

The relationships between malocclusion, fixed orthodontic appliances and periodontal disease. A review of the literature

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Aims: To review the literature on the periodontal implications of malocclusion and fixed orthodontic appliances.

Methods: The PubMed database was searched for original articles on 'orthodontics and gingivitis/periodontology/plaque/microbiology/histology', 'bracket and gingivitis/periodontology/plaque/microbiology/histology', 'crowding/spacing/overbite/overjet/open bite/crossbite traumatic occlusion and gingivitis/periodontology/plaque/microbiology/histology'. Only articles published between 1970 and 30 April 2007 were used. The search was augmented by screening the references cited in each paper for additional articles that might have been missed by the electronic search.

Results/Conclusions: Dental plaque is the primary cause of gingival inflammation and periodontitis. Conditions that encourage the growth and retention of dental plaque result in a localised gingivitis, which rarely progresses to periodontal disease. Only a few studies report attachment loss during orthodontic treatment. The contradictory findings on the impact of malocclusion and orthodontic appliances on periodontal health may be partly due to the selection of materials and differences in the research methods employed.

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Introduction

The number of patients undergoing orthodontic treatment with fixed appliances has increased steadily in recent decades.¹ The majority of patients have been treated to improve their dentofacial aesthetics, and only a minority have required treatment for medical or dental reasons. Several indices, such as the Index of Orthodontic Treatment Need, have been developed to objectively score orthodontic treatment need, but no malocclusion index has a periodontal component.² While crowded teeth are unsightly and frequently used to justify orthodontic treatment, they are also difficult to clean and might act as sites for the accumulation of dental plaque.

The aetiology and pathogenesis of periodontal diseases are known to be multifactorial, but dental plaque, not malocclusion, is the essential precursor.^{3,4} It could be argued that as orthodontic appliances, particularly the brackets and bands, promote the accumulation of plaque and make tooth cleaning

more difficult, they increase the risk of developing localised periodontal disease.

The aims of this paper are to review the published literature on the periodontal consequences of malocclusion and those parts of fixed orthodontic appliances (brackets and bands) that retain dental plaque in proximity to the periodontal tissues. A description of the impact of orthodontic treatment on the periodontium, both short- and long-term, is included.

Material and methods

The PubMed database was searched for original articles on: 'orthodontics and gingivitis/periodontology/plaque/microbiology/histology', 'bracket and gingivitis/periodontology/plaque/microbiology/histology', 'crowding/spacing/overbite/overjet/open bite/crossbite/traumatic occlusion and gingivitis/periodontology/plaque/microbiology/histology'. Only articles published between 1970 and 30 April 2007 were used.

The titles and abstracts of all potentially relevant articles were first reviewed. The full text versions of all relevant articles, articles with ambiguous titles and abstracts were then purchased and reviewed. The search was augmented by screening the references cited in each paper for additional articles that might have been missed by the electronic search.

Results

Pathogenesis

Dental plaque

Dental plaque is a complex biofilm that provides nutrients and protection for periodontopathic bacteria.⁵ It is the primary cause of gingival inflammation and periodontitis.⁶ The Gram-positive and mostly aerobic micro-organisms that initially colonise intra-oral hard surfaces are replaced by predominantly Gram-negative and anaerobic micro-organisms.^{7,8} *T. forsythia*, *P. gingivalis*, *A. Actinomycescomitans* and *P. intermedia* are found more frequently in patients with gingivitis and periodontitis than in healthy subjects.

The quantity and the quality of dental plaque are important factors in the onset of periodontitis. Many factors, such as the characteristics of intra-oral hard surfaces (surface roughness, surface free energy),^{9–11} have been found to be positively correlated with the rate of growth of dental plaque.¹² The presence of gingival inflammation will also promote the growth of plaque.^{13,14}

Host reaction

In 1976 Loesche¹⁵ postulated his 'specific plaque hypothesis' and stated that certain micro-organisms in the dental plaque were pathogenic, and that overgrowth of these species would result in increased gingival inflammation due to the host's immune response.

