

# **GRADUATION PROJECT**

# **Degree in Dentistry**

# THE IMPORTANCE OF OCCLUSAL STABILITY IN PERIODONTAL PATIENTS

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# SUMMARY

**Introduction**: The relation between periodontal inflammation and occlusal trauma remains an uncertain topic for the dental community. From the first studies on the subject published more than a century ago, to the most recent articles, the role of occlusion on the evolution and progression of periodontal disease remains unclear. Despite the numerous studies performed on animals and humans, it was never possible to achieve a statistically acceptable correlation between occlusion and periodontitis, due to several limitations arising regarding studies on those topics, in terms of lesions reproducibility, individualizing factors, and overlapping symptomatology.

**Objectives**: To remark the importance of occlusal stability during the treatment of periodontal disease in the long term.

**Materials and methods:** This article review study was carried out through the investigations of several databases on CRAI Dulce Chacon library and PubMed. Bibliographies of selected articles were also analysed, and relevant ones were included. A total of 34 articles were selected between case studies and articles reviews.

**Results**: 12 articles were included in the results section, 4 case studies regarding periodontal splinting, 2 case studies regarding occlusal adjustments, 6 literature reviews regarding occlusal stability and periodontitis. Regarding the case studies, an overall improvement after the managing of the occlusal instabilities was achieved at the end of the treatment.

**Conclusion:** Considering the multifactorial origin of periodontal disease, and the influence that occlusion has on the homeostasis and wellbeing of the periodontium, achieving occlusal stability will improve the outcome of the indicated and necessary periodontal treatment

Keywords: Dentistry, periodontitis, malocclusion, occlusal adjustments, periodontal splinting

# RESUMEN

**Introducción**: La relación entre la inflamación periodontal y el trauma oclusal sigue siendo un tema incierto para la comunidad dental. Desde los primeros estudios sobre el tema publicados hace más de un siglo, hasta los artículos más recientes, el papel de la oclusión en la evolución y progresión de la enfermedad periodontal sigue siendo poco claro. A pesar de los numerosos estudios realizados en animales y humanos, nunca fue posible lograr una correlación estadísticamente aceptable entre la oclusión y la periodontitis, debido a varias limitaciones que surgen en relación con los estudios sobre estos temas, en términos de reproducibilidad de las lesiones, factores individualizantes y sintomatología superpuesta.

**Objetivos**: Resaltar la importancia de la estabilidad oclusal durante el tratamiento de la enfermedad periodontal a largo plazo.

**Materiales y métodos**: Este estudio de revisión de artículos se llevó a cabo mediante la investigación de varias bases de datos en la biblioteca CRAI Dulce Chacón y PubMed. También se analizaron las bibliografías de los artículos seleccionados y se incluyeron los relevantes. Se seleccionaron un total de 34 artículos entre estudios de casos y revisiones de artículos.

**Resultados**: Se incluyeron 12 artículos en la sección de resultados, 4 estudios de casos sobre férulas periodontales, 2 estudios de casos sobre ajustes oclusales y 6 revisiones de literatura sobre estabilidad oclusal y periodontitis. En cuanto a los estudios de casos, se logró una mejora general después de la gestión de las inestabilidades oclusales al final del tratamiento.

**Conclusión**: Teniendo en cuenta el origen multifactorial de la enfermedad periodontal y la influencia que la oclusión tiene en la homeostasis y el bienestar del periodonto, lograr la estabilidad oclusal mejorará el resultado del tratamiento periodontal igualmente indicado y necesario.

**Palabras clave**: Odontología, periodontitis, maloclusión, ajustes oclusales, férulas periodontales.

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# **INTRODUCTION**

# Historical prospective

# **1.** Relation occlusal trauma – periodontal disease

The relation between occlusion and periodontal disease it's been and remains one of the most controversial topics in dentistry.

From the first publication of Karolyi in 1901, suggesting the implication of occlusal forces in the progression of periodontal diseases, several studies have been conducted on animals and human cadavers, investigating a possible causal relationship between infrabony pockets and trauma for occlusion.

Studies performed by Carranza in 1957, suggested that the disorganization of the periodontal tissue is a consequence of the occlusal trauma received, and will result in impaired repair function and play a role in the deepening of periodontal pockets already existing (1).

Subsequent studies in animals, performed by Bhaskar & Orban and Polson et al. (2), between 1955 and 1974, showed no clear association between occlusal trauma, gingival inflammation, or periodontitis. It's worth of notice though that animal occlusion differs quite a lot compared with human occlusion, as the function and physiology of mastication in humans and animals is very different, especially in terms of directions of forces, where for humans consist in mostly jiggling forces as a result of social and functional evolution and for animals the directions of forces are mostly unidirectional.

During the same years, Glickman proposed that occlusal trauma was instead of the causal factor an exacerbating factor, by promoting the penetration of gingival inflammatory exudate right in the periodontium apparatus (3,4). Nevertheless, the possibility to find signs of OT (occlusal trauma) and inflamed periodontium with no presence of vertical bone loss, imply the

non-exclusivity of those signs to OT. However, the presence of infrabony pockets are not always the results from trauma from occlusion (TFO), and it's possible for occlusal trauma and inflammation to occur simultaneously, even in the absence of pockets, meaning that these conditions are not exclusively indicative of trauma from occlusion (1).

Few years later, multiple studies performed on beagles dogs, aiming to prove a relation between the type of pocket, infrabony (vertical) or suprabony (horizontal), and the thickness of the surrounding alveolar bone undergoing occlusal trauma, but the presence of plaque in the deepest part of the pockets, invalidated this hypothesis, as the presence of infrabony pockets cannot always be attributed to trauma from occlusion alone (5).

In the last 40 years, thanks to the important technology advancements in terms of investigation and research, all the dental community agreed once for all that the principal etiological factor of periodontitis is plaque. Thanks to the previously performed studies implying a periodontal response from excessive occlusal forces, Davies and Harrel (6) proposed the implementation of occlusal adjustment for periodontally compromised teeth in order to relieve the occlusal load, thereby improving the prognosis, as he concluded that for regarding occlusal forces during periodontal treatment, they shouldn't exceed the adaptive capacity of the periodontium.(7)

The two major and most recent classifications of periodontal disease that were made, respectively in 1999 and 2018, during the two World Workshops held by the periodontal committed are reviewed in the following chapters, as some big changes, among which the impact of acquired risk factors such as occlusal trauma and excessive occlusal forces on the progression of periodontal pathology have been made (8).

### 2. Glickman concept

One of the most relevant concept regarding the role of occlusion in the advancement of PD, was made by Glickman between 1955 and 1965 (3,4,9). He stated that the spreading pathway

of a plaque-related gingival injury can be modified by forces of abnormal magnitude acting on the impaired tooth. Glickman classified two new zones regarding the periodontium, an irritation zone, including the interdental gingiva, marginal gingiva and gingival fibers, and a zone of co-destruction consisting of transeptal and alveolar crest fibers, periodontal ligament (PL), cementum, and bone complex.

He stated that trauma from occlusion (TFO) cannot induce gingival inflammation in the irritant zone, instead, the presence of inflammation is only due microbial plaque. Inflammation in this zone will lead to horizontal bone defect. The zone of co-destruction instead can be affected by both plaque, once the inflammation reaches the fibers bundle separating the two zones, and TFO. Those two factors can alter the composition and orientation of those fibers, promoting inflammatory exudates coming from the plaque-induced gingival inflammation straight to the zone of co-destruction. This will lead to the formation of infrabony or vertical defects(4,10).

