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Dental prostheses and tooth-related factors

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Abstract

Objectives: This narrative review summarizes the current evidence about the role that the fabrication and presence of dental prostheses and tooth-related factors have on the initiation and progression of gingivitis and periodontitis.

Findings: Placement of restoration margins within the junctional epithelium and supracrestal connective tissue attachment can be associated with gingival inflammation and, potentially, recession. The presence of fixed prostheses finish lines within the gingival sulcus or the wearing of partial, removable dental prostheses does not cause gingivitis if patients are compliant with self-performed plaque control and periodic maintenance. However, hypersensitivity reactions to the prosthesis dental material can be present. Procedures adopted for the fabrication of dental restorations and fixed prostheses have the potential to cause traumatic loss of periodontal supporting tissues. Tooth anatomic factors, root abnormalities, and fractures can act as plaque-retentive factors and increase the likelihood of gingivitis and periodontitis.

Conclusions: Tooth anatomic factors, such as root abnormalities and fractures, and tooth relationships in the dental arch and with the opposing dentition can enhance plaque retention. Restoration margins located within the gingival sulcus do not cause gingivitis if patients are compliant with self-performed plaque control and periodic maintenance. Tooth-supported and/or tooth-retained restorations and their design, fabrication, delivery, and materials have often been associated with plaque retention and loss of attachment. Hypersensitivity reactions can occur to dental materials. Restoration margins placed within the junctional epithelium and supracrestal connective tissue attachment can be associated with inflammation and, potentially, recession. However, the evidence in several of the reviewed areas, especially related to the biologic mechanisms by which these factors affect the periodontium, is not conclusive. This highlights the need for additional well-controlled animal studies to elucidate biologic mechanisms, as well as longitudinal prospective human trials. Adequate periodontal assessment and treatment, appropriate instructions, and motivation in self-performed plaque control and compliance to maintenance protocols appear to be the most important factors to limit or avoid potential negative effects on the periodontium caused by fixed and removable prostheses.

KEYWORDS

anatomy, classification, dental prostheses, dental restorations, gingivitis, periodontitis, tooth

The anatomy, position, and relationships of teeth within the dental arches are among the factors that have been associated[1] with plaque retention, gingivitis, and periodontitis. Factors related to the presence, design, fabrication, delivery, and materials of tooth-supported prostheses have been suggested to influence the periodontium, generally related to localized increases in plaque accumulation and, less often, to traumatic and allergic reactions to dental materials. This article reviews the role of tooth-related factors and dental prostheses on the initiation and progression of gingivitis and periodontitis.

MATERIALS AND METHODS

For this narrative review, PubMed database was searched for the time period from 1947 up to April 2017, with the strategy found on Table 1. The following filters were applied to the search results: clinical trial, review, guideline, randomized controlled trial, meta-analysis, systematic reviews, humans, and English. The articles obtained, including those referenced in a previous article,[1] were input into a reference manager software.[1] One reviewer (CE) screened titles and abstracts for potential inclusion and discarded duplicates. If title and/or abstract did not provide sufficient information regarding the article content, the article was obtained for review. The selected articles were then obtained in full text and saved as .pdf files in the reference manager database. One reviewer (CE) performed all text reading of the selected publications. When titles of referenced articles, not included in the electronic search, were identified as potentially related to the area of interest of this review, these articles' abstracts were obtained, reviewed for potential inclusion, included in the database, and their full text reviewed.

RESULTS

Biologic width (BW)

BW has been defined as the cumulative apical–coronal dimensions of the junctional epithelium (JE) and supracrestal connective tissue attachment (SCTA).[2] In a cadaver study, variable supracrestal tissue dimensions (i.e., histologic gingival sulcus [GS], JE, and SCTA) were recorded, with the SCTA exhibiting the most constant average dimension.[3] While JE and SCTA exhibited average dimensions within 0.5 to 1 mm when examined on different tooth surfaces,[4,5] this study[3] and others[6,7] showed that dimensions of JE and SCTA can vary considerably,[8] regardless of the association with other factors such as tooth type,[9] surface,[4,9] biotype,[5] loss of attachment,[3] presence of restorations,[4] and crown elongation,[10] so that it is impossible to clearly define a "fixed" biologic width dimension.[9] Biologic width dimensions (JE and SCTA) can only be assessed by histology.[3,4,11] Other methods, such as transgingival probing[10,12–14] and parallel profile radiography, can be used to clinically measure the dimensions of the dentogingival unit, but are not appropriate to measure the true biologic width.[6,15]

Buccal crown margins placed within the junctional epithelium and supracrestal connective tissue attachment have been associated with recession, and histologic evaluation of these sites demonstrated crestal bone loss and supracrestal connective tissue remodeling within 0 to 8 weeks.[16] However, this limited case series was not designed to correlate the observed histologic changes to plaque indices or other mechanisms that could document, in humans, the biologic rationale for the observed changes. Moreover, in a prospective clinical trial, comparing crowns with interproximal margins placed within varying distances from the alveolar bone crest (groups: I = < 1 mm between crown margin and alveolar crest, II = 1 to 2 mm, and III = > 2 mm) it was observed that, while the presence of supragingival plaque was not different among groups, papillary bleeding index (PBI) was greater in group 1, which was associated with increased probing depths (PD) and a clear encroachment of the crown margins within the supracrestal tissue attachment.[17] Given the limited available evidence in humans, it is not possible to determine if the negative effects on the periodontium associated with restoration margins located within the supracrestal tissue attachment is caused by bacterial plaque, trauma, or a combination of these factors.

Fixed dental restorations and prostheses

For class II restorations, gingival inflammation is significantly greater around subgingival margins compared with supragingival margins,[18] even when supragingival plaque levels are not significantly different from prerestoration levels.[19] Furthermore, PD around amalgam restorations with subgingival margins were found to be greater than around contralateral unrestored teeth.[20] Direct restorations with overhangs greater than 0.2 mm are associated with crestal bone loss.[21] Unfortunately, a large prevalence of overhanging amalgam restorations were found in several populations associated with increases in bleeding on probing (BOP) and PD which exceeded the values found at sites with well-fitting restorations and unrestored teeth.[22] The correlation between overhanging margins and PD, gingival inflammation,[23,24] and interproximal bone loss[25–27] was greater for larger overhangs.[28] The removal of the overhangs during scaling and root planing causes a resolution of the gingival inflammation[29] and a decrease in PD due to gingival recession (GR)[30] similar to the resolution of gingivitis.[31] From a microbiologic standpoint and similar to indirect restorations,[32] the elimination of amalgam overhangs during periodontal therapy caused a decrease of *Aggregatibacter actinomycetemcomitans* and increase of *Streptococcus mutans*.[33]

For indirect restorations, overhangs between 0.5 and 1 mm are associated with an increase in gingival inflammation[29] and a more apical crestal bone level, while overhangs of less than 0.2 mm are not.[32,34] Other studies showed that subgingival margins were associated with increased signs of gingival inflammation[35–42] and, at times, increases in PD.[43–47]

A clear association is found between periodontal health and patient compliance with self-performed plaque control and periodontal maintenance after prosthodontic therapy with fixed dental