The transition from a reversible gingivitis to an irreversible periodontitis is probably regulated by the bacteria-host reaction. Fransson et al.^{16,17} examined the host reaction to dental plaque in gingival specimens taken from young and old individuals, and reported that in the older subjects inflammation was more marked and there was a higher density of B-cells. They postulated that these differences were due to the different experiences of the young and old to microbial challenges.^{16,17}

Family studies suggest that environmental factors (e.g. oral hygiene and smoking) are major determinants of variance in adult periodontitis.¹⁸ Data from twin studies indicate that about half the population variance in periodontitis can be attributed to genetic factors.^{19–25} Moreover, there is mounting evidence that genetic variations in or near the cytokine genes may influence the inflammatory response in individuals with periodontitis.^{26–28}

Intra-arch orthodontic anomalies

Arch-length discrepancies

Despite the fact that relationships between arch length discrepancies and periodontal health have been studied frequently, there is little evidence that malocclusion and/or irregularity of the teeth affect gingival health. While it seems logical that removal of dental plaque is more difficult in crowded dentitions and that this might impact on periodontal health, modern concepts of periodontal health now emphasise the significance of the biologic, systemic and pro-inflammatory mediators in the development of periodontitis.²⁹

In teenagers a low, but statistically significant, correlation was found between tooth irregularity (tilting, rotation, displacement, crowding) and the plaque and gingival inflammation scores.³⁰ Tooth irregularity was less important than the extent of the plaque and calculus deposits.³⁰ Ashley et al.³¹ also provided some support for the concept of an association between incisor irregularity and gingivitis. They reported a stronger association occurred when a labio-lingually displaced incisor was overlapped by an adjacent tooth than when it was not overlapped by an adjacent tooth. In spite of this finding the level of oral hygiene seemed to be the most important factor.³¹

It would appear that irregular teeth are more difficult to clean and, therefore, more likely to develop gingivitis. In his study of army recruits Ainamo³² reported that periodontal disease was more marked adjacent to malaligned maxillary anterior teeth and less marked in the premolar areas. He concluded that there were no associations between malaligned teeth and periodontal disease at extremes of oral hygiene.³² But Ingervall,³³ who conducted a study of dental students with perfect oral hygiene who had refrained from interdental cleaning for 40 days, reported there was an equal increase in gingivitis in both the crowded

and non-crowded dentitions. He disagreed with Ainamo's conclusion that in individuals with average oral hygiene (brushing three times a day and no interdental cleaning) a crowded dentition did not enhance the accumulation of dental plaque and the development of gingivitis.

Ngom et al.³⁴ recently reported varying degrees of correlation between intra-arch relationships and periodontal health. The plaque and gingivitis indices were significantly correlated with crowding and displaced lower anterior teeth. Crowding in the lower dental arch was the only parameter that was significantly correlated with the severity of periodontal disease. A trend towards more favourable periodontal conditions in young subjects with a spaced dentition^{31,34–36} has also been reported, but the findings were not statistically significant. Other researchers have reported significant associations between the number of open contacts and gingival health in children.^{31,36} It is interesting to note that there was no evidence of a similar relationship in adults.^{37,38}

Alhaija and Al-Wahadni³⁹ recently investigated associations between mild crowding and periodontal disease in individuals with good oral hygiene. The average number of sites with labio-lingually displaced teeth overlapped or not overlapped by adjacent teeth was 1.81 ± 1.30 and 1.39 ± 1.10 respectively. They found no significant associations between crowding and periodontal disease in subjects with good oral hygiene. Their findings in subjects with good oral hygiene were supported by Ashley et al.³¹ However, Ashley et al. found significant associations between irregularity and gingivitis in subjects with moderate and poor oral hygiene,³¹ which disagrees with Ainamo's³² suggestion that when the oral hygiene was poor or non-existent, an association between crowding and gingivitis is less likely to be found.