## 3. Waerhaug concept

Few years after Glickman's studies, Waerhaug questioned it's results regarding the implications of TFO for the development of infrabony pockets(11). In his autopsy studies, Waerhaug was able to identify the existence of angular defects on teeth which were not subjected to occlusal trauma with the same frequency as teeth undergoing traumatic occlusion, partially invalidating Glickman's concept of irritation and co-destructive zones. The only correlation Waerhaug found, was the presence of subgingival plaque where alveolar crest height was reduced, implying that the appearance of loss of attachment is an exclusive feature of the bacterial colonization of the subgingival space and not related with occlusion.

The concepts of Glickman and Waerhaug are the pillars of the controversies raised in the last 50 years of research on the subject.

### Periodontium and occlusion

### 4. Periodontium and periodontitis

The term "periodontium" refers to all the tissues and structures that play a role in supporting and surrounding the teeth. It includes the gums (gingiva), the connective tissue fibers (periodontal ligament), the bone (alveolar bone), and the specialized calcified tissue (cementum) covering the tooth roots. Although each element of the periodontium has distinct anatomical and biochemical features, they function as an integrated unit. New studies have indicated that the extracellular matrix of a specific periodontal component can affect the cellular functions of confining periodontal structures, meaning that if one of those units get impaired, also the others can get affected, impairing the overall function and regenerative capacity (12).

Periodontitis is a multifactorial inflammatory disease which affects one or more periodontal structures. The main etiological cause of periodontitis is the accumulation of dental plaque, a sticky film of bacteria that forms on teeth and gum tissues. If plaque is not adequately removed by regular brushing and flossing, it can harden and form tartar (calculus), which cannot be removed by brushing alone. Tartar accumulation leads to the formation of deep pockets between the teeth and gums, which can act as a scaffolding for bacteria and cause inflammation and destruction of the periodontal tissues. Other factors that can contribute to periodontitis include smoking, hormonal changes, genetic susceptibility, certain medications, and systemic diseases such as diabetes (12).

# 4.1 Periodontal health

The World Health Organization (WHO) has put forth a definition of health that goes beyond simply the absence of illness or infirmity. According to this definition, health should be understood as a state of complete physical, mental, and social well-being. This definition can also be applied to periodontal health, which refers to the absence of inflammatory periodontal disease and the ability to function normally without any negative consequences resulting from past disease. However, while this definition is patient-focused, it may not be the most appropriate in terms of practical clinical-making decision. A more specific description of healthy periodontium could be the absence of clinical signs of periodontitis, meaning absence of inflammation and destruction of the periodontium. (13).

Some argue that the presence of anatomic alterations, such as periodontal pocketing or bone loss, suggests a history of disease and therefore cannot be considered an indication of true periodontal health. Others suggest that stability in the absence of inflammation may be considered a form of health, and that the emphasis should be on preventing further disease progression rather than reversing previous damage. Ultimately, the definition of periodontal health is complex and multifaceted, and must take into account not only the absence of clinical signs and symptoms, but also the patient's overall periodontal history and risk for future disease (14).

The first sign of periodontal disease is gingival inflammation around the gingival collar of the affected tooth, as shown in Figure 1. If the inflammation persist, with time, will lead to clinical attachment loss (CAL) and bone loss (BL) (15)



**Figure 1**: (a) Healthy gingiva, no inflammation present, (b) Early gingivitis, arrow pointing to an early inflammation process of the interdental papilla, (c) Chronic periodontitis, severe gingival inflammation, characterized by clinical attachment loss of tissues around the tooth, The arrow points to a deep periodontal pocket which was the result of the tissue lost. (15)

# 4.1.1 Determinants & risk factors

Current understanding of periodontal disease (PD) suggests that they should no longer viewed as straightforward microbial infections, but rather multifactorial conditions that result from a combination of factors. These factors include the composition of the microbiota, the host's immune and inflammatory responses, as well as environmental factors. As a result of the complex nature of periodontal diseases, evaluating the health of the periodontium solely based on plaque and bacteria present is not sufficient. Rather, a comprehensive evaluation of all factors that contribute to disease development plus the restoration and maintenance of health, is necessary (12).

Determinants of periodontal health can be grouped into three main categories: microbiological and host, which togethers form the first layer of determinants, and the environmental factors. Understanding controllable and uncontrollable predisposing and modifying factors is critical for achieving and maintaining clinical periodontal health(16). The summation of all those factors will give an idea regarding severity, progression, and prognosis of the ongoing periodontal disease, as shown in Figure 2.

#### CLINICAL DETERMINANTS OF PERIODONTAL HEALTH (12,14,16):

- 1. Microbiological determinants
  - A. Supragingival plaque composition
  - B. Subgingival biofilm composition
- 2. Host determinants
  - A. Local predisposing factors
    - Periodontal pockets
    - Dental restorations
    - Saliva
    - Tooth position and alveolar bone
    - Occlusion
  - B. Systemic modifying factors
    - Host immune function
    - Systemic health
    - Genetics

- 3. Environmental determinants
  - Smoking
  - Medications
  - Stress
  - Nutrition
  - Home and professional care
  - Nutrition



**Figure 2**: Susceptibility of the progression of periodontal disease. The balance between protective factors and risk factors will likely determine the prognosis (15)

### 5. 1999 vs 2018 classification

The 1999 classification only considers the severity and extension of the disease, classifying as 1) aggressive (localized and generalized), 2) chronic, 3) necrotizing and 4) as a manifestation of a systemic disease and was a primarily clinical and radiographic type of diagnosis that relied on the measurement of clinical parameters such as pocket depth, attachment level, and radiographic bone loss in order to determine the severity of periodontitis. This approach assumed that periodontitis was caused by a few specific pathogens and that the host response was relatively constant across individuals(17).

In contrast, the 2017 classification incorporates both clinical and individual factors in the diagnosis of periodontitis. This approach recognizes that periodontitis is a complex disease caused by multiple pathogenic bacteria and that the host response can vary widely across individual as individuals genetic, health, hygiene and harmful habits are unique for each patient.

Those factors had been included in the 2017 classification by adding four stages (I-IV) (Table 1, ANNEX), which assess complexity, severity, and extent of the disease, and three grading (a, b, c) (Table 2, ANNEX) which represent risk factors, rate of progression and responsiveness of the patient to the therapy(18).

Changes to the old classification also affected the role that occlusion plays in the development and progression: in the 1999 classification, occlusion played a significant role in the classification of periodontal diseases. Specifically, occlusal trauma was considered as "Developmental or Acquired Deformities and Conditions" (17).

In the 2017 classification instead, the role of occlusion in the classification of periodontal diseases is more specific. The classification recognizes occlusal trauma as a contributing factor in the progression of periodontitis, but it is not considered as a defining characteristic of any specific type of periodontal disease. However, there are situations where the decreased

adaptive capacity of a periodontally compromised tooth may let previously non harmful occlusal forces become harmful (second occlusal trauma) and presenting as a grade 2+ mobility. A situation like this, following the staging classification of the 2017 Workgroup, correspond to a stage IV (most severe) by definition(19).

Another important change regarding what was before the main diagnostic parameter, is the introduction of a new term: CAL (Clinical attachment loss). It's measured by circumferential probing erupted teeth from the bottom of the gingival margin or pocket to the cementoenamel junction (CEJ), and until today is considered one of the most precise indicators of disease progression and severity rather than the old "clinical attachment loss" parameter, which measure the distance from the gingival margin to the bottom of the pocket, as measured with a periodontal probe. Probing depth measurement still, is useful in identifying the presence of periodontal pockets, which are a sign of attachment loss and alveolar bone loss, hence it is still a useful parameter to consider (17).

