodontal pockets, gingival tissues, and salivary glands.

Inversely, periodontal disease may also be involved in the onset of AIDS-related pathological changes in the form of oral hairy leukoplakia and Kaposi's sarcoma. In individuals with AIDS, HIV-infected lymphocytes and monocytes are abundant at oral sites, including periodontal pockets, gingival tissues, and salivary glands. Direct interaction of these cells with periodontopathic bacteria and/or indirect interaction with soluble factors (e.g. butyric acid and

TNF-α) could induce local HIV-1 replication in the oral cavity. It is proposed that a cell in which viral transcription has been reactivated by a stimulus can spread throughout the body via the blood. In addition, TNF-α concentrations are known to be elevated in individuals with periodontal disease, which suggests that periodontal disease can act as a trigger for local and systemic breakdown of latent infection and may act as a risk factor for AIDS progression. **[**120 **]**

A study compared MMPs in gingival crevicular fluid (GCF) and saliva from HIV+ patients HIV− patients with adult periodontitis and controls. Results determined that polymorphonuclear-derived metalloproteinases in GCF and saliva from HIV+-patients were present in the activated form, and proposed that these activated enzymes may contribute to periodontal destruction in HIV+patients. **[**121**]**

A review by Pólvora et al (2018) highlighted key points on the interrelationship between HIV and periodontitis as follows :

• HIV infection acts as a modifying factor in periodontal diseases, and is frequently associated with the occurrence of acute periodontal diseases and exacerbation of preexisting chronic periodontitis.

• The great bacterial diversity and complexity in the oral microbiota of HIV-infected individuals seems to be related to the chronic periodontitis progression and severity.

• HIV infection may contribute to destruct the epithelium of oral mucosa, favoring the microbial translocation, which could generate a systemic inflammatory state.

• An overlap in immune activation due to HIV infection and chronic periodontitis would increase the systemic inflammatory state, thereby worsening the effector response and the subsequent clinical outcome of the patients.

• There is a need for biomarkers related to periodontal inflammation that may contribute to better understand the pathogenesis and progression of periodontal disease, and the search for therapeutic targets in HIV-1-infected patients.

• Inflamed gingival tissue may act as a reservoir of HIV-1 and could be considered as an obstacle to disease eradication. **[**122 **]**

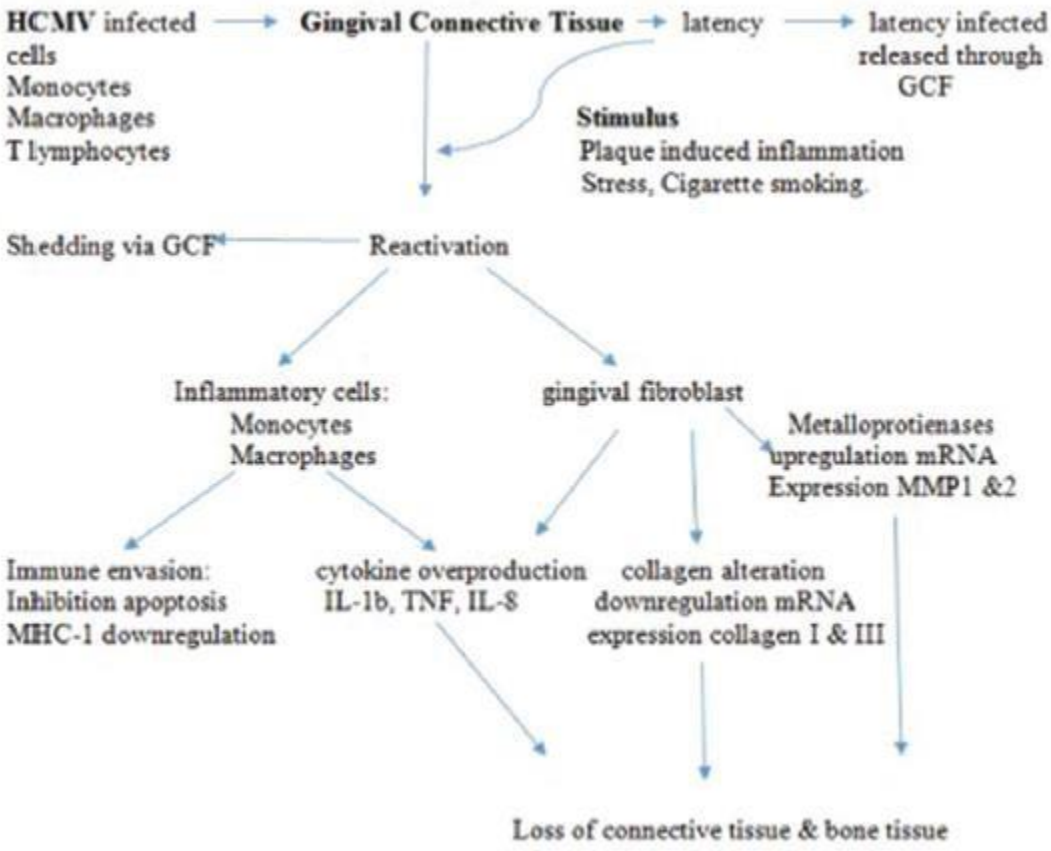
Human cytomegalovirus and periodontitis

Human cytomegalovirus is considered as one of the common causes of congenital and perinatal infections. HCMV infects epithelial cells, endothelial cells, smooth muscle cells, mesenchymal cells, hepatocytes, granulocytes, and monocyte-derived macrophages. Thus human cytomegalovirus is found in many body secretions including saliva, urine, semen, and breast milk. **[**123 **]**

The cytomegalovirus latent genome is carried into the periodontium by infected macrophages and T cells, and cytomegalovirus activation may subsequently give rise to infection of additional cell types. The down regulation of these cells because of the periodontal defense mechanisms may lead to bacterial superinfection resulting in enhanced virulence of resident bacteria. **[**108 **]**

An active cytomegalovirus infection in macrophages and T cells induces release of IL-1β and tumor necrosis factor (TNF) α. These proinflammatory mediators recruit antiviral inflammatory cells to the infection site but also induces osteoclast formation and production of matrix metalloproteinases (MMPs) which can lead to bone destruction. An active cytomegalovirus periodontal infection is associated to disease-active periodontitis, and the virus may also play an important role in other types of periodontal diseases such as aggressive periodontitis and refractory periodontitis. **[**124 **]**

The periodontal health was found to be associated with a median genomic detection rate of 8% for EBV and cytomegalovirus. There are certain studies, which have not detected viruses in periodontitis patients. **[**101**]**



Model linking cytomegalovirus to periodontal breakdown

Contreras et al. found the cytomegalovirus genome in gingival mononuclear cells (55%) and T-cells (20%) from periodontitis patients. They also found that Cytomegalovirus can infect and establish latency in gingival fibroblasts. **[**110**]**

Although biologically plausible, the extent to which cytomegalovirus participates in the destruction of the human periodontium is still a matter of research. Studies are needed to identify the environmental events and pathogenic pathways that trigger activation of cytomegalovirus in the peridontium, the possible link between cytomegalovirus reactivation and periodontitis disease activity, and the importance of anti- cytomegalovirus immunity in controlling periodontal disease. Such information may help to explain why cytomegalovirus and other ubiquitous herpesviruses may cause periodontitis only in a relatively small subset of individuals and teeth. **[**125**]**

Human papillomavirus and periodontitis

HPV belongs to Papilloma viridae family and is a double stranded, non enveloped DNA virus. HPV exihibits tropism for epithelial tissue thereby it can affect both skin and mucosa. HPV causes characteristic cytopathic effects called koilocytosis and proliferation of epithelial cells. Since proliferation and migration of the junctional epithelium is considered as a major hallmark of periodontal breakdown, these known biological effects of HPV might provide a link between role of viral infection and periodontal disease. **[**123**]**

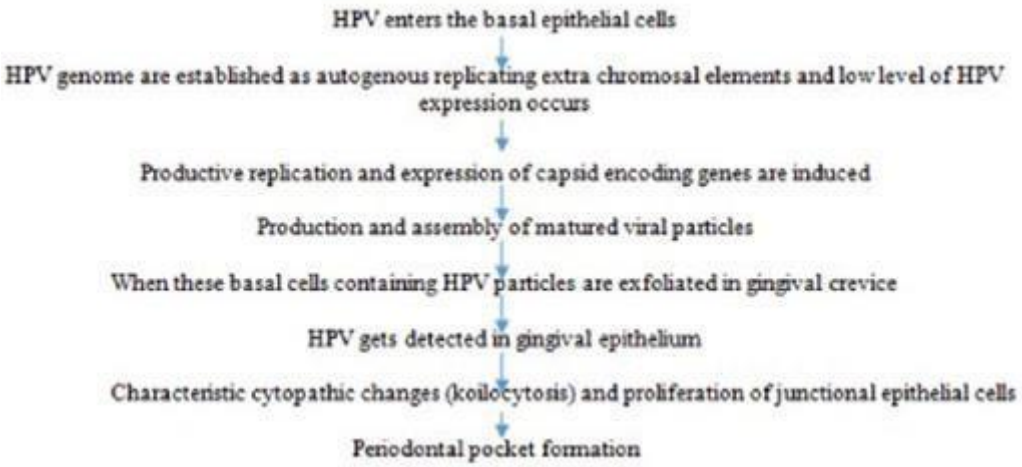


Figure : Model linking human papillomavirus (HPV) to periodontal breakdown.

The mere presence of viruses in periodontitis sites does not justify their role in the disease as viruses have also been detected in the healthy sites and viruses were not detected at all sites and in all studies.

Scientific evidence states “Association is not causation.” Thus, the second criterion of “elimination” further substantiates criteria of association. If the removal of an organism leads to resolution of the disease/lesion, causality may be surfaced. The synergistic pathogen concept reveals that microbes show great interdependence in periodontitis. The effect of the removal of one organism on the other microbes should not be overlooked. Modulation of viral prevalence by therapeutic intervention leads to the improvement in periodontal conditions and might confirm their role as putative pathogens. **[**101**]**

Various studies have pointed out considerable improvement in periodontal parameters along with combined bacterial and viral load reduction after mechanical periodontal therapy. This authenticates a synergistic role played by both pathogens or this might assign primary role to bacteria, whose removal led to decrease of the viral load simultaneously and vice versa.

Sunde et al. treated a patient, who exhibited refractory periodontitis and high Epstein– Barr virus subgingival copy counts, with the anti-herpesvirus drug, valacyclovir HCl, 500 mg twice a day for 10 days. The treatment suppressed subgingival Epstein–Barr virus to undetectable levels for at least 1 year and resulted in clinical improvement.125 Anti- herpesvirus chemotherapy has also shown to decrease the salivary viral load. A short course of valacyclovir, 2 g twice on the day of treatment and 1 g twice the following day, resulted in a significant decrease in the salivary occurrence of Epstein–Barr virus compared with controls. **[**101**]** With these studies, a cause-and-effect relationship between viruses and periodontal disease could be pointed out by proving the efficacy of antiviral therapy alone in achieving periodontal health.

Conclusion

Long-term studies with adequate sample size, well-designed randomized controlled trials, more sensitive and specific technological advancements to detect latent and activated viruses may provide sufficient evidence to implicate viruses as primary pathogens. Importance of the present literature cannot be undermined as it is rightly said that “absence of evidence is not the evidence of absence.” At the same time, a cause-and- effect relationship remains to be established. The possible involvement of viruses in the pathogenesis of periodontitis merits further investigation.

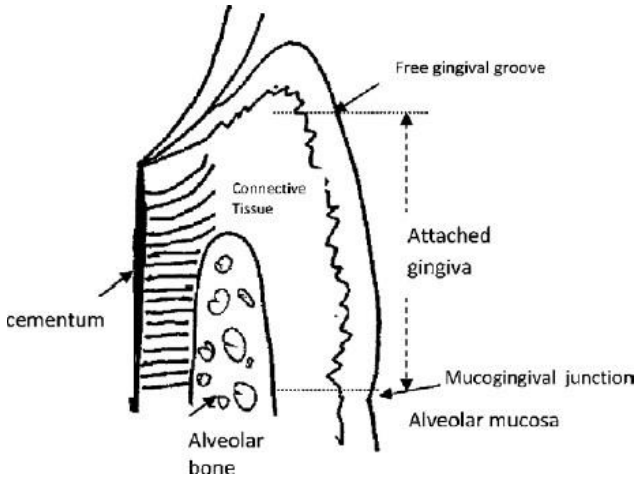
Width of Attached Gingiva

Introduction – Anatomical considerations

The gingiva is the part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the necks of the teeth. In an adult, normal gingiva covers the alveolar bone and tooth root to a level just coronal to the CEJ. The gingiva is divided anatomically into marginal, attached, and interdental areas. The width of the attached gingiva can be defined as the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or periodontal pocket (the width of attached gingiva can be estimated by subtracting the sulcus\pocket depth from the width of keratinised mucosa. **[**126**]**

The width of the attached gingiva on the facial aspect differs in different areas of the mouth. It is generally greatest in the incisor region (i.e., 3.5 to 4.5 mm in the maxilla, 3.3 to 3.9 mm in the mandible) and narrower in the posterior segments (i.e., 1.9 mm in the maxillary first premolars and 1.8 mm in the mandibular first premolars).

On the lingual aspect of the mandible, the attached gingiva terminates at the junction of the lingual alveolar mucosa, which is continuous with the mucous membrane that lines the floor of the mouth. The palatal surface of the attached gingiva in the maxilla blends imperceptibly with the equally firm and resilient palatal mucosa. **[**126**]**



Role of attached gingiva

The traditional roles attributed to this gingiva are because it is firm and resilient being attached to the underlying periosteum. Attached gingiva is important to dissipate the force of muscle pull and unattached mucosa, due to its mobility collects more plaque. Another advantage is that its epithelium is keratinised. It has been suggested that epithelial differentiation is determined by underlying connective tissue. It is also believed that this keratinised gingiva is more suitable to withstand the trauma. **[**127**]**

Width of attached gingiva and periodontal health

One of the requirements of a comprehensive periodontal examination is evaluation of the width of the attached gingiva. The aim of assessing the width of attached gingiva, as guided by conventional teaching, is to determine whether this width is ‘‘adequate’’. This prompted clinicians to ask what the significance of the width of attached gingiva was; how much can be considered adequate and what clinical relevance did these considerations have.

The amount of attached gingiva is deemed insufficient when stretching of the lip or cheek induces movement of the free gingival margin. Periodontists, historically, have indicated gingival augmentation to recreate this zone of attached gingiva. The early concept was that

of mastication and tooth brushing. In the early 1980s, Wennstrom et al. conducted a series of well designed experiments to prove that the attached gingiva and its width, have little role in maintaining periodontal health. Successive studies went on to prove that it is not the width but the volume of attached gingiva that is critical around restored or orthodontically moved teeth. Towards the end of the 1980s, the controversy around the significance of the width of attached gingiva had almost been resolved and clinicians were presented with clear guidelines to evaluate the width of attached gingiva and indications for gingival augmentation procedures. **[**128, 129**]**

There are two often quoted but contradictory studies done in the 1970s to explore the relationship between the width of the attached gingiva and gingival health. The results of the Lang and Loe study shows, ‘‘all surfaces with less than 2.0 mm of keratinised gingiva exhibited clinical inflammation.’’ **[**130**]** On the other hand, Miyasato et al. in their study of an experimental gingivitis in dental personnel demonstrated that areas of minimal width of attached gingiva may

not be prone to development of plaque induced inflammatory changes. **[**131**]** While interpreting these results, both studies reported only clinical findings as opposed to histological ones which are more objective in nature. The study by Lang and Loe is crosssectional in design implying association but no cause and effect relationship.

During this period, several surgical procedures were indicated based on the concept that a minimum width of attached gingiva is required to maintain periodontal health. However the then present literature was contradictory. In the early 1980s Wennstrom, Lindhe and Nyman Group conducted a series of three well designed experiments on the beagle dog model. During the course of the study, four different dentogingival units were studied. **[**128, 129**]**

• Normal attachment apparatus and normal width of attached gingiva;

• Normal attachment apparatus but narrow width of keratinised gingiva (no attached);

• Reduced attachment apparatus with narrow width of keratinised gingiva (no attached);

• Reduced attachment apparatus with normal\wide zones of attached gingiva (grafted sites).

The results of this experiment showed that in all four types, gingival health could be established and maintained. The same dentogingival units from the previous experiment were subjected to 40 days of plaque accumulation. No difference was seen in the histological sections with regard to size and apical extension of inflammation in the connective tissue. Following studies by Wennstrom and co-workers, not much controversy remained in literature regarding width of attached gingiva and periodontal health. It is clear that width of attached gingiva is not significant to maintain health in a healthy or reduced periodontium as long as plaque control is maintained. An ‘‘inadequate width of attached gingiva’’ is as resistant to plaque induced gingival inflammation as an adequate one. A narrow width of attached gingiva alone is not an indication for gingival augmentation.

Width of attached gingiva and recession

An association between lack of attached gingiva and recession has often been implied in the literature. Wennstrom monitored 26 test sites in six patients with little or no attached gingiva (surgically excised) for 5 years. Simultaneously, 12 sites, two per patient, with adequate width of

attached gingiva were included as control sites. They reported recession in two of the 26 test sites and three of 12 control sites. Four of these sites were in one patient. The clinical implication of this study is that there is no evidence to show that increasing the width of attached gingival under an area of recession will retard the progression of recession. **[**132**]**

These findings were corroborated by Lindhe and Nyman they followed 43 patients on maintenance therapy for 10–11 years. They concluded that changes in the position of the gingival margin followed a similar pattern in areas with and without keratinised gingiva. **[**133**]**

Collectively, the evidence suggests that areas with narrow width of attached gingiva are not more susceptible to recession. It is remarkable that as early as 1976, Baker and Seymour had brought forward an explanation regarding the pathogenesis of recession. Localised inflammation in a thin gingiva may involve the entire volume of gingival tissue and the consequent remodelling will lead to rapid recession of gingival margin. In contrast, in a thick gingiva this inflammatory lesion would be confined to only a part of the sulcus and not involve the ‘‘outer gingival tissue’’. This may predispose to pocket formation rather than recession. This was the beginning of recognition that thin gingival biotype is a risk factor for recession. **[**134**]**

Rajapakse et al. in a systematic review evaluated the influence of toothbrushing habits on gingival recession, while there is evidence that some toothbrushing factors may be associated with the development of gingival recession, no definitive conclusion could be drawn from the review. **[**135**]**

The challenges borne by the periodontium around a restored tooth differ from that around a natural tooth. The margins of a restoration are more prone to the accumulation of plaque. Stetler and Bissada found that the gingival index (GI) was elevated in areas with subgingival restorations and concurrent narrow width of attached gingiva. It is interesting to note that no significant difference in attachment levels and bone levels were seen. The authors suggested that tooth brushing is more difficult in areas of narrow zones of attached gingiva with subgingival restorations resulting in greater accumulation of plaque. Ericsson and Lindhe put forward the theory that, ‘‘The observations of more severe gingivitis in sites with subgingivally located

restorations should be related to the fact that the dimensions of gingival units are smaller not only in the apico-coronal but also in the buccolingual direction.'' **[**136**]**

We can now appreciate the evolution of literature as the concept of volume rather than width of gingiva tissue appears to be gaining credence. Goldberg thus concluded in his review article that in areas of subgingival margins, especially in aesthetic areas we require a minimum volume of attached gingiva. He also adds that the width of attached gingiva is significant when the patient reports an inability to brush at that site. **[**137**]**

The next question that arises is ''How much is adequate?''—the truth of the matter is that it is not the width but volume that is critical. As a rough guide, clinicians continue to use the Lang and Loe guideline as 2 mm of width of keratinised gingiva which equals 1 mm of width of attached gingiva as ''adequate''. This question of assigning a value to the width has been explored from a different perspective. Studies have shown that at least 1 mm thickness is required to prevent recession after scaling and root planing and get predictable results in procedures such as root coverage and guided tissue regeneration. **[**127**]**

Table 1 – Summary of key papers on keratinised/attached gingival on periodontal health.

Study	Design of study	Outcome	Conclusion and implication
Lang and Löe[2]	32 dental students underwent 6 weeks of supervised oral hygiene, width of KG, GI and gingival exudate were scored around plaque free sites	Sites with <2 mm KG had higher percentage of sites with clinical inflammation and gingival exudate	2 mm of keratinised gingiva is adequate to maintain gingival health
Miyasato et al.[14]	Gingival status of 16 dental personnel with adequate AG and minimal AG were compared. GI was compared in 6 subjects having contralateral sites with inadequate and adequate AG following a period of 25 days of no oral hygiene at sites	(i) No marked difference in GI in subjects with minimal AG or adequate AG; (ii) following a period of no oral hygiene, there was no sig difference in GI and plaque scores in areas with narrow or wide AG	It is possible to achieve gingival health even in the absence of adequate AG
Wennström and Lindhe[5] animal studies	7 beagle dogs, 4 different dentogingival units with varying widths of AG created followed by 40 days plaque accumulation	Clinical and histological investigations did not reveal any differences in the extent of inflammation	Gingival units without AG may not be more susceptible to inflammation than one with wide zone of attached gingival
Wennström[19]	5-Year monitoring of 26 sites deprived of AG compared with 12 control sites with adequate AG	7/26 test sites showed slight increase in AG, 2 sites showed reduced AG. 3 control sites showed reduced AG	In presence of good plaque control, lack of AG did not results in greater gingival recession

Freedman et al.[15]	18-Year follow-up and periodontal assessment in 17 subjects with inadequate KG	Change in width keratinised tissues: increased at 19/61 sites, reduced at 7 sites, unchanged at 35 sites	With good oral hygiene and gingival health, despite inadequate KG, KG may remain stable in the long term
Stetler and Bissada[23]	Two groups with $<$ or $\geq$ 2 mm (AG). 2 subgroups each with subgingival restoration and without subgingival restoration (control). Periodontal assessment carried out	(i) Subgingival restoration at teeth with narrow zone of AG have higher GI than teeth with wide zone of AG; (ii) no such sig. difference in GI was found in teeth with no subgingival restoration	

KG—keratinised gingiva; AG—attached gingiva; GI—gingival index.

Conclusion

Periodontal health depends on the integrity of the dentogingival unit with no relation to the quality or quantity of overlying gingiva. In summary, the general consensus is in the presence of good oral hygiene, the width of attached gingiva or gingival augmentation is not crucial for maintenance of gingival health.

The principles underlying the need for attached/keratinised mucosa around teeth have been elucidated successfully with the path breaking studies by Wennstrom et al. and supported by numerous studies since then. These remain our guide for clinical decision making when faced with sites lacking keratinised mucosa. Besides this, the need for augmentation has to be tailored to the particular clinical situation and patient's oral hygiene competence. Clinicians, till date are skeptical to accept that one may not need a certain width of keratinised mucosa around teeth and\or implants. One of the reasons may be that it is impossible to imagine ''adequate thickness'' without ''adequate width''. While current evidence point towards the clinical relevance of the thickness rather than the width of the keratinised tissue in determining soft tissue health and recession, the problem arises as it is more difficult to discern clinically the thickness as compared with measuring the width of attached gingiva. Therefore, the clinical impression that one needs a certain ''adequate width'' of attached mucosa may not be unfounded.

Occlusal Trauma and Periodontal Disease

It has been a topic of debate among dental care professionals, if trauma from occlusion is linked with periodontal disease or not. There are several schools of thought related to if trauma from occlusion is an etiological factor or cofactor for the occurrence of periodontal diseases. Excessive occlusal force is defined as occlusal force that exceeds the reparative capacity of the periodontal attachment apparatus, which results in occlusal trauma and/or causes excessive tooth wear (loss). Occlusal trauma is a term used to describe injury resulting in tissue changes within the attachment apparatus, including periodontal ligament, supporting alveolar bone and cementum, as a result of occlusal force(s). Occlusal trauma may occur in an intact periodontium or in a reduced periodontium caused by periodontal disease. 138

Classification of TFO

1. Physiologic or Traumatic Occlusion

Physiologic occlusion is a condition in which the system of forces acting upon the tooth during occlusion, are in a state of equilibrium and they do not and cannot change the normal relationship existing between the tooth and its supporting structures. In this, the occlusal pressure against the tooth is balanced by the resistance of periodontal tissues.