Staufer and Landmesser⁴⁰ investigated the risk of individuals with different types of anterior crowding developing periodontal disease, and whether the risk was associated with the severity of crowding. They reported there was a greater risk of chronic gingival inflammation in the anterior region in older individuals when the crowding exceeded the threshold value of 3 mm.

Mouth-breathing can also be a cofactor in the development of gingivitis in crowded dentitions. Jacobson and Linder-Aronson⁴¹ reported that mouth breathers

with crowding were more likely to develop gingivitis than nose breathers with crowded dentitions.

Inter-arch orthodontic anomalies

Open bite

Open bites and incompetent lips may lead to the development of a localised gingival recession around the upper incisors. In their study of 101 subjects who had not received orthodontic treatment Ngom et al.³⁴ reported that open bites were significantly, and positively, correlated with the plaque and gingivitis scores, the gingival bleeding index, loss of clinical attachment and pocket depth.³⁴ Machtei et al.⁴² compared the lengths of the clinical crowns, the amount of gingival recession, oral habits and the periodontal indices in children with untreated anterior open bites with a matched control group. Although the plaque indices in the two groups were similar the open-bite group had significantly longer clinical crowns and more gingival inflammation. This may be due to the greater virulence of dehydrated plaque. It has been suggested that a localised gingival recession may develop around the upper incisors in young children with an open bite and incompetent lips.^{41,42}

Crossbite

Excessive forces are exerted in centric relation and maximal occlusion on teeth in crossbite, especially when a 'forced bite' is present. Ngom et al.³⁴ reported a significant correlation between a crossbite and gingival recession,³⁴ which may be due to primary occlusal trauma. In centric relation and at the end of the closing phase during mastication, a tooth in crossbite may contact an opposing tooth prematurely. When this occurs the occlusal loads on the tooth in crossbite are unfavourable and may lead to gingival recession. While some researchers have reported higher periodontal scores in cases with crossbites, other researchers did not find any significant associations between crossbites and periodontal parameters.⁴⁴

Occlusal trauma

Although the influence of occlusal interferences on the health of the periodontium has been studied extensively, the evidence that occlusal interferences either initiate periodontal break-down or contribute to the progression of periodontal disease is inconclusive.^{45–47} Some researchers consider that

occlusal interferences/trauma are not a cause of periodontal disease,⁴⁸ but they may contribute to the progression of the disease.⁴⁷ Using evidence from an animal study Lindhe and Ericsson⁴⁸ argued that occlusal trauma played a role in the progression of periodontal disease. Indirect evidence from a randomised clinical trial of patients with periodontitis also suggested that occlusal trauma may contribute to the break-down of the periodontal tissues.⁴⁹ Bugett et al. reported a significant gain in attachment in patients with periodontitis who received conventional periodontal therapy and occlusal adjustment compared with those who received conventional therapy but no occlusal adjustment.⁴⁹

These findings do not justify prophylactic adjustment of the occlusion with orthodontic treatment to prevent periodontal disease. However, occlusal interferences may be a significant risk factor in the progression of existing periodontal disease, and removal of occlusal interferences/trauma may significantly improve the outcome achieved with periodontal treatment.⁴⁹ In healthy mouths occlusal trauma does not result in periodontal breakdown.

Overjet

Higher plaque scores and less alveolar bone have been found in individuals with an increased overjet. In a study of 19 year-old male military recruits Brojnaas et al.⁵⁰ compared recruits with overjets >8 mm with recruits with 'nearly ideal' occlusions. They reported that the alveolar bone height was 1 mm less in the upper incisor region and 0.4 mm less in the lower incisor region in the overjet group compared with the control group.⁵⁰ In a large, longitudinal study of 12 year-old Welsh children Davies et al.⁵¹ reported a significant association between the mean plaque score, bleeding on probing and anterior overjet.⁵¹ They reported that plaque accumulation and gingivitis occurred at the extremes of overjet, both positive and negative. Other researchers have reported similar results; significantly higher plaque scores were found in subjects with increased overjets.³⁴ Buckley's³⁰ failure to find a significant correlation between overjet and the plaque and gingival indices would appear to contradict these findings. However, Buckley's study is not directly comparable to the studies reported by Brojnaas and Davies;^{30,50,51} Buckley used three groups and his large overjet group had individuals with overjets only >3 mm.