The 2018 classification consider a patient a periodontal patient if we detect interdental CAL on two or more non-adjacent teeth or if we find CAL with pocketing >=3 mm in two or more teeth.

Bleeding on probing (BOP) remains a relevant clinical indicator to assess periodontal stability but does not directly influence the severity of classification (15).

# 6. Diagnosis

The diagnosis of periodontal disease involves a comprehensive evaluation of a patient's periodontal health, which should include(12):

Medical and dental history: A meticulous and detailed medical history is required for any type of dental treatment. The multi-etiological origin and the number of factors having a role in periodontitis makes it even more a crucial step for a proper diagnosis and management of the disease. Patient existing conditions, medications intakes, bleeding related problems, allergies, family history and patient's consumption of alcohol and tobacco are all possible factors to consider when dealing with periodontitis. Same goes for the dental history, where a detailed history of patient's oral hygiene habits, signs, and symptoms such as spontaneous bleeding and bad breath and taste should be recorded. There are several health-history forms available nowadays, the one proposed by the American Dental Association (ADA) (20) can be seen in Figure 3.

| Health History Form   | ADA American Der   | nta Association*           | Medical Information   | Please mark (X) your response t        | o indicate                 | if you have or have not had any of the fo   | dowing diseases or problem       | nı.              |
|---|--|----------------------------|---|--|----------------------------|---|----------------------------------|------------------|
| ( ) ) ) ) ) ( ) ) ) ( ) ) ) ) ( ) | Americas leading advo  | scate for oral nealth      | (Check DK if you Don't Know the answer to   | the question) W                        | ns No DK                   |   |                                  | Yes No DK        |
| emai: today's Lotte   |  |                            | Do you wear contact lenses?   | E                                      |                            | Do you use controlled substances (drugs)?   |                                  |                  |
| As required by law, our office adheres towritten policies and procedures to protect the priv  | acy of information about you that we create, receive or maintain   | . Your answers are for our | Joint Replacement. Have you had an orth<br>(hip, knee, elbow, finger) replacement?        | opedic total joint                     |                            | Do you use tobacco (smoking, snuff, chew,<br>if so, how interested are you in stopping?<br>Circle one: VERY / SOMEWHAT / NOT INTE | bidis)?                          |                  |
| additional questions concerning your health. This information is vital to allow us to provide a   | propriate care for you. This office does not use this information  | to discriminate.           | Date: If yes, have you ha   | a any complications /                  |                            | Do you drink alcoholic beverages?   |                                  |                  |
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| Matrix address  |  |                            | treatment with an antiresorptive agent (ik  | e Aredia", Zometa", XGEVA)             |                            | Premart?  |                                  |                  |
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| Do your ours bleed when you brush or floss?   | Do you have earaches or neck pains?  |                            | Congental reart osease (CHD)  |  |                            | Asthma  | Fainting spells or seizures      |                  |
| Are your teeth sensitive to cold, hot, sweets or pressure?  | Do you have any clicking, popping or discomfort in the jaw?  |                            | Unrepared, cyanotic CHD   |  |                            | Protectitis   | Neurological disorders           |                  |
| is your mouth dry?  | Do you brux or grind your teeth?   |                            | Hepared (completely) in last 6 month  |  |                            | Emotysena D D D   | If yes, specify:                 |                  |
| Have you had any periodontal (gum) treatments?  | Do you have sores or ulcers in your mouth?   |                            | Repared CHD with residual delects   | L                                      |                            | Soustmubie  | Sleep disorder                   |                  |
| Have you ever had orthodostic (braces) treatment?   | Do you wear dentures or partials?  |                            | Except for the conditions listed above, anti  | biotic prophylaxis is no longer recom  | mended                     | Titeroinsis D.D.D.  | Do you snore?                    |                  |
| Have you had any problems associated with previous dental treatment?  | Do you participate in active recreational activities?  |                            | for any other form of CHD.  |  |                            | Carcer/Cherrotherany/   | Mental health disorders          |                  |
| is your home water supply fluoridated?  | Have you ever had a serious injury to your head or mouth?  |                            | No. No. DV  |  |                            | Radiation Treatment   | Specify:                         |                  |
| Doyos drink bottled or filtered water?  | Date of your last dental exam:   |                            |   | Mitral valve omlanes                   |                            | Chest pain upon exertion  | Recurrent infections             |                  |
| If yes, how often? (Clask one) DAILYTH / WEEKLY TH / OCCASIONALLY TH  | What was done at that time?  |                            |   | forenation (                           |                            | Chronic pain  | Kidaw problems                   |                  |
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| what is the reason for your dental visit coday?   |  |                            | Heart attack  | Agenta                                 | 100                        | Gastrointestinal disease  | in neck                          |                  |
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| How do you feel about your smile?   |  |                            | Low blood pressure  | If yes, date;                          |                            | heartburn 🗌 🗆 🗆   | inganes                          |                  |
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| Are you now under the care of a physician?  | Have you had a serious liness, operation or been hospitalized  |                            | Name of physician or dentist making recom   | mendation:                             | ,                          |   | Phone: Adule reacefe             |                  |
| Physician Name: Phone: Include mea cade   | in the past 5 years?   |                            |   |  |                            |   | ( )                              |                  |
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| Has there been any change is your general beath within the past year?   | and/or dietary supplements:  |                            | I will not hold my dentist, or any other men  | nber of his/her staff, responsible for | any action                 | they take or do not take because of errors or   | omissions that I may have mad    | de in the        |
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| Form S600   |  |                            |   |  |                            |   |                                  |                  |
|   |  |                            |   |  | _                          |   |                                  |                  |

*Figure 3*: Health history form from ADA. It includes patient information, dental history, medications, allergies, surgical history, and chronic conditions (20) Clinical examination: Starting from the extraoral examination, we should first visually, then by palpation, assess TMJ, salivary glands and lymph nodes of head and neck, only then proceed with the intraoral exploration. All the soft tissue of the oral cavity should be inspected, looking for signs of possible premalignant lesions. Gingival inflammation is present in case of ongoing periodontal disease and is associated with swelling, redness, bleeding, and loss of scalloped gingival contour either due inflammation or bone loss. Regarding the dental and periodontal examination, signs of ongoing periodontitis include plaque accumulation, gingival recession, and tooth mobility. Subgingival invasion can be assessed by probing, which is another essential step giving us information about bleeding on probing (BOP), pocketing probing depth (PPD), and clinical attachment loss (CAL), as seen in Figure 4 (12,15).



**Figure 4**: (a) Periodontitis stages, from healthy (left), to advance (right). (b) clinical measurement of PPD: bottom of pocket to gingival margin (left). On the right, CAL measurement = PPD measurement + gingival marginal level lost (up to cemento-enamel junction) (15)

 Radiographic examination: The assessment of the existence of an inflammatory reaction of the PD should be complemented radiographically; periodontal ligament widening, root resorption and vertical bone loss are all pathological signs. A series of periapical x-rays and bitewings x-ray is the conventional way we should approach periodontal studies and further revaluations (Figure 5).



*Figure 5*: Example of radiographic periodontal examination series. In this case, 5 maxillaries periapical and 5 mandibular periapical, were taken. (15)

Other tests: In some cases, additional tests may be necessary to confirm the diagnosis
of periodontal disease, such as a bacterial culture or a genetic test to identify specific
bacteria or genetic factors that may increase the risk of developing periodontal disease.