Traumatic occlusion is where the damage produced in the periodontium is due to the overstress caused by the occlusion.

2. Acute or Chronic (depending on the duration of cause)

Acute trauma results from an abrupt occlusal impact such as that produced by biting on a hard object. In addition, restorations or prosthetic appliances that interfere with or alter the direction of occlusal forces on the teeth may induce acute trauma.

Chronic trauma most often develops from gradual changes in occlusion produced by tooth wear, drifting movement and extrusion of teeth combined with parafunctional habits such as bruxism and clenching, rather than as a sequelae of acute periodontal trauma.

3. Primary or Secondary (depending on the nature of cause)

Primary occlusal trauma is injury resulting in tissue changes from excessive occlusal forces applied to a tooth or teeth with normal periodontal support. It occurs in the presence of normal clinical attachment levels, normal bone levels, and excessive occlusal force(s).

Secondary occlusal trauma is injury resulting in tissue changes from normal or excessive occlusal forces applied to a tooth or teeth with reduced periodontal support. It occurs in the presence of attachment loss, bone loss, and normal/excessive occlusal force(s). **[**139**]**

Etiological factors Precipitating factors

The irritants and the devastating occlusal forces that further destroy the tissues are weakened by the predisposing factors. the precipitating factor is destructive occlusal forces. These forces when within normal range can be well adapted by the tooth supporting soft tissues. But when these forces exceed the adaptive capacity of tooth supporting tissues, pathologic changes can be seen in the soft tissues. These forces are normally described in terms of magnitude, direction, duration of application and frequency of application. **[**139**]**

Predisposing factors

Factors which take the place of those contributing to the histopathologic lesion are listed as developmental factors, functional mechanisms, and the systemic component. They can be divided into:

1. Intrinsic factors: Consist of the morphology of the roots, alveolar process, and the orientation of the occlusal surfaces and roots to the forces, in which the tooth gets exposed to.

2. Extrinsic factors: Consist of plaque, parafunctional activities, bone loss or loss of teeth, and malocclusion created iatrogenically.

Because trauma from occlusion is defined and diagnosed on the basis of histologic changes in the periodontium, a definitive diagnosis of occlusal trauma is not possible without block section biopsy. Consequently, multiple clinical and radiographic indicators are used as surrogates to assist the presumptive diagnosis of occlusal trauma.

Clinical signs	**Radiographic signs**
Mobility	Widening of the periodontal ligament(PDL) space
Positive fremitus test[12]	Thickening of the lamina dura in the apical region and in bifurcation areas
Occlusal prematurities[13]	Vertical destruction of the interdental septum with formation of infrabony defects
Wear facets	Radiolucence and condensation of the alveolar bone
Tooth migration	Root resorption
Fractured teeth	
Thermal sensitivity	

These clinical signs and symptoms may indicate other pathoses. For instance, loss of clinical attachment can affect the severity of mobility. Also, it is often very difficult to determine whether the wear facets are caused by functional contacts or parafunctional habits, such as bruxism. Therefore, differential diagnoses should be established. Supplementary diagnostic procedures, such as pulp vitality tests and evaluation of parafunctional habits, may be considered. **[**139**]**

Historical aspects

Ever since Karolyi (1901) postulated that an interaction may exist between "trauma from occlusion" (TFO) and "alveolar pyorrhea," different opinions have been presented in the literature regarding the validity of this claim. **[**140**]**

Using human autopsy material, it was concluded that gingival inflammation extending into the supporting bone was the cause of periodontal destruction. In a subsequent animal experiment, it was found that the excessive occlusal forces caused changes in the direction of the periodontal membrane fibers so that gingival inflammation passed directly into such areas. **[**141**]**

In the 1930s, Box (1935) and Stones (1938) reported experiments in sheep and monkeys, the results seemed to indicate that "TFO is an etiologic factor in the production of periodontal disease

in which there is vertical pocket formation associated with one or a varying number of teeth". **[**142, 143**]**

Glickman and Smulow proposed the theory in the early 1960s that inflammation progressed in an altered pathway in teeth subjected to occlusal trauma. The combined effect of occlusal trauma and bacterial plaque–induced inflammation was termed "co- destruction." **[**144**]**

This theory was then challenged by other investigators. Using human autopsy material again, the altered pathway of destruction was questioned because bacterial plaque was always present in close proximity to the site of periodontal destruction, and this suggested that inflammation and bone loss were associated with the presence of bacterial plaque rather than excessive occlusal forces. The historic studies used autopsy material that provided little or no information on the periodontal conditions and occlusal conditions of these study subjects. It was after the co-destruction theory was presented that researchers started to examine the concept of multiple risk factors that resulted in the initiation and progression of periodontal diseases. Waerhaug proved the involvement of TFO in the pathogenesis of infrabony pockets. **[**138**]**

Animal studies

In an attempt to prove a relationship between occlusion and periodontal disease, multiple animal studies with strict controls and designs were performed in the 1970s.

In two of the studies, it was found that when oral hygiene was maintained and inflammation was controlled, occlusal trauma resulted in increased mobility and loss of bone density without loss of connective tissue attachment, during the length of the study. If the occlusal forces were removed, the loss of bone density was reversible. In contrast, in the presence of plaque-induced periodontitis and occlusal trauma, there was greater

loss of bone volume and increased mobility, but loss of connective tissue attachment was the same as on teeth subjected to periodontitis alone. It was concluded that without plaque-induced inflammation, occlusal trauma does not cause irreversible bone loss or loss of connective tissue attachment. Therefore, occlusal trauma is not a causative agent of periodontitis. **[**145, 146**]**

None of the animal studies were able to reproduce all aspects of human periodontitis. In addition, the animal studies used excessive forces and were conducted for a relatively short duration (a few weeks to a few months). Nonetheless, the results from animal studies suggested that occlusal trauma does not cause periodontitis, but it may be a cofactor that can accelerate the periodontal breakdown in the presence of periodontitis.

Clinical studies

Tooth mobility has been described as one of the common clinical signs of occlusal trauma. However, increased tooth mobility may result from inflammation and/or bone loss or attachment loss alone. Progressive mobility may be suggestive of ongoing occlusal trauma, but assessments at different time points are necessary to make this determination. **[**139**]**

In an epidemiologic study, a group of subjects was re-examined for loss of periodontal clinical attachment after 28 years. It was found that baseline tooth mobility was a factor related to clinical attachment loss. Tooth mobility was also found to affect the results

following periodontal therapy. It was shown that teeth with mobility did not gain as much clinical attachment as those without mobility following periodontal treatment. Further, teeth with increased mobility demonstrated significantly more clinical attachment loss during the maintenance period. However, no association was drawn between mobility and occlusal forces. **[**147**]**

The relationship between cusps is an important factor in the transmission of occlusal forces to the periodontium. In an early retrospective study, the relationship between periodontal parameters and molar non-working contacts was examined. It was found that molar teeth with non-working contacts had greater probing depths and bone loss compared with those without non-working contacts. **[**148**]**

Conversely, other studies looked at occlusal disharmonies in patients with periodontitis and failed to find any correlation between abnormal occlusal contacts and periodontal parameters, including probing depth, clinical attachment level, and bone loss. Nevertheless, teeth with frank signs of occlusal trauma, including fremitus and a widened periodontal ligament space, demonstrated greater probing depth, clinical attachment loss, and bone loss. **[**149, 150**]**

A series of retrospective studies investigated the association between occlusal discrepancies and the progression of periodontitis in a private practice setting. All patients included had moderate to severe chronic periodontitis. These studies found that teeth with occlusal discrepancies had significantly deeper initial probing depths, more mobility, and poorer prognoses than those teeth without occlusal discrepancies. Multiple types of occlusal contacts, including premature contacts in centric relation, posterior protrusive contact, non-working contacts, combined working and non-working contacts, and the length of slide between centric relation and centric occlusion were associated with significantly deeper probing depths and increased assignment to a less favorable prognosis. In another cross-sectional epidemiologic study, the non-working side contact was also associated with deeper probing depth and more clinical attachment loss. **[**151-153**]**

Effects of excessive occlusal forces on gingival recession

Historically, it has been suggested that excessive occlusal force might be a factor in gingival recession and the loss of gingiva. The term “Stillman's cleft” is defined as narrow, triangular-shaped gingival recession on the facial aspect of the tooth. It was postulated that excessive occlusal force caused the Stillman's cleft. However, these historic references are based on uncontrolled clinical observations. **[**139**]**

By examining teeth with gingival recession, no correlation was identified between mobility and gingival recession. Compared with contralateral teeth without recession, teeth with recession showed either no or similar mobility. In a clinical investigation on the etiology of gingival recession, a positive association between occlusal trauma and gingival recession was reported; however, this association disappeared when tooth malposition was present. **[**154, 155**]**

In evaluation of the relationship between incisor inclination and periodontal status, labial gingival recession of the mandibular incisors was related to linguoversion. However, there was no further analysis of the functional occlusal relationship. **[**156**]**

A retrospective study also failed to establish a relationship between the presence of occlusal discrepancies and initial width of the gingival tissue or between occlusal treatment and changes in the width of the gingiva. **[**157**]**

Hence, existing data do not provide any solid evidence to substantiate the effects of occlusal forces on gingival recession.

Occlusal therapy as a treatment for TFO

Occlusal adjustment is defined as "reshaping the occluding surfaces of teeth by grinding to create harmonious contact relationships between the maxillary and mandibular teeth." The evidence linking occlusal adjustment to improvement in periodontal parameters is limited. In an earlier study, the flow rate and quality of gingival crevicular flow (GCF) after removal of occlusal interferences was examined in patients with advanced periodontitis. It was found that occlusal adjustment reduced the protein content and collagenase activity without affecting the quantity of GCF. **[**158**]**

Later, a well-controlled clinical trial was conducted to evaluate the effect of the occlusal adjustment on healing outcomes after periodontal treatment. In this study, half of the patients received occlusal adjustment by selective grinding before receiving surgical or non-surgical periodontal therapy. The other half did not receive occlusal adjustment. After healing, the group that received occlusal adjustment before periodontal treatment gained 0.4 mm improvement in mean clinical attachment levels compared with those without pre-treatment occlusal adjustment. **[**159**]**

During long-term periodontal maintenance, the parafunctional habits that are not treated with a bite guard and the presence of mobility were both associated with increased clinical attachment loss and tooth loss. **[**160**]**

In another study conducted in a private practice, the response of patients with periodontitis and occlusal discrepancies to occlusal adjustment was examined. Regardless of the periodontal treatment status, the probing depth of teeth with untreated occlusal discrepancies was increased by a mean of 0.066 mm/year while a decreased probing depth of 0.122 mm/year was noted on teeth with occlusal adjustment. **[**161**]**

Collectively, these clinical studies demonstrated the added benefit of occlusal therapy in the management of periodontal disease, but they do not provide strong evidence to support routine occlusal therapy. Clearly, occlusal therapy is not a substitute for conventional periodontal treatment for resolving plaque-induced inflammation. However, it may be beneficial to perform

occlusal therapy in conjunction with periodontal treatment in the presence of clinical indicators of occlusal trauma, especially relating to the patient's comfort and masticatory function. The patient's occlusion should be carefully examined and recorded before and after treatment. The occlusion of periodontally compromised teeth should be designed to reduce the forces to be within the adaptive capabilities of the reduced periodontal attachment. Overall, in the presence of occlusal trauma, occlusal therapy may slow the progression of periodontitis and improve the prognosis.

Conclusion

The role of occlusal trauma in the initiation and progression of periodontitis remains a controversial subject in periodontology. Because occlusal trauma can only be confirmed histologically, its clinical diagnosis depends on clinical and radiographic surrogate indicators which make clinical trials difficult. Over the years, studies have strongly indicated that excessive occlusal forces are not a causal factor in the initiation of periodontal disease. Plaque is the primary causal factor in periodontal disease and it is believed its control should be a priority in any periodontal treatment. Occlusal forces may be a cofactor in the progression of periodontal disease. Treatment of occlusal discrepancies may be a beneficial adjunct to routine periodontal therapy. Plaque control and proper oral hygiene are the primary factors which focus on elimination of inflammation from the periodontal tissues.

Hand Instruments vs Ultrasonic Instruments

According to the classic model proposed by Page & Schroeder, the development of gingivitis and its progression to periodontitis occurs in four stages. Clinical signs of gingivitis start to appear in the 'early lesion' (second stage). Up to the 'established lesion' (third stage), clinical signs of the disease can be reversed by disrupting and removing the microbial plaque biofilm. **[**162**]**

The most predictable way of disrupting the microbial plaque, reducing inflammation around the gingival margins and thus preventing gingivitis, is by mechanical disruption and removal of the microbial plaque community. Mechanical debridement consisting of scaling and root planing is an important procedure in the treatment of periodontal diseases. By root instrumentation, toxic substances can be removed from periodontally affected root surfaces resulting in the biologic / detoxic condition of the root surface, which is favorable for periodontal tissue healing. In addition to supragingival plaque control, subgingival plaque control by means of mechanical debridement is essential for elimination of the microbial causative factors of periodontal disease. Meticulous scaling and root planing is performed during the surgical and nonsurgical phases of periodontal treatment, as well as in the maintenance phase. **[**163**]**

The effectiveness of this procedure is highly dependent on the skill and ability of the individual to remove plaque from all the tooth surfaces.

Altering the subgingival microbiota to one compatible with periodontal health, or reducing the bacterial load and calculus deposits on tooth surfaces, can be achieved by hand scalers and curettes or ultrasonic scaling instruments, sonic and ultrasonic instrumentation, laser scaling, demineralization and chemical scaling. **[**164**]**

It is certain that hand instruments and ultrasonic scalers are used most frequently.

Manual instruments

Manual instruments for scaling are generally classified into five types: sickle, curette, file, hoe, and chisel types. The sickle and curette types are the most commonly used.

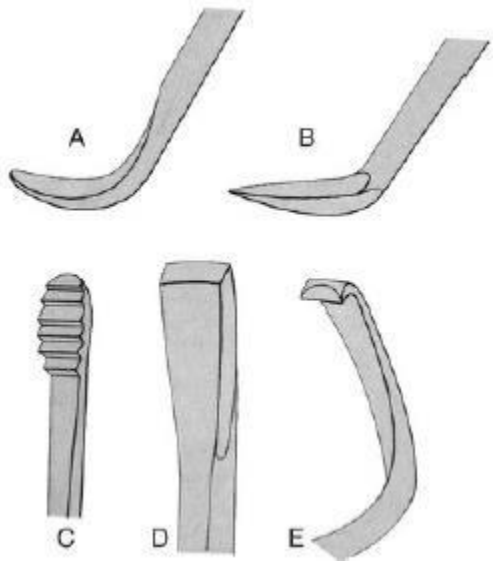
1. The sickle scaler is used for removal of supragingival calculus. The main sickle type scalers are University of Southern California sickle, Turner sickle, Jaquette sickle, and Morse sickle. The crosssection of the blade is triangular. Some sickle type scalers are designed for use in both anterior and posterior teeth.

2. The curette type is usually used for removal of subgingival calculus and root planing. Curette type scalers are more frequently used now than before. There are two basic types of curettes: universal and Gracey curette. These two types of curettes differ in the area-specificity, number of cutting edges, curve of cutting edge, and the angle of the face to terminal shank. Columbia curettes and Gothenburg curettes are representatives of the universal curette. Initially, Gracey curettes were available as a set of 14 instruments, but now mainly double-ended Gracey curettes (7 instruments) are used.

3. File type scalers are used to fracture or to crush calculus. This group includes a set of four scalers, for buccal, lingual, mesial and distal sites. The blade is narrow and used for access to deep and narrow pockets.

4. Hoe scalers are used for removal of subgingival calculus, and are a set of four scalers similar to the file type. They can be used for root planing and to remove calculus from the base of the pocket.

5. Chisel scalers, designed for proximal surfaces, are usually used in the anterior part of the mouth and therefore, the adaptation area is limited. **[**165**]**



5 basic scalin instruments - (A) Curette; (B) sickle; (C) file; (D) chisel; (E) hoe

Sharpness of the instrument and the instrument material are two instrument factors that can affect the efficiency of debridement.

Sharpness of the instrument

Most commercially available hand instruments require sharpening on a regular basis. For decades it has been accepted knowledge that periodontal instruments must be re- sharpened frequently. A few studies have compared changes in root-surface morphology based on the cutting edge of the instruments used.

O'Leary & Kafrawy recommended sharpening hand instruments after every five working strokes, Coldiron et al. after every 10 strokes and Rees et al after every 12 strokes. Zappa et al. found that after the first 20 strokes there was diminished hard-tissue removal and an increase in pressure applied per stroke. Even though all these studies show a decrease in instrument sharpness and effectiveness, very few clinicians sharpen their instruments every five to 20 strokes. Although instrument sharpening is the deciding factor for clinical effectiveness in achieving a clean and smooth root surface, sharpening the instrument every 5 to 20 strokes is not very practical and

results in destruction of the original contour of expensive instruments. Re-sharpening can weaken the scaler, causing breakage during function, or can create metal tags that are potentially harmful to the hard and soft tissues. Owing to the problems with instrument sharpening, clinicians and instrument manufacturers have been seeking instruments that can achieve better clinical effectiveness with less trauma to the hard and soft tissues and can be used for prolonged periods of time without sharpening. Several instruments with "edge retention" properties have been introduced to the market with the claims that these instruments need little or no sharpening, and allow unproblematic maintenance and display long-term effectiveness. Different metal alloys, including stainless steel, high-speed steel, carbon steel and tungsten carbide, have been shown to influence the efficacy and life expectancy of the instrument. **[**163**]**

Benfenati et al analyzed scanning electron microscopy images of root surfaces and found that blunt instruments produced smoother root surfaces compared with sharp instruments, even though they did not completely remove all the deposits on the root surface. A damaged curette created deep scratches on the root surface. In more recent studies, curettes have proven to create a relatively smooth surface morphology, as determined by profilometric findings. **[**166-168**]**

Instrument material

Sisera et al evaluated three different materials with edge-retention technology in comparison with a standard curette made of stainless-steel alloy. Of the three instruments tested, two had titanium nitride coating and one was made of cryogenically treated stainless steel. They simulated clinical conditions in the laboratory using bovine central incisors. The concurrent removal of dental hard tissue, at predetermined intervals (i.e. number of strokes), was evaluated to monitor the effectiveness and hard-tissue damage caused by the instruments. The surface roughness after use was also assessed. The authors found no statistically significant difference between the different instruments at different time points regarding the amount of tooth structure removed. It was concluded that although the manufacturers' claim for the titanium nitride-coated instruments and the tempered stainless-steel instrument about not requiring frequent sharpening over multiple usage was true, it was also true for the control curette, which was made of untreated stainless steel alloy. All instruments lost efficacy after being repeatedly treated with thermal and chemical sterilization. **[**169**]**

Another study examined the effect of repeated dry-heat sterilization and autoclave cycles on carbon-steel and stainless-steel curettes during scaling and root planing. Carbon-steel curettes were more likely to be affected by surface corrosion products and edge deterioration than were stainless-steel curettes.

Diamond-coated curettes have been introduced for scaling and root planing with conventional curettes. Eick et al evaluated the efficacy of an additional use of diamond- coated Gracey curettes on surface roughness, adhesion of periodontal ligament fibroblasts and detection of Streptococcus gordonii in vitro after conventional root planning. The authors found that the additional instrumentation with the diamond-coated curettes resulted in a two-fold increase in the number of attached periodontal ligament fibroblasts but not in the numbers of adhered bacteria. The authors concluded that conventional root planing with Gracey curettes followed by subsequent polishing with diamond-coated curettes, may result in a root surface that provides favorable conditions for adhesion of periodontal ligament fibroblasts without increasing microbial adhesion. **[**170**]**

Several studies have compared stainless-steel instruments with those of carbon steel, to see how the alloy mix affects the hardness of the cutting edge, and have reported conflicting results. In the study carried out by Tal and coworkers, the stainless-steel curettes showed significant edge attrition after 45 strokes compared with the high-speed steel, cemented-carbide steel and high-carbon steel instruments. **[**171**]**

Gorokhovsky et al. showed significantly less wear on instruments with a 10 multilayer titanium nitride/titanium coating compared with uncoated high-chromium stainless-steel scalers – wear resistance of the former was increased by at least 12.5 times and clinical usefulness extended from 3 months to 6– 11 months, depending on the rate of use. **[**172**]**

Powered instruments

Powered scalers utilized in debridement procedures are classified into sonic and ultrasonic instruments according to their working frequencies. The power setting that is normally tuned controls the length of the stroke, or amplitude. Sonic powered instruments operate at frequencies in the sonic range of 2–8 kHz (cycles per second) and are driven to vibrate by compressed air

striking a metal rod within the handpiece to produce audible oscillations that travel down to the attached scaling tip. The vibrating tip produces elliptical to orbital motions with all sides of the tip able to adapt to the root surface.

The two types of ultrasonic scalers are based on either magnetostrictive or piezoelectric mechanisms.

1. Magnetostrictive ultrasonic instruments are driven to vibrate by an electric current supplied to either a wire coil, metal stacks made of nickel–iron alloy, or to a ferrous rod in the handpiece, producing a magnetic field that causes the oscillation generator to change shape or dimension, creating the high vibrational energy that travels to the scaler tip. The piezoelectric scalers use electrical energy to electrosize crystals housed within the handpiece. The dimensional changes of these crystals cause the generation of high vibrational energy that travels to the tip. Magnetostrictive ultrasonic scalers have elliptical tip movement and operate between 18,000 and 45,000 cycles/second, much faster than sonic scalers, with an amplitude that ranges from 10 to 100 lm. All surfaces of the tip – front, side and back – are simultaneously active with the elliptical vibratory movement. The metal stack in the magnetostrictive scaler generates heat, and to prevent overheating it requires plenty of irrigation during scaling. It is recommended that the flow rate be at least 20–30 ml/min to prevent a temperature increase of more than 5°C that could potentially damage the pulp and dentin.

2. Piezoelectric devices do not generate much heat and require less irrigant; however, the cooler water might cause more sensitivity during the procedure. Piezoelectric ultrasonic scalers produce a linear vibratory movement that permits two lateral sides of the tip to be active, operating at 25,000–50,000 cycles/ second, with amplitude of 12–72 lm. The recommended technique for using the piezoelectric scaler is from a coronal to apical direction. **[**165**]**

Magnetostrictive and piezoelectric ultrasonic device

The irrigant used in these devices not only provides cooling at the treatment site, but creates acoustic turbulence, streaming and cavitation. Extreme conditions of pressure and temperature that destroy cell walls and kill bacteria are produced by the cavitation resulting from the formation and break down of microscopic bubbles (cavities) created as water passes through the handpiece. Water exiting the tip creates acoustic microstreaming and turbulence, further agitating and disrupting the content of the pocket. **[**165**]**

Hand instrumentation vs powered instrumentation Required time and clinical outcomes

A systematic review of controlled clinical trials, with 6 months or more of follow up, assessed the differences between ultrasonic, sonic and manual debridement for the treatment of chronic periodontitis. It was found that the mean gain in clinical attachment level, the mean reduction in probing depth and the mean reduction in bleeding on probing were similar for both machine-driven and hand instruments. Procedures using machine-driven instruments, however, took significantly less time (36.6% less than hand instrumentation) and caused less soft-tissue trauma but more root damage. **[**173**]**

A second systematic review, carried out by Needlemann et al., assessed supragingival and subgingival plaque removal using hand instruments (scalers and curettes) and powered instruments (sonic, ultrasonic, rotating devices and air-polishing devices). They found that

repeated oral-hygiene instructions showed similar effects to professional mechanical plaque removal using either technique. [174]

Early investigations demonstrated that hand instrumentation by curettes, as well as by very fine rotating diamonds, created the smoothest root surfaces, whereas "vibrating" instruments, such as sonic and ultrasonic scalers, as well as coarse diamonds, tended to roughen the root surface.165

Cobb found manual curettes more technique sensitive and time consuming but more efficient with increased probing depths.