Overbite

Increased overbite can result in the lower incisors impinging on the palatal mucosa or upper incisors impinging on the lower labial mucosa. Both conditions can result in recession and when this occurs orthodontic treatment is indicated.⁵² In less severe cases with no soft tissue damage, treatment may not be required. Bjornaas et al.⁵⁰ demonstrated a significant reduction in alveolar bone heights in the upper (0.7 mm) and lower (0.5 mm) incisor regions in army recruits with overbites >5 mm compared with age-peers with an 'nearly ideal' occlusions.⁵⁰ Ngom et al.³⁴ also reported significant positive correlations between increased overbite and attachment loss and pocket depth, but found no significant correlations between overbite and oral hygiene parameters. On the other hand, Buckley³⁰ did not find any relationship between overbite and periodontal health.

Orthodontic treatment

The amount of dental plaque increases when orthodontic brackets, bands and fixed appliances are placed.^{53–58} Most patients, even those with good oral hygiene, develop a generalised gingivitis following appliance placement, but usually there is no loss of attachment. Some studies have, however, reported loss of attachment during orthodontic treatment with fixed appliances.^{59–61}

Reliable indicators of early inflammation are the crevicular fluid flow ($\mu\text{l}/\text{min}$) and the composition of cytokines in this fluid.⁶² Several techniques for the collection of crevicular fluid have been described.⁶² Cytokines, which mediate the host immunological response to exogenous antigens, are also produced by connective tissue cells such as fibroblasts and osteoblasts and are involved in normal physiological turnover and bone remodelling. Cytokines are low molecular weight proteins (< 25 kDa) produced by cells that regulate or modify the action of other cells in an autocrine (acting on the cell of origin) or paracrine (acting on adjacent cells) manner. The definition includes the interleukins (ILs), tumour necrosis factors (TNFs), interferons, growth factors, and colony stimulating factors. A major difficulty in understanding cytokine biology is the sheer number and complexity of these factors. Another problem is that several factors such as IL-1 and TNF exhibit overlapping biological activities (redundancy) and many have multiple biological effects

pleiotropy). In clinical studies it may be difficult to distinguish between cytokine responses to inflammation and tooth movement during orthodontic treatment.

A clinical manifestation of the inflammatory reaction is the increase in pocket depth caused by gingival hypertrophy (pseudo-pocket formation). Various explanations for this reaction in orthodontic patients with fixed appliances have been suggested in the literature: allergic reactions, mechanical irritation, cytotoxic effects of the band/bracket or bonding material and changes in (subgingival) microbiology.

Comparisons of orthodontically treated and untreated groups have revealed some interesting findings. In a longitudinal study Sadowsky et al.⁶³ compared subjects who had received orthodontic treatment during adolescence with a matched group of subjects with malocclusions that had not been treated. The groups were matched for race, sex, age, socio-economic status, oral hygiene and dental awareness. Although the general prevalence of periodontal disease in the groups was similar, a detailed analysis of the groups disclosed that the orthodontically treated group had mild to moderate periodontal disease in the upper posterior and lower anterior regions. While orthodontic treatment in adolescence is not an important determinant of periodontal health in the long-term, some unfavourable periodontal changes may be found in orthodontic patients in adulthood. Further research is needed to elucidate whether these changes are due the type or duration of the orthodontic treatment. Conversely, lack of orthodontic treatment in adolescence does not appear to influence the development of periodontal disease in adult life.^{63,64}

A significant improvement in oral hygiene may follow orthodontic treatment. Davies et al.⁶⁵ recorded plaque indices, bleeding indices, and the degree of dental irregularity of the anterior teeth in orthodontically treated and untreated children. The children were re-examined three years after the baseline examination. They reported significant reductions in the plaque and gingivitis scores on all tooth surfaces in both groups at the 3-year examinations. The children who had received orthodontic treatment had greater reductions, but these appeared to be due to behavioural factors rather than to improved tooth alignment.