# 6.1 Periodontal charts

Periodontal charts are a useful instrument to monitor status and progression of the periodontal disease. The periodontal chart typically consists of a diagram of the patient's mouth, with each side of each tooth is represented as a separate area on the chart. To measure clinical attachment loss, the clinician should insert the probe in the deepest points of the

gingival sulcus and measure how many millimetres until the CEJ (Figure 4.b). Ideally, a healthy gum and periodontium should have a maximum CAL < 1 mm; measurements from 1 mm above imply gingival recession. To be considered a periodontal patient the chart should show(18):

- interdental CAL on two or more non-adjacent teeth or
- buccal or oral CAL of 3 mm or more with the presence of pockets of at least 3 mm for more than two teeth.

The amount of bleeding and inflammation present is also recorded, along with any signs of bone loss and tooth mobility.

## 7. Occlusal trauma & adaptive capacity of PDL

Occlusal trauma latest definition, from American academy of periodontology (AAP), is defined as an injury within the attachment apparatus of the periodontium due physiological or parafunctional forces, which lead to microscopic alterations of the periodontal components, resulting in alterations of its physiological and biological properties, which will manifest in an overall decrease of the adaptive capacity of the periodontium (19).

The periodontal ligament functions as a protective mechanism that absorbs and adjusts to the forces applied to the tooth's surface. However, variations in occlusal forces can lead to alterations in the periodontium (19).

The pattern and extension of the lesions are directly related to the mode of onset of those acting forces (acute or chronic), while the occlusal forces threshold for their manifestations depends on the healthiness of the periodontal apparatus (primary or secondary) (21).

# 7.1 Acute vs chronic OT

- Acute OT: lesion as the consequence of abrupt occlusal forces, such as biting something very hard, (bite an olive and find out it has a pit). It can also be caused by restorative or prosthetic interferences and prematurities, which can alter the direction of occlusal forces (12).
- Chronic OT: term used to describe changes in the periodontium resulting from gradual alterations in the occlusal forces generated by tooth friction, tooth drifting, and extrusion, which may be exacerbate by parafunctional habits. Unlike acute periodontal trauma, chronic occlusal trauma is more commonly observed and has greater clinical significance (12).

# 7.2 Primary vs secondary OT

Primary OT: is a form of trauma that arises when excessive occlusal forces are the main cause of periodontal damage or destruction, on a tooth with healthy support (Figure 6.1).

Some instances where the lesion can occur on periodontally healthy units include: 1) Restorations with inadequate occlusal contact; 2) the placement of a prosthetic device that puts excessive force on the teeth surrounding it; 3) due the drifting and extrusive movements of teeth into spaces caused by missing teeth; 4) orthodontic treatment that moves teeth into non physiological position (12,21,22).

 Secondary OT: describes a type of injury that results from normal or excessive forces applied to teeth with pre-existing damage or compromised periodontal ligament tissue (Figure 6.2), which can lead to further tissue changes, meaning that even the forces that wouldn't damage a healthy periodontium can result in tooth and bone damage in the moment those forces exceed the adaptive capacity of the PDL (12,21,22).



**Figure 6**: (1) Primary occlusal trauma, affecting a periodontally healthy tooth. As the attachment surface is intact up to CEJ, higher amount of occlusal forces can be withstand compared to a tooth which suffer attachment loss. (22)

# 7.3 Etiology of occlusal trauma

The etiology of TFO encompasses numerous factors, which can be broadly classified into two categories: predisposing and precipitating factors. The damaging occlusal forces are the main factors that trigger TFO, while there are other contributing factors that indirectly contribute to the development of this condition. These predisposing factors can be either intrinsic or extrinsic (23).

# 7.3.1 Precipitating factors

the primary etiology or precipitating factor of TFO is attributed to destructive occlusal forces. These forces are commonly characterized by their magnitude, direction, duration of application, and frequency of application (24).

- Magnitude: refers to when the amount of force applied to a tooth surpasses the normal range of forces it can withstand. As a natural adaptive response, certain changes may occur within the periodontal ligament. These alterations may comprise the expansion of the periodontal ligament space, an augmentation in the quantity and thickness of PDL fibers, and a rise in the compactness of the alveolar bone.
- Direction: The optimal direction to receive forces on our teeth is parallel to the tooth axis. In this way, the forces are distributer homogeneously around the whole ligament, maximizing the periodontal absorption of those forces. If the direction of those forces changes, like in situations of prematurities, interferences and tooth mobility, also the ability of the periodontium to withstand those forces is decreased, leading to possible injuries.
- Duration: of force application is also a critical factor in the development of TFO. In a situation where occlusal forces exceeding the adaptive capacity of the periodontium are affecting a tooth or group of teeth, it can result in damage of the PDL.
- Frequency: of force application is another crucial aspect in the development of TFO.
   The more frequently abnormal occlusal forces are applied to a tooth, the greater the damage they can inflict on the tooth-supporting structures within the periodontium, as compared to less frequent application.

# 7.3.2 Predisposing factors

There are various factors that can replace or supplement the primary etiology of occlusal trauma. These factors are generally divided into two categories: intrinsic and extrinsic factors, and include properties related to the dental development, functional occlusion, and systemic variabilities of the person. (24).

#### Intrinsic factors

- The alignment of the longitudinal axis of the tooth with respect to the direction of the forces exerted on it.
- Roots morphology, as the number of roots, dimension and structure play a critical role in determining how occlusal forces are dispersed. Teeth with sharper, narrowed, small, or fused roots generally present increased susceptible to occlusal trauma compared to those with longer, wider, and more robust roots.
- Quantitative and qualitative properties of the surrounding alveolar bone are a factor that can influence the development of TFO. The ability of the alveolar bone to absorb occlusal forces is critical in preventing damage to the toothsupporting structures. Alveolar bone with adequate quality and quantity is better equipped to withstand occlusal forces and prevent TFO (24).

#### - Extrinsic factors

- Irritants are factors that contribute to the development of TFO, with microbial plaque being the most significant. Other forms of irritation, having related consequences include open contact points, allowing food to get stuck in the interdental space, restorative treatments not respecting biological space and teeth preparations for full coverage with an inadequate finish line.
- Neuroses, such as bruxism, have probably the greatest influence regarding the appearance of abnormal occlusal stresses, and are among the commonest and severe triggers of TFO (24).

# 7.4 Occlusal interferences

Occlusal interferences are irregularities in the way the upper and lower teeth come together when the jaw is closed. These interferences can result from a variety of factors, such as misaligned teeth, dental restorations that do not fit properly, or changes in the bite due to tooth wear or loss. Occlusal interferences can lead to imbalanced forces on the teeth, when those forces are exceeded, trauma to the periodontal tissues could happen, and it could lead to the growth and advancement of periodontal disease (12,25).

# 7.1 Occlusal prematurities

Occlusal prematurities (or deflective occlusal contacts) are a type of occlusal interferences which specifically refer to premature contact between the teeth during biting or chewing.

When teeth come into contact prematurely, it can cause excessive force to be placed on the teeth and surrounding tissues. This can lead to occlusal trauma, damage of the supporting tissue and bone loss (12,25).

### 8. Tissue response to external forces

The response of tissues to increased occlusal forces occurs in three distinct stages: injury, repair, and adaptive remodelling of the periodontium(12,21).

