Chapter 36

Periodontal Changes and Oral Health

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1. Introduction

Periodontal disease is represented by inflammatory processes that affect the tooth's support / anchoring system and lead to tooth loss and negative effects on the oral health. Tooth loss and decreasing number of contacts between antagonist teeth was placed in relation to the educational level, marital status and incomes. Changes in periodontal status have been associated with oral factors and different systemic diseases.

2. Oral factors and periodontal changes

2.1. Caries, edentation and occlusal trauma

Maxillary represents a morphofunctional very complex entity, consisting of various components whose smooth work ensures performance of specific key like mastication, phonation, physiognomy and self-preservation, contributing in the same time, cooperation with other organs and tissues, to swallowing and respiration.

All physiological interrelationships of the components of the ensemble: the jaws including teeth with periodontal tissues, buccal mucosa, tongue, salivary glands, temporomandibular joint, neuromuscular complex and the veins that irrigate the functional territory providing nutrition, are directed by the central nervous system [1,2]. This dependence is due to the permanently received information by cortex from the entire reception network of maxillary. It satisfies the balance and health of each organ this way, in which the occlusion coordinates integrates.

It is acknowledged that any pain occurred in the maxillary area generated by a traumatogenic occlusion occurs not only locally, but also away from the mouth. Any alteration occurred in one of the components of the maxillary (under the action of certain triggers cumulated with the favoring ones), happens on a common ground: occlusion. On the other hand, disorganisation at the dental arches level, with a disruption of movement, can affect one of the elements of the same assembly, occurring dysfunctional disorders.

As teeth are dependent on their supporting tissues which keep them in the pockets of the maxillary bones, the periodontal complex depends on the activity of dental arches, normal occlusal function leading to a morphofunctional mechanical stimulation that manages the responsible biological mechanisms for the proper integrity of the periodontium [3].

The analysis of functional occlusion should be used as the basis for all conservative dental treatment, periodontal, prosthetic and orthodontic surgery.

Due to the functional differentiation structure of periodontal tissues and topographical situation of the two components (shell and supportive) defenses and resistance against aggressive risk factors direct or indirect is special, being conditioned by their character. On periodontal coating will act primarily micro-irritation local agents and at the periodontal support component factors of functional order, resulting dysfunction appearance. Risk factors that contribute to the disturbance or alteration action of periodontal can be classified into three big categories:

i. General favorable factors with dysmetabolic character that alters nutrition of the body, including the marginal periodontium.

ii. Local disturbing or precipitating factors that can be very divers, responsible for local trophicity change of periodontium.

iii. Aggravating regional factors that alter regional trophicity due to the presence of mandibular dysfunction caused by premature occlusal contacts and interference.

Studies performed on occlusal function and dysfunction shows that traumatic dental pain can be caused by traumatic injury to periodontal, cracks or fractures of teeth vital and a change of direction forces acting on periodontal dental units [1].

The occlusion was defined as the ratio of static contact between the two arches, regardless of the position occupied by the jaw to the cranium, unlike the interdental articulation, which requires dynamic contact of the dental arch.

Dynamic occlusal reports are made by the two arches during the performance of the stomatognathic system and during parafunctions. Occlusion is considered one of the three determinants of mandibular dynamics. In the same time, occlusion shows a anterior determinant (the front arches) and a posterior determinant (the arch side), between which there is a balance and interaction summarized in the context of mutual protection. Under this concept between the two determinants of occlusion there is a mutual protection, acting on static and dynamic phases of occlusion [4].

Dental occlusion is meant to stabilize the mandible position to the skull, participating in the development of systemic functions. Occlusal disorders occur as a result of dental anomalies of number, volume, position, dental crown injuries, dental migrations, edentulous, change of occlusion parameters, and secondary musculoskeletal joint dysfunction. Clinically it manifests as premature contacts (characterized by occlusion static phases), occlusal interference (in mandibular dynamic with dental contact), localized abrasion (at a tooth or dental group that takes over occlusal) or generalized to the whole arch.

Occlusal contacts can occur in the static and dynamic positions the jaw. Any occlusal contact that prevents uniform coaptation of support areas and occlusal contact points is called premature occlusal contact. Occlusal contact occurs early in the static occlusion (at the end of the terminal occlusion trajectory), or in the dynamic occlusion when the path of the jaw movement interferes with the dental contact.

Premature contact is always traumatizing for stomatognathic system elements. Traumatogenic capacity of a contact point depends on several factors such as the point of contact location, the size of the contact point, the state of the contact surfaces [5].

This way, if the point of contact is on a bigger surface, the friction force increases with it, and its pathogen potential. A reduced in size contact point, but between two rough surfaces can be as traumatic or even more than one big point on a polished surface, due to the high friction coefficient. Contacts may be multiple and symmetrical, while maintaining the mandible in a position close to or almost identical to the centric relation or intercuspation position without deviations above it, the rear or side. This rarely happens because the presence of small occlusal contacts creates what is called an occlusal instability. Clinical evaluation of static and dynamic occlusion cannot be made without registration cranio-mandibular relations. Within the tendency to establish maximum intercuspation contact (in patients with long-centric) and centric occlusion (in patients with point-centric), jaw moves from rest position, rising to the jaw with the action of high muscles [4].