Equivalent clinical outcomes have been shown in studies comparing ultrasonic units with hand scaling. A mean probing-depth reduction of 1.2–2.7 mm was observed with the use of ultrasonic instruments, and values similar to those were obtained with conventional hand instrumentation, showing a reduction of 1.29 mm for moderate pockets and 2.16 mm for deep pockets. [175, 176]

According to a systematic review conducted by Van der Weijden & Timmerman, subgingival mechanical instrumentation resulted in a mean attachment gain of 0.30–1.02 mm in pockets with an initial depth of up to 4 mm and a mean attachment gain of up to 1.58 mm in pockets with an initial depth of ≥7 mm. [177]

The literature on the physical effects of magnetostrictive and piezoelectric ultrasonic scaling devices on tooth surfaces has shown varying results. For example, Flemmig et al. reported that use of a magnetostrictive scaler for root debridement resulted in a rougher root surface compared with use of a piezoelectric device. [178] By contrast, Busslinger et al. showed that after root instrumentation, a piezoelectric device left a rougher surface than a magnetostrictive device. [179] Another study showed that root surfaces treated with a piezoelectric scaler using 200 g of lateral force were smoother than those treated with a magnetostrictive device with the same lateral force. [180]

Comparison of root-surface instrumentation using manual curettes, magnetostrictive ultrasonic scalers and rotary instruments demonstrated nonsignificant differences between the three groups in the amount of calculus remaining, loss of tooth substance and roughness of root surface after root planing; however, magnetostrictive ultrasonic scaling showed the lowest mean scores for the roughness/loss of tooth substance index, indicating less removal of cementum and fewer marks of instrumentation on the dentin surface. [181]

Kawashima et al. compared the effectiveness of two piezoelectric ultrasonic scalers and a hand scaler for subgingival scaling and root planing in vivo and found similar results, showing that the remaining calculus index did not differ significantly among the groups but the roughness/loss of tooth substance index was significantly lower for the groups treated with the piezoelectric ultrasound unit. **[**182**]**

Several investigators have reported that ultrasonic instruments can save 20–50% of time used for periodontal debridement, and cause less discomfort to the patient, while showing equal healing responses of the affected periodontium. Hand instruments yielded greater improvements in clinical parameters, such as bleeding on probing, compared with instrumentation using an ultrasonic system. Use of conventional Gracey curettes may result in higher substance loss, but significantly better calculus removal and smoother surfaces, compared with sonic and ultrasonic instrumentation. **[**183**]**

Around implant abutments, studies have observed that hand instrumentation produced much smoother surfaces with fewer irregularities and grooves compared with sonic and ultrasonic instruments.

Access to furcation areas

Bower has shown that in 81% of maxillary and mandibular molars the furcation entrance is 1.0 mm or less, and in 58% the diameter is 0.75 mm or less. The blade face-width of curettes used in scaling and root planing ranges from 0.75 mm to 1.10 mm, limiting movement of the blade within a space of the same size. **[**184**]**

Leon & Vogel showed that in Class I furcations, hand scaling and ultrasonic debridement have equivalent access and consequent effects on microbial outcome, whereas in Class II and Class III furcations, ultrasonic debridement is significantly more effective than hand scaling in decreasing the counts of motile rods and spirochetes and in maintaining decreased bacterial counts in these sites. Significantly thinner ultrasonic tips, measuring 0.55 mm, are smaller than the working ends of the smallest curettes, making them a superior choice for calculus removal at moderate and severe furcation sites. **[**185**]**

Effectiveness in elimination of virulent substances

Oosterwaal et al. showed equal outcomes in reducing the counts of rods, spirochetes and motile organisms with either manual or ultrasonic magnetostrictive scaling on the subgingival microbiota in periodontal pockets with probing depths of 6–9 mm. 186 Baehni et al. compared the effects, on the subgingival microbiota, of scaling using a piezoelectric instrument with scaling using a sonic instrument and reported no differences between the two techniques in microscopy or culture observations. **[**187**]**

The efficacy of ultrasonic scalers on the removal of endotoxin has also been investigated. Nishimine & O'Leary found that ultrasonic scaling resulted in average residual endotoxin values (i.e. 16.8 ng/ml) approximately eight times higher than those after hand scaling (i.e. 2.09 ng/ml). **[**188**]** Smart et al. found endotoxin levels of <2.5 ng per root after debridement with a magnetostrictive ultrasound unit, which was enough to allow fibroblast reattachment. **[**189**]**

Eick et al. did not find any additional bacterial adhesion after instrumentation with diamond-coated curettes compared with Gracey curettes alone. Studies have shown that initial bacterial adhesion always occurs on surface irregularities. **[**190, 191**]**

However, a study comparing hand instrumentation with Er:YAG lasers and ultrasonics showed that the roughest root surface with the greatest amount of adhesion of Streptococcus sanguinis was obtained after hand instrumentation. **[**192**]**

Effectiveness in cell attachment

Apart from producing a smooth surface free of bacteria, another goal of scaling and root planing is to facilitate fibroblast cell attachment on the root surface. Studies have shown that a very low number of fibroblasts attach on untreated root surfaces with periodontal disease.

Some studies have found no difference between rotary instruments and hand scaling. In the study by Eick et al. the number of fibroblasts attached doubled when the surface was treated with diamond-coated curettes. The fibroblast orientation suggested that moderate roughness of the root

surface was beneficial for cell attachment. More studies are needed to evaluate the relationship between bacterial adhesion and the attachment of fibroblast cells on root-surface roughness. **[**190**]**

In view of the above studies we can say that various studies performed under different conditions and in different models have concluded that neither hand nor mechanical instruments are superior in removing subgingival deposits.

Conclusion

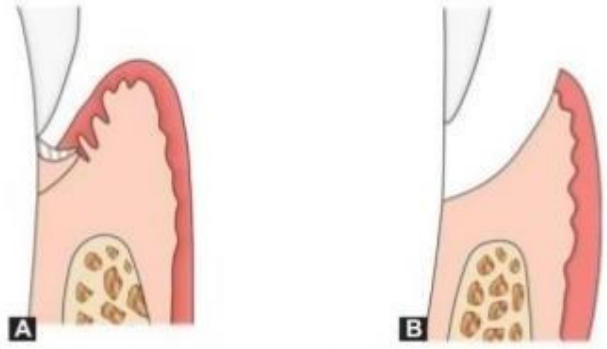
Power-driven instruments have many advantages over the manual scalers. However, many studies have demonstrated that hand and power-driven instruments are equally effective in reducing the probing depth, attaining attachment level gains and reducing inflammation by removal of plaque bacteria, calculus, and endotoxin. Long- term randomized controlled studies are also required to examine the efficacy of any newly designed scaling instruments.

Gingival Curettage

Gingival curettage is a surgical procedure designed to remove the soft tissue lining of the periodontal pocket with a curette leaving only a gingival connective tissue lining.

Scaling: Instrumentation of the crown and root surfaces of the teeth to remove plaque, calculus, and stains from these surfaces.

Root planing: A treatment procedure designed to remove cementum or surface dentin that is rough, impregnated with calculus, or contaminated with toxins or microorganisms. **[**191**]**



Gingival curettage with a curette

Types of curettage

Curettage can be classified based on the instruments/equipments that are being used to eliminate the epithelium.192

1. Gingival curettage: Consists of removal of inflamed soft tissue lateral to pocket wall

a. Subgingival curettage: It is a procedure that is performed apical to epithelial attachment

b. Inadvertent curettage: Curettage that is done unintentionally during scaling and root planing

2. Surgical curettage

3. Chemical curettage

4. Ultrasonic curettage

5. Laser curettage

Gingival curettage, as originally conceived, was designed to promote new connective tissue attachment to the tooth, by the removal of pocket lining and junctional epithelium and hence has a number of advantages :

Advantages of curettage

1. Improved root visualization

2. Complete removal of sulcular epithelium and epithelial attachment

3. Minimal gingival trauma

4. No loss of keratinized gingiva

Disadvantages of curettage

1. Difficult to determine apical extent of epithelial attachment, as it is a closed procedure.

2. It does not afford the improved root surface access and visibility gained with flap surgery that is needed to achieve complete mechanical removal of plaque, calculus, and biofilm.

Gingival curettage is often performed simultaneously with SRP procedure, which is aimed at the complete removal of bacteria, biofilm, calculus, and diseased root structure to achieve a biologically acceptable root surface. This makes it difficult to determine separate effects of each procedure.

Short- and long-term clinical trials have confirmed that gingival curettage provides no additional benefit when compared to SRP alone in terms of probing depth reduction, attachment gain, or inflammation reduction **[**193-195**]** or when performed as part of the scaling and root planning. **[**196**]**

In a study by Ramfjord et al to assess 4 different periodontal therapy (pocket elimination or reduction surgery, modified Widman flap surgery, subgingival curettage, and scaling and root planing), there was no statistically significant difference among the results following the various procedures. For 1-3 mm probing depth, scaling and root planing, as well as subgingival curettage

led to significantly less attachment loss than pocket elimination and modified Widman flap surgery. For 4 - 6 mm pockets, scaling and root planing and curettage had better attachment results than pocket elimination surgery.195 Results from recent longitudinal studies for up to 6½ years indicate that even in deep pockets scaling and root planing alone will have as favorable response as surgical methods including removal of crevicular epithelium and chronically inflamed connective tissues. **[**197**]**

Another study by Lindhe et al established that results after scaling and root planing are not significantly influenced by soft tissue curettage. **[**198**]**

The actual result obtained with curettage is most often a long junctional epithelium, which is the same result obtained with SRP alone. The theoretical clinical advantage of curettage over SRP alone was eliminated when new connective tissue attachment was shown to be an unattainable goal in a study to determine the effect of four periodontal regenerative procedures on the connective tissue attachment level (modified Widman flap procedure, the modified Widman flap procedure combined with transplantation of previously frozen autogenous red marrow and cancellous bone, the modified Widman flap procedure in combination with implantation of beta tricalcium phosphate, and periodic root planing and soft tissue curettage). The data revealed that healing following the four different regenerative procedures resulted in the reformation of an epithelial lining (long junctional epithelium) along the treated root surfaces, with no new connective tissue attachment. **[**199**]**

A review of the literature reveals that other methods for gingival curettage that have been reported, using sodium sulfide, phenol camphor, antiformin, and sodium hypochlorite, ultrasonic devices have the same goal, which is complete removal of epithelium. But there are no reports showing that these alternative methods of epithelial removal have any clinical or microbial advantage over mechanical instrumentation with a curette. **[**200**]** The goals of laser curettage are epithelial removal, as with previous methods, and, in addition, bacterial reduction. A short-term study reported that Nd:YAG laser treatment did not produce statistically significant bacterial reduction. **[**201**]**

This was subsequently confirmed in a multicenter study of laser curettage, which reported that bacterial reduction was not often achieved. **[**202**]**

Laser assisted procedures have demonstrated reduced probing depth, gain in clinical attachment and microbiological benefits, but these studies could not report the effect of curettage when performed without scaling and root planing. **[**203-206**]**

These findings indicate that despite advances in technology, gingival curettage, as a clinical procedure, fails to consistently provide any advantage over SRP alone for the treatment of chronic periodontitis. The American Academy of Periodontology, in its Guidelines for Periodontal Therapy, did not include gingival curettage as a method of treatment. This indicates that the dental community as a whole has started to regard gingival curettage as a procedure with no clinical value. **[**200**]**

Conclusion

Based on current studies, gingival curettage, by whatever method performed, should be considered as a procedure that has no additional benefit to SRP alone in the treatment of chronic periodontitis. These studies provide convincing evidence that SRP alone produces results that are clinically equivalent to curettage with SRP. When these findings are considered, it must be concluded that curettage is a procedure which provides historic interest in the evolution of periodontal therapy but has no current clinical relevance in the treatment of chronic periodontitis.

Full Mouth Disinfection

Periodontal therapy is primarily focused on the reduction/elimination of periodontal pathogens, in combination with the re-establishment (often by surgical pocket elimination) of a more suitable environment (less anaerobic) for beneficial microbiota, because the susceptibility of the host cannot be modulated at a clinical level (with the possible exception of anti-inflammatory medications). [207] Several studies indicate that the presence of periodontal pathogens (persisting or re-established after treatment) is associated with a negative clinical outcome of periodontal treatment. [208-211]

After mechanical debridement, the subgingival microbial load (colony-forming units / ml) decreases to 0.1% of pretreatment levels. However, only 1 week later, the periodontal pocket becomes recolonized by a similar number of bacteria, fortunately of a less pathogenic nature. The origin of these bacteria is still a matter of debate. The multiplication of the remaining bacteria within the pocket, or within either the junctional or pocket epithelium and/or within the dentinal tubules, is considered to be the major cause of this subgingival recolonization. [207]

Supragingival plaque has also shown to play a significant role in the subgingival recolonization of periodontal pockets. As such, bacteria in the saliva or on the tongue, tonsils or oral mucosa also can have an impact on the subgingival recolonization of pockets after periodontal therapy. [212]

The traditional modality of nonsurgical therapy is to perform scaling and root planing according to jaw quadrant (Q-SRP) at a series of appointments. However, as most bacterial species exist not only in periodontal pockets but also colonize several other oral niches and the oropharyngeal area, such as the mucosa, the tongue, the tonsils and the saliva, they could be transmitted from one of their niches to the subgingival environment, leading to the reinfection of treated periodontal pockets. [213 One-stage, full-mouth disinfection

With the perspective of reinfection of treated pockets, a one-stage, full-mouth disinfection procedure, was proposed by the research group headed by Quirynen, at the Catholic University at Leuven, Belgium, as a new treatment strategy. [214]

Aim

• The aim of the full-mouth disinfection approach was to eradicate, or at least suppress, all periodontal pathogens in a very short time span, not only from the periodontal pockets but from the entire oropharyngeal cavity (mucous membranes, tongue, tonsils and saliva).

• Delay the recolonization of the treated pockets by bacteria from untreated sites/niches (called crosscontamination or intra-oral translocation), until better healing of the pockets is achieved. **[**214**]**

The one-stage full-mouth disinfection concept consists of a combination of several therapeutic efforts.

• Full-mouth scaling and root planing (the entire dentition in two visits within 24 h, i.e. two consecutive days) to reduce the number of subgingival pathogenic organisms.

• An additional subgingival irrigation (three times, repeated within 10 min) of all pockets with a 1% chlorhexidine gel in order to suppress the remaining bacteria.

• Tongue brushing by the patient with a 1% chlorhexidine gel for 1 min to suppress the bacteria in this niche.

• Chairside mouth rinsing by the patient with a 0.2% chlorhexidine solution for 2 min to reduce the number of bacteria in the saliva and in the pharynx, including the tonsils (by gargling or via the use of a local spray), prior to and after each session of root planing.

• Optimal oral hygiene, supported during the first 2 months by a 0.2% chlorhexidine mouthrinse to retard the recolonization of the pockets. **[**214**]**

“Proof of principle” experiments

The impact of a one-stage, full-mouth disinfection procedure was explored in four prospective studies conducted by a research group at the Catholic University at Leuven. The studies were designed as “proof of principle” experiments. **[**215-218**]**

In the control group, the recolonization of the treated pockets was provoked by the long time-interval before completion of the debridement of all quadrants (in total 6 weeks) and the lack of oral hygiene in the untreated quadrants. Furthermore, only patients with severe periodontitis (periodontal pockets ± 7 mm) and with a significant amount of supragingival and subgingival plaque and calculus were selected. In other words, the probability of crosscontamination was very high. In the test group, by contrast, a debridement of all periodontal pockets within 2 consecutive days, together with the extensive use of chlorhexidine in all niches, was applied with the goal of extensive reduction of the bacterial load within the oropharynx.

All four studies reported significantly greater improvements of clinical outcomes in the test group, including - a significant additional reduction in probing depth (up to 1.5 mm for single-rooted teeth and up to 1.0 mm for multirooted teeth for initial pockets ±7 mm), a significant additional gain in clinical attachment level (up to 1.7 mm for single rooted teeth and up to 1.5 mm for multi rooted teeth for pockets initially ±7 mm) and a significantly greater reduction in bleeding upon probing.The studies also showed statistically significant additional reductions in the prevalence of periodontopathogens, especially subgingivally, and to a lesser extent in the other intra-oral niches, the latter especially during the period when the patients were rinsing with chlorhexidine.

All the above-mentioned studies clearly indicated that when the opportunity for intraoral translocation of periodontopathogens was reduced, the outcome of nonsurgical periodontal therapy could be improved. An indirect impact via a change in the supragingival plaque that may gradually extend subgingivally seems to be a more reasonable explanation.

However, bacteria can also be translocated subgingivally by contaminated oral hygiene aids and ⁄or dental instruments (which can penetrate the pocket). Several studies reported that toothbrushes used in a daily regimen harbor a complex microbiota, including periodontopathogens, cocci, Haemophilus spp. and fungi, and Streptococcus mutans, and most of these bacteria survived for 48 h or even longer on these toothbrushes. **[**219,220**]**

Studies

In one study, Pawlowski et al. left three teeth in one quadrant untreated, while all other teeth were scaled and root planed. The untreated sites showed a significant probing depth reduction

and gain in attachment, and the number of Treponema denticola and P. intermedia species, counted in the subgingival flora of these sites, were reduced for up to 12 weeks. Also, these authors suggested that up to half of the improvements observed following scaling and root planing may be a result of factors other than the removal of plaque, calculus and irritants. **[**221**]**

Clinical data obtained with different protocols in different centres involving a full-mouth concept did not show any significant differences between test group (full mouth scaling within 24hrs) and control group (full mouth scaling and root planning extended over 1 week/2 weeks). The microbiological analyses included in some of these studies also revealed only minor additional improvements with the full-mouth approach.222-225 Authors Teughels et al defend the efficacy of FMD by listing the drawbacks of studies which proved FMD non-significant.

The authors stated that these studies differ significantly from the Leuven trials -

• They did not include proper disinfection of the intra-oral niches (besides the periodontal pockets), did not use a strong antiseptic during the initial healing time, and ⁄ or reduced the probability of intraoral translocation of bacteria by giving instruction on optimal oral hygiene prior to therapy. Part of the success of the one- stage, full-mouth disinfection protocol might be explained by the extensive use of chlorhexidine.

• Optimal oral hygiene should only performed in the treated quadrants, in other words, a significant amount of plaque in at least one quadrant remained for up to 6 weeks after debridement of the first quadrant, thus allowing enough time for bacterial translocation.

• The best results with the one-stage, full-mouth disinfection protocol were recorded in deep pockets, but most of the studies enrolled patients with only moderate periodontitis.

None of the studies performed at other centres reported any negative outcomes of a full- mouth strategy, and most studies indicated a significant reduction in treatment time, especially after full mouth ultrasonic debridement.

In a study by Koshy, where a less potent disinfection (1% povidine iodine irrigation) protocol was used, resulted in 26% more sites with a reduction in pocket depths to <5 mm when compared with a staged approach. **[**226**]**

A systematic review published in 2008, in which meta-analyses were conducted, confirmed that both the traditional quadrant approach and the newer full-mouth debridement were equally effective; no statistically significant differences were observed between the methods. The FMD approach resulted in only modest additional improvements of probing depth and clinical attachment compared with conventional treatments for sites with an initial probing depth of 5–6 mm in single-rooted teeth. **[**227**]**

Another systematic study review concluded that FMD or FMS did not provide clinically relevant advantages over the conventional strategy. **[**228**]**

A systematic review comparing FMD and Q-SRP concluded that FMD yielded statistically significant differences in terms of clinical attachment level gain in single and multirooted teeth with moderate pockets vs. Q-SRP. A secondary objective of the review was to examine whether the degree of post-treatment discomfort in the FMD group was more serious than in the Q-SRP group. No significant differences were observed. **[**213**]**

Several studies have been performed in an attempt to assess the results of the application of this therapy, but the comparison between studies is difficult due to the variety of methodologies used in research, such as: homogeneity of the allocated groups, inclusion criteria and adopted exclusion criteria, periodontal disease classification mode, changes in treatment, use and dosage of chlorhexidine and others.

Conclusion

The one-stage, full-mouth disinfection concept has shown significant additional clinical and microbiological improvements with nonsurgical periodontal therapy. The concept has no disadvantages and/or risks for the patient. The clinician and the patient therefore can only gain via a better outcome of the mechanical debridement, reduced need for surgery, and more efficient treatment and time management, with less travelling or absence from work for the patient. A scientific explanation for the success of this concept has not yet been obtained. Reduction in the probability of bacterial cross- contamination, optimal combination/application of antiseptics may be contributing factors. More research is needed to explore in greater detail the potential of the onestage, full-mouth disinfection, and to improve its applicability and benefits.

Periodontal – Endodontic Controversy

The relationship between the periodontium and the pulp was first discovered by Simring and Goldberg in 1964. Over the past century, the dental literature has consistently reflected a controversy related to the effect of periodontal disease on the dental pulp and more recently the effect of pulpal necrosis on the initiation and progression of marginal bone loss. Pulpal and periodontal problems are responsible for more than 50% of tooth mortality today. An endo-perio lesion can have a varied pathogenesis which ranges from quite simple to a relatively complex one. Several questions have been raised in lieu of this controversy - Is periodontal disease a cause of pulp necrosis? Can a pulpless tooth be the cause of periodontal disease? The answers to these basic questions are of utmost clinical importance, as the appropriateness of treatment planning hangs in the balance. These lesions often present challenges to the clinician as far as diagnosis and prognosis of the involved teeth are concerned. **[**229, 230**]**

Development

Dental pulp and periodontium have embryonic, anatomic and functional inter- relationships. They are ectomesenchymal in origin, the cells from which proliferate to form dental papilla and follicle, which are the precursors of the pulp and periodontium, respectively. They are separated by the formation and development of tooth bud from the overlaying ectoderm into enamel and dentine. **[**231**]**

The embryonic development gives rise to anatomical connections which remain throughout the life of the tooth. The apical foramen decreases in size as the proliferation of the Sheath of Hertwig continues. It remains patent and serves as the communication on which the pulpal tissues rely for nutrition and nervous innervation. As the root develops, ectomesenchymal channels get incorporated, either due to dentine formation around existing blood vessels or breaks in the continuity of the Sheath of Hertwig, to become accessory or lateral canals. The majority of accessory canals are found in the apical part of the root and lateral canals in the molar furcation regions. Tubular communication between the pulp and periodontium may occur when dentinal tubules become exposed to the periodontium by the absence of overlying cementum. **[**232**]**

These are the pathways that may provide a means by which pathological agents pass between the pulp and periodontium, thereby creating the endo-perio lesion.

Pathways of communication

There are various pathways for the exchange of infectious elements and irritants from the pulp to periodontium or vice versa, leading to the development of endodontic periodontal lesions. **[**230**]**

Pathways of developmental origin (anatomical pathways):

• Apical foramen, accessory canals/lateral canals

• Congenital absence of cementum exposing dentinal tubules

• Developmental grooves

Pathways of pathological origin:

• Empty spaces on root created by Sharpey's fibers

• Root fracture following trauma

• Idiopathic root resorption - internal and external

• Loss of cementum due to external irritants.