- Stage I: Tissue Injury

The first stage, known as the injury stage, occurs when excessive occlusal forces cause damage to the tissue. If the forces are not chronic and constant, our organism will try to fix the lesion, repairing the injured tissues. In cases where instead, normal occlusal forces cannot be re-established, it will cause the PDL to widen up, impairing the bone around it. In this type of situation, we should expect radiographically and clinically angular bony defects without the presence of pocket. Rotation of the tooth is another consequence of decreased bone attachment especially for uni-radicular teeth, and according to the direction of the forces and type of occlusal contact it can produce one or multiple side of pression, and one or multiple sides of tension, as the human occlusion is characterized by jiggling forces. For multi-radicular teeth instead, the furcation area is the one which is more affected by excessive occlusal forces. In case of PDL lesion, the reparative capacity of the periodontium is compromise, until those harmful forces will be removed (12,21).

Stage II: Repair

The reparative stage happening in the PDL, will start once the forces that previously were larger than the adaptive capacity of the periodontium will stop of decrease under the PDL adaptive capacity. The reparative process starts by the removal of compromised tissue and the formation of new one, which will try to repair the damage. The forces only cause damage when they exceed the adaptive capacity of the tissue (12,21).

Stage III: Adaptive remodelling

During the remodelling stage, the PDL changes its structure in response to the traumatic forces. This happen when Stage II is unable to keep up with the destruction. The periodontal ligament widens, and the bone undergoes angular defects without the formation of pockets. This widening of the periodontal ligament and bone defects allow for cushioning of the forces, inhibiting further damage to the tissues. This stage may also involve increased vascularization.

Histometric analysis can differentiate the three stages of traumatic lesions. In the Stage I, the resorptive amount is predominant over the formation, and on contrary, in Stage II we can appreciate increase in osteogenesis and decrease in resorptive portion.

After this stage, remodelling, resorption, and formation return to normal (12,21).

# **OBJECTIVES**

#### MAIN OBJECTIVE

- To remark the importance of occlusal stability during the progression and treatment of periodontal disease in the long term.

#### SECONDARY OBJECTIVE

- To emphasize the importance of diagnosing and treatment of occlusal trauma in periodontal patients.

# METHODOLOGY

The research was carried out through CRAI Dulce Chacon library and PubMed, including databases such as Academic search ultimate, Dentistry & oral science, and Medline complete.

To develop the investigating question, PICO methodology has been applied as follow:

- Do periodontal patients undergoing occlusal adjustments/splinting therapy present an increase in clinical parameters compared with the ones who don't?

The search equation used was as follow: ((periodontal disease) AND ((occlusal trauma) OR (malocclusion)) and ((tooth splinting)) or (occlusal adjustments))

The references of the articles selected have also been analysed and the most relevant articles and studies have also been included.

Articles older than 10 years old have been used for an historical introduction on the subject, while only the articles from the last 10 years have been used to develop the rest of the paper.

- Inclusion criteria:
  - Case studies, case report, literature review regarding periodontitis, malocclusion, and its management.
- Exclusion criteria:
  - In-vivo studies older than 10 years old, studies regarding implants.

### RESULTS

### 9. Data collection process

The identification of relevant studies included in the results section was carried out through CRAI Dulce Chacon library and PubMed. From an initial amount of 3538 results, 12 were excluded as duplicated, 3526 were excluded based on title. Of the remaining 282, 263 were not retrieved after abstract analysis, and 4 studies regarding periodontal splinting, 2 studies regarding occlusal adjustments on periodontal patients, and 6 literature review regarding the overall role of occlusion regarding periodontal disease progression were included in order to provide a comprehensive overview on the status of this subject.

The flow diagram regarding articles identification, screening, and inclusion can be found in the ANNEX section of this paper (ANNEX, Figure 7).

### **10.** Regarding tooth splinting

In the four studies included in this section, a total of 183 periodontally compromised patients received tooth splinting for a total of 549 mandibular and 148 maxillary teeth splinted. The result table regarding the included splinting studies can be found in the ANNEX section (Table 3).

Sonnenschein et al. in 2017 (26) assessed survival rate and stability of splinted lower anterior teeth of periodontal patients (where at least one of the lower anterior group presented mobility, CAL  $\geq$ 5mm and RBL  $\geq$  50% ) undergoing periodontal treatment over a period of time ranging from 3 to 15 years. Out of the 39 patients included in this study, a total of 162 teeth were splinted. None of the 162 splinted teeth was lost for 6 years, having the first tooth loss at the 7<sup>th</sup> year and the second at the 12<sup>th</sup> due problems occurred during the devitalization treatment. Regarding the periodontal parameters of the splinted teeth, at baseline, the average CAL was 5.61mm, radiographic bone loss (RBL) = 72%, gingival bleeding index (GBI)

=12.8% and polymerize chain reaction (PCR) 35.9%. During the follow up, an overall improvement of the periodontal situation was seen, as the follow up record showed a CAL of 5.09mm, RBL = 63%, GBI = 6.1% and PCR = 34.02%. Regarding splint stability, the survival rate after 3 years was 74.4%, and after 10 years 67.3%.

Another recent cohort study performed by Graetz et al. (27) assessed 57 periodontal patients regarding splint survivability over an average of 11 years. Those 57 patients had received active periodontal treatment (APT) (scaling & root planning and flaps surgeries when needed), supportive periodontal treatment (SPT) and splinting, for a total of 227 teeth splinted, 148 of which located in the maxilla and 79 on the mandible, with an average of 4.1 splinted tooth per patient. The indexes have been recorded in two different times, T1 before the splinting procedure, and T2, after one year. The mean periodontal probing depth (PPD) regarding splinted teeth went from 5.4mm in T1 to 3.2 in T2; the comparison between PPD evolution and progression at T1 and T2 in the different dental zones for both group A and B can be seen in Figure 8.



Worsend between T1 - T2
 Stable between T1 - T2
 Improved between T1 - T2

*Figure 8*: Evolutionary comparison of periodontal probing depth (PPD) between splinted and non-splinted teeth by dental sector (27)

In the same way, bone loss evolution can be found in Figure 9.



*Figure 9*: Evolutionary comparison of bone loss (BL) between splinted and non-splinted teeth by dental sector (27)

171 splinted teeth out of the 227 (75%) needed to be repaired during the time of this study, finding smokers, lower anterior region and not having antagonist contacts having a significant association.

Splinted and non-splinted teeth didn't show any difference in terms of risk factors regarding tooth loss, while tooth position, patient age, pockets >6mm showed up to have a relation.

Regarding the second study conducted by S. Sonnenschein et al. in 2022 (28), 26 periodontal patients, each one requiring lower canine-to-canine splinting were randomly selected in two groups. Group A consisting of 12 patients undergoing splinting after full mouth disinfection (FMD) and periodontal treatment (PT) procedure, and group B, consisting of 14 patients who received splinting before FMD and PT. Both groups were examined before treatment and after 1 year; the indexes were calculated 4 times, two before treatment (T1), measuring the whole denture (overall) and lower anterior sector (teeth 33-43), and the same two 1 year after

treatment (T2). The parameter analysed, resumed in Table 4 (ANNEX) for group A and Table 5 (ANNEX) for group B are CAL, PPD, BOP, PCR and GBI.

Regarding probing depth, only the lower anterior pockets have been recorded at T1 and T2, and it can be seen in Figure 10.



After one year of treatment, there were no tooth loss regarding the lower anterior sector, and only one case of debonding.

The study realized by Zhang et al. in 2023 (29), consist in a retrospective assessment of 61 periodontal patients, where 26 received standard posterior sector SRP, 19 posterior sector modified Widman flap, and 11 posterior sector guided tissue regeneration. Surgical treatment was not administered to any of the anterior regions. On average, each patient had 2.6 teeth that were splinted, for a total of 161 splinted teeth, 67 of which splinted with resin + mesh strip and 94 only with composite resin.