Microbiology

Orthodontic appliances create a favourable environment for the accumulation of microbiota and food residues, which may cause caries or periodontal disease. Naranjo et al.⁵⁸ observed a change in the micro-organisms populating the subgingival dental plaque after the placement of brackets. The plaque gingivitis scores increased significantly. Levels of *P. gingivalis*, *P. intermedia*/*P. nigrescens*, *T. forsythia*, and *Fusobacterium* species were significantly elevated after bracket placement as compared with the untreated control group. Super-infecting micro-organisms such as *E. cloacae*, *K. oxytoca*, *K. pneumoniae*, and *S. marcescens* were also found in the treatment group.⁵⁸ Lee et al.⁶⁶ succeeded in detecting significant differences in the subgingival dental plaque retrieved from areas of gingivitis in patients with and without fixed appliances: *T. forsythia*, *T. denticola*, and *P. nigrescens* were more common in the orthodontic patients than in the control patients.⁶⁶ These results indicate that following placement of fixed appliances, the pathogenic micro-organisms in the dental plaque increased with a consequential increase in gingivitis. No differences in periodontal pocket depth were found. Changes in the types of micro-organisms were restricted to the dental plaque on the teeth supporting the orthodontic appliance.^{58,66}

Local changes occur in the microbiota and periodontal tissues following placement of an orthodontic appliance. Huser et al.⁶⁷ carried out clinical and bacteriological examinations at the beginning of orthodontic treatment and up to 90 days after placement of banded orthodontic appliances. They reported increased plaque and bleeding scores on the banded teeth as compared with the control sites. The probing depth, however, remained within normal values in the test and control groups. They also examined the composition of the dental plaque with dark-field microscopy, and found increased percentages of spirochetes, motile rods, filaments, and fusiforms at the test sites after banding. Over the 90 days there were no significant changes in the distributions of micro-organisms in the control group.⁶⁷ In two small groups of banded and control patients Diamanti-Kipioti et al.⁶⁸ observed an increase in black pigmented bacteria without an increase in the gingival and plaque indices. The mean pocket depths increased significantly from baseline levels in the experimental group, but not the control group.⁶⁸ In a

similar study Petti et al.⁶⁹ compared the influence of fixed and removable orthodontic appliances on the supra- and subgingival microflora. Their data indicate that while gingivitis and periodontitis did not occur in patients with good oral hygiene during the first six months of treatment, changes in the oral microbiota over time suggest that patients with fixed appliances risk developing gingivitis and periodontitis.⁶⁹ The abovementioned studies show that changes in the microbial environment after the placement of fixed orthodontic appliances are accompanied by increased gingivitis.

The effects of the different materials used in modern orthodontics on the microbiota and periodontal health have not been studied thoroughly. Türkkakraman et al.⁷⁰ investigated the influence of different methods of archwire ligation (steel ligatures versus elastomeric rings) on the intra-oral microbial flora and periodontal health. Although teeth ligated with elastomeric rings exhibited slightly higher numbers of micro-organisms than the teeth ligated with steel ligature wires, the differences were not statistically significant. There were no differences in the gingival index, plaque index or pocket depths of the bonded teeth. However, the gingivae around the teeth ligated with elastomeric rings were prone to bleeding.⁷⁰ Van Gastel et al.,⁵³ in a randomised clinical trial with split-mouth design, compared the growth of plaque, clinical periodontal parameters and the crevicular fluid flow on teeth bonded with two different bracket types with non-bonded control teeth. They showed a significantly higher number of aerobes as well as anaerobe colony forming units (CFU) on the bonded teeth compared to the non-bonded controls. There were also significant differences between the teeth bonded with the different brackets. Most important of all was the significant difference in the ratio CFU aerobe/CFU anaerobe because this indicates the pathogenicity of the dental plaque layer. They concluded that the design of an orthodontic bracket system can make the oral environment less favourable for the development of plaque.