Pathways of iatrogenic origin:

• Exposure of dentinal tubules following root planing

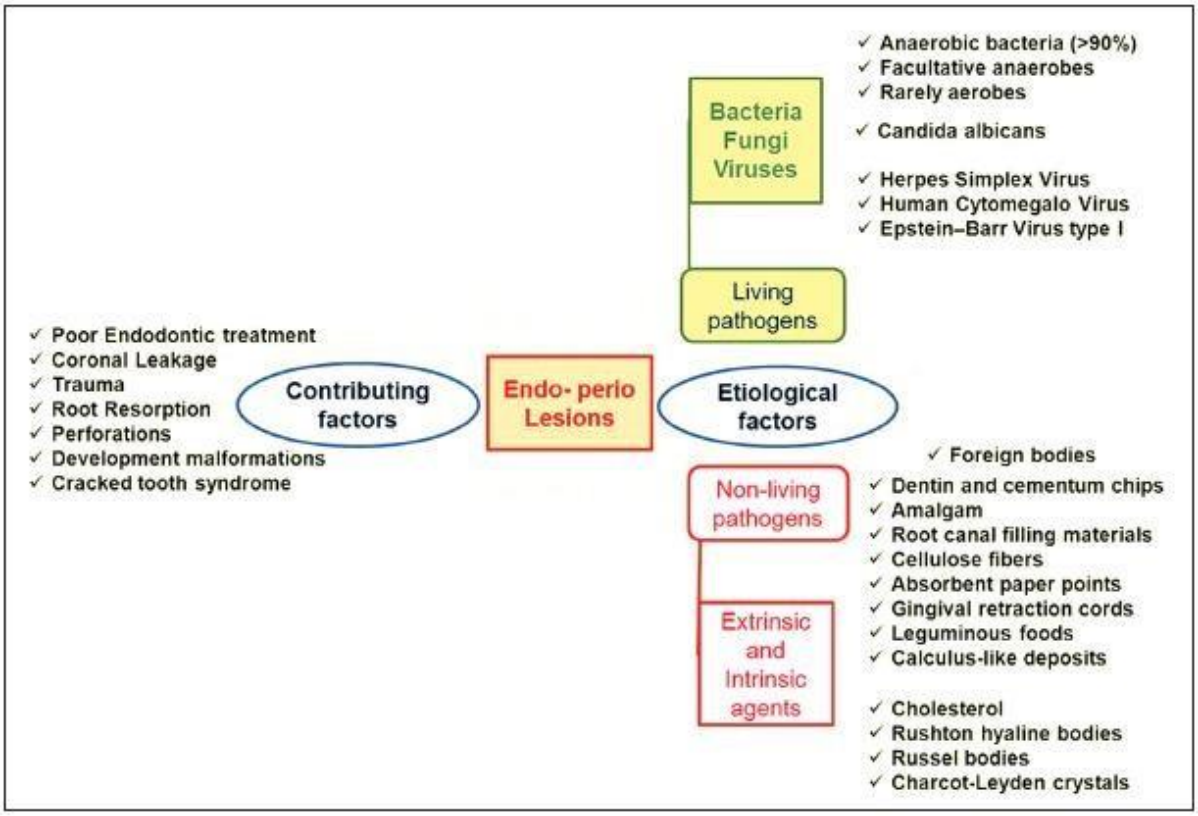
• Accidental lateral root perforation during endodontic procedures

• Root fractures during endodontic procedures.

Demonstration of the presence of such pathways is commonly identified as evidence that specific periodontal disease must have some effect on the health of the dental pulp.

Etiological factors

Following is a summarized illustration that enumerates the etiological and contributing factors that eventually may lead to an endo-perio lesion. **[**230**]**



Classification of endodontic-periodontic lesions

The most accepted classification of endodontic-periodontal lesions based on pathology of origin was proposed by Simon et al as follows:

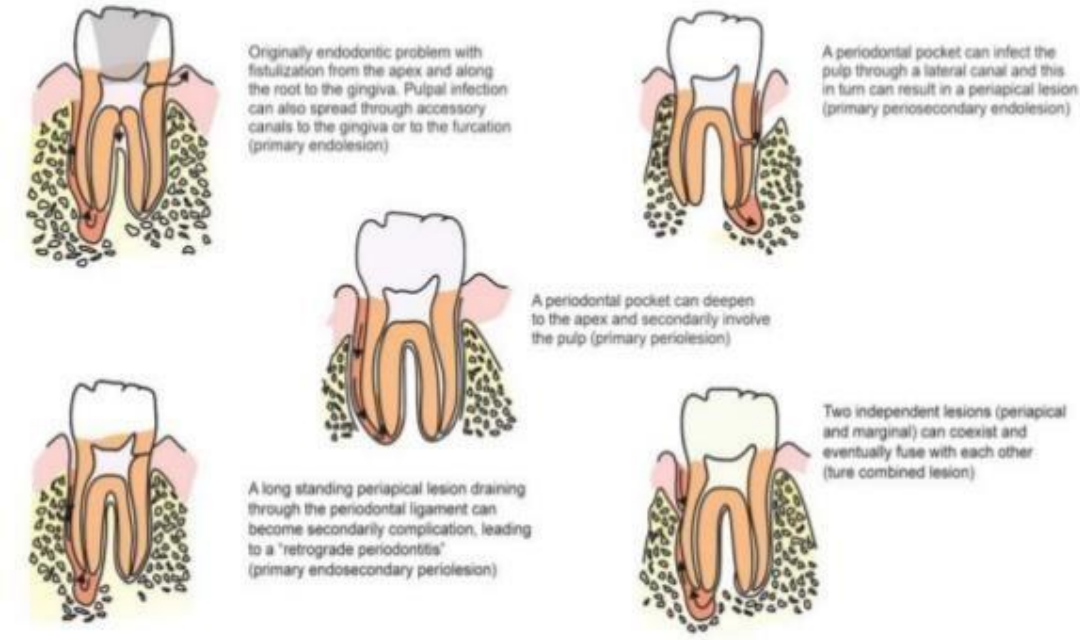
• Primary endodontic lesions

• Primary periodontal lesions

• Primary endodontic lesions with secondary periodontal involvement

• Primary periodontal lesions with secondary endodontic involvement

• True combined lesions. **[**233**]**



Diagrammatic representation of various types of endodontic-periodontal lesions

Effect of periodontal infection on endodontium

A vital pulp is very resistant to microbial invasion. In a healthy pulp, penetration of the surface by oral bacteria is relatively slow or may be blocked entirely. It has been found that the pulp has a quite sophisticated vasculature system with a network of capillary beds, pre capillary sphincters and arteriovenous shunts, which provides a significant capacity for the pulp to survive. **[**231**]**

The formation of bacterial plaque on denuded root surfaces, following periodontal disease, has the potential to induce pathologic changes in the pulp through lateral or accessory canals. This process, the reverse of the effects of a necrotic pulp on the periodontal ligament, has been referred to as retrograde pulpitis. **[**234**]**

Major changes seen in relation to endodontium after a tooth is inflicted of periodontal disease are as follows:

1. Atrophic changes: The pulp tissue of a periodontally involved tooth has cells which are small and have more collagen depositions than normal. Due to impaired nutrition, the pulp cells slowly

degenerate. The death of the cell is so gradual that morphologic evidence sometimes appears to be lacking. The cause of these atrophic changes is the disruption of blood flow through the lateral canals, which leads to localized areas of coagulation necrosis in the pulp. These areas are eventually walled off from the rest of the healthy pulp tissue by collagen and dystrophic mineralization. With slowly advancing periodontal disease, cementum deposition may act to obliterate lateral canals before pulpal irritation occurs.

2. Inflammatory changes: The causative agents of periodontal disease are found in the sulcus and are continually challenged by host defenses. An immunologic or inflammatory response is elicited in response to this microbiologic challenge. This results in the formation of granulomatous tissue in the periodontium. When periodontal disease extends from the gingival sulcus towards the apex, the inflammatory products attack the elements of the periodontal ligament and the surrounding alveolar bone. A clear cut relationship between progressive periodontal disease and pulpal involvement, however, does not invariably exist.

3. Resorption: Resorption of the sides of the roots is frequently found adjacent to the granulation tissue overlying the roots. When the periodontal lesions are deep, resorption may also be found within the root canals, often opposite lateral canals, and at the apical foramen. Since this resorptive process extends into the dentin peripherally towards the pulp, and the activating factors are produced from the periodontal lesion, a name which reflects the etiology of this phenomenon, peripheral inflammatory root resorption (PIRR) was proposed. **[**235**]**

Over the past century, the dental literature has consistently reflected a controversy related to the effect of periodontal disease on the dental pulp. Some studies have suggested that the effect of periodontal disease on the pulp is atrophic and degenerative in nature including a decrease in number of pulp cells, an increase in dystrophic calcifications, fibrosis, as well as a direct inflammatory effect and therefore, periodontal disease and periodontal treatments should be regarded as potential causes of pulpitis and pulpal necrosis. On the other hand, many studies have demonstrated that periodontal disease or sequelae of periodontal treatment does not affect the pulp. It has also been advocated that periodontal disease has no effect on the pulp, unless it extends all the way to the tooth apex, the dental pulp is capable of surviving significant insults and that the effect of periodontal disease as well as periodontal treatment on the dental pulp is negligible. **[**236-240**]**

Kirkham examined 100 periodontally involved teeth and found that only 2% had lateral canals located in a periodontal pocket. **[**241**]**

Tagger & Smukler removed roots from molar teeth so extensively involved with periodontal disease that root amputation was required, and found that none of the pulps of the resected roots showed inflammatory changes. **[**242**]**

Haskell et al. also removed roots from maxillary molars with total or nearly total periodontal involvement and found no inflammatory cells or very few inflammatory cells present in the pulps of the periodontally involved resected roots. **[**243**]**

Czarnecki & Schilder performed a histological study of intact, caries-free teeth and compared the pulps of teeth which were periodontally within normal limits with teeth which had periodontal disease. The pulps in the intact, caries-free, periodontitis group

were all histologically within normal limits regardless of the severity of the periodontal disease. In the same study they found that only teeth with extensive decay or extensive restorations showed evidence of pulp pathosis. **[**240**]**

A case report by Torabinejad & Kiger of a patient with extensive periodontal disease supports the position that advanced periodontal disease has little or no effect on the pulps of humans. **[**239**]**

Blomlof et al created defects on root surfaces of intentionally extracted monkey teeth with either open or mature apices. The root canals were either infected or filled with calcium hydroxide and replanted back in their sockets. After 20 weeks, marginal epithelial downgrowth was found on the denuded dentin surface of the infected teeth. Noxious elements of pulpal origin including inflammatory mediators and bacterial byproducts may leach out through the apex, lateral and accessory canals, and dentinal tubules to trigger an inflammatory response in the periodontium including an early expression of antigen presentation. **[**244**]**

Ross & Thompson evaluated the progress of 100 patients with maxillary molar furcation involvement over a period of 5–24 years. Sixty-two of the patients were followed for over 10 years. Of the 387 maxillary molars, 79% had at least 50% or less bone support around one root prior to periodontal treatment. Only 4% (14 of 380 vital teeth) required root canal treatment

subsequent to periodontal therapy, and it was the opinion of the authors that in all cases the need for root canal treatment resulted from caries or pulp degeneration under restorations. None were ascribed to the effects of the advanced periodontal disease on the pulp. Two per cent of the teeth in this study had root canal treatment prior to periodontal therapy for reasons unknown to the authors. **[**245**]**

Bergenholtz & Nyman evaluated 52 patients with advanced periodontal disease over a 4 to 13 year period. Of 417 nonabutment teeth, 60% had crestal bone level in the apical two-thirds of the root. 3% (14 of 417 teeth) required root canal treatment during the recall period. The reasons cited by the authors were progression of periodontal disease to involve the root apices in four teeth, decay into the pulp in five, one with internal resorption, two with crown fractures, and two for unknown reasons. **[**237**]**

Jaoui et al. studied patients with advanced periodontal disease for 5–14 years after completion of active periodontal treatment. Of the 571 teeth that did not have root canal treatment at the time of completion of periodontal treatment, only one tooth (0.175%) required root canal treatment over the 5- to 14-year recall period. **[**238**]**

Langeland et al presented evidence that periodontal disease must extend all the way to the apex of a tooth before an accumulation of plaque in the area of the apical foramen or foramina can cause significant pulp involvement and suggested that pulpal insults through patent dentinal tubules or the occasional exposed lateral canal have relatively insignificant effects on the ability of the dental pulp to survive. **[**236**]**

Effect of endodontic infection on periodontium

Historically, the effect of periodontal disease on the dental pulp has been a source of discussion for the better part of the past century. Only in recent years has the reverse been discussed - the potential effect that a tooth with a necrotic pulp or a tooth that has had root canal treatment may pose as a risk factor in the initiation of periodontal disease, the progression of periodontal disease, and the resolution of periodontal pockets.

Many studies have stated that a pulpless tooth with a periapical lesion promotes the initiation of periodontal pocket formation, progression of periodontal disease, and interferes with healing of a

periodontal lesion after periodontal treatment. The presumed pathway is primarily through patent dentinal tubules. **[**246-248**]**

Jansson et al assessed the effect of endodontic pathogens on marginal periodontal wound healing of denuded dentinal surfaces surrounded by healthy periodontal ligament. Their results showed that in infected teeth, the defects were covered by 20% more epithelium while the noninfected teeth showed only 10% more connective tissue coverage. They concluded that pathogens in necrotic root canals may stimulate epithelial downgrowth along denuded dentin surfaces with marginal communication and thus augment periodontal disease. **[**246**]**

In a second paper Jansson et al state, 'Mean probing depths for each tooth were approximately 0.2mm deeper in teeth with the same degree of radiographic attachment in the presence of angular destructions when periapical pathology was present compared to teeth without periapical pathology'. In an evaluation of clinical radiographs, Jansson et al. state that teeth with periapical lesions had lost significantly more proximal marginal bone, approximately 2mm. Relative bone loss is difficult to evaluate from the data presented. **[**247**]**

In their 1995 paper, Jansson et al. extrapolate their data to estimate that the rate of marginal proximal radiographic bone loss for teeth with active periapical lesions in periodontitis-prone patients is 0.19 mm/year vs. 0.06mm/year for teeth with no periapical lesion or where there is evidence of reduction in lesion size. **[**248**]**

Harrington et al mentioned another parameter which may influence clinical impressions related to the dental pulp from early histological observations. They explained the importance of adequate fixation of pulp tissue to be a challenge, and artifacts resulting from inadequate fixation continue to be described as evidence of pathosis. **[**234**]**

Ehnevid et al found that there was no correlation between periapical pathosis and mean pocket depth reduction for nonsurgical treatment of vertical marginal defects, nor was there any correlation between periapical pathosis and mean pocket depth reduction after surgical management of either horizontal or vertical defects. **[**249, 250**]**

In a later study, Jansson & Ehnevid evaluated the periodontal status of mandibular molars. They reported that the mean periodontal probing depth of a nonroot-filled molar with a periapical lesion

was 0.7mm deeper than corresponding teeth with no periapical lesions, and that the mean probing depth difference at proximal sites was 0.2mm. **[**251**]**

Diagnostic characteristics of endo-perio lesions

The differential diagnosis of endodontic and periodontal diseases can sometimes be difficult but it is of vital importance to make a correct diagnosis so that the appropriate treatment can be provided.252

Lesion	Pain	Swelling	Periodontal pocketing	Radiographic	Vitality
Primary endodontic	Moderate to severe	Possible	None unless sinus tract	Possible periapical radiolucency	Non-vital
Primary endodontic secondary periodontic	Moderate to severe	Likely	Evident or sinus tract	Radiolucency from apex to sulcus, decreased crestal bone height	Non-vital
Primary periodontic	None to moderate	Possible	Moderate	Decreased crestal bone height	Vital
Primary periodontic–secondary endodontic	None unless acute endo	Possible	Severe	Bone loss approaching apex	Vital
Combined pulpal–periodontal	Moderate to severe	Likely	Severe, connects with periapex	Bone loss extending to apex	Non-vital

Effect of treatment Periodontal procedures

The aforementioned clinical research studies by Ross & Thompson, Bergenholtz & Nyman and Jaoui et al. evaluated patients who presented with advanced periodontal disease, received what was considered to be appropriate periodontal treatment, and received follow-up maintenance care for periods ranging from 4 to 24 years. There were 1,623 teeth in the combined studies which were treated for advanced periodontal disease and were assumed to have vital pulps at the completion of treatment and the beginning of the recall period. Four per cent (67 of 1623 teeth) required root canal treatment subsequent to periodontal disease, periodontal treatment, and follow-up periodontal care. The cause of pulp necrosis could be identified by the clinicians in most cases. Recurrent decay resulting in pulp exposure was the primary cause. Extension of periodontal disease to involve the root apices is also cited as a reason for root canal treatment.

Scaling and root planing

This procedure removes the bacterial deposits. However, improper root planning procedures can also remove cementum and the superficial parts of dentin, thereby exposing the dentinal tubules to the oral environment. Subsequent microbial colonization of the root dentin may result in bacterial invasion of the dentinal tubules. As a consequence, inflammatory lesions may develop in the pulp. The initial symptom is sharp pain of rapid onset that disappears once the stimulus is removed.

The increase in intensity of pain may be explained by one or both of the following two reasons.

• The smear layer formed on the root surface by the scaling procedures will be dissolved within a few days. This, in turn, will increase the hydraulic conductance of the involved dentinal tubules and decrease the peripheral resistance to fluid flow across dentin. Thereby, pain sensations are more readily evoked.

• Open dentinal tubules serve as pathways for diffusive transport of bacterial elements in the oral cavity to the pulp, which is likely to cause a localized inflammatory pulpal response. Vigorous root planing may remove cementum and expose numerous dentinal tubules through which etiological agents may enter and inflame the pulp. **[**253**]**

It seems that the pulp is usually not directly affected by periodontal disease until recession has opened an accessory canal to the oral environment. At this stage, pathogens penetrating from the oral cavity through the accessory canal into the pulp may cause a chronic inflammatory reaction and pulp necrosis. However, as long as the accessory canals are protected by sound cementum, necrosis usually does not occur. In addition, if the microvasculature of the apical foramen remains intact, the pulp will maintain its vitality. The effect of periodontal treatment on the pulp is similar during scaling and root planing or periodontal surgery if accessory canals are severed and/or opened to the oral environment. In such cases microbial invasion and secondary necrosis of the pulp can occur. **[**236**]**

Acid etching

Root conditioning using citric acid during periodontal regenerative therapy helps to remove bacterial endotoxin and anaerobic bacteria and to expose collagen bundles to serve as a matrix for new connective tissue attachment to cementum. Though beneficial in the treatment of periodontal disease, citric acid removes the smear layer, an important pulp protector. Cotton and Siegel reported that citric acid, when applied to freshly cut dentine, has a toxic effect on the human dental pulp. However, several other studies have concluded that pulpal changes after the application of citric acid does not show any significant changes in the pulp. **[**254**]**

From these studies and from many other recall studies in the periodontics literature, it appears that periodontal treatment, as well as periodontal disease, has almost negligible effect on the dental pulp.

Endodontic procedures

In 1979 Nyman & Lindhe evaluated a group of patients who had lost 50% or more periodontal bone support. After periodontal and restorative treatment they were followed for a period of 5–8 years. In comparing bone height measurements of patients who had both an endodontically treated abutment and a vital abutment tooth, they found that the bone height was maintained equally well around the root-filled teeth as around the vital teeth. **[**255**]**

Sanders et al. reported in 1983 that after the use of freeze-dried bone allografts 65% of the teeth that did not have root canal treatment showed complete or greater than 50% bone-fill in periodontal osseous defects, while only 33% of the teeth which had root canal treatment prior to the periodontal surgical procedure had complete or greater than 50% bone-fill. **[**256**]**

Miyashita et al. recently used a paired sample in which the test tooth had been endodontically treated or not treated but had a periapical radiolucency, but not the control tooth. The selected patients had minor or no signs of periodontal disease. The distance from the cemento–enamel junction to the marginal bone level was measured using intraoral radiographs. A somewhat larger loss (mean value 0.1mm) of alveolar bone support was found in test teeth vs. the controls, but the difference was not statistically significant and the study failed to show a correlation between a reduced marginal bone support and endodontic status. **[**257**]**

In contrast to the preceding clinical studies, McGuire & Nunn attempted to relate disease etiology and progression of periodontal disease with a pretreatment-assigned prognosis, and found that some commonly accepted clinical parameters did not accurately predict a tooth's survival. Their statistical model had predicted that endodontic involvement would be associated with the probability that the prognosis for such a tooth would worsen over time. In their clinical study, however, the actual outcome was that none of the 131 teeth lost from a total of 2,509 teeth had endodontic involvement. Endodontic involvement at the time of periodontal treatment planning therefore was determined not to be a significant clinical factor associated with tooth loss. In the Jaoui et al. study, tooth loss was 2% of the 911 periodontally involved teeth and the overall failure rate of the 340 endodontically treated teeth was 1.2%. **[**258-260**]**

A series of papers by the groups of Jansson & Ehnevid concluded that root canal treatment should be completed before periodontal therapy and that root canal treatment should be accomplished at a very high technical level. It has also been found that the periapical trauma may occur by over instrumentation during shaping and cleaning of the root canal, extrusion of irrigants, sealer and gutta percha points that may hinder new bone, cementum and connective tissue repair. Therefore, precautions should be taken when periodontal therapy has to be followed by endodontic treatment.

Johnson and Orban showed that periodontal disease that remained after unsuccessful endodontic therapy cleared up after successful endodontic therapy. Several authors have also shown the remission of severe periodontal bone loss after endodontic therapy alone. Simring and Goldberg postulated that endodontic therapy is indicated in the treatment of terminal periodontal disease that does not respond to periodontal therapy. Many studies in the literature indicate that combined periodontal and endodontic therapy is essential for successful healing of a periodontal-endodontic lesion. Most authors agree that both forms of therapy are essential for successful healing of combined lesions. However, the problem arises over which lesion came first and which caused or perpetuated the clinical problem. **[**261**]**

Conclusion

A perio-endo lesion can have a varied pathogenesis which ranges from quite simple to relatively complex one. Having enough knowledge of these disease processes is essential in coming to the correct diagnosis. Because the primary aetiology is infection, endodontic treatment is directed at control and elimination of the root canal flora by working in a sterile way. The presence of a combined endodontic-periodontal lesion will always result in a compromised situation following treatment. Even with apparently successful treatment, the tooth will still be compromised as there is likely to be some gingival recession and loss of periodontal attachment and bone support. It is of utmost importance that the patient maintains good oral hygiene and obtains regular professional care for this region. The tooth anatomy and the etiology of endodontic periodontal lesions offer a strong base for establishing a correct diagnosis. Due to the complexity of these affections, an interdisciplinary approach with a good collaboration between endodontists, periodontologists and microbiologists, is recommended.

Root Biomodification

Repair of the periodontium and the regeneration of periodontal tissues remains a major goal in the treatment of periodontal disease and is an area still in need of major research attention. Research regarding periodontal therapy has made it clear that standard treatment techniques do not result in periodontal regeneration once root exposure occurs. It has become apparent that, if the goal of periodontal regeneration is to be realized, the problem of regeneration needs to be approached from a biological perspective. **[**262**]**

One important consideration in periodontal regenerative attempts is the root surface which has become exposed to the oral cavity as a result of periodontitis. In regenerative attempts the root surface functions as one of the wound margins and must provide an appropriate surface for cell attachment and fibre development if regeneration is to occur. **[**263**]**

Changes in the pathologically exposed root surface are well documented.

Root surface changes

The normal root is rich in collagen, with extrinsic and intrinsic fibers that form a renewable connection to the adjacent alveolar bone.

Alterations in the root surface due to pathological causes include:

• Loss of collagen fibre insertion

• Contamination of the root surface by bacteria and or endotoxins

• Alterations in mineral density and composition.

• Also they may lack the necessary chemotactic stimuli for migration of cells capable of producing periodontal regeneration. **[**264**]**

The exposed root surface, as a result of periodontitis will undergo substantial alterations and may no longer serve as an appropriate substrate for cell attachment and fibre development.

The goal of root surface biomodification is to determine the alterations of disease root surface that would create an appropriate and hospitable surface for cell attachment and eventually development of fibre attachment. **[**265**]**

Methods of root conditioning

1. **Mechanical modification of root surface**

This includes removal of cementum, removal of softened dentin, or the smoothening of surface irregularities.