TABLE 1 Electronic search strategy used for the study

Topic	Search strategy		Search strategy
Biologic width	("biology"[MeSH Terms] OR "biology"[All Fields] OR "biologic"[All Fields]) AND width[All Fields]	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Fixed dental restorations and prostheses	("Crowns"[Mesh:NoExp] OR "Dental Prosthesis Design"[Mesh:NoExp] OR "Dental Restoration Failure"[Mesh] OR "Dental Restoration, Permanent" [Mesh:NoExp] OR "Dental Veneers"[Mesh])	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Dental materials	("dental materials"[Pharmacological Action] OR "dental materials"[MeSH Terms] OR "dental materials"[All Fields]) NOT ("dental implants"[MeSH Terms] OR "dental implants"[All Fields] OR "dental implant"[All Fields] OR "dental prosthesis, implant-supported"[MeSH Terms] OR "implant-supported dental prosthesis"[All Fields] OR "dental prosthesis, implant supported"[All Fields])	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Removable dental prostheses	("Dentures"[MeSH] OR "Dental Clasps"[MeSH])	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Enamel pearls	Enamel pearl [All Field]	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Cervical enamel projections	("neck"[MeSH Terms] OR "neck"[All Fields] OR "cervical"[All Fields]) AND ("dental enamel"[MeSH Terms] OR ("dental"[All Fields] AND "enamel"[All Fields]) OR "dental enamel"[All Fields] OR "enamel"[All Fields]) AND ("projection"[MeSH Terms] OR "projection"[All Fields] OR "projections"[All Fields] OR "forecasting"[MeSH Terms] OR "forecasting"[All Fields])	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Developmental grooves	grooves[All Fields]	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])

(Continues)

TABLE 1 (Continued)

Topic	Search strategy		Search strategy
Tooth and root fractures	"tooth fractures"[MeSH Terms] OR ("tooth"[All Fields] AND "fractures"[All Fields]) OR "tooth fractures"[All Fields]	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Root resorption	"Tooth Root/pathology"[MAJR] AND Root Resorption/pathology	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Tooth position	("malocclusion"[MeSH Terms] OR "malocclusion"[All Fields]) AND ("tooth"[MeSH Terms] OR "tooth"[All Fields]) AND position[All Fields])	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Root proximity	("tooth root"[MeSH Terms] OR ("tooth"[All Fields] AND "root"[All Fields]) OR "tooth root"[All Fields]) AND proximity[All Fields]	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])
Open contacts	"Diastema"[MAJR] OR Open contacts	AND	(Periodontitis OR Periodontal Diseases OR Gingivitis OR Gingival Diseases) NOT ("case reports"[Publication Type] OR "comment"[Publication Type] OR "editorial"[Publication Type] OR "interview"[Publication Type] OR "letter"[Publication Type] OR "news"[Publication Type] OR "newspaper article"[Publication Type])

prostheses.[47–49] In a prospective clinical trial where patients were instructed and motivated on adequate measures of self-performed plaque control, plaque levels and gingival inflammation were not significantly different between teeth that received crowns and controls.[50] Similarly, in a cohort of patients who were seen for periodontal maintenance every 1 to 6 months, no difference in plaque and gingival indices were found between crowned and non-crowned teeth regardless of the position of the crown margins,[51] a finding also reported by other studies.[52–54]

While porcelain veneers were not associated with changes in plaque levels and gingival inflammation for as long as 7 years after delivery,[55–59] gingival recession can be a common consequence of other fixed prosthodontic therapies.[60–62] Prosthodontic procedures required for the fabrication of fixed prostheses can negatively affect the periodontium. Procedures and/or materials such as crown preparation, gingival displacement during impression,[63,64] impressions, provisional prostheses,[65] and luting agents[66] may be contributing factors for the development of gingivitis, gingival recession, and periodontitis. The placement of provisional crowns causes an increase in plaque retention regardless of the resin material used for the prosthesis.[65] In another study[67] where all crown margins were designed in a subgingival location during crown preparation, only 82% of them were still located subgingivally at crown delivery. This suggests that the actual crown margin location was less of a contributing etiologic factor affecting the occurrence and magnitude of recession than the prosthetic procedures required to design and record the crown margin position. In a short-term randomized, multicenter, controlled trial, different methods of gingival displacement produced different magnitudes and frequency distributions of gingival recession, and most of the recession occurred before

final crown delivery.[68] The anatomy of the periodontium of teeth receiving crowns should be evaluated to minimize the likelihood of gingival recession because the presence of an initial shallow PD and narrow band of gingiva negatively influenced the level of periodontal attachment after crown delivery.[69] These studies point out the critical importance of including a complete periodontal assessment prior to prosthodontic manipulations when studying the response of the periodontium to indirect restorations.[60]

The available literature supports the conclusion that a direct restoration with subgingival margins can be associated with localized gingivitis and increases in PD. A direct or indirect restoration with overhanging margins can be associated with localized gingivitis, increase in PD, and interproximal bone loss, especially for larger overhangs. These changes are likely caused by the overhang acting as a plaque-retentive factor and causing a qualitative shift toward a subgingival cultivable microflora more characteristic of periodontitis.

From cross-sectional studies, it can be concluded, especially when self-performed plaque control and periodontal maintenance measures are not mentioned, that an indirect restoration subgingival margin is associated with gingivitis. However, in longitudinal studies, where self-performed plaque control and periodontal maintenance measures are described and patient compliance is achieved, subgingival prosthesis margins do not appear to act as plaque-retentive factors that cause gingivitis. Based on the available evidence, it appears that plaque control by the patient and compliance with periodontal maintenance is of paramount importance to maintain the health of the periodontium when subgingival margins are adopted in the prosthetic design. Permanent changes to the periodontium, such as gingival recession, could occur when subgingival margins are adopted for prosthesis design; however, they appear to be mostly related to trauma to the periodontium exerted by the procedures, instruments, and materials required to place and record the margins in a subgingival location, rather than the nominal position of the margin.

Dental materials

Different dental materials, their surface characteristics, and location in relation to the gingiva have been associated with variable periodontal responses.[70–73] However, this response could be potentially affected, not only by the type of material, but also by the surface characteristics, such as surface-free energy and roughness, among others, that act as confounding variables. For the latter, a minimum roughness threshold (Ra < 0.2 μm) has been suggested, with increases in plaque retention expected above this threshold, but no reduction for lower Ra values.[74] Similarly, when different alloys were used to fabricate onlays[75] and other types of prostheses,[50] they showed similar levels of plaque and gingival inflammation. Roughness changes, resulting from polishing, scaling, or patient-related factors are material-specific and data on resultant plaque accumulation as a function of the change in Ra is scarce.[76] Teeth restored with a variety of dental materials, when compared with enamel, had similar plaque levels, gingival inflammation, interleukin (IL)-1α, IL-1β, and IL-1ra levels, but most important, in a 10-day gingivitis experiment, showed no difference for the same parameters.[49,77] Similar clinical gingival reactions in periodontally healthy patients were also seen when comparing class V restorations of composite resin or calcium aluminate/silicate material.[78–82] These findings appear also valid when different restorative materials are used to rebuild part of the tooth anatomy during mucogingival surgical procedures.[83–88] Therefore, available evidence demonstrates that different dental materials act similarly to enamel as plaque-retentive factors to initiate gingivitis.