Histology

In a longitudinal study of gingival biopsies from the buccal sites opposite molars Zachrisson demonstrated that, shortly after the insertion of bands, the gingival connective tissue was infiltrated by inflammatory

cells.⁵⁵ The dominant cell type in the first month was lymphocytes, but subsequently plasma cells dominated. The topography of the leukocytic infiltrate also differed: plasma cells were located more centrally in the connective tissue, while lymphocytes and PMNs were located more superficially, close to the epithelium. After band removal the inflamed gingival lesions gradually recovered, but acanthosis of the sulcular epithelium persisted in some areas.

Diedrich et al.⁷¹ also described the histopathological picture of an established gingival lesion. It consisted of a detached and severely damaged epithelial attachment, subgingival plaque, massive leukocyte infiltration (mainly lymphocytes, PMNs and plasma cells) and loss of collagen fibres, resulting in destruction of the intra-papillary fibre apparatus. The transeptal fibres, the final protective barrier opposing microbial infiltration of the alveolar bone, were infiltrated to varying degrees by cells. The cellular infiltration was particularly marked interdentally because of the subgingival position of the band margins. At one of these sites the connective tissue attachment was severely damaged, resulting in apical migration of the pocket epithelium, leading to a transition from chronic gingivitis to an initial irreversible periodontal lesion.⁷¹ The cement gaps of the orthodontic bands were analysed and were widest at the cervical margins (average gaps of 0.28 mm). In 85 per cent of the band margins defects in the cement margins were colonised with loosely dispersed dental plaque.⁷¹

Conclusions/clinical implications

1. Dental plaque is the primary cause of gingival inflammation and periodontitis. Transition from a reversible gingivitis to an irreversible periodontitis is regulated by the bacteria-host reaction, which may be studied by analysis of the crevicular fluid. Conditions that encourage the growth and retention of dental plaque increase the risk of developing localised periodontal disease. The gingivitis that develops during orthodontic treatment,^{57,58,65,67,81} is not likely to develop into a periodontitis. Only a few studies report attachment loss during orthodontic treatment.^{59–61}

2. Crowding has no adverse effects on periodontal health in cases with excellent oral hygiene, but in cases with average oral hygiene,^{31–33} crowding promotes the accumulation of dental plaque, which may

impact detrimentally on the health of the periodontium.^{31–33,40,43} In the majority of cases with poor oral hygiene crowding has no effect on periodontal health.^{31–33} It is difficult to determine when gingivitis due to crowding will progress to adult periodontitis.

3. Orthodontic closure and restorative closure of a diastema are contraindicated because both can have negative effects on periodontal health, especially in adults in their third and fourth decades of life.^{31,34–36,72}

4. A localised gingival recession may develop around the upper incisors in children with an anterior open bite and incompetent lips, possibly because lip incompetence leads to dehydration of the plaque.^{41,42} A positive association has been found between an open bite, the plaque score and periodontal health.^{34,42}

5. Crossbites have no detrimental impact on plaque scores and periodontal health.^{34,44}

6. Occlusal trauma does not initiate periodontal breakdown, but it may play a role in the progression of periodontal disease.^{45,46,73} Removal of occlusal trauma may result in a significant gain in attachment in patients with periodontitis.

7. Orthodontic treatment of severe overjets (>7 mm) and overbites (>5 mm) may be justified from a periodontal point of view.^{34,50,51} Long-term studies are required to clarify the periodontal implications of an increased overjet and overbite.

8. Periodontal screening should be an essential part of the orthodontic diagnosis.⁷⁴ Before any orthodontic treatment is started patients should have excellent oral hygiene and the periodontium should be healthy. Loss of attachment before orthodontic treatment is not a contraindication for orthodontic treatment in a healthy mouth.^{75–80}

9. The contradictory findings on the impact of malocclusion and orthodontic appliances on periodontal health may be due to the selection of material and to differences in the research methods employed.

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