Although, the effectiveness of scaling and root planing has been well documented, its efficacy in making the root surface disease free has been questioned, since such root modification may not completely remove contaminated cementum particularly in apical areas. Scaling and root planing eliminates the mineralized debris but leads to the formation of a thin residous smear layer that delays the adhesion of new fibroblasts and connective tissue on periodontally involved root surfaces. Smear layer is an amorphous, granulated, and irregular layer covering the root surface when observed under scanning electron microscope (SEM). **[**266**]**

Mishra et al, have evaluated the effects of hand instruments, ultrasonic scaling, and Er:YAG lasers on smear layer formation. Most of the specimens that were treated with hand instruments (66.7%) and ultrasonic scaling (80%) were covered by a smear layer, while it was not observed in most of the specimens treated with laser (60%). A significant difference was found in the presence of the smear layer between ultrasonic scaling and laser treatments. A smear layer will inevitably cover the instrumented surface. **[**267**]**

The success of mechanical biomodification may depend on several other factors. The tooth type and the severity and nature of the periodontal disease or it may be related to the amount of local non mineralized and mineralized deposits on the root surface, which requires more or less effort by the operator to attain a clinically smooth root surface. The age of the patient and the caries experience of the involved teeth, as well as diet- and oral hygiene-related issues (e.g., toothpaste, mouth rinses, and the consumption of low-pH beverages), are additional factors that may

influence the conditions of radicular dentin and, consequently, the amount of smear layer formed. **[**266**]**

Thus alternate approaches were suggested to overcome the limitations inherent in the mechanical root cleansing therapy.

2. **Chemical methods**

Because the presence of a smear layer is unsuitable for reattachment of periodontal connective tissue, the purpose of surface demineralization is to recreate a biologically active substrate for periodontal cellular reattachment. Therefore, chemical conditioning agents are often used to help remove root surface impurities including minerals and cytotoxic materials derived from bacterial products.

Various chemical agents have been employed for root biomodification. **[**263**]**

• Citric acid

• Tetracycline hydrochloride

• Fibronectin

• Laminin

• EDTA

• Sodium hypochlorite

• Sodium deoxycholate

• Stannous fluoride

• Hydrochloric acid

• Chlorhexidine

• Formalin

• Cohn’s plasma fraction IV

• Cetyl pyridinium chloride & sodium - N- Lauroyl sarcosine

• Zinc iontophoresis

• Bile salts & plasma fractions

• Phosphoric acid

• Maleic acid

• Lactic acid

• Polyacrylic acid

The mechanism by which these chemicals operate on the root surface is not well understood, but it has been hypothesized that demineralizing agents act by exposing collagen fibres within the root matrix thereby facilitating attachment by other fibres in the periodontium, and/or by decontaminating the root surface via elimination of endotoxin and bacteria, and/or by removal of the root debris allowing for the unobstructive attachment of regenerative cells to the root surface. The rationale for this approach was that a major requirement of regeneration of connective tissue attachment to a denuded, periodontitis affected root is migration and attachment of connective tissue cells to the root surface. **[**268**]**

As early as 1833, Marshall presented a case of pocket eradication with “presumable clinical reattachment” after the use of aromatic sulfuric acid. In the 1890s; Stewart described the use of acids in conjunction with the mechanical removal of calculus and cementum. The potential of acid demineralization of root surfaces as an adjunct to new attachment procedures gained popularity following studies by Urist (1965) that suggested that dentin following acid demineralization possessed inductive properties. **[**269-271**]**

Citric acid (1%)

It was suggested for smear layer removal by Register in 1973 and has been studied extensively. Citric acid consistently enhances root features, thought to be relevant in the regeneration of periodontal tissues: Exposing collagen, inducing mesenchymal cell differentiation, extracting endotoxins and other toxic products, accelerating cementogenesis and widening dentinal tubules.

Register and Burdick reported reattachment of collagen fibres to previously denuded root surfaces following treatment of root surface with citric acid, in a study conducted on 1000 teeth in 50 dogs and 15 cats. **[**272**]**

Ririe et al. studied healing of periodontal connective tissue after surgical wound and application of citric acid in dogs. The demineralized sites showed enhanced, connective tissue healing with rapid and consistent establishment, devoid of initial embedding of collagen fibres in newly formed cementum. **[**273**]**

A.M. Polson and M.P. Proye studied the effect of citric acid treatment of the denuded root resulted in new connective tissue attachment, and the response appeared to be dependent upon early establishment of fibrin linkage with the root surface. **[**274**]**

Cole et al. examined the effects of citric acid in a pilot study after replaced flap surgery. A probing attachment level gain of 2.1 mm for the acid-treated teeth resulted, compared to 1.5 mm for controls. **[**275**]**

On the other hand, several studies have reported controversial results regarding the use of root surface conditioning. Sture Nyman, Lindhe, and Karring studied the healing following surgical treatment and root demineralization in monkeys with periodontal disease and reported that citric acid conditioning of the root dentin surface did not promote cementum formation and new connective tissue attachment. **[**275**]**

Mark S.C Jr and Mehta N.R in a study that compared the effects of a comprehensive surgical plaque control procedure with or without citric acid treatment for generalized and localized effects on gingival height, probing pocket depth and attachment level, concluded that there is no added clinical advantage of citric acid conditioning of the roots during treatment of periodontitis. **[**277**]**

Tetracycline

Tetracyclines are a group of bacteriostatic antimicrobials effective against a wide range of organisms.

Tetracyclines have a low pH in concentrated solution and this can act as a calcium chelator resulting in demineralization. Tetracyclines possess several unique antibacterial characteristics that may contribute to their efficacy in periodontal therapy -

• It enhances attachment and growth of gingival fibroblasts, thus facilitating regeneration.

• It has anti-collagenase activity.

• It has anti-inflammatory properties.

• It has high substantivity.

• It inhibits parathyroid hormone induced bone resorption.

Another beneficial effect of tetracycline conditioning was that the drug was released in a biologically active concentration for 48 hours and upto 14 days after application. Various types of tetracyclines have been suggested, but tetracycline hydrochloride used for at least 30 seconds, has proven most effective in removing smear layer and opening dentinal tubules. They are generally used as a 0.5% solution at a PH of 3.2 and is applied for 5 minutes. The solution is prepared by adding 1 standard ml of sterile water to the contents of each capsule, then thoroughly mixing the two. The material is applied with lateral pressure using passive burnishing technique using a sterile cotton pledget. It was demonstrated that 10 or 100 mg/ml solutions of tetracycline hydrochloride were sufficiently concentrated to remove the smear layer and expose a regular pattern of open dentinal tubules.

Terranova et al. suggested that treatment of dentin surfaces with Tetracycline HCL increase binding of fibronectin which subsequently promotes the attachment and growth of gingival fibroblasts. These findings suggest that tetracycline and fibronectin may be used to treat periodontally involved tooth surfaces. **[**278**]**

Alger et al. carried out a study to determine how the treatment of human tooth roots with tetracycline HCL and fibronectin during periodontal surgery influences the attachment of the gingiva to the root surface. Tetracycline and fibronectin group demonstrated some reattachment whereas the tetracycline treated group showed greater connective tissue attachment. **[**279**]**

However, long etching time of 3 minutes and above has been shown to impair periodontal healing. In another study by Lewis et al, there were no significant differences in clinical parameters b/w tetracycline treated & control sites. **[**280**]**

Isik et al. compared different tetracycline HCl concentrations of 0, 10, 25, 50, 75, 100, 125 and 150 mg/ml for root conditioning and found that concentration between 50 mg/ml and 150 mg/ml showed a statistically significant opening of dentinal tubules at 1,3 and 5 minutes. **[**281**]**

Ethylene diamine tetra-acetic acid

Use of acidic agents to demineralize the root surface had a drawback of adversely affecting the surrounding tissues. So, a chemical agent that could remove the smear layer and demineralize the tooth surface at neutral pH was required. EDTA exerts its demineralizing effect through chelating divalent cations at neutral pH. Studies have shown that application of 18% EDTA on root surface improves fibroblast attachment and migration on the root surface and also facilitates development of an oriented fibre attachment system between the demineralized surfaces. **[**282, 283**]**

However, the clinical effects of EDTA as a root conditioning agent are not evident and its use is controversial.

Results of other studies which used 24% of EDTA (pH 7.2) and applied it to root surfaces for 23 minutes showed no difference in probing depth, clinical attachment level and probing bone levels between EDTA treatment and control root surfaces. **[**284, 285**]**

A meta-analysis of 28 clinical trials on the use of chemical root conditioning agents was performed by Mariotti. The three trials of EDTA included in the review did not show significant effects on clinical attachment level (CAL) and probing depth (PD); one study was nonsurgical, while another was not randomized or controlled. **[**268**]**

In another meta analysis by Liu et al on the effect of EDTA, it was concluded that the effect of EDTA is not beneficial for improving the levels of PD and CAL. **[**286**]** Considering the extreme lack of evidence, additional studies are needed to evaluate the role of EDTA as a root conditioning agent in periodontal surgery.

Fibronectin

Fibronectin is a high molecular weight extracellular matrix glycoprotein. It is involved in many cellular processes, including tissue repair, embryogenesis, blood clotting, and cell migration/adhesion. It has a chemoattractant effect on fibroblasts and mesenchymal cells and it also promotes cell adhesion to both collagen and scaled root surfaces. One important function of fibronectin is that it acts as a non specific opsonin. It binds to actin and DNA, thus promoting cellular and tissue debris removal by macrophages. It has been shown that the application of fibronectin to partial demineralized root surfaces enhances new attachment and cell proliferation from periodontal ligament and supra crestal area. Caffesse RG et al. suggested that the application of Fibronectin to partially demineralized roots has been shown to significantly enhance the effects of demineralization with regard to new attachment and enhance cell proliferation from periodontal ligament and supra crestal area. **[**287**]**

Raul G et al. evaluated 46 patients after treatment and reported significant gains in clinical attachment and probing depth reduction when citric acid and fibronectin were used.288 Wikesjo et al. studied root surface demineralization with tetracycline HCL and topical fibronectin application in furcation defects of beagle dogs. The authors observed that tetracycline had the potential to induce connective tissue repair but application of fibronectin does not have any effect on connective tissue repair. **[**289**]**

Laminin

While collagen has some adhesion promoting activity, laminin has been demonstrated to have potent actions on cells - stimulating cell adhesion, growth, differentiation, and migration. It has been shown that laminin promotes gingival epithelial chemotaxis and in addition, movement of gingival fibroblasts. The affinity of laminin and fibronectin are not same towards mineralized surface. A mineralized surface attracts laminin which favours epithelial proliferation whereas a demineralized surface attract fibronectin and favours fibroblast proliferation.

Terranova et al have demonstrated that laminin promotes epithelial cell adhesion and growth to tetracycline and glycoprotein conditioned surfaces. **[**290**]**

Recent studies indicate that laminin binds strongly to type IV collagen and proteoglycans, but poorly to other collagen types. Other chemical agents that have been used for root biomodification lack sufficient amount of evidence to both understand the mechanism and also to determine their efficacy.

Platelet rich plasma

The concept behind PRP application for periodontal regeneration is to obtain high density platelet concentrate from patient's own blood and then applying this concentrate in the area of periodontal wound healing where regeneration is desired. Platelet derived growth factor (PDGF) is a major mitogen for fibroblasts, smooth muscle cells, and other cells. Platelets synthesize a mixture of the three possible PDGF isoforms (70% AB, 20% BB, 10% AA). It has been shown that PDGFAB is a potent stimulator of DNA synthesis in fibroblasts. Keceli et al. used PRP as root bio-modifier in an SCTG procedure but no difference could be found between SCTG and SCTG+PRP.291, **[**292**]**

Enamel matrix proteins

The enamel matrix proteins are involved in early tooth development and play a vital role during formation of cementum, periodontal ligament and alveolar bone. Application of enamel matrix proteins on root surface creates a biological environment similar to that during tooth development favouring periodontal regeneration.

In recent years, biomodification of root surface with Enamel Matrix Protein during surgery, following demineralization with EDTA has been introduced. It is based on the biologic concept that the application of Enamel Matrix Protein (amelogenins) may promote periodontal regeneration as it mimics events that take place during the development of periodontal tissues. Clinical trials have been conducted for the assessment of the effectiveness of Enamel Matrix Protein regarding its ability to improve periodontal health. In vitro (cell culture) exposure of fibroblasts to EMD's was done to find out fibroblast response to these proteins. It was found that EMD's enhanced proliferation of PDL cells, but not epithelial cells. Total protein production by PDL cells was increased and mineralized nodule formation of PDL cells was also increased. Other

potential mechanisms which potentiate periodontal regeneration upon EMD application include increase attachment of periodontal ligament fibroblasts to diseased root surfaces, increased production of growth factor, limiting epithelial down growth, and increased matrix formation by affecting fibroblast mRNA levels for synthesis of matrix proteoglycans and hyaluronic acid. **[**293, 294**]**

Recombinant human growth factors

Various growth factors which are believed to contribute to periodontal regeneration include the platelet derived growth factor (PDGF), insulin like growth factor (IGF), transforming growth factor (TGF), epidermal growth factor (EGF), fibroblast growth factor (FGF), and bone morphogenetic protein (BMP). In general, these growth factors promote proliferation of fibroblasts from the periodontal ligament and favour bone formation. Research has provided evidence for improved cellular response following growth factor application. **[**263**]**

The effect of human platelet derived growth factor- BB on attachment of periodontal ligament cells on root surfaces was investigated. The results demonstrated that citric acid combined with platelet derived growth factor-BB showed better results than EDTA and tetracycline hydrochloride on attachment of periodontal ligament cells on root surfaces. **[**295**]**

In another study it was seen that EMD and TGF-β1 may play an important role in periodontal regeneration. EMD induced PDL fibroblast proliferation and migration, total protein synthesis, ALP activity, and mineralization, while TGF-β1 increased cellular adhesion. However, the combination of both factors did not positively alter PDL fibroblast behaviour. **[**296**]**

In an in- vitro study it was seen that the combination of Enamel Matrix Derivative (EMD) and PDGF-BB produces greater proliferative and wound-fill effects on PDL cells than each by themselves. If these combined effects can be translated clinically, one may see greater regeneration in periodontal defects with this combination. PDGF-BB in concentrations equal to or greater than 50 ng/ml demonstrated a significant stimulation of PDL cells adherence to periodontal diseased root surface. Hence it has an important role in promotion of the PDL healing and can be useful in clinical application for the promotion of regeneration of the periodontal tissue. **[**297**]**

Hyaluronic acid (HA)

HA is an essential component of the periodontal ligament matrix and plays various important roles in cell adhesion, migration and differentiation mediated by the various HA binding proteins and cell-surface receptors such as CD44. Hyaluronan has numerous roles in the initial inflammatory stages such as the provision of a structural framework via the interaction of Hyaluronan with the fibrin clot, which modulates host's inflammatory and extracellular matrix cell infiltration into the inflamed site.HA has also been studied as a metabolite or diagnostic marker of inflammation in the gingival crevicular fluid (GCF) as well as a significant factor in growth, development and repair of tissues. **[**263**]**

Hakansson, et al. suggested role of Hyaluronan in migration and adherence of polymorphonuclear leukocytes and macrophages at the inflamed site and the phagocytosis and killing of invading microbes. HA accelerates the bone regeneration by means of chemotaxis, proliferation and successive differentiation of mesenchymal cells. It shares bone induction characteristics with osteogenic substances such as bone morphogenetic protein-2 and osteopontin. Hyaluronan has also shown to induce the production of proinflammatory cytokines by fibroblasts, keratinocytes, cementoblasts and osteoblasts which promote the inflammatory response and consequently stimulate hyaluronan synthesis by endothelial cells. **[]**298

Lasers

Recently, lasers have been recommended as an alternative or adjunctive therapy in the control and treatment of periodontally diseased root surface. Lasers are capable of sterilizing the diseased root surface and thus ultimately promoting cell reattachment. Hess and Myers said that the removal of root surface contaminants with these techniques allows for the elimination of inflammation and possible attachment to adjacent hard tissue. **[**263**]**

Hibst et al. gave a first description of effects of Er: YAG laser on dental hard tissues. It is a very promising laser system because the emission wavelength of 2.9 µm coincides with the absorption peak of water resulting in good absorption in all biological tissues including enamel and dentin. **[**299**]**

Yamaguchi et al. have demonstrated the ability of Er: YAG laser to remove lipopolysaccharides from root surfaces, facilitate removal of smear layer after root planing, remove calculus and cementum and leave a surface similar to an acid etched appearance. **[**300**]**

Vamsi Lavu et al. conducted a literature review on using Er: YAG laser for root biomodification and concluded that Erbium lasers shows benefited outcomes when used as a root modifier owing to its anti-bacterial action, predictable calculus removal and minimal root substance removal favouring cell attachment. **[**301**]**

However, a lower degree of calculus removal with the Er:YAG laser than with scaling and root planing has also been noted in another in vivo study. **[**302**]**

R. Fekrazad et al. conducted an in-vitro Study to evaluate fibroblast attachment in root conditioning with Er, Cr:YSGG Laser Versus EDTA and found Er, Cr:YSGG laser conditioning can promote enhance fibroblast attachment on dentinal root surfaces more than EDTA. **[**303**]**

Nd:YAG laser was developed by Geusic in 1964 and it has been proposed as an instrument with great potential for effective root preparation. Use of Nd:YAG lasers come with certain limitations for the treatment of dental hard tissues. They cause thermal side effects such as cracking or charring at the target site and also pulpal damage unlike Er:YAG laser. Application of the Nd: YAG laser to root surfaces results in. **[**263**]**

Patel et al. were the first to develop CO2 laser. CO2 lasers are capable of ablating calcified tissues effectively. However, they have the same limitations of thermal side effects such as cracking or charring at target site and pulpal damage like the Nd: YAG laser. **[**304**]**

Misra V, et al in an in vitro study evaluated the effect of CO2 laser on the periodontally involved root surface and observed that laser irradiation of 1 second at 3W completely removed the smear layer with minimal change in the diameter of the dentinal tubules. **[**305**]**

Pant V, et al in an in vitro study observed the attachment behavior of human PDL fibroblasts on periodontally involved root surface after conditioning with CO2 laser and compared its efficacy with chemical conditioning agents, namely tetracycline HCL, citric acid and EDTA, using scanning electron microscopy and found that CO2 laser irradiation for 1.0 s promotes

comparatively better attachment of periodontal ligament fibroblast on dentinal root surfaces than the conventional chemical conditioning agents used in the study. **[**306**]**

R Crespi et al. in a randomized clinical trial comparing modified widman flap surgery with that of coronally advanced flap surgery with Co2 laser root conditioning found that coronally advanced flap + Co2 laser resulted in statistically significant result in terms pocket reduction at sites ≥7 mm. **[**307**]**

Root canal irrigants as root biomodifier

The use of intracanal irrigant on periodontally affected root surface was first suggested by B. Houshmand et al. using MTAD (root canal irrigant) as a root conditioner. He suggested that a statistically significant difference were seen in smear layer removal from periodontally affected root surface when compared with saline. **[**309**]**

C. Tandon et al. concluded that MTAD as a root biomodifier have a significant role in periodontal wound healing and future new attachment both in vitro and in vivo. **[**310**]**

Shewale A, Gattani D , in an In vitro study studied the potential of a root canal irrigant containing Chlorhexidine and EDTA as a root biomodifier and concluded that it was efficient in removing smear layer from periodontally affected root surface. **[**311**]**

Current views

In a systematic review on efficacy of chemical root surface biomodifiers in the treatment of periodontal disease, Angelo Mariotti concluded that chemical modifiers like EDTA, citric acid or tetracycline provided no significant clinical benefit to regeneration in patients with chronic periodontitis. In another systematc review by Oliveira GH, Muncinelli EA, citric acid, EDTA and laser therapy were used as biomodifying agents along with free gingival graft, subepithelial connective tissue graft plus coronally advanced flap and semilunar coronally repositioned flap for root coverage. They concluded that biomodification provided no additional benefit in terms of the evaluated clinical parameters. **[**311**]**

Karam PS, et al., 2016 conducted a systematic review on root surface modifiers and subepithelial connective tissue graft for treatment of gingival recessions and concluded that none of the products evaluated (citric acid, EDTA, PRP, lasers and EMD) showed evident benefits in clinical outcomes. Test and control groups presented similar outcomes related to root coverage and periodontal parameters, with no significant differences between them. The exception was root biomodification with the Nd:YAG laser, which impaired root coverage and had a detrimental effect on clinical outcomes. **[**312**]**

Conclusion

It is well established that the periodontally diseased root surface does not favour regeneration of the periodontium due to its surface characteristics. Demineralization has been shown to alter the diseased root surface, creating a more acceptable surface that can influence events in wound healing. However evidence to date suggests no clear conclusion that the use of root conditioning agents to modify the root surface provides any benefit on clinical significance for regeneration in patients with chronic periodontitis. Histologic evidence seems to suggest that new connective tissue attachment and some limited regeneration may result from root surface conditioning. However, this histologic healing pattern does not result in significant improvement in clinical conditions beyond non-acid treated control sites. These results should be interpreted in light of the inability of clinical measurements to delineate the type of new attachment that has resulted post- treatment. In the future clinical procedures which rely on biological principles for root biomodification may create successful regenerative method using root conditioning agents.

Periodontal Dressing

Wound healing is a complex and dynamic process of restoring cellular structures and tissue layers. This biologic process can be broadly divided into 3 distinct phases – i.e., inflammatory, proliferative and remodeling. Within these 3 phases, a complex and coordinated series of events takes place. The culmination of wound healing results in the restoration of normal structure and formation of the injured tissue. Louis Pasteur stated, "The germ is nothing. It is the terrain in which it is found that is everything." Factors that influence wound healing must be addressed in a holistic fashion, looking, as Pasteur suggested, at the terrain in which the wound is found. One may thus infer that the environment in which a wound heals plays a critical role. This favorable environment can be, in part, created by a surgical dressing. **[**313**]**

A surgical dressing allows for uninterrupted healing to occur and also contributes to the protection of the surgical area and prevention of wound damage and infection. The first surgical dressing was patented by E. P. Lesher in 1953 (US Patent 2632443). Similarly, a surgical dressing is also utilized after periodontal surgical procedures. These dressings are applied around the necks of the teeth and adjacent tissue to cover and protect the surgical wound after periodontal surgery. Zentler in 1918 first reported the use of a periodontal dressing in the form of iodoform gauze. This marked the beginning of a trend toward using periodontal dressings after surgery. A. W. Ward in 1923 invented the Wondrpak, using the word "pack" in this context for the first time. **[**314-316**]**

Controversy on the terms "pack" or "dressing"

At one stage in the development of periodontal therapy a packing material was used therapeutically to help eliminate the periodontal pocket. Thus at that time the term pack was used. With the advent of modified surgical techniques for pocket elimination and use of postsurgical dressing to cover the exposed wound surface the term periodontal dressing is more appropriate and is found more commonly in literature. Both names can be used interchangeably. **[**317**]**

Ideal properties of dressings

• The dressing should be soft, but still have enough plasticity, and flexibility to facilitate its placement and adaptation.

• Setting of the dressing should be within reasonable time.

• After setting, the dressing should have sufficient rigidity to prevent fracture and dislocation.

• It should have a smooth surface after setting to prevent irritation to the mucosa, cheeks lips.

• It should have bactericidal properties and prevent excessive plaque formation

• It should not interfere with healing.

• It should not induce allergic reaction.