Metal ions and metal particles can also be released from dental alloys and can be found locally within plaque, the periodontum, and in several organs and tissues. While several of these ions (nickel [Ni], palladium [Pd], copper [Cu], titanium [Ti] among others) have been shown, via in vitro studies, to potentially affect cell count, viability, function, and the release of inflammatory mediators, their influence on gingivitis and periodontitis is largely unclear.[89] Metal ions and particles, especially Ni and Pd, have also been associated with hypersensitivity reactions which might clinically appear as gingivitis, localized in the area of gingival contact with the dental material that does not respond to adequate measures of plaque control, and contact stomatitis, often with a lichenoid-type appearance.[90–93] For patients who have shown allergic reactions to dental alloys, very limited evidence suggests that the replacement of these prostheses with zirconia-based protheses was associated with a resolution of the allergic reaction.[94]

Removable dental prostheses

In cross-sectional studies, where no information is present on the level of self-performed plaque control and periodontal maintenance or where clearly heterogeneous baseline periodontal conditions are present,[95] partial removable dental prostheses (RDPs) have been associated with increased prevalence of caries, gingivitis, and periodontitis.[96–100] A study has shown no changes in PD, but increases in plaque levels and gingival inflammation in patients wearing RDPs.[101] Other authors have reported that when the patient was adequately instructed on self-performed plaque control and seen at frequent periodic maintenance visits, there was a decrease in plaque levels and gingival inflammation.[102] A recent study showed no difference in PD, BOP, gingival recession, microbial count, and species between teeth that supported RDPs and teeth that did not.[103] Longitudinal studies of distal extension RDPs indicate that a favorable periodontal prognosis may be expected provided the following conditions are satisfied: 1) periodontal disease, if present, is treated and an adequate preprosthetic plaque control regimen established; 2) periodontal health and oral hygiene are maintained through self-performed plaque control measures[104] and periodic maintenance appointments,[105] and 3) patient's motivation is reinforced to enhance compliance to self-performed plaque control and periodontal maintenance.[106–112] Therefore, we can conclude that, if plaque control is established, the prostheses are correctly designed and regularly checked, and indicated maintenance procedures are performed,

RDPs do not cause greater plaque accumulation, periodontal loss of attachment, or increased mobility.[113–118] On the other hand, if patients do not adequately perform plaque control and attend periodic maintenance appointments, removable dental prostheses, including overdentures,[118–127] could act as plaque-retentive factors and indirectly cause gingivitis and periodontitis. In addition, especially distal extension RDPs, when not properly maintained and relined, have the potential to apply greater forces and torque to the abutment teeth, causing a traumatic increase in mobility.[107]

Tooth anatomy and position

Cervical enamel projections (CEP) and enamel pearls (EP)

Tooth anatomic factors, such as CEP and EP, have been associated with furcation invasion, increased PD, and loss of clinical attachment.[128,129] The extent of CEP extension toward the furcation area can be classified into three classes, with grade I described as "distinct change in cemento-enamel junction (CEJ) attitude with enamel projecting toward the furcation;" grade II, "the CEP approaching the furcation, but not actually making contact with it;" and grade III, "CEP extending into the furcation proper."[130] Prevalence of CEP for all extracted teeth varies, depending on the report, from 25% to 35.5% and 8% to 17% in mandibular and maxillary molars, respectively.[130–135] When controlling for the presence of furcation invasion (FI), CEP were found in 82.5% and 17.5% of molars with and without FI, respectively,[136] with prevalence for CEP associated with FI ranging from 63.2% to 90%[130,137,138] and only one study finding no greater significant association between CEP compared with FI.[134] While the prevalence of grade III CEP varies in the literature from 4.3% to 6.3%, these types of CEP might be more detrimental to the furcation periodontal tissues than grade I and II CEP.[136,139]

Enamel pearls are generally spheroidal in shape, occur in roughly 1% to 5.7% of all molar teeth,[140–142] vary in dimension from 0.3 to 2 mm, and occur most often isolated on a tooth, potentially localized in the furcation area of molars.[133,142–144] EP can act as a plaque-retentive factor when periodontitis progresses to the point that they become part of the subgingival microbial ecosystem.

Developmental grooves

The most frequent developmental groove appears to be the palatal groove, most often located in the maxillary lateral incisor with a prevalence of 1% to 8.5% at the subject level and 2.2% at the tooth level.[145] Forty-three percent of grooves do not extend more than 5 mm apical to the CEJ and only 10% are present 10 mm or more apical the CEJ.[146] The mechanism suggested for developmental grooves to initiate periodontal disease is related to plaque retention that causes localized gingivitis and periodontitis.[133,145,147–150] Grooves are also present on other teeth[151,152] and mostly in the interproximal areas, with few of these grooves extending to the tooth apex.[153]

Tooth and root fractures

Tooth fractures

If tooth fractures occur coronal to the gingival margin and do not extend to parts of the tooth surrounded by periodontal tissues, they do not initiate gingivitis or periodontitis, unless the surface characteristics of the fracture area predispose to greater plaque retention.

Root fractures

Root fractures can be classified based on the trajectory of the fracture (vertical, transverse, or oblique), their extent (complete or incomplete), location (apical, midroot, or cervical regions) and on the healing/repair mode.[154] While fractures located within the midroot and apical regions were shown in a 10-year study to have a very favorable prognosis (78% and 89% tooth survival, respectively), fractures located within the cervical one-third of the root had a significantly worse prognosis for tooth retention (33%).[154–156] Since fractures located within the cervical third of a root have a more likely possibility of being colonized by subgingival plaque, they can act as plaque-retentive factors and indirectly cause gingivitis and periodontitis. In addition, they can directly traumatize the surrounding periodontium due to mobility of the fractured tooth surfaces. Limited short-term evidence suggests that fractures located within the anatomic crown or slightly into the cervical third of the root can be successfully repaired with adhesive techniques and that periodontal parameters, such as plaque index, gingival index (GI), PD, and clinical attachment level, are not different than control teeth.[157–159] Vertical root fractures are defined as longitudinal fractures that might begin on the internal canal wall and extend outward to the external root surface. They occur most often on endodontically treated teeth, although they can be present on non-endodontically treated teeth, especially molars and premolars, as a result of apical extensions of coronal tooth fractures.[160] A localized pocket, with loss of attachment and bone is usually associated with the fractured tooth[161] and extends to variable lengths along the fracture line.[162,163] Narrow, deep, V- or U-shaped osseous defects are generally seen during surgical exposure of the fractured area with bone resorption and inflammation related to bacterial infection from the gingival margin and root canal system.[164,165]

Root resorption

Root resorption can be classified into surface, inflammatory, replacement resorption,[166,167] and depending on its location, as internal or external, cervical or apical.[168,169] When root resorption is located within the cervical third of the root, it can easily communicate with the subgingival microbial ecosystem. Plaque retention at such sites can cause gingivitis and periodontitis. Cemental tears are localized areas of cementum detachment from the underlying dentin and can potentially lead to localized periodontal breakdown, although the biologic mechanism involved has not been elucidated.[170,171]

Tooth position

Cross-bite,[172,173] misalignment/rotation of a tooth,[174] and crowding of the maxillary[175] and mandibular anterior sextant[176] have been shown to be associated with increased plaque retention[176] and gingivitis, greater PD, and bone[177] and clinical attachment loss.[178] However, other studies assessing the effect of crowding on the periodontium did not find an association with plaque retention and gingivitis.[179–181] Tooth position and periodontal biotype and their interaction[182] can also be factors that influence the likelihood of mucogingival deformities, as it has been shown that a thin periodontal biotype has a significantly thinner labial bone plate, narrower gingival width, and greater apico-coronal distance between the CEJ and the alveolar crest.[183] In subjects who exhibit trauma related to tooth brushing[184–187] or tooth malposition within the alveolar process,[187,188] a greater risk for gingival recession can be present. Tooth anatomy, and specifically the shape of the tooth and their approximation, have been shown to affect the height of the interproximal papilla.[189]