Dynamic occlusion analysis is performed through a test movement printed to the jaw, and also during mastication, phonation and deglutition movements. The analysis of the test movements (retrusion, protrusion, left and right laterality) often reveals the presence of occlusion blockages or of some traumatic sliding slopes. The retrusion movement performed between maximum intercuspation and centric relation can be blocked by some premature contacts, thus preventing the mandible excursion to centric relation during deglutition. The protrusion can register early contacts in the lateral area which would prevent the previous guidance of the occlusion on the retro incisive slope. The deeper is the occlusion, the larger is the trajectory of the protrusion. The premature contact points of the previous area in the protrusion movement prevent balanced contact of the whole frontal group in the guide movement, creating an overload of the teeth which keep the contact [6].

The test movements with left and right side orientation can reveal an inequality of the trajectories due to movement blocking through occlusal obstacles or due to their different orientation depending on the inclined planes which produce them. In the laterality movement there is recorded the most intense traumatogenic activity at the level of the interferences that

may occur on the inactive or swing area, by turning the mandibular into lower grade leverage, therefore, more traumatic. In many cases, laterality or protrusion movement also causes a slight mobilization of the teeth which are in premature contact [4].

Occlusal force action on periodontal unit depends on the intensity, duration, direction of force, and the effects are also influenced by the state of tissue over which the force acts. The periodontal occlusal trauma is the degenerative injury that occurs when the occlusal forces exceed the adaptive capacity of the supporting tissue. Given that the dento-dental gearing is a cusp – fossa kind, the efforts leading is made in the long axis of the teeth. In normal circumstances, this condition is achieved by the way in which the teeth are implanted in the dental alveoli since their arcade eruption and through the manner in which are realized the dento-dental contacts in centric relation and maximum intercuspation, moments in which the masticatory pressures are maximum. In the teeth and periodontal tissue normal function, a particular importance has the correct position of dental organs, this being possible due to the presence of the balance between the multiple factors that are interrelated (presence of dental arcade integrity, the nature of the existing relations between adjacent teeth through contact areas and their character, dental morphology and cusps inclination, physiological mesial migration of teeth, physiological abrasion of teeth and their axial tilt, biological resistance of the healthy periodontium, lips, cheeks and tongue tone.

In the moment of integrity loss of dental arches, there occur multiple changes not only in the expense of odonto-periodontal units, but also for other components of the stomatognathic system, which demonstrates once again the present physiological interrelations between constituents of the complex.

Closely related to biomechanical homeostasis specific of the periodontium, the whole structural complex of the dento-maxillary system is conditioned by a series of morphological and functional elements that fit in the principle of inclined planes, among which the most important ones would be:

- Maximum and proximal vestibule-oral convexities of teeth ensure self-defense and self-stimulation of marginal periodontium through its protection against micro traumas that occur during mastication.
- The specific morphology of the frontal inferior teeth, vestibule-oral flattened in the third incisal and mesial-distal cervical narrowed to align them in a circle, defends the periodontium from pressures coming from the vestibule to the oral.
- Teeth roots' number and conical shape avoid the uneven application of the cementum-alveolar walls.
- Oral tilt of lower molars and vestibular tilt of the upper ones direct the masticatory pressures in the axial direction of these teeth while concentrating inwards massive facial.

When masticatory pressures are routed and transmitted in the long axis of the teeth, the ligaments that form the periodontal membrane are not crushed, but they act almost entirely, and are subject to forces with functional direction that determine their uniform extent, in functional limits with trophic effect on alveolar bone. After force's cessation, ligaments return

to their spiral resting shape [7]. Taking into account that masticatory pressures are not permanent, periodontal ligaments are submitted to a real functional gymnastics, functional tasks interspersed with periods of rest, which stimulates periodontium, periodontal membrane and alveolar bone, and keeps it in normal parameters.

Local causes of traumatic occlusion with its negative consequences on the dento-maxillary are varied:

- Untreated caries, besides pulpal and periapical complications, they may also lead to occlusal disharmony and dysfunction by horizontal migration of proximal caries teeth or of their neighbors disturbing occlusal curves, vertical migration of antagonist teeth, of one tooth with occlusal caries or which considerably reduced the height of its crown, tipping of neighboring or antagonist caries teeth.

Through the loss of interdental contact point due to proximal caries, the fibrous foods can directly damage the marginal periodontium, which leads to periodontal damage and possible installation of a secondary occlusal trauma.

- The edentation without prosthesis acts by cancelling the dental arches continuity, due to loss of contact points, interrupting the continuity of the over alveoli ligaments system that normally form a connecting strap between teeth.

Also, the horizontal migration of teeth which border the edentulous breaches makes possible the spaces appearance between teeth and traumatic food impact of interdental gums papillae.

- Dental iatrogenic is often represented by inadequate fillings, inappropriate prosthetic marginal axial or transversal or which does not restore correctly proximal contact surfaces or natural convexities vestibule-oral and /or determine at the level of soft tissue rejection reactions, caused by prosthetic material.

The erroneous occlusal articular balancing compromised by grinding the occlusal stops and slopes guide altering vertical occlusal dimension and interdental space also produce occlusal dysfunction.