• It should have acceptable taste and odour. **[**318**]**

Types of periodontal dressings

Periodontal dressings are generally classified into three types:

1. Zinc oxide eugenol dressings

2. Zinc oxide non eugenol dressings

3. Those containing neither zinc oxide nor eugenol like:

a) Cyanoacrylates
b) Tissue conditioners
c) Dressings which contain antimicrobial agents
d) Photo curing periodontal dressings. **[**313**]**

Name, type and composition of each commercially available dressing

Sr. No.	Name	Type	Composition
1	Ward's Wondrpak	Eugenol dressing	Powder – zinc oxide, powdered pine resin, talc & asbestos Liquid – isopropyl alcohol 10%, clove oil, pine resin, pine oil, peanut oil, camphor & coloring materials
2	Kirkland formula	Eugenol dressing	Zinc oxide, resin, zinc acetate, eugenol, tannic acid and olive oil.
3	Coe-Pak	Noneugenol dressing	Two pastes First paste – zinc oxide, added oils, gums & lorothidol Second paste – unsaturated fatty acids & chlorothymol
4	Cross Pack	Noneugenol dressing	Colophony powder, zinc oxide, tannic acid bentonite & powdered neomycin sulphate
5	Peripac	Noneugenol dressing	Calcium sulphate, zinc oxide, zinc sulphate, acrylic type of resin & glycol solvent
6	Septopack	Noneugenol dressing	Amyl acetate, dibutyl phthalate, butyl polymetacrylate, zinc oxide, zinc sulphate
7	PerioCare	Noneugenol dressing	Two pastes First paste – paste of metal oxides in vegetable oil Second paste – gel of rosin suspended in fatty acids
8	Perio Putty	Noneugenol dressing	Methylparabens, propylparabens, benzocaine
9	Periogenix™	Noneugenol dressing	Perfluorodecalin, purified water, glycerin, hydrogenated phosphatidylcholine, cetearyl alcohol, polysorbate 60, tocopheryl acetate, benzyl alcohol, methylparaben, propylparaben, & oxygen
10	Cyanoacrylate dressings	Other	n-Butyl cyanoacrylate
11	Light cure dressings	Other	Silicon dioxide crystalline – quartz, hydrophobic amorphous fumed silica, urethane dimethacrylate resin
12	Collagen dressing	Other	Type I collagen derived from bovine tendon mixed with cancellous granules
13	Stomato adhesive dressing	Other	Gelatin, pectin, sodium carboxymethylcellulose and polysio polysiobutylene

Effects of periodontal dressings

The effects of a dressing can be perceived as physical effects and therapeutic effects.

Physical effects

With the advent of flap repositioning, advocated by Ariaudo and Tyrell, it was established that the periodontal dressing could be used as a stent. It was Prichard who stated that a dressing was

to be used to prevent postoperative hemorrhage and to protect the wound area from contact with food, concluding that a dressing "has no other virtue." Later, Manson said that a dressing is applied to protect a healing wound from saliva and trauma, thus producing comfort and enhancing healing. **[**319-321**]**

Wikesjo et al also described elevated sensibility of healing during the first few hours and days, especially in the process of fibrin attachment to the root surface. They stated that a dressing protected the coagulum from forces exerted during talking and chewing and prevented its detachment from the root surface. **[**322**]**

Subsequently, Plagman recommended the covering of the wound area for 3-4 days with a periodontal pack in addition to suturing, because the dressing prevented food debris from impacting in the interdental spaces. He assumed that the coagulum had to be stabilized so that movements of the healing epithelium were prevented and an untroubled attachment to hard tissues was guaranteed. **[**323**]**

To summarize, we can say that the list of physical benefits of a periodontal dressing includes protection of the postsurgical wound from postoperative trauma, saliva, and food debris and stabilization of the blood clot. Secondly, it limits the entry of bacteria and other microorganisms which may cause infection and other complications. Furthermore, it has been suggested that it acts as a splint for loose teeth and to immobilize newly positioned grafts and flaps. Finally, a dressing may control postoperative discomfort in the early stages of healing.

Therapeutic effects

Ward advocated the use of a periodontal dressing to bypass pain, infection and root sensitivity and to prevent formation of caseous deposits on the root surface. He felt a dressing would also act to provide temporary support after gingivectomy. **[**316**]**

Orban used a zinc oxide eugenol dressing and observed that better healing occurred after gingivectomy if the dressing was changed every 2 to 4 days for 10 to 14 days. However, he also noted that if the dressing was left in place in excess of 12 days, delayed healing occurred. **[**324**]**

Box and Ham described the use of a zinc oxide eugenol dressing after performing a chemical curettage for the treatment of necrotizing ulcerative gingivitis. This significantly improved the clinical parameters. **[**325**]**

Bernier and Kaplan reported that the use of a dressing facilitates the healing process. They indicated that the dressing's function as a surface barrier provided the primary benefit, while the constituents of the dressing appeared to be of secondary importance. **[**326**]**

Blanqui stated that the purpose of a periodontal dressing was to control postoperative discomfort, allowing tissue healing under aseptic conditions, preventing reestablishment of a periodontal pocket and desensitizing denuded cementum. **[**327**]**

The use of isobutyl cyanoacrylates, self curing and light curing packs led Bhaskar et al to consider instant hemostasis as one of its main advantages. Greensmith and Wade using a split mouth surgical technique, evaluated healing after reverse bevel flap procedures with or without a dressing. They concluded that the application of a dressing led to statistically slightly better results, as indicated by a shallower pocket and lower gingival index in spite of a slight increase in inflammation. In the same year Asboe- Jörgensen et al discussed the use of dressings after periodontal surgery in terms of improved patient comfort. **[**328-330**]**

To summarize, the therapeutic effects of a dressing include control of bleeding or hemostasis, improvement in clinical periodontal parameters, desensitization of denuded root surface and prevention of reestablishment of periodontal pockets. Evidence supporting the use of periodontal dressings.

Several authors contributed in providing evidence towards the establishment of the beneficial effects of using periodontal dressing.

Ariaudo and Tyrell in their study supported the use of periodontal dressings as they helped in protection of wound from mechanical trauma, stability of the surgical site during healing process. Prichard suggested that periodontal dressings increased patient comfort during healing, helped in good adaptation to underlying gingival and bony tissue, prevention of postoperative hemorrhage or infection, decreasing tooth hypersensitivity, protecting the clot from forces applied during speaking or chewing, preventing gingival detachment from the root surface. Wikesjo et al

mentioned that periodontal dressings prevented flap displacement in apically repositioned flaps and provided additional support in free gingival grafting procedures.

Sigusch et al found that periodontal wound dressing had a positive effect on clinical long- term results. **[**331**]**

Sachs et al supported use of dressing in retention of an apically positioned flap to prevent physical coronal displacement, provide additional support to stabilize a free gingival graft. They stated that periodontal dressing helped in protecting denuded bone from further injury, may act as a template for healing by preventing formation of excess granulation tissue. **[**332**]**

Evidence not in favour of periodontal dressings

Loe and Silness stated that in the absence of dressing, complete healing still takes place. Stahl et al undermined the use of periodontal dressings as it accumulates plaque and irritates healing tissue and hence repair might be improved if a dressing is not used as it. Greensmith and Wade said that the use of dressing causes more pain and swelling but less sensitivity and eating difficulty when no dressing is used. Harpenau found no difference in clinical parameters when periodontal dressings were not used. **[**333-335**]**

Kidd and Wade concluded that there was greater pain experience, plaque accumulation, subsequent microbial invasion and that nonpack areas showed better wound healing and lesser pain scores. **[**336**]**

Jones and Cassingham attributed disadvantages of dressings like possibility of displacing the flap, entrapping of sutures beneath dressings, forcing the dressing material under the flap during placement. **[**329**]**

Checchi and Trombelli found no statistical differences in pain scores and number of analgesics consumed between the pack and nonpack groups. Postoperative pain was found to be higher in patients with dressing. **[**331**]**

Heaney and Appleton tested the effect of periodontal dressings when placed in periodontally healthy mouths, using either Coe-Pak or Wondrpak. They found that while the dressings caused

little damage to the periodontium, they were associated with more inflammation than undressed areas.

Allen and Caffesse found no difference in PD, CAL and gingival inflammation. Wampole et al found a 24% incidence of transient bacteremia in patients during postoperative dressing change. This finding was felt to be of significance in medically compromised cases, especially those with a history of rheumatic heart disease or bacterial endocarditis. **[**332**]**

Bose et al found that pronounced swelling increases plaque accumulation, inflammation and GCF and that patients reported difficulty in eating. **[**337**]**

Studies assessing antibacterial properties of periodontal dressings

To enhance healing and prevent infections, the addition of antibiotics to dressings has been evaluated. The earliest reports outlining the use of tetracycline are by Fraleigh and of zinc bacitracin, by Baer et al. In 1972, Grant et al discussed this subject and stated that the possible advantages of the use of bactericidal and bacteriostatic drugs in periodontal dressings had not been fully investigated and pointed out the possibility of sensitization and allergy, and the potential development of candidiasis with the use of these drugs. **[**338-340**]**

Though the addition of these agents is beneficial, there are a few authors who claim that their addition may be harmful. Heaney et al suggested the removal of the dressing within 7 days of application, as antimicrobial agents used in conjunction with dressings may allow for selective inhibition of microorganisms and bring about variations in complex oral microbiota. Two possible problems may occur: emergence of resistant organisms and opportunistic infections. **[**338**]**

Romanow found that clinical signs of candidiasis occurred when using tetracycline in dressings and that bacitracin was found to enhance the growth of yeasts. **[**341**]**

In 1983, Breloff and Caffesse tested the effect of Achromycin applied underneath a dressing and showed that topical Achromycin had no beneficial effect on healing. Plüss showed that significantly less plaque formed under periodontal packs with chlorhexidine powder than under control packs. In evaluation of healing process, O'Neil revealed that tested periodontal dressings

(Coe-Pak, Cross-Pak, Peripac, Septo-Pak, ZOE) had no antibacterial properties, and ZOE had minimal antifungal properties. [332]

In some in vitro studies, antibacterial properties of periodontal dressings against bacterial plaque have been reported to be inconsistent. [343-345]

Studies assessing periodontal dressing cytotoxicity

An in vitro cell culture technique suggested that the solubility of the leachable toxic substances in cell culture medium is an important factor responsible for various behaviors of dressings.345

Haugen et al introduced Wondrpak as the most irritating product, followed by Coe-Pak and Peripac. They found that under laboratory conditions, fresh samples of Coe-Pak and Wondrpak cause more hemolysis than other products, and the cytotoxicity of Coe-Pak increases with time.

Nezwek et al and Wennberg et al in their in vitro studies, investigated tissue reactions to some periodontal dressings. They reported that the greatest inflammatory reaction was caused by Wondrpak. Also, Wennberg et al showed that when the contact period increased to 3 days, Peri-pac showed a more severe tissue reaction than Wondrpak. Smeekens et al in an animal study, suggested that the products that contain eugenol trigger greater inflammatory reactions, although this increase was not significant in other studies. [346, 347]

By using scanning electron microscopy and L-929 cell media, the cytotoxicity of some periodontal dressings was assessed. They showed that all of the materials had an insignificant toxic effect on L-929 cell lines, and Sne-Pack and Coe-Pak dressings were smoother than ZOE.348

Baer and Wertheimer, Haugen and Mjör and Saito et al in their studies (36-38) showed that periodontal dressings can cause greater inflammatory infiltration on the bone and the inflammatory reaction is greater when the dressing is directly placed on the bone compared with the time when it is placed on the periosteum.

The question of whether we need to use a dressing for all surgical procedures remains open. The fact that complete healing can take place even without a dressing, provided the surgical area is

kept clean, and that there is no difference in healing between dressed and nondressed wounds, lends support to the theory that not all surgical areas need to be “packed”. Other factors such as the presence of inflammation seemed to influence the rate of wound healing to a larger extent than the use of a dressing. Conflicting reports exist in the literature, as these factors are based on patient responses and thus are not objectively evaluated, because of the subjective criteria usually employed.

The answer to this controversy, though still open to debate, is probably that the choice of use of a periodontal dressing is a matter of individual preference and the judgment of the operator. It is, however, prudent to use a dressing for stabilization of free gingival grafts and protection of donor site, retention of an apically positioned flap, protection of the denuded bone from further injury, protection of the graft site in periodontal regeneration and to facilitate retention of drugs delivered locally in the subgingival sites.

Conclusion

There appears to be no consensus regarding the absolute indication for the use of periodontal dressings after a surgical procedure. However, the literature does elaborate on the benefits of application of a dressing postsurgically. Moreover, no periodontal dressing material has been shown to exhibit all of the ideal properties – both physical and therapeutic. Further research to improve biomaterial properties may lead to a more universal applicability.

Splinting

Physiological/normal tooth mobility may be defined as the slight displacement of the clinical crown of a tooth, that is allowed by the resilience of an intact and healthy periodontium, under the application of a moderate force. Increased tooth mobility/hypermobility could be defined as the clinically perceptible tooth movement or displacement in bucco-lingual (palatal), mesio-distal and/or vertical (apico-coronal) direction, when the tooth is exposed to a small force (e.g. 0.25 N). **[**349-351**]**

Over the past decades, an extensive bulk of studies, the majority of which were animal experiments, conclusively documented that the main causes of tooth mobility are the widening of periodontal ligament and/or the destruction of supporting periodontal tissues, most usually resulting from the presence of active or treated periodontitis (presence of non-inflamed periodontium with reduced periodontal support), either alone or combined with trauma from occlusion. **[**352-356**]**

Physiologic or normal tooth mobility depends basically on:

1. The quality or "viscoelestic" properties of the periodontal tissue

2. The anatomical characteristics such as the amount of supporting alveolar bone and the width of the periodontal ligament space.

3. Other factors such as number, shape and length of the roots or the intrinsic elasticity of the tooth itself may also be considered. **[**357**]**

Increased Tooth Mobility:

Some physiologic phenomenon are also associated with increased tooth mobility, for example:

1. Tooth eruption, due to the incomplete maturation of the periodontal membrane during the process.

2. Pregnancy, as a result of the hormonal influences on collagen and vascular structures of the ligament tissues.

3. The greatest tooth mobility is observed upon arising, and decreases during the day. **[**358**]**

Literature indicates that the main pathological reasons for tooth mobility are primary and secondary occlusal trauma, and progressive mobility, migration/drifting due to periodontal and/or periapical causes. **[**359**]**

Occlusal trauma/trauma from occlusion

It is described as trauma to the periodontium from functional or parafunctional forces causing damage to the attachment apparatus of the periodontium by exceeding its adaptive and reparative capacities.

Primary traumatism: It is the production of mobility in a tooth with normal support subjected to a force in excess of physiologic limits.

Secondary traumatism: It is the production of mobility by normal forces in a tooth with weakened support. When local and intrinsic factors such as inflammation and metabolic disturbance are present, normal forces may produce mobility in a tooth with a full osseous support. These forces can either be acute (an abrupt change in the occlusal forces) or chronic (gradual change).

The reduction of mobility is an important objective of periodontal therapy. Root planing, curettage, oral hygiene, and surgery may cause teeth to tighten as inflammation is resolved. Occiusal adjustment, periodontal orthodontics, and restorative dentistry may alter occiusal relationships and redirect forces, thereby reducing traumatism.

Increasing the support of loose teeth may also increase their firmness; the device used for such treatment is the splint.

Splinting is one of the periodontal therapeutic procedures which is miserably understood and decisive to implicate. Proper knowledge regarding its implications will go far in deciding for/ against the use of splints. The value of splinting has been debated for a long time. Most of the benefits of splinting have been reported from clinical observations than from scientific evaluations. The need to stabilize periodontally involved mobile teeth has resulted in the development of numerous types of splints, which allow for maximum repair of the periodontium during and after periodontal therapy. But any attempts to perform splinting techniques without

adequate diagnostic techniques in oral diagnosis, periodontal analysis or occlusal analysis can often lead to misapplication of these procedures. **[**360**]**

Splint

Definitions:• Any apparatus or device employed to prevent motion or displacement of fractured or movable parts. – Hallmen et al.

• An appliance for immobilization or stabilization of injured or diseased parts – Glickman.

• Dental Splint: An appliance designed to immobilize and stabilize mobile loose teeth. **[**360**]**

Clinical rationale for splinting:

• To control parafunctional or bruxing forces.

• Distribution of forces received by any one tooth on to a number of teeth.

• Stabilization of mobile teeth during surgical, especially regenerative, therapy. Mobile teeth may not respond as well to reattachment procedures, until splinted.

• Stabilization of a periodontally compromised tooth when more definitive treatment is not possible.

• Prevention of the supra-eruption of an unopposed tooth to eliminate the potential for the development of periodontal problems.

• Stabilization of loose teeth to restore the patient's psychological and physical well-being.

• Splinting during or following periodontal therapy is useful and beneficial for controlling the effects of secondary trauma from occlusion. Also, it improves the patient's comfort and function.

• To control the progressive tooth mobility. **[**361, 362**]**

In the past, the use of splinting of periodontally compromised teeth was contentious. The presumption was that the use of splints to control tooth mobility was required to control gingivitis,

periodontitis, and pocket formation. It was assumed that mobility had a direct relationship to attachment loss and vertical osseous defect formation. Another assumption was that increasing tooth mobility was a direct consequence of traumatic occlusion, bruxism, and clenching. Consensus also pointed to the fact that even normal physiologic functions including mastication and swallowing contributed to tooth mobility. [363]

A number of periodontal clinical trials investigated these assumptions. When teeth were occlusally overloaded and other variables that contribute to periodontal disease were controlled, it was difficult to produce gingivitis, periodontitis, or pocket formation. [364, 365] In another study, it was reported that there is no correlation between splinting and reduced tooth mobility during initial periodontal therapy. Control of tooth mobility with splinting after osseous surgery did not reduce mobility of the individual teeth. [366]

However, other studies report that tooth mobility can be controlled and managed with splinting and will improve periodontal prognosis. [367-369]

With such conflicting data, it is very difficult for the clinician to decide whether to use splinting or not, what degree of mobility can be managed or extraction is the ultimate cure.

For this the clinicians should be able to segregate and identify splints, so that they can make the best use of them in various clinical circumstances.

Type	Details	Usage
Short-term temporary splint	Worn for < 6 months	During active periodontal treatment; may or may not lead to another type of splinting
Medium-term provisional splint	For months to several years	For diagnostic purposes; usually lead to more permanent types of stabilization
Long-term permanent splint	Maintain long-term stability	Worn indefinitely and may be either removable or fixed type (Lemmerman, 1976)
External splints	E.g., ligature wires; night guards; interim fixed prostheses	**Ligature wire** Used for mobile anterior teeth E.g., Dead-soft round stainless steel wires (0.25-0.30 mm) or brass wires **Night guards:** Recommended in patients with history of bruxism and clenching Stabilize teeth following selective occlusal adjustment Heat polymerized poly (methyl methacrylate) occlusal splint is commonly used (Mikami,1977) **Interim fixed prostheses:** Used in periodontally compromised teeth until a definitive treatment plan is made. Restores esthetics and restores occlusal scheme to incorporate a definitive prosthesis in future. Added advantage is it provides time for evaluation of design and occlusal form before deciding to proceed with definitive restoration (Malone and Koth, 8th ed)
	Composite resin restorative material with or without wire or fiber inserts	Composite resin restorative materials Increasing the bond strength of composite to enamel as well as dentine has led clinicians to attempt splinting of very mobile teeth. Preferred in splinting of anterior teeth for esthetic reasons. In order to enhance the shear stress, the composite is reinforced with high strength, bondable, biocompatible, aesthetic color and easily manipulated neutral fiber.
Internal splints	Composite or fiber-reinforced composite material used as internal splints	Referred to as intra-coronal splints. Composite resin restorations can be placed in adjoining teeth and cured to eliminate any interproximal separation Can be further reinforced with metal wires, glass-reinforced fibers or pin. (Barzilay, 2000)

Among the above mentioned splints, most authors generally do not recommend internal splints.370

Types and material details of various splints

Type	Trade name	Material details
Fiber reinforced composite splint	Splint-It®-Pentron	Fiber splints are available in three unique designs for a variety of stabilization and reinforcement procedures The high strength glass and polyethylene fibers are pre-impregnated with a special resin ensuring complete saturation within each strand and eliminating the need to apply bonding agent The resin-treated fibers provide versatility, in addition to substantial strength and ease of placement **Unidirectional fiber strip:** The 3 mm wide unidirectional glass fiber strip is ideal for stabilizing mobile teeth, repairing dentures, and reinforcing temporary bridges **Woven fiber strip:** The 2 mm wide woven glass fiber strip easily tucks into interproximal contacts, adapts effortlessly to malaligned teeth, and stays in place due to its lack of memory **Braided rope strip:** The 1 mm wide braided polyethylene rope is ideal for use when lingual space is limited, and may also be used as a post
	Open weave glass fiber ribbon	Have been adapted to compensate for the unique structural design of periodontal splints Has an inherent ability to dissipate stresses and prevent crack propagation, which is not seen with the unidirectional glass fibers (Giordano, 2000; Vallittu, 1998)
	Ribbond (Eminkahyagil, 2006; Clinical Research Associates, 1997)	Advantages of this material include ease of manipulation and adaptation to dental contours during the bonding process, as it is a relatively easy and fast technique (no laboratory work is needed) In the case of fracture, the appliance can be easily repaired Now also available as thinner higher modulus (THM) Ribbond. This material is thinner than the regular Ribbond and has higher flexural strength. Its thinness allows the operator to adapt it more closely to the teeth. Developed by Dr. David Rudo Woven using spectra polyethylene fibers in a leno weave configuration It is lock stitched and cross-linked
Unidirectional pre-impegrated glass fibers	E.g., Prepreg (Giordano, 2000; Vallittu, 1998)	Unidirectional fibers oriented in multiaxial plane (e.g., 0°, +45°, -45°) stitched together Glass fiber reinforcing materials are available as resin-impregnated (pre-preg), fiber-reinforced glass fibers, in contrast with polyethylene fibers, and have to be protected from environmental damage These materials are esthetic and have translucency similar to castable glass-ceramics such as OPC and Empress The glass fibers can pose a health risk. They are small enough to be inhaled and deposited in the lungs, resulting in a silicosis-type problem.
Open weave glass fibers	(Giordano, 2000; Vallittu, 1998)	Can be used with both polyester and epoxy resins Open weave glass fiber design has been adapted to compensate for the unique structural design of periodontal splints Has an inherent ability to dissipate stresses and prevent crack propagation, which is not seen with unidirectional glass fibers
Provisional fixed partial prosthetic splint	Heat processed acrylic resin splint (Renggli and Schweizer, 1994; Pollack, 1999)	In certain situations occlusal rehabilitation is complex in nature. In such situations, provisional prosthetic splints play greater role Allows patient and periodontist to evaluate restorative treatment planning Material of choice to fabricate a provisional splint is heat processed acrylic resin
Definitive fixed partial prosthetic splint	Crown and bridge prosthesis (Renggi, 1984; Seigel, 1999)	Serve additional purpose of splinting the abutment and other supporting teeth Conventional crown and bridge prostheses fulfill this requirement very well if adequate abutment teeth are included Optional resin-bonded splint can be designed if anatomy and situation of the teeth are not conducive to slender cement retained prosthesis

Selecting abutment teeth for splinting

While selecting an abutment tooth for splinting one should always consider the pericemental area of an abutment tooth. Ante postulated that "The total periodontal membrane area of the abutment teeth must equal or exceed that of the teeth to be replaced." Also, "The length of the periodontal membrane attachment of an abutment tooth should be at least one-half or two-thirds of that of its normal root attachment." Moreover, teeth with mobility/widened periodontal ligament should be avoided as abutments for splinting. **[**371**]**

Effects of splinting on oral and periodontal health Splinting and oral hygiene:

Splinting makes oral hygiene procedures difficult. Therefore, to ensure the longevity of the connected teeth, special attention must be given to instructing the patient about enhanced measures for oral hygiene after placement of the splint prosthesis. Effective personal plaque control, professional caries risk assessment, and periodontal maintenance are crucial to the longevity of the splint and health of the splinted teeth. **[**370**]**

Splinting and periodontal repair:

In a study on rhesus monkeys to determine the effect of splinting on hyperocclusion, it was observed that forces applied to one tooth in a splint are distributed over the entire unit, that is, all the teeth included in that splint, thus reducing the occlusal load on a periodontally compromised tooth and facilitating the distribution of occlusal forces over a larger periodontal surface. Thus, it was concluded that splinting of tooth helps in redistributing the occlusal forces over a larger area. It was also observed that the areas of root bifurcation and trifurcation are more susceptible to excess occlusal forces. In a study to determine the effect of initial preparation and occlusal adjustment on tooth mobility, it was observed that for teeth with initial mobility of greater than 0.2 mm there was a decrease in tooth mobility up to 20%. **[**356**]**

Many authors believed that mobile teeth may inhibit "periodontal repair." Fixed splinting was advocated believing that it would reduce the mobility of individual teeth during healing, but studies have shown otherwise in the following manner.

1. Splinting of the teeth will not prevent or retard apical downgrowth of plaque (in fact, it will increase) and associated attachment loss.