From a starting point of 2.7 PI and 85% of BOP, after 5 years from the treatment the results were 0.9 PI and 13% BOP. PD improved from 4.31mm to 2.93mm and call from 5.02mm to 4.58mm. Contrary, RBL didn't show changes before and after the follow up. The survival rate of the splints was 83.9% after 3 years and 70.2% after 5 years, showing splints without mesh

more incline to breakage compared to the reinforced ones, with a total repair ratio of 0.2/patient. The VAS score regarding satisfaction increased from 3.3 to 7.9 at the end of the treatment (29).

### 11. Regarding occlusal adjustments

Two recent in vivo studies regarding malocclusion and periodontitis progression have been selected to be included in this review section. Both conducted by Meynardi et al., respectively in 2016 and 2018 (30,31).

The first one, realized in 2016 consisted in 260 periodontal patients divided into group 1 and group 2. Group 1, containing 81 subjects undergoing APT alone, and group 2 undergoing APT as well as occlusal adjustments. Consistent plaque samples were retrieved from all the 260 patients and monitored monthly during 6 months in order to identify bacterial profile evolution for both groups. Starting from the third month, group 2 showed a significant increase in proportion of cocci content (74%) which remain constant up to the last month, while group 1 coccus content at third month was 48%, lowering to 26% at the last month, implying the role of occlusal traumas in restabilise a pathogenic bacterial profile, as cocci is an indicator of periodontal health (30).

In his second case study, a 60 years old female patient, shows sign of periodontal inflammation (BOP, alveolar bone deterioration, mobility) on the maxillary anterior region. Plaque samples, which have been collected monthly for two years, also suggested a periodontal profile. After an occlusal evaluation, she was also diagnosed with dento-prosthetic malocclusion due a distal functional precontact which was inducing an aseptic inflammatory reaction, especially on teeth 11 and 21. The treatment of choose consisted in a re-establishment of a harmonious occlusal equilibrium in maximum intercuspidation, followed by antero-maxillary SRP. After having removed the precontact, BOP and probing indexes showed a restored periodontal health together with plaque profile values (31).

# 12. Regarding literatures reviews

The most relevant outcomes and conclusions regarding the literatures review included can be found in Table 7 in the ANNEX and Discussion section.

# 13. Limitations

The several limitations regarding studies on humans are one of the reasons this subject is subjected to so many controversies. Different nature of the studies, lacking of a control groups, different numbers of participants are only some of the overall limitations For all the studies included, only compliant patients were part of inclusion criteria, and the intensity and frequency of dental appointments and oral hygiene were increased compared to the average, smokers and other deleterious habits were not proportionally realistic, the materials and modalities of treatment performs were also different according to the different studies and the different patient needs, measurements were performed by different clinicians, in some situations multiple measurements has been taken, and regarding inclusion criteria, even if resulted similar among the studies, they were not identical, making impossible any type of statical analysis between the various studies (26–31).

### DISCUSSION

### **14.** Splinting

Despite the numerous controversies regarding tooth splinting, and the lack of studies aiming to study the implications of splinting periodontally compromised teeth, the results obtained from Sonnenschein et al. 2017 and 2022 study (26,28) showed a significant survival rate of splinted teeth, with only two teeth lost after 7 and 12 years, due to endodontic complications during the treatment, out of the 162 total splinted teeth. Contrary, the study performed by Graetz et al. in 2019 (27) counted 26 teeth loss at T2 out of 227, but this study didn't include only mandibular anterior group like the first study did (148 teeth were actually splinted on the maxillary group). In fact, he noticed that non-anterior teeth presented a significant increased risk of tooth loss due to the higher occlusal forces exerting on them compared to anterior teeth, which was also associate with the level of probing depth and bone loss. This can also be seen from the number of repairs needed, as for Sonnenschein et al. the survival rate of splinting was 67% after 10 years, while for Graetz et al. was 24.7% at 11. A similar result regarding splint and tooth survival was found also by Zhang in 2023 (29), with 65.2% survival mandibular anterior splint and no tooth loss during the follow-up period. Regarding the splinting material, no associations have been found between composite resin only and composite resin + fiber glass, while Zhang noticed a significant increased risk of breaking for composite resin alone compared with composite resin + mesh grid.

Regarding periodontal parameters, a noticeable improvement has been detected for all the four studies: mean PPD decreased from 3.39mm to 2.04mm for the first studies of Sonnenschein et al., from 5.4mm to 3.2mm for the study of Graetz et al. (2019), for the second study of Sonnenschein et al., from 3.7mm (group A, splint before FMD) and 3.3mm (group B, splint after FMD) both decreased to 2.3mm, and for the 2022 study of Zhang et al. (2023) PPD decreased from 4.31mm to 2.93mm.

While the RBL indexes remained stable for Graets et al. and Zhang et al. studies before and after treatment, an improvement from 72% to 63% was obtained by Sonnenschein et al.; the reason behind this apparent improvement could be the limited number of periapical x rays available due the retrospective nature of this study (the x rays were available only for 10 out of 39 patients) and an overall better values at baseline of 61% and 34% for Graetz et al. and Zhang et al. combined with longer observation time.

Regarding the sequence of treatment (splinting before or after periodontal treatment) the study of Sonnenschein et al. showed an overall better outcome for patients who had receive splinting prior to periodontal treatment. Even though splinting teeth after periodontal treatment (PT) can be associated with an easier access to the subgingival area, an early stabilization of periodontal teeth, which will lead to decrease tooth mobility could help to achieve a more consistent blood clot formation subsequent to PT. Nevertheless, the higher proportion of diabetic patients in the group receiving splinting after periodontal therapy may have affected this result and perform firstly PT in some cases has led to great decrease in tooth mobility and therefore splinting was not anymore indicated.

Plaque accumulation and challenging oral hygiene for the patient are others concerning factors to consider prior to perform splinting, especially when treating advanced periodontal patient (26–29). Nonetheless, the studies of Zhang and Garez concluded that splinting was only very slightly affecting plaque accumulation and didn't find any relation between splinting / plaque indexes or periodontal destruction of adjacent teeth, While Sonnenschein et al. noticed that even though periodontal parameters, including plaque and bleeding indexes were decreased after 12 months, gingivitis and therefore BOP were still present, despite the significant improvement of PPD.

Regarding patient perception, all the studies reported a consistent recovery in terms of masticatory comfort after the collocation of the splint, and specifically, the study conducted by Sonnenschein et al. in 2022 reported a quicker improvement of oral health related quality

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of life (OHRQoL) for the group of patient who received splinting prior to PT, and the group who received splinting after PT settle at the same value after 12 months (28).

# 15. Occlusal adjustments

Regarding occlusal adjustments, Meynardi et al. in 2016 and 2018 performed two studies regarding the relation between malocclusion and plaque profile of patients undergoing periodontal disease (30,31). According to Meynardi, focusing on plaque composition before the appearance of the traditional signs and symptoms (gingival recession, bone loss, tooth mobility, inflammation...) can allow an early onset detection for periodontal and gingival disease.

In his 2016 study, he clearly demonstrated a better outcome in terms of bacteria profile, as a pathogenic bacteria profile was re-established starting from the third month after the traditional periodontal treatment (SRP) for the group which didn't undergone occlusal analysis and occlusal adjustment therapy, suggesting that sanitizing subgingival margin is not the only priority when dealing with periodontal disease (30).