- Alteration of the morphology of dental crowns by pathological abrasion produces a broadening of the occlusal plane, constituting a cause of overload of odonto-periodontal units by masticatory forces.

- Primary malposition of some permanent teeth which erupt vicious in the three spatial planes. In this category, there are included anomalies of position: infra, over, predental, retrodental, vestibular or oral of some teeth, different combinations of malposition: mesio-vestibular position and distovestibular position.

- Isolated dental anomalies of form and volume can be generating occlusal interference by inconsistencies that appear in reports to other normally developed teeth. It produces a change in the position of teeth, bone implant base change, changing cuspid plans finally affecting occlusion reports.

- Occlusal vicious habits and tics are multiple and a source of risk factors for a dysfunctional maxillary. The most common harmful habits are: onychophagy (nail biting), biting objects (pipe, rubber, glasses frames), the practice of keeping and tighten between teeth, needle, nails, pencil, while working.
- Dentoalveolar fractures or maxillary bones may lead to dysfunctional occlusal interference. When the traumatic accident caused significant displacement of bone fragments of jaw, they rarely can be reduced in such a manner that there will be no significant occlusal changes after consolidation.
- Multiple parafunctions are the most common sources of occlusal dysfunction, bruxism holding priority. It is conditioned by the existence of occlusal disharmonies caused by premature dental contacts and occlusal interferences with an important role for multi-causal dysfunctional factors of the maxillary.

With a change of direction of force, normal pressure of normal muscle contractions become traumatic for periodontal membrane crushed between the tooth and alveolar wall it has no irrigation and normal metabolism anymore, and on the other hand, not all ligaments take functional tasks. Besides periodontal membrane suffering appears a harmful effect on alveolus: pressure causes bone lysis. Bone lysis always occurs in the way the force that causes pressure on the bone acts, which results in a stronger inclination of the tooth, like this appears traumatic periodontal conditions. By tilting the teeth it can escape the occlusal pressures making even dental contact to disappear; periodontal pain does not disappear with the disappearance of dental tooth contact as lack of stimuli periodontium undergoes hyaline degeneration of hypofunction [8]. Following these considerations to set the concept of primary occlusal trauma which means the harmful effect of occlusal forces on initial healthy periodontal when the direction, intensity or duration of occlusal force are beyond functional parameters: direction outside the long axis of the tooth, too long time, too much intensity. In this context, it should be emphasized that most studies are in agreement that the primary occlusal trauma (in the absence of superimposed etiologic factors, inflammatory, degenerative-dystrophic) does not causes periodontal disease but isolated periodontal lesions. Experience has shown that when the periodontium is weakened, initial periodontal suffering having other causes than occlusal, occlusal requests, even with optimal direction, the long axis of the teeth, even if they are intermittent, or even if the intensity normal, all lead to a periodontal trauma [9,10,11]. In this case, it is a secondary occlusal trauma in which the occlusal forces act on a previously weakened tooth periodontium. Obviously for already weaken teeth periodontium faulty forces within that direction, intensity or duration, have bad effects. Great difficulty occurs when, after periodontal is affected by an occlusal trauma, inflammatory component is superimposed, because at the moment it is hard to tell whether it is a primary or secondary occlusal trauma.

We can describe the three stages of occlusal trauma: stage of aggression, stage of repair and periodontal adaptation stage. During the stage of aggression collagen and osteogenic activity is inhibited, so that when the injury is not too strong to stimulate repair possibilities. If the trauma is not excessive, overcoming repair potential has serious periodontal consequences. If trauma is not excessive, it can reach the third stage, the periodontal adaptation. [12].

Disorders at the level of occlusive parameters characterized by shortening and cutting of occlusal areas, their artificial or mixed incorrectly realization, discontinuities, incorrect reconstructions of retroincisal slope, changes in the integrity and shape of support and guidance cusps, altered occlusion curves, uneven occlusal plane, are important factors of occlusal dysfunction, resulting in changes in jaw dynamic patterns, with muscle and joint response, taking into account the role of the dental determinant in achieving mandibular dynamics.

It is worth noting that the teeth in occlusal trauma, especially those with pathological abrasion, fractures may occur in low varnish areas, which can go up to an aspect of,, shelling " of dental crown [4].

True cuneiform lesions (mylolysis) are missing carious dentin being located strictly in the varnish. These are considered by many authors as pathognomonic lesions for teeth in occlusal trauma [13,14]. These lesions with lack of dental hard tissues are located on the vestibular side. The section looks like an obtuse angle open to the mouth vestibule. The lesion affects hard coronary tissues but extends to the root cementum, in the same time with marginal periodontal retraction [5,15,16]. The color of the cuneiform lesions walls is slightly modified and they have a hard consistency, heat sensitivity or chemical is inconsistent and injury has a slow progress. If you are creating a five grade cavity lesion evolves rapidly while getting the characteristics of dental cavity. Occlusal obstacles and / or occlusal parafunctions often cause appearance of pathological abrasion [13]. It should be clinically noted how the abrasion is dependent on other factors. It is demonstrates that the patient's age, degree of abrasion of tooth of specific subject, the presence of eccentric abrasion (which betrays occlusion function) are factors that cause pathological abrasion [4].