2. Splinting of mobile teeth before scaling and root planing (SRP), and elimination of potential SRP- induced trauma to the mobile teeth did not have any adjunctive effect on healing.

3. Tooth mobility increases initially after surgery and subsequently decreases by 24 weeks to about pre-surgical values. Splinting did not reduce the mobility of individual teeth and also did not have any influence on bone and attachment level after osseous surgery.

4. Splinting of mobile teeth did not have any effect on mobility reduction after initial therapy.

5. Attachment levels and bone levels were similar in splinted and non-splinted teeth following osseous surgery.374

Effect of splint material and thickness on tooth mobility

Although current guidelines for the treatment of mobile teeth and traumatic injuries recommend the use of 'flexible' splints, the specific definition of what is considered flexible versus rigid has not been clearly defined, leaving the clinician with a wide range of options for this critical factor.

Kwan et al. quantified and compared the effect of eight different splints on tooth mobility after extraction and replantation using a human cadaveric model. The experimental splints included 30-pound test monofilament nylon composite and six wire-composite splints made of 0.012" (0.3 mm), 0.016" (0.4 mm), or 0.020" (0.5 mm) diameter stainless steel (SS) or nickel titanium (NT) wires. Following strict selection criteria (complete root maturation, lack of periodontal disease, normal bone levels, and crown integrity), a maxillary central incisor was atraumatically extracted and splinted with eight different splints. These eight splints were applied five times each, and tooth mobility was measured between the pre-split and the post-split measurements quantified using Periotest.

Significant less tooth mobility with direct composite splint compared to all other splints and no differences between nylon-composite and wire composite splints were observed. The authors also suggested that nylon and SS or NT wires up to 0.016" (0.4 mm) diameter are significantly more

flexible than direct composite splints and thus may be better suited for the splinting and management of traumatized teeth. **[**373**]**

Hereby, we should believe that splinting mobile teeth may act as an adjunct to periodontal treatment and maintenance and hence should be recommended after selecting the right splint for the right procedure based on the discretion of the advantages and disadvantages of each. A splint should be designed in such a way that it attracts the least plaque and calculus, is able to be retained for the specified time, is able to carry out its designated function, and does not interfere with healing and esthetics.

Conclusion

Provided all the factors are considered and proper maintenance therapy is recommended, splints are becoming an integral part of periodontal therapy and maintenance. However, it should be noted that splinting itself will not eliminate the cause of tooth mobility. They are only an aid in stabilizing the mobile tooth, and mobility may revert once the splints have been removed. Hence, splinting is an essential adjunct in addition to cause-related therapy in the treatment of mobile teeth.

Based on the available data it could be observed that splinting can be considered so as to increase the longevity of periodontally compromised teeth with advanced mobility. However, further research is still required to come to a definitive conclusion about the exact role of splints, and patient selection criteria for splinting in periodontal treatment.

Frenectomy

Aesthetic concerns have led to an increasing importance in seeking dental treatment, with the purpose of achieving perfect smile. The continuing presence of a diastema between the maxillary central incisors in adults, has often been considered as an aesthetic problem. The presence of an aberrant frenum being one of the aetiological factors for the persistence of a midline diastema, the focus on the frenum has become essential. **[**375**]**

The frena may also jeopardize the gingival health by causing a gingival recession when they are attached too closely to the gingival margin, either because of an interference with the proper placement of a toothbrush or through the opening of the gingival crevice because of a muscle pull. **[**376**]**

There has been a controversy among researchers regarding the need of frenectomy and the time of the surgery.

Anatomy

The maxillary labial frenum is a normal anatomic structure in the oral cavity, usually triangular in shape, extending from the maxillary midline area of the gingiva into the vestibule and mid-portion of the upper lip. It consists of epithelium, collagen fibres, blood vessels, nerves and sometimes few elements of minor salivary glands and isolated stratified muscle fibers. The frenum is a dynamic and changeable structure, which tends to have variations in size, shape, and position of attachment during the different stages of growth and development. It is found to be smaller in length, thicker and more inferiorly attached in children. Henry et al, in their histological study, concluded that there are also elastic fibres which extend sometimes to the whole length of the frenum, even perforating the periosteum. Those authors considered that the harmful effect of the frenum is due to the presence of the elastic and collagen fibres, while no evidence of substantial differences in composition of normal and abnormal fraena were identified. **[**377**]**

Implications of a pathological frenum

Miller characterized as “pathological” a frenum which is -

• Uncommonly wide

• When there is insufficient attached gingival zone in the midline

• When the interdental papilla moves by stretch of the frenum. **[**378**]**

An abnormal labial frenum has been implicated in functional and aesthetic problems, such as a maxillary midline diastema. Two ways were suggested in which the frenum may cause it -

• In the first way, the bulk of the frenum fibres, retaining their embryological connection with the incisive papilla, will physically prevent approximation of central incisors.

• Alternatively, these fibres will interrupt the fibres of the periodontal ligament between the central incisors and produce a weak link in the chain of fibres that join the teeth from one end of the arch to the other.

High frenum insertion can lead to gingival recession due to the tension which is applied on the tissues during normal functions, such as speaking, chewing, and laughing. Moreover, a frenum that encroaches on the gingival margin and prevents the closure of space between the maxillary central incisors creates an area for food impaction and difficulty in plaque removal. The poor oral hygiene, due to difficulty in tooth brushing results in inflammatory periodontal destruction. Aesthetics could be affected as well in cases of a high smile line. Finally, a big and high attached frenum could eliminate lip movement.

Diagnosis

The abnormal frena are detected visually by applying tension over the frenum to see the movement of the papillary tip or the blanch which is produced due to ischaemia in the region. The frenum is characterized as pathogenic when it is unusually wide or when there is no apparent zone of the attached gingiva along the midline or the interdental papilla shifts when the frenum is extended. **[**379**]**

Treatment

Frenectomy is complete removal of the frenum, including its attachment to underlying bone, and it may be required in the correction of an abnormal diastema between the maxillary central incisors. Frenotomy is relocation of the frenum, usually in a more apical position.

Various surgical techniques have been described for the management of the abnormal upper labial frenum. Some of the most cited surgical techniques are :

Type of technique	Advantages	Disadvantages
V-shaped/Archer incision/ diamond incision[45]	Easy to perform	Scar tissue formation Loss of papilla High relapse rate
Z-plasty[45]	Less scar formation	Surgically demanding More aggressive/morbidity
Vestibular sulcus extension[45]	-	High relapse rate
Morselli et al[53]	Less tissue contracture Less scar formation Less healing time	Surgically demanding
Bagga et al[54]	Advanced esthetic results Minimal scar tissue formation	Performed only in cases of adequate attached gingiva

Over the years, the relationship between the maxillary midline diastema and the labial frenum has been the subject of much controversy and confusion. In the 1939, Hirschfield advocated frenectomy as a mucogingival procedure to eliminate the aforementioned pathologic situations caused by an abnormal frenum attachment. There is still a controversy among researchers concerning the need for it at all, as well as the right time for frenectomy. **[**379**]**

The frenum by orthodontic approach

The presence of the maxillary labial frenum has a great significance for the orthodontic community, since it is considered to be the commonest causative factor for a maxillary midline diastema. An abnormal frenum has also been accused of being a great danger for relapse after orthodontically treated diastema. Consequently, maxillary labial frenectomy was considered for many years as the indicated treatment for maxillary midline diastema. There has been a controversy even among orthodontists concerning the need at all, and the timing for a frenectomy.

Some orthodontists support a viewpoint that there is a need for an early removal of the frenum, so as to prevent any obstacles to complete diastema closure. Other orthodontists propose to close the diastema first, and then carry out frenectomy in the hope that the resultant scar tissue will hold together the teeth in close apposition. A third body of clinicians rarely, if ever, considers

surgical removal of the frenum. They prefer to combat the undeniably increased relapse potential when a diastema is closed, by using bonded retainers on the two central incisors. **[**379**]**

Literature offers a great variety of opinions during years and it is obvious that they differ a lot concerning the etiology of a persisting diastema, such as to the possibility of promoting closure of a diastema by means of frenectomy. At the beginning it was thought that the labial frenum interfered with the closure of the midline diastema. This belief resulted in misdiagnosis and unnecessary surgical intervention of the frenum.380, 381 Adams suggested that there is a specific type of frenum which interrupts the continuity of interdental fibre, forms the factor that inducts the reaction for the development of the diastema. Although, he stated that there is a need of presence of other causative factors. Campbell et al11 stated the same. **[**382**]**

Shashua and Artun43 found that there is a relationship between abnormal frenum and the width of the maxillary midline diastema. **[**383**]**

Edwards supported the presence of a strong but not absolute correlation between the frenum and the upper midline diastema. **[**384**]**

Gardiner made a survey of 1000 children 5-15 years old. 80% of the cases with midline diastema were associated with a prominent frenum. He took this finding as an evidence to support the opinion that the frenum is often a contributory cause of midline diastema. **[**385**]** Angle concluded that the presence of an abnormal frenum is a cause for midline diastema. **[**386**]**

James used a sample of 10 girls 12-22 years old with medial diastema. A year after frenectomy, a reduction was noted in 8 cases. He assumed that frenectomy leads to a reduction of the diastema. **[**387**]**

Tait stated that the frenum has no effect to the maxillary central incisors. Ceremelo concluded that the frenum is not related to the presence or the width of the diastema. **[**379**]** Bergstorm et al stated that the long term potential for spontaneous diastema closure, in patients with abnormal frenum, has no difference even if there has been a frenectomy, or not. **[**388**]**

Popovich et al suggested that the presence of the diastema leads to the abnormal frenum, and not the adverse. **[**375**]** Some studies support frenectomy to be carried out, so as the scar tissue will hold the teeth together.

It is important to emphasize on the fact that frenectomy has clinical validity only after the eruption of all 6 permanent teeth if it failed to close the diastema, and then only in conjunction with orthodontic treatment. So after the eruption of all 6 permanent teeth, orthodontic appliances are used to close the diastema. Most authors believe that during the primary dentition phase, surgical intervention of the labial frenum is not recommended. **[**379**]**

The frenum by periodontal approach

The labial frenectomy must be examined by the aspect of periodontists as well. As early as 1939, Hirschfield observed a relationship between the attachment of the frenum and periodontal disease. It has been reported that when the attachment of a maxillary frenum is very pronounced and also exhibits a crestal insertion point close to the gingival margin of the incisors, it can retract the marginal gingiva or papilla, thus contributing to the initiation or progression of periodontal disease. Furthermore, oral hygiene procedures may be complicated and the accumulation and retention of plaque may be promoted when the periodontal pocket is pulled and opened, allowing food debris to enter more readily.386 Mirko et al found that certain types of maxillary frenum influence periodontal condition. The periodontal resistance was significantly lower in cases of gingival, papillary, and papillary-penetrating types of maxillary frenum attachment in persons with pathologic changes in the papilla in comparison to persons with the same type of attachment but with healthy papilla. Additionally, another study revealed that the correlation between a maxillary frenum with crestal attachment and the gingival recession was more pronounced in men than women. **[**389, 390**]**

In contrast, Addy et al reported that plaque and bleeding scores of the maxillary incisors decreased when increasing the proximity of the frenum to the gingival margin. Therefore, they support that the position of the maxillary frenum is not relevant for plaque accumulation and gingivitis. A clear cause-and-effect relationship between the presence of an abnormal maxillary frenum and gingival recession are lacking. **[**391**]**

For years, clinicians targeted in removing the frenum or deepening the vestibule. Today, it is approved that the presence of an adequate zone of attached gingiva is the basic factor. When there is an adequate zone of attached gingiva, even a high frenum attachment does not constitute dangerous factor for the beginning and the process of periodontal disease. On the other hand, in

the case of inadequate zone of the attached gingiva, the draw of the frenum and muscle attachment cannot be balanced, there is inability of good and atraumatic oral hygiene, and this is a fact that usually leads to gingival recession.

Consequently, there exist anatomic (not adequate zone of attached gingiva), biologic (inflammation, inability forgood oral hygiene) and functional (inability for protection during chewing procedure) factors that lead to the decision of frenectomy.392 Many periodontists tend to use frenectomy in which there is partial removal of the frenum and relocating it to a more apical position. This technique leads to an acceptable solution of the problem and to the movement of the frenum more apically.

Conclusion

The maxillary labial frenum is a normal anatomic structure in the oral cavity, formed by mucous membrane and connective tissue. Although it is a normal structure, its presence has been associated with some unpleasant and even pathological situations. A surgical removal of the frenum is indicated in order to prevent these situations or facilitate orthodontic closure of the diastema. As shown in the literature there has been a controversy among researchers regarding the need of frenectomy and the time of the surgery.

Many orthodontists support the idea that even in cases of an abnormal frenum we should wait the eruption of all 6 permanent anterior teeth first, and carry out the procedure if the eruption of all 6 permanent teeth has failed to close the diastema. They also state that the relapse of orthodontically treated diastema caused by an abnormal frenum, which had not been excised, is a rare phenomenon. On the other hand, periodontists accuse the pathologic frenum for causing unpleasant situations cited previously, such as gingival recession and advocate frenectomy. Lastly, it is important to remember that the final decision is taken by patients. The duration and the cost of the treatment are two basic factors. Patients rarely compromise with expensive and long-term procedures, especially if these include orthodontic treatment which affects aesthetics. A final decision should be taken keeping all these factors in mind.

Application of Lasers

In the past 100 years there has been extensive development of the mechanical cutting devices used in dentistry. From the end of the 20th century until now, there has been a continuous upsurge in the development of laser-based dental devices based on photo- mechanical interactions. It has been nearly 50 years since the first laser device was produced in 1960 by Maiman. **[**393**]**

In the medical field, lasers have been successfully used since the mid-1960s for precise photocoagulation of the retina. The first report of laser application in dentistry was reported for the treatment of dental caries, published in Nature in 1964 by Goldman et al. Since then, studies on the use of the neodymium-doped yttrium aluminium garnet (Nd:YAG), carbon dioxide lasers, erbium lasers and others have been publishes for various dental treatments. **[**394**]**

What is a laser?

A laser is a device that produces coherent electromagnetic radiation. The term "LASER" is well known as the acronym for Light Amplification by Stimulated Emission of Radiation. The term laser was first reserved for visible light, but now it is used for any type of electromagnetic radiation produced in this way. Hence, we may say microwave laser (instead of maser), or infra-red laser (instead of iraser). In fact, most lasers intended for medical and dental use operate in the red to infra-red spectra of light. **[**395**]**

Laser light is produced by pumping (energizing) a certain substance, or gain medium, within a resonating chamber but three factors are important for the final characteristics of the laser light: composition of the gain medium, source of pump energy, and design of the resonating chamber. In addition, both the laser-delivery system (e.g. optical fiber or articulated arm with mirrors) and the application tip are of paramount importance clinically, as they may determine the ease of use, range of applications and energy efficiency of a laser system. **[**395**]**

When biological tissue is irradiated with laser light, four types of interactions may occur: reflection, scattering, absorption, or transmission. The type of interaction that predominantly takes place depends largely on the wavelength of the laser. **[**394**]**

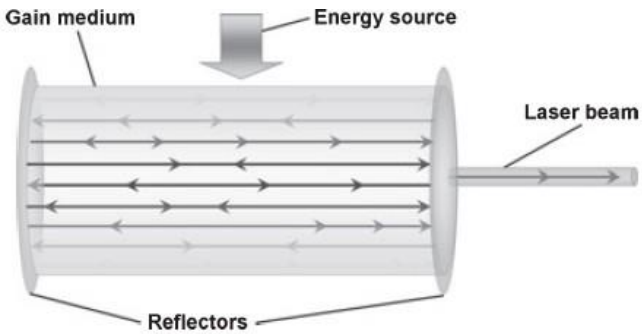


Fig. 2. Schematic drawing showing the main components of a laser. The gain medium is pumped by an external energy source. The gain medium then emits photons, which bounce back and forth between the reflectors. Part of the radiation is allowed to exit through an aperture in one of the reflectors, resulting in the laser beam.

Classification of lasers

Criteria	Types	Examples*
Output energy	Low-output, soft, or therapeutic	Low-output diodes
	High-output, hard, or surgical	Diodes, CO_2, Nd:YAG, Er:YAG, Er,Cr:YSGG
State of the gain medium	Solid-state	Nd:YAG, Er:YAG, Er,Cr:YSGG, KTP
	Gas	HeNe, Argon, CO_2
	Excimer	F_2, ArF, KrCl, XeCl
	Diode	GaAlAs, InGaAs
Oscillation mode	Continuous-wave	CO_2, Diodes
	Pulsed-wave	CO_2, Diodes, Nd:YAG, Er:YAG, Er,Cr:YSGG, KTP

*Not an inclusive list.

Lasers can also be classified into two types depending on their wavelength, as follows:

• Types where the laser light penetrates the tissue more deeply (such as Nd:YAG and diode lasers),

• Types where the laser light is absorbed in the superficial layers (such as CO2, Er:YAG and Er,Cr:YSGG lasers)

Advantages of using lasers in the periodontal therapy

1. Less pain

2. Less need for anesthetics (an advantage for medically compromised patients)

3. No risk of bacteremia

4. Excellent wound healing; no scar tissue formation

5. Bleeding control (dependent on the wavelength and power settings);

6. Usually no need for sutures Use of fewer instruments and materials and no need for autoclaving (economic advantages)

7. Ability to remove both hard and soft tissues

8. Lasers can be used in combination with scalpels (however, the laser is a tool and not a panacea). **[**394**]**

Disadvantages of using lasers in periodontal therapy include

1. Relatively high cost of the devices

2. A need for additional education (especially in basic physics)

3. Every wavelength has different properties

4. The need for implementation of safety measures (i.e. goggle use, etc.).

Risks and precautions in clinical use of lasers

1. Caution before and during irradiation.

Use of glasses for eye protection (patient, operator and assistants)

Precautions for inadvertent irradiation and reflection from shiny metal surfaces

Protection of patient's throat and oral tissues outside the target site Accurate foot pedal control

Adequate high speed evacuation to capture the laser plume.

2. Risk of thermal injury during interaction with the tissues Understanding of the penetration depth of each laser

Thermal injury to the root surface, gingival tissue, pulp and bone tissue Effective use of water spray to minimize heat generation

3. Risk of excessive tissue destruction by direct ablation and thermal side effects Excessive ablation of root surfaces and gingival tissue during pocket irradiation Destruction of the attachment apparatus at the bottom of pockets during pocket irradiation

Bone and root surface alterations during gingival soft tissue surgery or pocket irradiation

Damage of the tooth enamel by inadvertent irradiation **[**395**]**

Lasers vs conventional therapy

Gingivectomy, gingivoplasty and frenectomy are the most popular procedures carried out using lasers. One animal study has reported that compared with conventional scalpel surgery, laser surgery produces less pain with the oral soft tissue incision. All the major advantageous properties of lasers come to the benefit of the treatment and its outcome. **[**396**]**

Use of electrosurgery also facilitates easy tissue incision accompanied with a strong hemostatic effect. The major concern is the potential risk of thermal damage to the underlying periosteum and alveolar bone by direct contact of the electrosurgical tip during gingival tissue management, leading to necrosis of bone or delayed wound healing. The pulpal pain experienced by the patient as a result of direct contact of the electrosurgical tip on the root surface during the procedure is also a concern when the local anesthesia is insufficient. Compared with electrosurgery, lasers have a higher comfort level in patients, resulting in less operative and postoperative pain and fewer complications. **[**397, 398**]**

The CO2 laser, provides rapid and simple vaporization of soft tissues with strong hemostasis, which produces a clear operating field and requires no suturing. The Nd:YAG and diode lasers, can be used to cut and reshape soft tissues; however, these lasers have greater thermal effects,

leaving a relatively thicker coagulation area. The Er:YAG laser is also effective for soft tissue surgery, although, the hemostatic effect is weaker than for other lasers, but the healing of the laser wound is relatively fast and comparable to that of a scalpel wound. **[**399-402**]**

Esthetic gingival procedures

Lasers can be applied in esthetic procedures such as recontouring or reshaping of gingiva and in crown lengthening. In particular, the Er:YAG laser is very safe and useful for esthetic periodontal soft tissue management because this laser is capable of precisely ablating soft tissues using various fine contact tips, and the wound healing is fast and favourable owing to the minimal thermal alteration of the treated surface.403, 404 Depigmentation is another indication for laser use in esthetic treatments. The CO2, diode and Nd:YAG lasers can treat melanin pigmentation effectively. However, in areas of thin gingiva, these lasers have a risk of producing gingival ulceration and recession as a result of their relatively strong thermal and/or deeply penetrating effects. In these situations, the Er:YAG laser is more useful and safe for melanin depigmentation. Following melanin depigmentation in dogs using the Er:YAG laser, the width of the thermally affected layer in gingival connective tissue has been reported to be approximately 5–20 μm. In addition, the Er:YAG laser can be utilized to remove metal tattoos. **[**403**]**

Nonsurgical pocket therapy Conventional root debridement

Access to areas such as furcations and grooves is limited owing to the complicated root anatomy. Furthermore, conventional mechanical debridement using curettes is still technically demanding and time-consuming, and power scalers sometimes cause discomfort and stress in patients as a result of noise and vibration. **[**406**]**

The benefits of lasers, such as ablation, bactericidal and detoxification effects, as well as photo-biomodification, have been reported to be useful for periodontal pocket treatment, and the application of lasers has been suggested as an adjunctive or alternative tool to conventional periodontal mechanical therapy. **[**402**]**

Removal of subgingival calculus

The CO2 laser cannot be used for calculus removal because this laser readily causes melting and carbonization on the dental calculus. The Nd:YAG laser is also basically ineffective for calculus removal when a clinically suitable energy is employed. Unlike these lasers, the Er:YAG laser is capable of easily removing subgingival calculus without a major thermal change of the root surface in vitro. The level of calculus removal by this laser is similar to that of ultrasonic scaling. A similar performance for calculus removal has been reported with the Er,Cr:YSGG laser.