Root proximity

Root proximity (RP) in the maxilla is most prevalent between the first and second molar and between the central and lateral incisors; in the mandible, it is generally seen between the central and lateral incisors.[190,191] However, RP has been defined and measured in different ways in the literature, therefore producing inconsistent conclusions on its effect on the periodontum.[192,193] More recently, however, a longitudinal 10-year clinical study concluded that, while an interproximal root distance (IRD) of mandibular central and lateral incisors > 0.8 mm was not associated with a more apical position of the interproximal bone, an IRD > 0.8 mm was associated, even when controlled for age, smoking, plaque, and calculus, with interproximal crestal bone loss, and sites with IRDs < 0.6 mm were 28% and 56% more likely to lose > 0.5 mm and > 1.0 mm of bone during 10 years, respectively.[194] Based on the limited evidence, we are not able to conclude which are the biologic mechanisms underlying this increased bone loss.[194] To standardize the location and magnitude of RP, a classification has been proposed that defines the location of the measured site of RP (cervical, middle, or apical third of the root) and divides the severity of the RP into type 1: > 0.5 to ≤0.8 mm; type 2: > 0.3 to ≤0.5 mm; type 3: ≤0.3 mm.[190]

Open contacts

The presence of adequate proximal tooth contacts is considered important to prevent food impaction between teeth.[195] From a periodontal standpoint, while the presence of open contacts was not a factor directly associated with increased GI and PD, the statistically greater occurrence of food impaction at sites with open contacts was associated with increased PD in these areas.[196,197]

CONCLUSIONS

Tooth anatomic factors, root abnormalities and fractures, and tooth relationships in the dental arch and with the opposing dentition can enhance plaque retention. Restoration margins located within the gingival sulcus do not cause gingivitis if patients are compliant with self-performed plaque control and periodic maintenance. Tooth-supported and/or tooth-retained restorations and their design, fabrication, delivery, and materials have often been associated with plaque retention and loss of attachment. Hypersensitivity reactions can occur to dental materials. Restorations margins placed within the junctional epithelium and supracrestal connective tissue attachment can be associated with inflammation and, potentially, recession. However, the evidence in several of these areas, especially related to the biologic mechanisms by which these factors affect the periodontium, is inconclusive. This highlights the need for additional well-controlled animal studies to elucidate biologic mechanisms, as well as longitudinal, prospective human trials. Adequate periodontal assessment and treatment, instructions and motivation in self-performed plaque control, and compliance with maintenance protocols appear to be the most important factors to limit or avoid potential negative effects on the periodontium associated with fixed and removable prostheses.

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[1]Thomson Reuters, New York, NY.

REFERENCES

1. Blieden TM. Tooth-related issues. *Ann Periodontol*. 1999;*4*:91–97.
2. American Academy of Peridontology. Glossary of periodontal terms. 2001. Available at: https://www.perio.org. Accessed July 30, 2016.
3. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. *J Periodontol*. 1961;*32*:261–267.
4. Vacek JS, Gher ME, Assad DA, Richardson AC, Giambarresi LI. The dimensions of the human dentogingival junction. *Int J Periodontics Restorative Dent*. 1994;*14*:154–165.
5. Rasouli Ghahroudi AA, Khorsand A, Yaghobee S, Haghighati F. Is biologic width of anterior and posterior teeth similar. *Acta Med Iran*. 2014;*52*:697–702.
6. Alpiste-Illueca F. Dimensions of the dentogingival unit in maxillary anterior teeth: a new exploration technique (parallel profile radiograph). *Int J Periodontics Restorative Dent*. 2004;*24*:386–396.
7. Barboza EP, MonteAlto RF, Ferreira VF, Carvalho WR. Supracrestal gingival tissue measurement in healthy human periodontum. *Int J Periodontics Restorative Dent*. 2008;*28*:55–61.
8. Gargiulo A, Krajewski J, Gargiulo M. Defining biologic width in crown lengthening. *CDS Rev*. 1995;*88*:20–23.
9. Schmidt JC, Sahrmann P, Weiger R, Schmidlin PR, Walter C. Biologic width dimensions – A systematic review. *J Clin Periodontol*. 2013;*40*:493–504.