As stated already, periodontium and alveolar bone are physiologically modulated mainly by occlusion, hence the outcome that malocclusion and traumatic occlusal forces may be responsible for vascular, cellular, and metabolic alteration at alveolar and surrounding tissue level, which if not treated properly, could lead to a further re-establishment of a pathogenic microbial profile (24).

Regarding Meynardi case study of 2018, he was able to treat an already established periodontal and gingival inflammation affecting the two maxillary central incisors of a patient, by mean of occlusal analysis and adjustments prior to periodontal therapy. Even though the plaque analysis showed a profile compatible with periodontal disease, the occlusal analysis performed showed a distal precontact displacement of tooth 24, causing an occlusal trauma on teeth 11 and 21 (31).

After one month from the removal of the distal precontact and the perform of SRP on the antero-superior sector, both periodontal and bacterial profile confirmed the disappearance of the periodontal and gingival inflammation.

Overall, both his studies concluded that management of occlusal disharmonies should be an integrated part of periodontal treatment and maintenance, as the non-removal of those can lead to a reestablishment of a pathogenic bacteria profile, despite successful periodontal treatment and improved oral hygiene, and that plaque sampling is a cheap and consistent way to not only diagnosis presence or absence of certain conditions, but to also monitor the effectiveness of adjunctive treatments, such as occlusal adjustments (30,31).

### **16.** Management

As stated already, the main etiological factor of periodontal disease is plaque (1,21,23,24,32,33).

The role of occlusion regarding the progression and evolution of periodontal disease however, is still under debate; even though some associations between TFO and periodontal disease progression was noticeable towards the numerous studies on animals and in vivo (32,33), a scientific agreement regarding their relation has not been found yet (1,33).

Tooth mobility is a common consequence of both, periodontal disease, and occlusal trauma. A tooth presenting mobility is characterized by a widened periodontium, and increased periodontal parameters compared to a non-mobile tooth. Even though the etiological causes are quite different (plaque accumulation for periodontitis, excessive occlusal forces for occlusal trauma) the manifestation of eithers conditions include inflammation of the periodontium, with consequent attachment loss and increased tooth mobility, which if untreated could lead to an even further worsening of the overall attachment level, and so on. Tooth mobility is also one very important indicator regarding the overall outcome for a periodontal therapy, as it was shown that teeth with less mobility shown a better outcome in terms of CAL compared to mobile teeth or even teeth undergoing regenerative procedures during SPT (33).

Detection of occlusal trauma, however, has its own limitations in terms of diagnosis, as it can be confirmed only histologically, hence, focussing on occlusal discrepancies, such as posterior protrusive contacts, prematurities in centric relation and non-working contact was an easier and more practical option. Even tough numerous inconsistencies and contradictory results have been obtained in terms of significance and statistical analysis, teeth showing signs of occlusal trauma (fremitus, widening of periodontal ligament, hypercementosis, secondary dentin invading the pulp chamber space) showed a worst periodontal profile both at baseline and at treatment done, with an increase in PPD, BL, and CAL (1,32,33).

As agreed by all the studies included, periodontal disease treatment should be aiming on plaque control and oral hygiene, and whenever signs of occlusal trauma, excessive mobility or uncomfortable mastication for the patient should be present, the best approach is to manage periodontitis and occlusal trauma as two distinct pathologies (1).

Management of occlusal trauma may include occlusal adjustments (coronoplasty), occlusal reconstruction, managing of parafunctional habits such as bruxism by mean of bite-planes, orthodontic intervention, and tooth extraction (1,21,23,24).

Coronoplasty consist in a selective reduction of occlusal anatomy in order to optimize the occlusal relationship between the opposing arches in centric and eccentric relation, in order to establish an harmonious occlusal scheme and remove or redirecting traumatic occlusal forces to decrease periodontal stress (24). It will provide trophic conditions for the whole masticatory system. The idea is to remove all the occlusal supracontacts by creating multiple and simultaneous new ones, so teeth will receive more homogeneously spread forces during functional movement, while also TMJ and muscles will be subject to less fatigue.

The steps can be resumed as (24):

- Elimination of retrusive prematurities
- Elimination of supracontacts interfering with posterior mandibular closing
- Optimization of intercuspal relation (by maximizing and simultaneous contacts)
- Elimination of posterior protrusive contacts to stabilize incisal relation
- Reduction of medio-trusive prematurities
- Depending on the occlusal scheme of election, elimination of latero-protrusive prematurities

Once the elimination of traumatic forces is established, a clinical re-evaluation of tooth mobility and tissue lost should be perform, and in case of persistent advanced mobility and patient discomfort during mastication, a good option to consider is to perform a splinting of the affected tooth/teeth (34), especially the anterior sector, as already shown by Graetz et al. posterior teeth splints are more incline to fail due the higher amount of masticatory forces (27). Plaque accumulation and difficult oral hygiene at the splint area remain a cause of concern, even though plaque indexes remain overall stables in most of the studies presented. Therefore, patient oral hygiene motivation and increase of frequency of dental appointments may be advisable.

Regarding parafunctional habits, such as bruxism, especially in case of active periodontal disease, the loss of tissue and attachment, hence the reduced adaptive capability of the tooth, may imply an increase of the strength and occurrence of traumatogenic occlusal forces onto the periodontium, resulting in an amplification of the damage and the mobility. In those situations, an occlusal appliance would be very beneficial for the patient, as a raised posterior tooth contact (by mean of the biteplate thickness) will facilitate anterior guidance and eccentric mandibular movements while leaving masticatory muscles and TMJ in a relaxed centric relation. Occlusal appliances will not eliminate bruxism itself but are a very effective way of protecting the masticatory apparatus by decreasing or eliminating certain pathogenic occlusal forces (12).

### CONCLUSIONS

All the previously carried out investigations concluded that excessive occlusal forces alone do not trigger the development of periodontal diseases. However, the periodontal ligament is a highly vascularized tissue, which allows the absorption of applied forces by hydrodynamic damping. Consequently, we can deduce that processes such as occlusion, which creates physical pressure on the teeth, leads to a complicated interaction between various biological molecules with distinct compositions and roles, as well as the diverse cells of the various tissue present in the periodontal support compartment. This implies that occlusion is the principal factor that triggers the homeostatic response within the periodontal supporting complex, and in situations where the periodontal ligament should be already compromised by an existing plaque related periodontitis, meaning a decrease in the overall adaptive capacity of the periodontium, it could lead to an increased periodontal inflammatory response, increasing tooth mobility, and in terms of homeostatic response, as a cascade reaction will activate osteoclastic activity and others proinflammatory cytokines, it could lead to a progressive alveolar bone and periodontal atrophy.

Even though a direct correlation between occlusal force and periodontal destruction could not be demonstrated, the benefits of achieving a non-pathogenic occlusion and a proper tooth stability were shown in terms of periodontal parameters, teeth longevity and patient comfort, suggesting that, beside the contradictory finds, can be beneficial for a better outcome of the periodontal treatment.