However, a lower degree of calculus removal with the Er:YAG laser than with scaling and root planing has also been noted in another in vivo study. **[**406-410**]**

Regarding thermal generation, the deeply penetrating type of lasers, such as diode and Nd:YAG lasers, carry the risk of intrapulpal temperature elevations during laser irradiation on the root surface. With the Er:YAG laser, the use of water coolant can effectively prevent thermal generation during laser scaling while not compromising the efficiency of laser scaling. A recent animal study showed that no adverse effects were observed histologically in the pulp tissue of roots following root debridement using an Er:YAG laser during flap surgery. **[**412, 413**]**

Table 3. *In vivo* studies on laser application in the treatment of periodontitis: removal of Plaque and Calculus and Effects on the Tooth Surface

Author and year (reference)	Laser	Laser parameters	Study design	Experimental group	Control group	Observation period	Findings
Non-surgical treatment							
Cobb et al. 1992 (24)	Nd:YAG	1.75–3.0 W, 20 Hz	Clinical (8 patients, 18 teeth)	Laser, Laser + RP, RP + Laser	Untreated	Immediately after treatment	Low effectiveness of laser for calculus removal, but decreased numbers of bacteria in laser-treated sites
Gold & Vilardi 1996 (48)	Nd:YAG	1.25, 1.75 W 62.5, 87.5 mJ/pulse 20 Hz	Clinical (6 patients, 24 teeth)	Laser		6 weeks	The complete removal of pocket lining epithelium without necrosis or carbonization of underlying connective tissue
Schwarz et al. 2001 (131)	Er:YAG	ED: 71, 83, 94 and 106 J/cm^2/pulse*, 10 Hz	Clinical (40 teeth)	Laser		Immediately after (*in vivo* & *in vitro*)	Smooth root surface morphology after laser scaling *in vivo* not comparable to the marked morphological changes *in vitro*. The surface alterations were not related to the energy setting used
Schwarz et al. 2003 (139)	Diode	655 nm, 1.8 W ED: 0.63 J/mm^2/pulse*	Clinical (24 teeth)	Laser	SRP with hand scaler	Immediately after (*in vivo* & *in vitro*)	Remaining debris and alternation of root surface such as grooves and cratering following laser irradiation
Schwarz et al. 2003 (139)	Er:YAG	ED: 19.4 J/cm^2/pulse*, 10 Hz	Clinical (24 teeth)	Laser	SRP with hand scaler	Immediately after (*in vivo* & *in vitro*)	Selective subgingival calculus removal using a fluorescent calculus detection system. Smooth and homogeneous root surface morphology following laser treatment

Eberhard et al. 2003 (38)	Er:YAG	ED: 26 J/cm^2/ pulse*, 10 Hz	Clinical (12 patients, 30 teeth)	Laser	SRP with hand scaler	Immediately after	Lower effectiveness of laser for calculus removal but the obvious conservation of the underlying cementum in laser-treated sites
Crespi et al. 2006 (27)	CO_2	ED: 15 J/cm^2/ pulse*, 10 Hz	Clinical (15 patients, 40 teeth)	Laser	SRP with hand scaler	Immediately after	Clinical use of Er:YAG laser achieved plaque and calculus removal, providing a rough surface morphology
Schwarz et al. 2007 (144)	Er:YAG	ED: 10.2, 12.8, 15.4, 18.0 or 20.4 J/cm^2/ pulse, 10 Hz	Animal (5 dogs)	Laser	SRP with ultrasonic device	6 months	Significantly greater new cementum formation with inserting collagen type I fibers along the root surfaces in laser sites treated with higher ED than control sites
Yukna et al. 2007 (186)	Nd:YAG	3 W, 20 Hz	Clinical (6 patients, 12 teeth)	Laser	SRP with hand scaler	3 months	Connective tissue attachment and cementum formation in all laser-treated sites

Table 3. Continued

Author and year (reference)	Laser	Laser parameters	Study design	Experimental group	Control group	Observation period	Findings
Surgical treatment							
Williams et al. 1995 (180)	CO_2	ED: 41.28 J/cm^2/pulse, 20 Hz	Animal (2 dogs)	Laser for degranulation	Manual curette	0, 3, 7, 14, 21 and 28 days	Mean times required for procedure in control sites were faster. Necrosed tissue or carbonized debris were phagocytosed
Centty et al. 1997 (22)	CO_2	8 W, 20 Hz	Clinical (5 patients)	OFD + laser irradiation to outer and inner aspects of mucoperiosteal flap	OFD	Biopsy during the surgery	Laser eliminated significantly more sulcular epithelium in comparison with conventional periodontal surgery
Crespi et al. 1997 (28)	CO_2	13 W, 40 Hz and 2 W, 1 Hz in defocus mode	Animal (6 dogs)	Laser for degranulation and root surface irradiation	GTR/SRP	6 months	Significantly greater formation of new periodontal ligament, cementum and bone in laser-treated sites
Gopin et al. 1997 (50)	CO_2	6 W, 20 Hz	Animal (2 dogs)	Laser, laser + RP for root surface treatment	Hand scaler and untreated	28 days	Inhibitation of periodontal tissue attachment to irradiated root surface by residual char
Mizutani et al. 2004 (94)	Er:YAG	ED: 18.8 or 14.5 J/cm^2/pulse, 10 Hz	Animal (6 dogs)	Laser for degranulation and root debridement	Hand scaler	3 months	Significantly greater new bone formation in the laser group than the control group. Resorption of the affected layer on the lased bone and root surface during the healing process

Root surface alterations

The CO2 laser readily carbonizes the root cementum, and cyan-derived toxic products, such as cyanamide and cyanate ions, have been clearly detected on the carbonized layer by chemical analysis using infrared spectroscopy. The residual char layer has been demonstrated to inhibit periodontal soft tissue attachment in vivo, and thus focused CO2 laser irradiation is contraindicated for root surface treatment. **[**394**]**

Regarding the Nd:YAG laser, surface pitting and crater formation with charring, carbonization, melting and crater production have been reported after irradiation in vivo, even when irradiation was performed parallel to the tooth surface. Also, a decrease in the protein ⁄ mineral ratio and potential alteration of the surface by protein by-products, have been reported in Nd:YAG laser-treated cementum. Regarding the diode laser, lasing dry or saline moistened root specimens resulted in no detectable alterations. However, the blood-coated specimens showed severe damage, depending on the irradiating conditions. In the case of the Er:YAG laser, several studies have described a characteristic morphological change of the root surface after irradiation. The Er:YAG laser treated root surface under water coolant has been reported to have a micro-irregular appearance without cracks or thermal side effects, which are usually observed after treatment with a CO2 or Nd:YAG laser. The superficial layer of the root surface ablated by Er:YAG laser irradiation presented a minimal affected layer with characteristic microstructure and staining and without major compositional or chemically deleterious changes of the root cementum and dentin studies demonstrate that when a suitable energy is selected, the root surface, after Er:YAG laser irradiation of the diseased surfaces, seems to offer better conditions for the adherence of fibroblasts in vitro than that after mechanical scaling alone. **[**414, 415**]**

Table 4. Clinical studies on laser application in non-surgical treatment of periodontitis

Author and year (reference)	Laser parameters	Study design	Experimental group	Control group	Observation period	Findings
Nd:YAG laser						
Radvar et al. 1996 (116)	0.5 or 0.8 W, 10 Hz	RCT (11 patients, 80 sites)	Laser alone at 0.5 and 0.8 W	SRP and untreated	6 weeks	No clinical or microbiological improvements on laser-treated sites
Ben Hatit et al. 1996 (17)	0.8–1.5 W, 8–15 Hz	RCT (14 patients, 150 sites)	SRP + Laser	SRP	Immediately after, 2, 6 and 10 weeks	Significantly reduced post-therapy levels of bacteria following adjunctive laser therapy
Neil & Mellonig 1997 (101)	2 W, 25 Hz	RCT, split-mouth design (10 patients, 186 teeth)	SRP + Laser	SRP and untreated	6 months	No significant difference in clinical improvements between SRP + laser therapy and SRP alone, but the laser-treated sites showed a tendency to improve steadily until 6 months post-therapy, different from the SRP alone group
Liu et al. 1999 (83)	3 W, 20 Hz	RCT, split-mouth design (8 patients, 52 sites)	Laser alone Laser + SRP 6 weeks later SRP + Laser 6 weeks later	SRP	12 weeks	Less effectiveness of laser treatment in comparison to SRP in reduction of interleukin-1β
Miyazaki et al. 2003 (93)	2 W 20 Hz	RCT (18 patients, 41 sites)	Laser	US	1, 4 and 12 weeks	Significant clinical improvements following laser and US therapies. Significant decrease of *P. gingivalis* and amount of interleukin-1 in the laser-treated sites, similar to the US sites
Noguchi et al. 2005 (104)	2 W, 10 Hz	RCT (16 patients, 135 sites)	Laser alone Laser + local minocycline Laser + povidone-iodine	Untreated	1 and 3 months	Greater reduction of bacteria on laser + minocycline sites than laser alone and sham-treatment sites

Diode laser						
Moritz et al. 1997 (96)	2.5 W, 50 Hz	Controlled study (50 patients)	SRP + Laser	SRP	1 and 2 weeks	Higher bacterial reduction in SRP + laser sites than SRP alone sites
Moritz et al. 1998 (95)	2.5 W, 50 Hz	RCT (50 patients)	SRP + Laser	SRP + H_2O_2 rinse	6 months	Significantly higher bacterial, BOP and PD reduction on laser-treated sites than SRP + H_2O_2 sites
Borrajo et al. 2004 (18)	2 W, pulsed	RCT (30 patients)	SRP + Laser	SRP alone	6 weeks	No additional improvements in adjunctive application of laser in comparison to SRP alone
Kreisler et al. 2005 (72)	1 W, CW	RCT, split-mouth design (25 patients)	SRP + Laser	SRP alone	3 months	Greater reduction of PD and increase of attachment gain in adjunctive application of laser in comparison to those of SRP alone
CO_2 laser						
Miyazaki et al. 2003 (93)	2 W, CW non-contact	RCT (18 patients, 41 sites)	Laser (irradiation to external surface)	US	1, 4 and 12 weeks	No decrease of *P. gingivalis* and amount of interleukin-1 following laser sites in comparison to the significant decrease in US sites
Er:YAG laser						
Watanabe et al. 1996 (175)	ED: 11.3 J / cm^2 / pulse,* 10 Hz	Case series (60 patients, 60 sites)	Laser		4 weeks	Safe and effective calculus removal and subsequent pocket depth reduction at 4 weeks
Schwarz et al. 2001 (132)	ED: 18.8 or 14.5 J / cm^2 / pulse,* 10 Hz	RCT, Split-mouth design (20 patients, 660 sites)	Laser	SRP	6 months	Clinical improvements following laser therapy were similar to or a little better than those of SRP therapy

Schwarz et al. 2003 (138)	ED: 18.8 or 14.5 J/cm^2/pulse,* 10 Hz	RCT, split-mouth design (20 patients, 660 sites)	Laser	SRP	2 years	Clinical improvements following laser therapy could be maintained until 2 years
Sculean et al. 2004 (147)	ED: 18.8 or 14.5 J/cm^2/pulse,* 10 Hz	RCT, split-mouth design (20 patients, 1306 sites)	Laser	US	6 months	Clinical improvements following laser therapy were similar to those of US treatment
Tomasi et al. 2006 (167)	ED: 18.8 J/cm^2/pulse,* 10 Hz	RCT, split-mouth design (20 patients, 160 sites)	Laser	US	6 months	At 1 month following therapy, laser-treated sites showed significantly greater clinical improvements; however, similar results in comparison with US therapy was observed at 4 months post-therapy. No differences microbiologically were observed between laser and US treatments; however, faster healing and less discomfort during treatment were observed in the laser-treated group than in the US-treated group in the maintenance treatment
Crespi et al. 2007 (31)	ED: 16 J/cm^2/pulse,* 10 Hz	RCT, split-mouth design (25 patients, 1200 sites)	Laser	US	2 years	Significantly greater clinical improvements of laser therapy than US therapy at 1 and 2 years post-therapy
Derdilopoulou et al. 2007 (36)	ED: 18.8 or 14.5 J/cm^2/pulse,* 10 Hz	RCT (72 patients, 288 sites)	Laser	SRP, US and Sonic scaling	6 months	Lower bacterial reduction in laser-treated sites than in US sites. Also, US was more pleasant than laser therapy

Periodontal pocket treatment

One of the possible advantages of laser treatment of periodontal pockets is the debridement of the soft tissue wall. Conventional mechanical tools are not effective for the complete curettage of soft tissue.

Gold & Vilardi reported the safe application of the Nd:YAG laser (1.25 and 1.75 W, 20 Hz) for removal of the pocket-lining epithelium in periodontal pockets without causing necrosis or carbonization of the underlying connective tissue in vivo. Use of an Nd:YAG laser in a laser-assisted new attachment procedure has been advocated to remove the diseased soft tissue on the inner gingival surface of periodontal pockets (Food and Drug Administration 510 k clearance K030290). A case series by Yukna et al. reported that the laser assisted new attachment procedure could be associated with cementum-mediated new connective tissue attachment and apparent periodontal regeneration on previously diseased root surfaces in humans. Thus, adjunctive or alternative use of laser treatment in periodontal pockets may promote more periodontal tissue regeneration than conventional mechanical treatment. **[**416, 417**]**

Surgical pocket therapy

In order for a periodontal surgical procedure to be successful with optimal tissue regeneration, it is necessary for the root surface and bone defect to be completely debrided and decontaminated. Laser application is effective in debriding areas of limited accessibility, such as deep intrabony defects and furcation areas where mechanical instruments can-not eliminate microbiological etiologic factors. Laser irradiation can facilitate complete debridement of the defect as a result of its ablation effect as well as improved accessibility when there is contact of the tip of the laser. Crespi et al.used the CO2 laser in a defocused mode (13 W, 40 Hz) for the treatment of experimentally induced Class III furcation defects in dogs following flap surgery and reported that laser treatment promoted the formation of new periodontal ligament, cementum and bone. In addition, the CO2 laser (8 W and 20 Hz) has been shown to increase the effectiveness of periodontal therapy through an epithelial exclusion technique in conjunction with conventional flap surgery procedures. **[**418**]**

The Er:YAG laser has also been shown to be effective and easy to use for granulation tissue removal and root surface debridement during surgical procedures. Sculean et al. reported that application of the Er:YAG laser during the treatment of periodontal intrabony defects with access flap surgery is effective and safe with significant clinical improvements at six months following surgery, however, the laser treatment was equally effective as the mechanical debridement alone.419 In a study, Gaspirc et al. reported the long-term clinical outcome comparing the Er:YAG laser-assisted periodontal flap surgery with conventional treatment using the modified Widman flap procedure. In this investigation, the reduction of pocket depth and the gain of clinical attachment level were significantly greater in the laser group at 6–36 months after surgery. **[**420**]** Schwarz et al. also confirmed that regeneration therapy using an enamel matrix protein derivative was equally effective on the root surface irradiated with an Er:YAG. **[**421**]**

Table 5. Clinical studies on laser application in the surgical treatment of periodontitis

Author and year (reference)	Laser parameters	Study design	Experimental group	Control group	Observation period	Findings
CO_2 laser						
Centty et al. 1997 (22)	8 W, 20 Hz	Randomized, controlled study, split-mouth design	OFD + laser irradiation to outer and inner aspects of mucoperio-steal flap	OFD	Biopsy during the surgery	Laser eliminated significantly more sulcular epithelium in comparison with conventional periodontal surgery
Er:YAG laser						
Schwarz et al. 2003 (133)	ED: 14.5 J/cm^2/pulse, 10 Hz	Randomized, controlled study, split-mouth design	OFD + laser + EMD irradiation for degranulation and root debridement	OFD + EMD EDTA root conditioning	6 months	No significant difference in clinical improvements between EMD + laser therapy and EMD with EDTA root conditioning
Sculean et al. 2004 (148)	ED: 14.5 J/cm^2/pulse, 10 Hz	Randomized, controlled study, split-mouth design	OFD + laser irradiation for degranulation and root debridement	OFD	6 months	No significant difference in clinical improvements between OFD + laser therapy and OFD using hand and ultrasonic instruments
Gaspirc et al. 2007 (46)	100, 140 or 180 mJ/pulse, 10 or 20 Hz	Randomized, controlled study, split-mouth design	MWF + laser irradiation to intrabony defect, root surface and flap	MWF	5 years	Adjunctive application of laser showed significantly greater reduction of PD and increase of attachment gain in comparison to MWF alone until 3 years after surgery

Osseous surgery

Bone recontouring and reshaping are often part of periodontal surgical therapy to establish the physiologic anatomy of the alveolar bone and to allow for an optimal gingival contour after surgery. Commonly employed conventional instruments for bone surgery are mechanical rotary instruments that use carbide or diamond burs, hand instruments such as chisels and files and ultrasonic instruments.

Over the years, the use of erbium lasers has become popular for bone surgery. They reduce the risk of collateral damage, improve the comfort of both patients and surgeons by markedly reducing the noise and eliminating the vibration associated with the mechanical cutting and grinding of bone tissue. Nevertheless, despite the advantages of lasers over mechanical instruments, some issues still hinder a broader use of lasers in bone surgery. These include the reduced cutting efficiency of lasers compared with mechanical instruments, lack of depth control and the effects of the laser on the surrounding irradiated tissue. **[**394**]**

Characteristics of the irradiated bone

Concerns have been raised regarding the use of lasers in bone surgery as a result of the lack of information regarding the nature of the remaining irradiated tissue. Reports showed that irradiation of bone with a CO2 laser leads to severe carbonization and melting. Fourier transform infrared spectra of bone surfaces showed that the extremely high temperatures produced by CO2 laser irradiation cause denaturation of proteins and formation of toxic by-products. **[**422**]**

Likewise, Er:YAG laser irradiation of hard tissues without water coolant may result in superficial charring and the formation of toxic substances.

Such toxic substances may delay the healing process an Er:YAG laser with saline irrigation results in minimal thermal changes and no toxic substance production. One characteristic of the Er:YAG-lased bone tissue that must be considered is the thin affected layer of lased tissue that remains after irradiation. A morphological analysis of this layer showed that it contains irregularities that contribute to entrapment of the initial components of the early healing process and therefore the lased bone heals more quickly compared with bone treated using

Table 6. *In vitro, in vivo* and clinical studies of laser application on bone tissue

Author and year (reference)	Type of study	Laser	Laser parameters	Purpose of application	Findings
Nelson et al. 1989 (102)	*In vivo* (rabbit)	Er:YAG	ED: 12.7 J/cm^2/pulse, 10 Hz	Irradiated tissue characteristics	Er:YAG laser found useful as a cutting tool albeit with delay in healing
McKee et al. 1993 (88)	*In vivo* (rat)	CO_2	3, 7, 9 W, CW ED: 153, 357, 459 J/cm^2, respectively	Cutting efficiency and irradiated tissue characteristics	Novel alternative method for exposing unerupted dental tissues. Irradiated tissue presented densely packed or coagulated collagen fibrils and a separation of old bone and new bone
Lewandrowski et al. 1996 (82)	*In vivo* (rat)	Er:YAG	53 mJ/pulse, ED: 60 J/cm^2/pulse, 1 Hz	Cutting efficiency and irradiated tissue characteristics	Comparable thermal damage in laser and mechanically cut bone. Satisfactory osteointegration of screws in holes made by the laser. Normal fracture healing
Krause et al. 1997 (69)	*In vivo* (rat)	CO_2	ED: 40 to 2062 J/cm^2	Irradiated tissue characteristics	Distinct layer of residual carbonized tissue and thermal necrosis
Friesen et al. 1999 (44)	*In vivo* (rat)	CO_2, Nd:YAG	CO_2: 8 W (1368 J/cm^2) Nd:YAG with coolant: (1368 J/cm^2) Nd:YAG without coolant: (571 J/cm^2)	Irradiated tissue characteristics	Delayed healing. Presence of residual char in the osseous defect. Beneficial effects of water coolant in regards to the healing response
Sasaki et al. 2002 (125)	*In vivo* (rat)	CO_2, Er:YAG	Er:YAG: 1 W (100 mJ/pulse, ED: 35.4 J/cm^2/pulse,* 10 Hz), saline irrigation CO_2: 1 W, CW	Cutting efficiency and irradiated tissue characteristics	Er:YAG laser treatment resulted in tissue removal, absence of charring and presence of a characteristic tissue with two distinct sublayers: a superficial, greatly altered layer showing great amount of cracking and a deep, less affected layer with more discrete cracking. CO_2 laser treatment produced charring and almost no tissue removal
Sasaki et al. 2002 (126)	*In vivo* (rat)	CO_2, Er:YAG	Er:YAG: 1 W (100 mJ/pulse, ED: 35.4 J/cm^2/pulse,* 10 Hz), saline irrigation CO_2: 1 W, CW	Irradiated tissue characteristics	Er:YAG laser produced a characteristic rough tissue and entrapment of fibrin, suggesting a suitable environment for the first events of bone healing. CO_2 laser produced melting and carbonization, and toxic substances that may jeopardize the healing process
Rupprecht et al. 2003 (122)	*In vivo* (rabbit and minipig)	Er:YAG	300–2000 mJ 1, 10, 15 and 20 Hz	Depth control	Highly selective ablation of bone when applied with a closed-loop control system

Table 6. Continued

Author and year (reference)	Type of study	Laser	Laser parameters	Purpose of application	Findings
Abu-Serriah et al. 2004 (2)	Clinical	Er:YAG	700 mJ, 10 Hz	Cutting efficiency	Er:YAG laser bone ablation was more time consuming than bur drilling
Pourzarandian et al. 2004 (114)	*In vivo* (rat)	CO_2, Er:YAG	Er:YAG: 1 W (100 mJ/pulse, ED: 35.4 J/cm2/pulse,* 10 Hz), saline irrigation CO_2: 4 W, CW	Irradiated tissue characteristics	Er:YAG laser irradiation caused a faster initial bone healing. CO_2 laser irradiation produced thermal necrosis and residual char layer
Ivanenko et al. 2005 (60)	*In vivo* (dog)	CO_2	80 mJ/pulse	Cutting efficiency and irradiated tissue characteristics	Effective osteotomy was achieved with absence of tactile feedback, no aggravating thermal side effects and no healing delay. Healing process comprised a complete bony rearrangement of the osteotomy gap with newly built lamellar Haversian bone 22 days after laser surgery
Wang et al. 2005 (174)	*In vivo* (rabbit)	Er,Cr:YSGG	ED: 80 J/cm^2/pulse, 20 Hz	Irradiated tissue characteristics	Er,Cr:YSGG laser cutting produced effective hemostasis, minimal delay of healing and minimal thermal damages to adjacent tissues
Mizutani et al. 2006 (94)	*In vivo* (dog)	Er:YAG	ED: 10.0 J/cm^2/pulse, 20 and 30 Hz	Irradiated tissue characteristics	The affected layer produced by laser was scarcely detected in the bone tissue at 3 months postsurgery. Significantly more bone formation was seen in the laser-treated sites
Papadaki et al. 2007 (110)	*In vitro*	Er:YAG	ED: 63.6, 127, 191 and 255 J/cm^2	Cutting efficiency	Er:YAG laser osteotomies were shown as feasible. Bone cutting was smooth and caused no carbonization
Youn et al. 2007 (184)	*In vitro*	Free electron	ED: 21.2 and 42.4 J/cm^2/pulse, 10 Hz	Cutting efficiency	Ablation efficiency was dependent on wavelength. The wavelength of 6.1 μm provided the highest ablation efficiency
Stubinger et al. 2007 (160)	Clinical	Er:YAG	ED: 64 or 177 J/cm^2/pulse,* 12 Hz	Cutting efficiency and depth control	Laser osteotomies were efficient and precise, with satisfactory healing and no thermal damage, but the procedures were time consuming and offered no depth control
Stubinger et al. 2007 (161)	Clinical	Er:YAG	ED: 64 or 177 J/cm^2/pulse,* 12 Hz	Cutting efficiency	Laser produced precise bone ablation without any visible negative thermal side effects, but the procedures were more time consuming

Conclusion

The application of lasers has been recognized as an adjunctive or alternative approach in periodontal therapy. Soft tissue surgery is one of the major indications of lasers. CO2, Nd:YAG, diode, Er:YAG and Er,Cr:YAG lasers are generally accepted as useful tools for these procedures.

Laser treatments have been shown to be superior to conventional mechanical approaches with regards to easy ablation, decontamination and hemostasis, as well as less surgical and postoperative pain in soft tissue management. Laser or laser-assisted pocket therapy is expected to become a new technical modality in periodontics. The Er:YAG laser shows the most promise for root surface debridement, such as calculus removal and decontamination.

Concerning the use of lasers for bone surgery, CO2 and Nd:YAG lasers are considered unsuitable because of carbonization and degeneration of hard tissue. Currently, the Er:YAG laser is safe and efficient for periodontal bone surgery when used concomitantly with water irrigation.

Further studies are encouraged to understand in more detail the effects of lasers on biological tissues, including the periodontium, in order to ensure their safe and effective application during periodontal treatment. Among lasers currently available, the Er:YAG laser seems to provide the most suitable characteristics for various types of periodontal treatment.

Summary

In the last 50 years, there have been many technological advances in the methods used for the clinical examination of periodontal tissues. We now have a better understanding of the etiological factors associated with periodontitis, the mechanism involved in periodontal diseases and the inter relationship between patient factors and treatment outcomes. This has been made possible due to the constant debate and dialogue surrounding various concepts and procedures, introduction of new concepts and repudiation of certain beliefs and theories. Critical evaluation of both old and new concepts, has unpinned and changed the field of education and teaching in periodontology, as well as encouraged great volumes of research and investigative work.

The subject of periodontology considers a broad range of issues that represent contemporary controversies in the discipline. Controversies exist in various subjects like classification of periodontal diseases, pathogenesis of periodontal diseases including the role of occlusion, periodontal-endodontic controversy, lasers in periodontics and many more. A sincere attempt has been made in giving holistic conclusions to these various controversies through this book. However, the conclusions drawn are not the final word on these topics of discussion and debate.

In the near future one may expect the current controversial topics to get general agreement. However, it is also possible that new controversial topics may arise.

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