10. Lanning SK, Waldrop TC, Gunsolley JC, Maynard JG. Surgical crown lengthening: evaluation of the biological width. *J Periodontol*. 2003;*74*:468–474.
11. Xie GY, Chen JH, Wang H, Wang YJ. Morphological measurement of biologic width in Chinese people. *J Oral Sci*. 2007;*49*:197–200.
12. Novak MJ, Albather HM, Close JM. Redefining the biologic width in severe, generalized, chronic periodontitis: implications for therapy. *J Periodontol*. 2008;*79*:1864–1869.
13. Ursell MJ. Relationships between alveolar bone levels measured at surgery, estimated by transgingival probing and clinical attachment level measurements. *J Clin Periodontol*. 1989;*16*:81–86.
14. Greenberg J, Laster L, Listgarten MA. Transgingival probing as a potential estimator of alveolar bone level. *J Periodontol*. 1976;*47*:514–517.
15. Galgali SR, Gontiya G. Evaluation of an innovative radiographic technique—parallel profile radiography—to determine the dimensions of dentogingival unit. *Indian J Dent Res*. 2011;*22*:237–241.
16. Tarnow D, Stahl SS, Magner A, Zamzok J. Human gingival attachment responses to subgingival crown placement. Marginal remodelling. *J Clin Periodontol*. 1986;*13*:563–569.
17. Gunay H, Seeger A, Tschernitschek H, Geurtsen W. Placement of the preparation line and periodontal health—a prospective 2-year clinical study. *Int J Periodontics Restorative Dent*. 2000;*20*:171–181.
18. Schatzle M, Land NP, Anerud A, Boysen H, Burgin W, Loe H. The influence of margins of restorations of the periodontal tissues over 26 years. *J Clin Periodontol*. 2001;*28*:57–64.
19. Gullo CA, Powell RN. The effect of placement of cervical margins of class II amalgam restorations on plaque accumulation and gingival health. *J Oral Rehabil*. 1979;*6*:317–322.
20. Jansson L, Blomster S, Forsgardh A, et al. Interactory effect between marginal plaque and subgingival proximal restorations on periodontal pocket depth. *Swed Dent J*. 1997;*21*:77–83.
21. Bjorn AL, Bjorn H, Grkovic B. Marginal fit of restorations and its relation to periodontal bone level. I. Metal fillings. *Odontol Revy*. 1969;*20*:311–321.
22. Pack AR, Coxhead LJ, McDonald BW. The prevalence of overhanging margins in posterior amalgam restorations and periodontal consequences. *J Clin Periodontol*. 1990;*17*:145–152.
23. Kells BE, Linden GJ. Overhanging amalgam restorations in young adults attending a periodontal department. *J Dent*. 1992;*20*:85–89.
24. Lervik T, Riordan PJ, Haugejorden O. Periodontal disease and approximal overhangs on amalgam restorations in Norwegian 21-year-olds. *Community Dent Oral Epidemiol*. 1984;*12*:264–268.
25. Jansson L, Ehnevid H, Lindskog S, Blomlof L. Proximal restorations and periodontal status. *J Clin Periodontol*. 1994;*21*:577–582.
26. Parsell DE, Streckfus CF, Stewart BM, Buchanan WT. The effect of amalgam overhangs on alveolar bone height as a function of patient age and overhang width. *Oper Dent*. 1998;*23*:94–99.
27. Laurell L, Rylander H, Pettersson B. The effect of different levels of polishing of amalgam restorations on the plaque retention and gingival inflammation. *Swed Dent J*. 1983;*7*:45–53.
28. Jeffcoat MK, Howell TH. Alveolar bone destruction due to overhanging amalgam in periodontal disease. *J Periodontol*. 1980;*51*:599–602.
29. Rodriguez-Ferrer HJ, Strahan JD, Newman HN. Effect of gingival health of removing overhanging margins of interproximal subgingival amalgam restorations. *J Clin Periodontol*. 1980;*7*:457–462.
30. Gorzo I, Newman HN, Strahan JD. Amalgam restorations, plaque removal and periodontal health. *J Clin Periodontol*. 1979;*6*:98–105.
31. Caton J, Bouwsma O, Polson A, Espeland M. Effects of personal oral hygiene and subgingival scaling on bleeding interdental gingiva. *J Periodontol*. 1989;*60*:84–90.
32. Lang NP, Kiel RA, Anderhalden K. Clinical and microbiological effects of subgingival restorations with overhanging or clinically perfect margins. *J Clin Periodontol*. 1983;*10*:563–578.
33. Roman-Torres CV, Cortelli SC, de Araujo MW, Aquino DR, Cortelli JR. A short-term clinical and microbial evaluation of periodontal therapy associated with amalgam overhang removal. *J Periodontol*. 2006;*77*:1591–1597.
34. Bjorn AL, Bjorn H, Grkovic B. Marginal fit of restorations and its relation to periodontal bone level. II. Crowns. *Odontol Revy*. 1970;*21*:337–346.
35. Newcomb GM. The relationship between the location of subgingival crown margins and gingival inflammation. *J Periodontol*. 1974;*45*:151–154.
36. Paniz G, Nart J, Gobbato L, Chierico A, Lops D, Michalakis K. Periodontal response to two different subgingival restorative margin designs: a 12-month randomized clinical trial. *Clin Oral Invest*. 2016;*20*:1243–1252.
37. Bader J, Rozier RG. The effect of crown receipt on measures of gingival status. *J Dent Res*. 1991;*70*:1386–1389.
38. Bader JD, Rozier RG, McFall WT, Jr, Ramsey DL. Effect of crown margins on periodontal conditions in regularly attending patients. *J Prosthet Dent*. 1991;*65*:75–79.
39. Al-Wahadni AM, Mansour Y, Khader Y. Periodontal response to all-ceramic crowns (IPS Empress) in general practice. *Int J Dent Hyg*. 2006;*4*:41–46.
40. Valderhaug J, Heloe LA. Oral hygiene in a group of supervised patients with fixed prostheses. *J Periodontol*. 1977;*48*: 221–224.
41. Felton DA, Kanoy BE, Bayne SC, Wirthman GP. Effect of in vivo crown margin discrepancies on periodontal health. *J Prosthet Dent*. 1991;*65*:357–364.
42. Reitemeier B, Hansel K, Walter MH, Kastner C, Toutenburg H. Effect of posterior crown margin placement on gingival health. *J Prosthet Dent*. 2002;*87*:167–172.
43. Boeckler AF, Lee H, Psoch A, Setz JM. Prospective observation of CAD/CAM titanium-ceramic-fixed partial dentures: 3-year follow-up. *J Prosthodont*. 2010;*19*:592–597.
44. Boeckler AF, Lee H, Stadler A, Setz JM. Prospective observation of CAD/CAM titanium ceramic single crowns: a three-year follow up. *J Prosthet Dent*. 2009;*102*:290–297.
45. Hey J, Beuer F, Bensel T, Boeckler AF. Single crowns with CAD/CAM-fabricated copings from titanium: 6-year clinical results. *J Prosthet Dent*. 2014;*112*:150–154.
46. Larato DC. Effects of artificial crown margin extension and tooth brushing frequency on gingival pocket depth. *J Prosthet Dent*. 1975;*34*:640–643.
47. Heschl A, Haas M, Haas J, Payer M, Wegscheider W, Polansky R. Maxillary rehabilitation of periodontally compromised patients with extensive one-piece fixed prostheses supported by natural teeth: a retrospective longitudinal study. *Clin Oral Invest*. 2013;*17*:45–53.
48. Glossary of prosthodontic terms (GPT9). *J Prosthet Dent* 2017;*117*(5S):e1-e105. https://doi.org/10.1016/j.prosdent.2016.12.001.
49. Konradsson K, Claesson R, van Dijken JW. Dental biofilm, gingivitis and interleukin-1 adjacent to approximal sites of a bonded ceramic. *J Clin Periodontol*. 2007;*34*:1062–1067.
50. Morris HF. Veterans Administration Cooperative Studies Project No 147. Part VIII: plaque accumulation on metal ceramic restorations cast from noble and nickel-based alloys. A five-year report. *J Prosthet Dent*. 1989;*61*:543–549.
51. Carnevale G, di Febo G, Fuzzi M. A retrospective analysis of the perio-prosthetic aspect of teeth re-prepared during periodontal surgery. *J Clin Periodontol*. 1990;*17*:313–316.
52. Di Febo G, Bedendo A, Romano F, Cairo F, Carnevale G. Fixed prosthodontic treatment outcomes in the long-term management of patients with periodontal disease: a 20-year follow-up report. *Int J Prosthodont*. 2015;*28*:246–251.