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# ANNEXES

**Table 1**: 2017 classification of periodontal disease according to the severity and complexity of

the case. (18)

| Periodontitis           | stage  | Stage I  | Stage II   | Stage III  | Stage IV  |
|-------------------------|--|--|--|--|---|
|                         | Interdental CAL at<br>site of greatest<br>loss | 1 to 2 mm  | 3 to 4 mm  | ≥5 mm  | ≥5 mm   |
| Severity                | Radiographic bone<br>loss                      | Coronal third<br>(<15%)  | Coronal third<br>(15% to 33%)                                    | Extending to mid-third of root and beyond  | Extending to mid-third of root and beyond   |
|                         | Tooth loss                                     | No tooth loss d  | ue to periodontitis  | Tooth loss due to<br>periodontitis<br>of ≤4 teeth  | Tooth loss due to periodontitis<br>of $\geq 5$ teeth  |
| Complexity              | Local  | Maximum probing<br>depth ≤4 mm<br>Mostly horizontal<br>bone loss | Maximum probing<br>depth ≤5 mm<br>Mostly horizontal<br>bone loss | In addition to stage II<br>complexity:<br>Probing depth ≥6 mm<br>Vertical bone loss ≥3 mm<br>Furcation involvement<br>Class II or III<br>Moderate ridge defect | In addition to stage III<br>complexity:<br>Need for complex rehabilitation<br>due to:<br>Masticatory dysfunction<br>Secondary occlusal trauma (tooth<br>mobility degree ≥2)<br>Severe ridge defect<br>Bite collapse, drifting, flaring<br>Less than 20 remaining teeth<br>(10 opposing pairs) |
| Extent and distribution | Add to stage as<br>descriptor                  | For each stage, desc   | ribe extent as localized   | (<30% of teeth involved), g  | eneralized, or molar/incisor pattern  |

# **Table 2**: 2017 classification of periodontal disease according to the progression rate expected

#### due clinical determinants. (18)

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

| <b>Table 3</b> : Overview of included studies regarding splinting of periodontally compromised teeth |                |          |                     |                           |
|--|----------------|----------|---------------------|---------------------------|
| Author   | Study type     | Patients | Objectives          | Outcomes                  |
| 2017,  | Retrospective  | n = 39   | Evaluate tooth      | Splinting mobile anterior |
| S. Sonnenschein  | Case study     |          | loss and stability  | teeth in conjunction      |
|  |                |          | of splinted         | with periodontal therapy  |
|  |                |          | anterior            | may improve tooth         |
|  |                |          | mandibular teeth    | survivability             |
| 2019,  | Cohort         | n = 57   | Assess              | Splinting periodontally   |
| C. Graetz  |                |          | survivability of    | compromised teeth         |
|  |                |          | splinted            | presenting high mobility  |
|  |                |          | periodontally       | on compliant patients     |
|  |                |          | compromised         | showed an increase in     |
|  |                |          | teeth               | tooth survivability       |
| 2022,  | Randomized     | n = 34   | Compare lower       | It was impossible to      |
| S. Sonnenschein  | clinical trial |          | anterior tooth      | determine the optimal     |
|  |                |          | splint efficacity   | timing for splinting of   |
|  |                |          | when done before    | mobile teeth during       |
|  |                |          | or after FMD        | systematic periodontal    |
|  |                |          |                     | therapy. Slightly better  |
|  |                |          |                     | result when done before   |
|  |                |          |                     | SRP                       |
| 2023,  | Retrospective  | n = 61   | Assessment of       | Long terms survivability  |
| Y. Zang  | Case study     |          | survivability and   | if combined with          |
|  |                |          | stability for lower | traditional periodontal   |
|  |                |          | anterior splinted   | therapy                   |
|  |                |          | periodontally       |                           |
|  |                |          | compromised         |                           |
|  |                |          | teeth               |                           |

| Table 4       |       |       |
|---------------|-------|-------|
| Group A       | (T1)  | (T2)  |
| CAL (overall) | 5.2mm | 4.2mm |
| CAL (33-43)   | 6.1mm | 4.9mm |
| PPD (overall) | 3.9mm | 2.5mm |
| PPD (33-43)   | 3.7mm | 2.3mm |
| BOP (overall) | 61%   | 22%   |
| BOP (33-43)   | 63%   | 28%   |
| PCR (overall) | 54    | 40    |
| PCR (33-43)   | 76    | 60    |
| GBI (overall) | 9     | 3.5   |
| GBI (33-43)   | 8.5   | 7.2   |

| Table 4 - 5: Evolution of analyzed parameters of group A (table 4) and group B (table 5) at the | ie |
|---|----|
| beginning (T1) and end of treatment (T2) (28)   |    |

| Table 5       |       |       |
|---------------|-------|-------|
| Group B       | (T1)  | (T2)  |
| CAL (overall) | 4.7mm | 4mm   |
| CAL (33-43)   | 5.4mm | 4.7mm |
| PPD (overall) | 3.6mm | 2.6mm |
| PPD (33-43)   | 3.3mm | 2.3mm |
| BOP (overall) | 53%   | 20%   |
| BOP (33-43)   | 66%   | 15%   |
| PCR (overall) | 48    | 27    |
| PCR (33-43)   | 66    | 57    |
| GBI (overall) | 15    | 3.4   |
| GBI (33-43)   | 16    | 2.7   |

| Table 6: Overview of included studies regarding occlusal adjustments on periodontally |            |          |               |                        |
|---|------------|----------|---------------|------------------------|
| compromised teeth   |            |          |               |                        |
| Author  | Study type | Patients | Intervention  | Outcomes               |
| 2016,   | Case       | n = 260  | G1 = SRP      | G2 bacteria microflora |
| F. Meynardi   | control    | G1 = 81  | G2 = SRP + OA | analysis showed        |
|   |            | G2 = 179 |               | consistent increase in |
|   |            |          |               | coccus content (non-   |
|   |            |          |               | pathogenic) than G1    |
| 2018,   | Case study | n = 1    | Perform       | OA led to a reduced    |
| F. Meynardi   |            |          | occlusal      | inflammation improving |
|   |            |          | analysis and  | the recovery time      |
|   |            |          | OA as an      |                        |
|   |            |          | adjunctive to |                        |
|   |            |          | SRP           |                        |

**Table 7**: Overview of included studies review regarding the role and correlation between occlusion and periodontitis

|                 |                | -                |  |
|-----------------|----------------|------------------|--|
| Author          | Methods        | Aim of study     | Main outcomes                          |
| 2016,           | Article review | Define role of   | Causa factor of PD is plaque,          |
| R. Sanadi       |                | occlusion in PD  | occlusal forces may be a cofactor      |
| 2017,           | Article review | Clarify role of  | Lack of studies clarifying TFO role    |
| D. Singh        |                | TFO regarding    | regarding attachment loss              |
|                 |                | PD               | Occlusal forces can induce changes     |
|                 |                |                  | in periodontium and bone               |
| 2018,           | Article review | Evaluate OT role | OT is not the initiating factor of PD. |
| J. Fan          |                | in development   | Small evidence that OT alters PD.      |
| (2017 Workshop) |                | and progression  | Reducing tooth mobility can            |
|                 |                | of PD            | improve periodontal therapy            |
| 2019,           | Article review | Clarify role of  | TFO may play a role in the apical      |
| E. Passanezi    |                | occlusion in     | spreading of plaque and                |
|                 |                | chronic          | inflammation, resulting in             |
|                 |                | periodontal      | attachment and bone loss               |
|                 |                | disease          |  |
| 2020,           | Article review | Investigate      | To achieve periodontal health,         |
| J. Mahendra     |                | effect of        | elimination of periodontal             |
|                 |                | traumatic        | inflammation and traumatic forces      |
|                 |                | occlusal forces  | is critical.                           |
|                 |                | over PD          |  |
| 2020,           | Article review | Emphasize the    | Traumatic occlusion in certain         |
| A. Nirola       |                | role of TFO as a | cases can exacerbate PD                |
|                 |                | risk factor      |  |
|                 |                | during           |  |
|                 |                | periodontal      |  |
|                 |                | treatments       |  |