Studies show that reducing the masticatory field by edentation accelerates abrasion. Local hyperacidity (by diet or acid regurgitation) can lead to erosion (as opposed to abrasion) [17]. Presence of enamel dystrophy and dysplasia, in one word the quality of dental hard tissues is an important factor that causes tooth wear [18].Another extremely important factor that can cause tooth wear is the abrasive capacity of prosthetic restoration materials.Isolated clinical examination makes it virtually impossible to determine the rate of pathological abrasion. Therefore it is prudent that in such cases to make exploratory therapeutic methods (selective grinding, temporary dentures) before major restaurateurs interventions. In this way, the dentist is able to identify more precisely the primary determinant of pathological abrasion and abrasion evolution speed.General pathological abrasion-is the abrasion inconsistent with biological age. The generalized pathological abrasion is a major sign of dysfunctional occlusal [18,19].

Periodontal pockets do not occur in primary occlusal trauma, but usually in secondary, on a periodontium already affected in the presence of infectious and local irritative factors. As long as the inflammation is limited to the gum, it is not aggravated by traumatic occlusal forces, but when the inflammatory process spreads to desmodontium own tissue, the occlusal trauma becomes a co-destruction factor of support structures, protecting the periodontal pockets of bones. A periodontal pockets is pathological deepening of the gingivodental fosse which is

formed gradually, resulting the destruction of tooth support tissue and its mobility, finally leading to its expulsion of [19].

Destructive alveolar processes represent another consequence of the occlusal trauma phenomenon. Alveolar bone, despite of its appearance rigidity, is less stable than periodontal tissue, as is continuous-changing structure by obvious resorption phenomenon in the pressure area and by apposition ones manifested within traction territory. In the case of occlusal trauma, the destructive effect on alveolar bone is directly proportional to the overload degree, their frequency and duration being inversely proportional to the resistance of the tissue. On such a field, under the action of repeated occlusion constraints, the negative effects of occlusal trauma occur more easily, periodontal disease having a fast and serious evolution. In conjunction of any occlusal trauma caused by bruxism amid a normal gum, first the bone destruction presents the characteristic of an aseptic process, lytic, of some areas that cannot be radiologically detected yet. In later stages, due to parafunction persistence, destructive phenomena complicate, the blood nutrition being even more deficient, due to prolonged action of pressure forces, amid local irritations (tartar and plaque) will contribute to the failure of the epithelial barrier to invasion of microorganisms and toxins. The existing bone bags, along with the gum ones installed will progress simultaneously, adding also the gingival retraction [20]. Another result of the occlusion dysfunction is represented by the opening of the interproximal contacts. The consequence of periodontal changes caused by occlusal trauma is represented by dental mobility, dental migrations, and gingivorragia. Mobility is due to an occlusal trauma exerted on that tooth, the tooth receiving abnormal forces which pressure it during protrusion and laterotrusion movements.

The more frequently there are affected the monoradiculars that are subjected to occlusal trauma producing bone lysis in the support periodontium level. Because of dental mobility is difficult to detect when occlusal trauma occurs, requiring consideration of occlusion, both in centric relation and maximum intercuspation and also in protrusion and laterotrusion movements. Pathological tooth migration is a phenomenon that occurs due to poor periodontal structure, exacerbating existing traumatic occlusion with more pronounced effect of paraxial transmission of masticatory forces so harmful to the entire dento-maxillary system. Changing the position of one or more teeth causes 'contact rupture' between them, creating spaces (trema, diastema) favoring mechanical injury of epithelial insertion with papilla inflammation often accompanied by bleeding. Implantation of the pluriradiculars is more favorable for the capacity of trauma resistance when the roots are divergent. In fact, all aspects mentioned above influence the capacity of occlusal trauma resistance, making a normal request to appear as supraliminal, emphasizing the traumatogenic character of occlusal forces [3, 21].

Coating or superficial periodontium injuries take various clinical forms depending on the intensity, duration and direction on which occlusal trauma manifests. Occlusal trauma can cause a progressive denudation of teeth roots, characterized by moving the gum to the tooth apex. There are two sets of gum retractions: one which is detected on physical examination, another one hidden, and a part of the root being covered by the inflamed wall of a periodontal pocket. It should be noted that gingival retraction may involve all insertion area from the level

of dental package, or only partially. The most common areas are the vestibular and oral of one tooth, of a group of teeth or even of a complete dental arch.

Traumatic dental hygiene habits can worsen the gingival recessions at the level of vestibular teeth face, this being associated with the occurrence and emphasis of cuneiform injuries, which is a pathognomonic sign that the tooth / teeth in question are in occlusal trauma [22].

Occlusal trauma causes and aggravates the gingival retraction, thus accelerating the initial epithelial proliferation by a local irritation, clinical form known as Mc Call's garlands or festoons. It also can reveal injuries as cracks (Stillman's fissures). These identities are pathological bag bottoms in which the ulcerative process developed, they could spontaneously cicatrize or persist in the form of deep fissures with rolled edges [23, 24, 25]. Papilla and gingivitis occurring as a result of opening the cervical interproximal space arise as a consequence of the loss of dental contact points in the presence of partial edentation which are accompanied by migrations, tipping or translations of the limiting dental units to edentulous breaches. The opening of interproximal space allows food particles penetration, thus injuring the gingival papillae.