53. Guncu MB, Cakan U, Muhtarogullari M, Canay S. Zirconia-based crowns up to 5 years in function: a retrospective clinical study and evaluation of prosthetic restorations and failures. *Int J Prosthodont*. 2015;*28*:152–157.
54. Muller HP. The effect of artificial crown margins at the gingival margin on the periodontal conditions in a group of periodontally supervised patients treated with fixed bridges. *J Clin Periodontol*. 1986;*13*:97–102.
55. D'Arcangelo C, De Angelis F, Vadini M, D'Amario M. Clinical evaluation on porcelain laminate veneers bonded with light-cured composite: results up to 7 years. *Clin Oral Invest*. 2012;*16*:1071–1079.
56. Gurel G, Morimoto S, Calamita MA, Coachman C, Sesma N. Clinical performance of porcelain laminate veneers: outcomes of the aesthetic pre-evaluative temporary (APT) technique. *Int J Periodontics Restorative Dent*. 2012;*32*:625–635.
57. Jiang T, Hong W, Zhang Q. Two-year clinical trial of resin-bonded fixed partial dentures incorporating novel attachments. *Int J Prosthodont*. 2005;*18*:225–231.
58. Pippin DJ, Mixson JM, Soldan-Els AP. Clinical evaluation of restored maxillary incisors: veneers vs. PFM crowns. *J Am Dent Assoc*. 1995;*126*:1523–1529.
59. Reid JS, Kinane DF, Adonogianaki E. Gingival health associated with porcelain veneers on maxillary incisors. *Int J Paediatr Dent*. 1991;*1*:137–141.
60. Sola-Ruiz MF, Lagos-Flores E, Roman-Rodriguez JL, et al. Survival rates of a lithium disilicate-based core ceramic for three-unit esthetic fixed partial dentures: a 10-year prospective study. *Int J Prosthodont*. 2013;*26*:175–180.
61. Sjogren G, Lantto R, Tillberg A. Clinical evaluation of all-ceramic crowns (Dicor) in general practice. *J Prosthet Dent*. 1999;*81*:277–284.
62. Pelaez J, Cogolludo PG, Serrano B, Lozano JF, Suarez MJ. A prospective evaluation of zirconia posterior fixed dental prostheses: three-year clinical results. *J Prosthet Dent*. 2012;*107*:373–379.
63. Polat NT, Ozdemir AK, Turgut M. Effects of gingival retraction materials on gingival blood flow. *Int J Prosthodont*. 2007;*20*:57–62.
64. Ruel J, Schuessler PJ, Malament K, Mori D. Effect of retraction procedures on the periodontium in humans. *J Prosthet Dent*. 1980;*44*:508–515.
65. Luthardt RG, Stossel M, Hinz M, Vollandt R. Clinical performance and periodontal outcome of temporary crowns and fixed partial dentures: a randomized clinical trial. *J Prosthet Dent*. 2000;*83*:32–39.
66. Orug BO, Baysallar M, Cetiner D, et al. Increased antibacterial activity of zinc polycarboxylate cement by the addition of chlorhexidine gluconate in fixed prosthodontics. *Int J Prosthodont*. 2005;*18*:377–382.
67. Koke U, Sander C, Heinecke A, Muller HP. A possible influence of gingival dimensions on attachment loss and gingival recession following placement of artificial crowns. *Int J Periodontics Restorative Dent*. 2003;*23*:439–445.
68. Einarsdottir E, Lang NP, Aspelund T, Pjetursson BE. A multicenter randomized, controlled clinical trial comparing the use of displacement cords, an aluminum chloride paste, and a combination of paste and cords for tissue displacement [published online ahead of print 2017]. *J Prosthet Dent*. https://doi.org/10.1016/j.prosdent.2017.03.010.
69. Stetler KJ, Bissada NF. Significance of the width of keratinized gingiva on the periodontal status of teeth with submarginal restorations. *J Periodontol*. 1987;*58*:696–700.
70. Wang JC, Lai CH, Listgarten MA. *Porphyromonas gingivalis*, *Prevotella intermedia* and *Bacteroides forsythus* in plaque subjacent to bridge pontics. *J Clin Periodontol*. 1998;*25*:330–333.
71. Weishaupt P, Bernimoulin JP, Lange KP, Rothe S, Naumann M, Hagewald S. Clinical and inflammatory effects of galvano-ceramic and metal-ceramic crowns on periodontal tissues. *J Oral Rehabil*. 2007;*34*:941–947.
72. Willershausen B, Kottgen C, Ernst CP. The influence of restorative materials on marginal gingiva. *Eur J Med Res*. 2001;*6*:433–439.
73. Ababnaeh KT, Al-Omari M, Alawneh TN. The effect of dental restoration type and material on periodontal health. *Oral Health Prev Dent*. 2011;*9*:395–403.
74. Quirynen M, Marechal M, Busscher HJ, Weerkamp AH, Darius PL, van Steenberghe D. The influence of surface free energy and surface roughness on early plaque formation. *J Clin Periodontol*. 1990;*17*:138–144.
75. Walter MH, Wolf BH, Schmidt AE, Boening KW, Koch R. Plaque, gingival health and post-operative sensitivity in titanium inlays and onlays: a randomized controlled clinical trial. *J Dent*. 2001;*29*:181–186.
76. Bollen CM, Lambrechts P, Quirynen M. Comparison of surface roughness of oral hard materials to the threshold surface roughness for bacterial plaque retention: a review of the literature. *Dent Mater*. 1997;*13*:258–269.
77. Konradsson K, van Dijken JW. Interleukin-1 levels in gingival crevicular fluid adjacent to restorations of calcium aluminate cement and resin composite. *J Clin Periodontol*. 2005;*32*:462–466.
78. Konradsson K, van Dijken JW. Effect of a novel ceramic filling material on plaque formation and marginal gingiva. *Acta Odontol Scand*. 2002;*60*:370–374.
79. van Dijken JW, Sjostrom S, Wing K. Development of gingivitis around different types of composite resin. *J Clin Periodontol*. 1987;*14*:257–260.
80. van Dijken JW, Sjöström S. The effect of glass ionomer cement and composite resin fillings on marginal gingiva. *J Clin Periodontol*. 1991;*18*:200–203.
81. van Dijken JW, Sjostrom S. Development of gingivitis around aged restorations of resin-modified glass ionomer cement, polyacid-modified resin composite (compomer) and resin composite. *Clin Oral Invest*. 1998;*2*:180–183.
82. Litonjua LA, Cabanilla LL, Abbott LJ. Plaque formation and marginal gingivitis associated with restorative materials. *Compend Contin Educ Dent*. 2012;*33*:e6-e10.
83. Cairo F, Pini-Prato GP. A technique to identify and reconstruct the cementoenamel junction level using combined periodontal and restorative treatment of gingival recession. A prospective clinical study. *Int J Periodontics Restorative Dent*. 2010;*30*:573–581.
84. Santamaria MP, Ambrosano GM, Casati MZ, Nociti FH, Jr, Sallum AW, Sallum EA. Connective tissue graft and resin glass ionomer for the treatment of gingival recession associated with noncarious cervical lesions: a case series. *Int J Periodontics Restorative Dent*. 2011;*31*:e57-e63.
85. Santamaria MP, Ambrosano GM, Casati MZ, Nociti FH, Jr, Sallum AW, Sallum EA. Connective tissue graft plus resin-modified glass ionomer restoration for the treatment of gingival recession associated with non-carious cervical lesion: a randomized-controlled clinical trial. *J Clin Periodontol*. 2009;*36*:791–798.
86. Santamaria MP, Casati MZ, Nociti FH, Jr, et al. Connective tissue graft plus resin-modified glass ionomer restoration for the treatment of gingival recession associated with non-carious cervical lesions: microbiological and immunological results. *Clin Oral Invest*. 2013;*17*:67–77.
87. Santamaria MP, da Silva Feitosa D, Casati MZ, Nociti FH, Jr, Sallum AW, Sallum EA. Randomized controlled clinical trial evaluating connective tissue graft plus resin-modified glass ionomer restoration for the treatment of gingival recession associated with non-carious cervical lesion: 2-year follow-up. *J Periodontol*. 2013;*84*:e1-e8.
88. Santamaria MP, Queiroz LA, Mathias IF, et al. Resin composite plus connective tissue graft to treat single maxillary gingival recession