Local examination reveals the presence of gingival inflammation that may be associated with bleeding. In advanced stages there is a junction of the vestibular and oral gums, accompanied by a slight extrusion of the affected tooth. Interradicular space dissection is characterized by roots denudation, gingival epithelium covering the limbus bone top retreating. Reaching bifurcation or trifurcation root is generally due to deepening of vestibular gingivodental or oral channel [26, 27].

Any indiscriminate therapeutic act in terms of ignorance or underestimation the capacity features and adaptive limit capacity, respectively of teeth defense, periodontium, temporomandibular joint, jaw bones, neuromuscular and vascular complexes, is likely to confuse the morphofunctional balance of dento-maxillary system, thus prejudicing the treated subject through iatrogenesis.

2.2. Malocclusions

The interrelation between the periodontal health status, the presence of dento-maxillary anomalies and the orthodontic treatment remains a controversial issue in the literature [28], reflected in the great diversity of the findings of studies that address this issue. Some researchers promote the idea that the presence of dento-maxillary anomalies is a risk factor in the development of periodontal pathology: [29-34];

The dento-maxillary anomalies may represent a risk factor in producing chronic marginal periodontitis as they maintain the periodontal inflammation, while changing the intensity and direction of occlusal forces. Other periodontal changes as insufficient attached gingiva width and low height of the alveolar bone were also observed and associated with the presence of dento-maxillary anomalies in general, or a single misaligned teeth [35, 36], as well as people with evident dento-maxillary anomalies were discovered to whom periodontal changes were minimal or nonexistent [37].

2.2.1. Dento-alveolar disharmony (DAD) with crowding

They are a risk factor for the presence of septic inflammation, because due to the disparity between mesial-distal sizes of permanent teeth and corresponding alveolar arches' perimeter, various dental malposition occur, localized mainly in incisor-canine region (Figure 1), which causes retention of food debris and plaque, and difficulty in removing them by self-cleaning or artificial cleaning [38]. This correlation is weaker in the maxilla compared to the mandible [39].

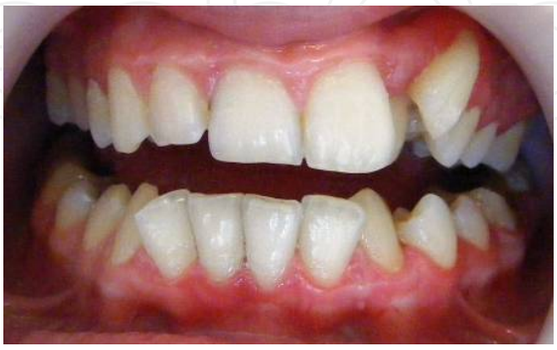

Figure 1. Gingival inflammation with papillae hypertrophy in lower and upper incisors, thin periodontium at 23 (eruption in buccal position) in a patient with dental crowding

The fact that malocclusion with crowding is a risk factor in the development of periodontal pathology is supported by studies that have reported the existence of a strong correlation between the presence of this anomaly and the occurrence of periodontal pockets, [40]; [41]; or the reduction of alveolar bone[42].

Anatomical conditions specific to this anomaly are unfavorable because interdental septa are thin, interdental papillae are laminated, with low volume and with poor blood circulation, unfavorable for a good gingival-periodontal nutrition [43].

2.2.2. Dento-alveolar disharmony (DAD) with spacing

DAD cause periodontal adaptive phenomena such as: hiperkeratinized epithelium, gingival chorion fibrosis, flattening dental papilla (which become a plateau or even concave aspect) (Figure 2). The presence of this anomaly may favor direct trauma on interdental papillae by food fragments.

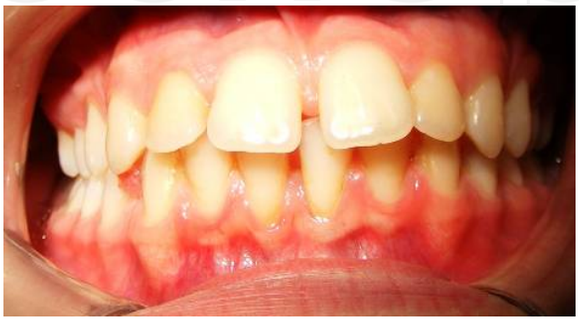

Figure 2. Gingival retractions with a thin periodontium at 31, 41, aplatized papillae between 11 and 21

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Many specialized studies could not establish any positive correlation between incongruence with spacing and periodontal parameters in conditions of a rigorous hygiene, so that there are authors who consider that the indication for closure of interdental spaces is aesthetic rather than for periodontal dental health maintaining. [39]

2.2.3. Open bite

In the anterior open bite teeth are not functionaly requested during mastication (missing the food cut), and the self-cleaning phenomenon is absent favoring installation of gingival inflammation and hyperplastic changes. In contrast, lateral teeth are in occlusal contact and they are overworked during masticatory effort, they being almost in a state of permanent occlusal trauma due to the transfer of mandibular movements' previous guide of the lateral teeth [44].

We thus witness the periodontal space widening, gingival retraction emergence and horizontal bone atrophy of these teeth.

According to Macht and Zubery [45], in this syndrome we are witnessing a significant increase in the gingival inflammation, consequence of the enhancing virulence of the dehydrated plaque (due to lack of labial competence), and an increase in the length of the clinical crowns of incisors, which may suggest that open bite predisposes patients to the development of gingival retractions localized in the incisor segment (Figure 3).

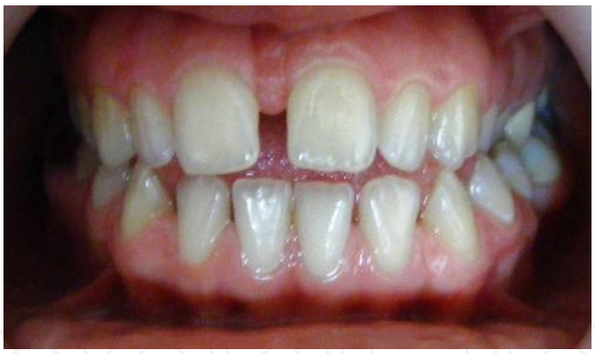


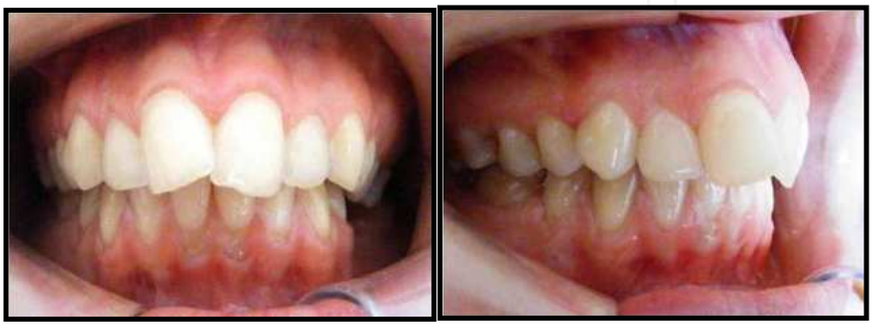
Figure 3. Open bite in a 19 years patient-thin periodontium predisposed at gingival recessions

2.2.4. Deep bite (class II division 2 malocclusion)

In the deep bite syndrome, anterior teeth don't have stable occlusal stops, and their implantation remains normal just as long as inflammation isn't installed due to the presence of plaque. When gingival-periodontal injuries of microbial cause occur, the anterior teeth implantation degrades, it begins a process of accelerated active eruption of the anterior inferior teeth, with the possibility of their direct trauma to the incisive upper periodontium [43], and the progression of periodontal lesions. Deep bite syndrome will lead in these conditions to increased periodontal pocket depth and marginal gingival retractions appearance [39]

2.2.5. Overbite (class II division 1 malocclusions)

Due to inocclusion lips and upper lip hypotrophy, bacterial accumulation occurs in the anterior dental area, the immunological role of saliva is reduced, and on long-term increases the frequency of periodontal lesions [46]. Similar to open bite, in the anterior dental regions, a fragile periodontal can be structured (Figure 4), prone to periodontal lesions because of unstable interdental contacts. The same fragile periodontium can be observed in the side areas, due to unilateral or bilateral crossbite (consequences of a different degree of compression of the two jaws).



(a) (b)

Figure 4. Fragile periodontium with gingival recessions in lower incisors (a-frontal b-lateral view) in a patient with maxillary compression

There is no concordance of views regarding correlations between sagittal inocclusion (overjet) and periodontal parameters. Authors such as Davies et al. 1991, [47] or Geiger et al., 1976, [48] support the existence of a significant correlation between plaque index, periodontal diseases and severe anterior overjet (> 6 mm), while Buckley, 1981 [49] considers that there is no significant correlation between the presence of overjet and plaque index, or gingival inflammation index.

According to Torres et al., 2006, [50] an increase in the plaque index occurs only in subjects with sagittal inocclusion > 6, and after Bjornaas et al. 1994 [51], to adulthood, in the presence of a severe sagittal inocclusion (overjet ≥8mm), there is a reduction of alveolar bone level with ≈0,96mm in the upper anterior area, and with ≈0,35 mm in the lower area.

2.2.6. Mandibular prognathism (class III malocclusions)

In mandibular prognathism, the lingual pressure (of the protrude tongue and low positioned) continuously exercised on the lower incisors' lingual face and the occlusal trauma due to anterior crossbite can lead to important vestibulo-version of the mandibular incisors with fine periodontal biotypes inducing and the presence of a very thin vestibular cortical bone, located away from the cemento-enamel junction.

Transferring anterior guide on the lateral teeth, found in this group of anomalies, leads to the lateral dental area overloading concurrent to a less loading of the front area (no food incision)

[44]. Therefore we can expect the emergence of periodontal changes like horizontal bone atrophy and epithelial insertion's descent [52].

Periodontal changes occur early in reverse gear and consist of the occurrence of significant gingival retraction of the lower incisors' vestibular face ("disposal trench"), possibly a tooth mobility, following a permanent occlusal trauma.

These periodontal changes can regress spontaneously if orthodontic treatment is instituted early [53].