associated with non-carious cervical lesion: randomized clinical trial. *J Clin Periodontol*. 2016;*43*:461–468.
89. Gursoy UK, Sokucu O, Uitto VJ, et al. The role of nickel accumulation and epithelial cell proliferation in orthodontic treatment-induced gingival overgrowth. *Eur J Orthod*. 2007;*29*:555–558.
90. Geurtsen W. Biocompatibility of dental casting alloys. *Crit Rev Oral Biol Med*. 2002;*13*:71–84.
91. Muris J, Scheper RJ, Kleverlaan CJ, et al. Palladium-based dental alloys are associated with oral disease and palladium-induced immune responses. *Contact Dermatitis*. 2014;*71*:82–91.
92. Mizoguchi S, Setoyama M, Kanzaki T. Linear lichen planus in the region of the mandibular nerve caused by an allergy to palladium in dental metals. *Dermatology*. 1998;*196*:268–270.
93. Bruce GJ, Hall WB. Nickel hypersensitivity-related periodontitis. *Compend Contin Educ Dent*. 1995;*16*:178–184.
94. Gokcen-Rohlig B, Saruhanoglu A, Cifter ED, Evlioglu G. Applicability of zirconia dental prostheses for metal allergy patients. *Int J Prosthodont*. 2010;*23*:562–565.
95. da Fonte Porto Carreiro A, de Carvalho Dias K, Correia Lopes AL, Bastos Machado Resende CM, Luz de Aquino Martins AR. Periodontal conditions of abutments and non-abutments in removable partial dentures over 7 years of use. *J Prosthodont*. 2016;*25*:1–6.
96. Drake CW, Beck JD. The oral status of elderly removable partial denture wearers. *J Oral Rehabil*. 1993;*20*:53–60.
97. Kern M, Wagner B. Periodontal findings in patients 10 years after insertion of removable partial dentures. *J Oral Rehabil*. 2001;*28*:991–997.
98. Markkanen H, Lappalainen R, Honkala E, Tuominen R. Periodontal conditions with removable complete and partial dentures in the adult population aged 30 years and over. *J Oral Rehabil*. 1987;*14*:355–360.
99. Mojon P, Rentsch A, Budtz-Jorgensen E. Relationship between prosthodontic status, caries, and periodontal disease in a geriatric population. *Int J Prosthodont*. 1995;*8*:564–571.
100. Tuominen R, Ranta K, Paunio I. Wearing of removable partial dentures in relation to periodontal pockets. *J Oral Rehabil*. 1989;*16*:119–126.
101. Isidor F, Budtz-Jorgensen E. Periodontal conditions following treatment with distally extending cantilever bridges or removable partial dentures in elderly patients. A 5-year study. *J Periodontol*. 1990;*61*:21–26.
102. Ribeiro DG, Pavarina AC, Giampaolo ET, Machado AL, Jorge JH, Garcia PP. Effect of oral hygiene education and motivation on removable partial denture wearers: longitudinal study. *Gerodontology*. 2009;*26*:150–156.
103. Costa L, do Nascimento C, de Souza VO, Pedrazzi V. Microbiological and clinical assessment of the abutment and non-abutment teeth of partial removable denture wearers. *Arch Oral Biol*. 2017;*75*:74–80.
104. Tinanoff N, Manwell MA, Zameck RL, Grasso JE. Clinical and microbiological effects of daily brushing with either NaF or SnF2 gels in subjects with fixed or removable dental prostheses. *J Clin Periodontol*. 1989;*16*:284–290.
105. Berg E. Periodontal problems associated with use of distal extension removable partial dentures — A matter of construction. *J Oral Rehabil*. 1985;*12*:369–379.
106. Vanzeveren C, D'Hoore W, Bercy P. Influence of removable partial denture on periodontal indices and microbiological status. *J Oral Rehabil*. 2002;*29*:232–239.
107. Akaltan F, Kaynak D. An evaluation of the effects of two distal extension removable partial denture designs on tooth stabilization and periodontal health. *J Oral Rehabil*. 2005;*32*:823–829.
108. Bergman B, Hugoson A, Olsson CO. Caries, periodontal and prosthetic findings in patients with removable partial dentures: a ten-year longitudinal study. *J Prosthet Dent*. 1982;*48*:506–514.
109. Milward P, Katechia D, Morgan MZ. Knowledge of removable partial denture wearers on denture hygiene. *Br Dent J*. 2013;*215*(10):E20. https://doi.org/10.1038/sj.bdj.2013.1095. Published online ahead of print November 14, 2013.
110. Chandler JA, Brudvik JS. Clinical evaluation of patients eight to nine years after placement of removable partial dentures. *J Prosthet Dent*. 1984;*51*:736–743.
111. McHenry KR, Johansson OE, Christersson LA. The effect of removable partial denture framework design on gingival inflammation: a clinical model. *J Prosthet Dent*. 1992;*68*:799–803.
112. Ogunrinde TJ, Dosumu OO, Shaba OP, Akeredolu PA, Ajayi MD. The influence of the design of mandibular major connectors on gingival health. *Afr J Med Med Sci*. 2014;*43*:29–33.
113. Maeda Y, Kinoshita Y, Satho H, Yang TC. Influence of bonded composite resin cingulum rest seats on abutment tooth periodontal tissues: a longitudinal prospective study. *Int J Prosthodont*. 2008;*21*:37–39.
114. Dhingra K. Oral rehabilitation considerations for partially edentulous periodontal patients. *J Prosthodont*. 2012;*21*:494–513.
115. Bergman B. Periodontal reactions related to removable partial dentures: a literature review. *J Prosthet Dent*. 1987;*58*:454–458.
116. Kratochvil FJ, Davidson PN, Guijt J. Five-year survey of treatment with removable partial dentures. Part I. *J Prosthet Dent*. 1982;*48*:237–244.
117. Petridis H, Hempton TJ. Periodontal considerations in removable partial denture treatment: a review of the literature. *Int J Prosthodont*. 2001;*14*:164–172.
118. Kapur KK, Deupree R, Dent RJ, Hasse AL. A randomized clinical trial of two basic removable partial denture designs. Part I: comparisons of five-year success rates and periodontal health. *J Prosthet Dent*. 1994;*72*:268–282.
119. Budtz-Jorgensen E. Effects of denture-wearing habits on periodontal health of abutment teeth in patients with overdentures. *J Clin Periodontol*. 1994;*21*:265–269.
120. Budtz-Jorgensen E. Prognosis of overdenture abutments in elderly patients with controlled oral hygiene. A 5 year study. *J Oral Rehabil*. 1995;*22*:3–8.
121. Toolson LB, Smith DE, Phillips C. A 2-year longitudinal study of overdenture patients. Part II: assessment of the periodontal health of overdenture abutments. *J Prosthet Dent*. 1982;*47*:4–11.
122. Carlsson GE, Hedegard B, Koivumaa KK. Late results of treatment with partial dentures. An investigation by questionnaire and clinical examination 13 years after treatment. *J Oral Rehabil*. 1976;*3*:267–272.
123. Davis RK, Renner RP, Antos EW, Jr, Schlissel ER, Baer PN. A two-year longitudinal study of the periodontal health status of overdenture patients. *J Prosthet Dent*. 1981;*45*:358–363.
124. Ettinger RL, Jakobsen J. Periodontal considerations in an overdenture population. *Int J Prosthodont*. 1996;*9*:230–238.
125. Ettinger RL, Taylor TD, Scandrett FR. Treatment needs of overdenture patients in a longitudinal study: five-year results. *J Prosthet Dent*. 1984;*52*:532–537.
126. Gomes BC, Renner RP, Antos EW, Baer PN, Carlson M. A clinical study of the periodontal status of abutment teeth supporting swinglock removable partial dentures — A pilot study. *J Prosthet Dent*. 1981;*46*:7–13.
127. Keltjens HM, Schaeken MJ, van der Hoeven JS, Hendriks JC. Effects of chlorhexidine gel on periodontal health of abutment teeth in patients with overdentures. *Clin Oral Implants Res*. 1991;*2*:71–74.
128. Matthews DC, Tabesh M. Detection of localized tooth-related factors that predispose to periodontal infections. *Periodontol 2000*. 2004;*34*:136–150.
129. Lim HC, Jeon SK, Cha JK, Lee JS, Choi SH, Jung UW. Prevalence of cervical enamel projection and its impact on furcation involvement