There is no uniformity of opinion or about the association between anterior crossbite with different periodontal parameters. Ngom et al. researches 2005, [39] have reported the presence of a significant correlation between anterior crossbite and the percentage of gingival retraction, but not with the plaque index and the gingival pockets depth, while Hashim and Al-Jasser's researches, 1996, [54] have found a significant correlation between crossbite, the plaque index and the periodontal pocket depth. The difference between the two studies may be due to differences in age and dental hygiene of the subjects investigated. Silness and Roynstrand, 1984, [55] opines that the crossbite teeth show more frequently signs of periodontal disease compared to those dealing a normal occlusion.

2.2.7. Congenital malformations of the lip, maxilla and palate(clefts)

Next to specific anatomical defects, the delays in the formation and timing of tooth eruption, the need for long orthodontic treatment [56, 57] and the presence of prosthetic restorations are factors contributing to the reduction of the alveolar bone level in areas adjacent to dehiscence [58].

Multiple dental malposition, segmental alveolar gaps, soft tissue folds made before palatoplasty, the presence of scar tissue or oro-nasal communications persisting after surgical closure of the defect, make oral hygiene maintenance a difficult task, increasing risk and progression of periodontal disease [59, 60, 61].

Comparing the periodontium from patients with cleft lip and cleft palate, to the one from patients with cleft palate only, Gaggl et al., 1999, [62 found that the first have a predisposition to deep periodontal destructions in the teeth adjacent to the splicing area, while in patients that only have cleft palate, clinical peridental appearance may be similar to that of subjects without malformations. However, Dewinter and Quirynen state that the periodontium of the teeth from the splicing zone or near it, in patients with unilateral cleft, can cope relatively well to a long orthodontic treatment or to a combined periodontal-orthodontic treatment [63].

2.3. Orthodontic treatment

The three main reasons justifying the need for orthodontic treatment are: to improve facial and dental aesthetics, oro-dental health and the normal oral functions [36].

In the absence of periodontal diseases and in the presence of a proper oral hygiene, a well led orthodontic treatment should not have, on long-term, significant effects on periodontal

supportive structures. According to Graber et al., 2005, [64] it is possible to occur a decrease in the alveolar bone's volume and height, as an adaptive process to the trauma.

The main clinical periodontal effects that can be seen in the oral cavity after insertion of orthodontic appliances are: gingival hyperplasia, marginal gingival retraction, irreversible loss of bone support and excessive fibrous tissue that prevents complete closure of the post extraction spaces [65].

2.3.1. Gingival overgrowth (hypertrophy and hyperplasia)

A periodontal change frequently observed during orthodontic treatment, especially with fixed appliances, is the emergence of gingival overgrowth [66].

Scope, they can be localized or generalized, but seem to be more common in mandibular incisors region (67, 68) (Figure 5).

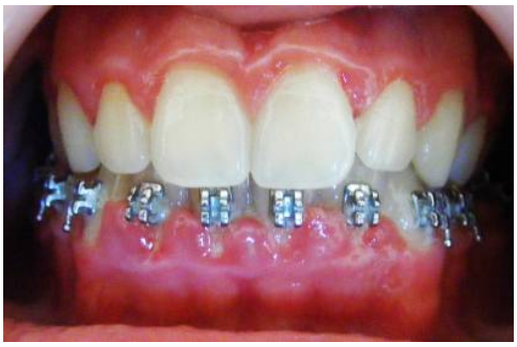


Figure 5. Gingival overgrowth with inflammation and plaque accumulation in the lower incisors

Other authors (69) believe that overgrowths may be marginal, diffuse, papillary, or discrete and have four degrees of severity:

- 0-no gingival overgrowth;
- I – gingival overgrowth extended only to the dental papilla;
- II-gingival overgrowth covering the papilla and marginal gingiva;
- III-gingival overgrowth covers three quarters or more of the dental crown.

Since gingival overgrowth is a factor limiting or preventing orthodontic tooth movement, it often requires its removal by gingivectomy, which removes all fibrous tissues around the tooth and at the same time allows gum's reshaping or remodeling [69]. After gingivectomy, periodontal condition is improving, so the orthodontic mechanic's normal course is possible. If it does not prevent the effectiveness of orthodontic treatment and causes no discomfort to the patient, gum volume enlargement can be removed after the completion of orthodontic treatment, if it does not regress spontaneously.

2.3.2. Marginal gingival retractions and losses of bone support

Sometimes, the incidence of gingival recessions in patients with a fixed orthodontic appliance can be up to 10% [70]. In addition, repeated trauma on marginal gingiva by teeth movements and plaque accumulation, inherent with the application of orthodontic appliances, can lead to the formation of marginal gingival retractions. Moreover, mucogingival problems prior to the initiation of orthodontic treatment could be exacerbated by the application of orthodontic force [70]. It seems that the lower incisors are the teeth most likely to develop marginal gingival retractions, the mechanism of their occurrence being the excessive force applying, which does not allow bone's repairing or remodeling during teeth movement with the existence of a thin or non-existent vestibular cortical and an inadequate or absent keratinized gum [40].

2.3.3. Assesment of periodontal changes using immunological analysis in Gingival Crevicular Fluid (GCF)

During the initial phase of orthodontic treatment, orthodontic forces induce a response to the mechanical stress from periodontium and a net of events it is produced: angiogenesis, aseptic inflammation and periodontal remodeling [71].

Gingival crevicular fluid (GCF) is used to determine the presence and levels of biomarkers expressed during the first phase of orthodontic treatment, this cascade of substances comprising cytokines, metalloproteinases and other mediators of complex transformations in the periodontium [72, 73].

In studies funded by the grant ID573/2008 of the Ministry of Education and Research of Romania, we measured the levels of Pentraxin-3 (PTX3), Thrombospondin1 (TSP1), Lipocalin2/Matrix metalloproteinase 9 (MMP9/NGAL) complex and Matrix metalloproteinase 9 (MMP9) in GCF at different time points of the first 2 weeks of orthodontic treatment, to determine the relationship between these values and theirs implication in inflammation and angiogenesis balance, in the situation of a good control of the bacterial plaque [74, 75].

GCF samples were collected from orthodontic patients requiring upper canine distalization with first premolar extraction. For the orthodontic appliance, there are placed brackets Roth 0.018 inch (GAC Intl, Bohemia, USA) with 0.012 inch NiTi archwire (GAC Intl, Bohemia, USA) and a laceback made from 0.010 inch stainless wire, placed and activated 21 days after the premolar extraction.

Using the statistical analysis, our results show a change in time of PTX3, TSP1, MMP9/NGAL and MMP9 levels in GCF of patients with this method of orthodontic treatment and suggest their stronger involvement in inflammation and angiogenesis processes in PDL during orthodontic periodontal remodeling, in the situation of a healthy periodontium and a good control of the bacterial plaque.

2.3.4. Assessment of periodontal changes using immunohistochemical analysis of gingival tissue

The gingival overgrowth as a reaction of the orthodontic treatment was longtime considered by the clinicians as an inflammatory result of the retention of the bacterial plaque by the

orthodontic devices. Clinical observations showed that the gingival overgrowth appear also in patients with good oral hygiene, without any clinical signs of gingival inflammation.

Our studies [76,77] in the grant ID573/2008 funded by the Ministry of Education and Research in Romania showed that the gingival overgrowth during the fixed orthodontic treatment appears at the beginning, without any inflammatory signs, as a result of the mechanical stress and periodontal remodeling during the orthodontic movement, the MMP8 and MMP9 acting as indicators of this situation.The inflammation of gingiva occurs as a consequence of the accumulation of the bacterial plaque favorized by the orthodontic devices.

Gingivectomy was performed in patient with gingival overgrowth in the first eight weeks of the orthodontic treatment and the material obtained was used for histologic and immunohistochemical study.

3. Systemic factors and periodontal changes

3.1. Diabetes and obesity

Diabetes mellitus (DM) and periodontitis are both chronic inflammatory disorders and which enhances their severity, worsen each other prognosis and share a number of pathogenic mechanisms with common inflammatory mediators which have been investigated as possible biomarkers of disease status.These improved diagnostic efforts resulting from utilization of biomarkers should enable optimal treatment planning, also assist in monitoring clinical response to treatment and more focused prevention of common human conditions. The most important inflammatory mediators linked to initiation and progression of periodontal disease is a complex network of pro-inflammatory cytokines, matrix metalloproteinases (MMPs) and prostaglandins [78]. The vast majority of studies of cytokines, adipokines and other mediators in periodontitis and diabetes have been small-scale clinical studies using GCF(gingival crevicular fluid), saliva or gingival tissues samples which have focused on limited number of mediators and many inconclusive because of limitation in study design. Nevertheless there are promising data on certain mediators such IL-1β, IL-6, TNF-α and emerging data on RANKL and OPG; these are likely to have a central role in the pathogenesis of periodontitis in diabetic patients [79, 80]. Complex interactions between individual mediators and emergent pathways, for example, synergy in cytokine signaling, will not be apparent from simple disease association studies of a limited number of molecules [79, 80].

There are studies suggesting that pro-inflammatory cytokines which induce chronic inflammatory diseases including periodontitis, could increase insulin resistance [81, 82]. Both TNF-α and IL-6 are produced in adipose tissue, and a large quantity of circulating IL-6 is derived [83]. There are also studies which correlate periodontitis to obesity [84, 85]. These directions of research suggest that obesity, diabetes and periodontitis may be related to each other.

Effective periodontal treatment in patients with DM significantly reduced GCF [86, 87] and serum levels of several mediators, such as IL6, TNF, adiponectin [88, 89, 90], MMP2, MMP9 [91], thus leading to reduced systemic inflammation.

In the effort to establish a pathway for the periodontitis-DM-obesity co-morbidity, some studies have determined genetic polymorphisms for IL6 and IL1 [92, 93]. These cytokines have been previously measured in blood and GFC from patients with these diseases.

Because fatty tissue serves as a reservoir for inflammatory cytokines, an increase in body fat may determine an increase of the inflammatory response of the host in periodontal disease [94].

In 2014, a study concerning the low fibers rich and fat poor diet for 8 weeks, demonstrated an improvement of the periodontal disease's markers, their levels returning to the initial value after follow-up period [95]. A study proposed the hyperinflammatory state observed in obesity, determined by the increase in cytokine levels, as mechanism to explain this relation [96].

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