in mandibular molars: a cone-beam computed tomography study in Koreans. *Anat Rec*. 2016;*299*:379–384.
130. Masters DH, Hoskins SW. Projection of cervical enamel into molar furcations. *J Periodontol*. 1964;*35*:49–53.
131. Grewe JM, Meskin LH, Miller T. Cervical enamel projections – Prevalence, location, and extent, with associated periodontal implications. *J Periodontol*. 1965;*36*:460–465.
132. Tsatsas B, Mandi F, Kerani S. Cervical enamel projections in the molar teeth. *J Periodontol*. 1973;*44*:312–314.
133. Shiloah J, Kopczyk RA. Developmental variations of tooth morphology and periodontal disease. *J Am Dent Assoc*. 1979;*99*:627–630.
134. Leib AM, Berdon JK, Sabes WR. Furcation involvements correlated with enamel projections from the cementoenamel junction. *J Periodontol*. 1967;*38*:330–334.
135. Bissada NF, Adbelmalek RG. Incidence of cervical enamel projections and its relationship to furcation involvement in Egyptian skulls. *J Periodontol*. 1973;*44*:583–585.
136. Hou GL, Tsai CC. Relationship between periodontal furcation involvement and molar cervical enamel projections. *J Periodontol*. 1987;*58*:715–721.
137. Hou GL, Tsai CC. Cervical enamel projection and intermediate bifurcational ridge correlated with molar furcation involvements. *J Periodontol*. 1997;*68*:687–693.
138. Zee KY, Bratthall G. Prevalence of cervical enamel projection and its correlation with furcation involvement in Eskimos dry skulls. *Swed Dent J*. 2003;*27*:43–48.
139. Blanchard SB, Derderian GM, Averitt TR, John V, Newell DH. Cervical enamel projections and associated pouch-like opening in mandibular furcations. *J Periodontol*. 2012;*83*:198–203.
140. Moskow BS, Canut PM. Studies on root enamel (2). Enamel pearls. A review of their morphology, localization nomenclature, occurrence, classification, histogenesis and incidence. *J Clin Periodontol*. 1990;*17*:275–281.
141. Kao MC, Lin CL, Kung CY, Huang YF, Kuo WC. Miniature endoscopic optical coherence tomography for calculus detection. *Appl Opt*. 2015;*54*:7419–7423.
142. Risnes S. The prevalence, location, and size of enamel pearls on human molars. *Scand J Dent Res*. 1974;*82*:403–412.
143. Goldstein AR. Enamel pearls as a contributing factor in periodontal breakdown. *J Am Dent Assoc*. 1979;*99*:210–211.
144. Cavanha AO. Enamel pearls. *Oral Surg Oral Med Oral Pathol*. 1965;*19*:373–382.
145. Withers JA, Brunsvold MA, Killoy WJ, Rahe AJ. The relationship of palato-gingival grooves to localized periodontal disease. *J Periodontol*. 1981;*52*:41–44.
146. Kogon SL. The prevalence, location and conformation of palato-radicular grooves in maxillary incisors. *J Periodontol*. 1986;*57*:231–234.
147. Gound TG, Maze GI. Treatment options for the radicular lingual groove: a review and discussion. *Pract Periodontics Aesthet Dent*. 1998;*10*:369–375.
148. Lejnes KN, Lie T, Selvig KA. Root grooves – A risk factor in periodontal attachment loss. *J Periodontol*. 1994;*65*:859–863.
149. Anderegg CR, Metzler DG. Treatment of the palato-gingival groove with guided tissue regeneration. Report of 10 cases. *J Periodontol*. 1993;*64*:72–74.
150. Andreana S. A combined approach for treatment of developmental groove associated periodontal defect. A case report. *J Periodontol*. 1998;*69*:601–607.
151. Dababneh R, Rodan R. Anatomical landmarks of maxillary bifurcated first premolars and their influence on periodontal diagnosis and treatment. *J Int Acad Periodontol*. 2013;*15*:8–15.
152. Gher ME, Vernino AR. Root morphology—Clinical significance in pathogenesis and treatment of periodontal disease. *J Am Dent Assoc*. 1980;*101*:627–633.
153. Albaricci MF, de Toledo BE, Zuza EP, Gomes DA, Rosetti EP. Prevalence and features of palato-radicular grooves: an in-vitro study. *J Int Acad Periodontol*. 2008;*10*:2–5.
154. Andreasen JO, Ahrensburg SS, Tsilingaridis G. Root fractures: the influence of type of healing and location of fracture on tooth survival rates – An analysis of 492 cases. *Dent Traumatol*. 2012;*28*:404–409.
155. Andreasen JO, Ahrensburg SS, Tsilingaridis G. Tooth mobility changes subsequent to root fractures: a longitudinal clinical study of 44 permanent teeth. *Dent Traumatol*. 2012;*28*:410–414.
156. Cvek M, Mejare I, Andreasen JO. Healing and prognosis of teeth with intra-alveolar fractures involving the cervical part of the root. *Dent Traumatol*. 2002;*18*:57–65.
157. Eichelsbacher F, Denner W, Klaiber B, Schlagenhauf U. Periodontal status of teeth with crown-root fractures: results two years after adhesive fragment reattachment. *J Clin Periodontol*. 2009;*36*:905–911.
158. Giachetti L, Bertini F, Rotundo R. Crown-root reattachment of a severe subgingival tooth fracture: a 15-month periodontal evaluation. *Int J Periodontics Restorative Dent*. 2010;*30*:393–399.
159. Grossmann Y, Arauz-Dutari J, Chogle SM, Blatz MB, Sadan A. A conservative approach for the management of a crown-root fracture. *Quintessence Int*. 2006;*37*:753–759.
160. Chan CP, Lin CP, Tseng SC, Jeng JH. Vertical root fracture in endodontically versus nonendodontically treated teeth. A survey of 315 cases in Chinese patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1999;*87*:504–507.
161. Nicopoulou-Karayianni K, Bragger U, Lang NP. Patterns of periodontal destruction associated with incomplete root fractures. *Dentomaxillofac Radiol*. 1997;*26*:321–326.
162. Lommel TJ, Meister F, Gerstein H, Davies EE, Tilk MA. Alveolar bone loss associated with vertical root fractures. Report of six cases. *Oral Surg Oral Med Oral Pathol*. 1978;*45*:909–919.
163. Luebke RG. Vertical crown-root fractures in posterior teeth. *Dent Clin North Am*. 1984;*28*:883–894.
164. Lustig JP, Tamse A, Fuss Z. Pattern of bone resorption in vertically fractured, endodontically treated teeth. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000;*90*:224–227.
165. Meister F, Jr, Lommel TJ, Gerstein H. Diagnosis and possible causes of vertical root fractures. *Oral Surg Oral Med Oral Pathol*. 1980;*49*:243–253.
166. Andreasen JO. External root resorptions: its implication in dental traumatology, pedodontics, periodontics, orthodontics, and endodontics. *Int Endodont J*. 1985;*18*:109–118.
167. Carrotte P. Endodontics: part 9. Calcium hydroxide, root resorption, endo-perio lesions. *Br Dent J*. 2004;*197*:735–743.
168. Bartok RI, Vaideanu T, Dimitriu B, Varlan CM, Suciu I, Podoleanu D. External radicular resorption: selected cases and review of the literature. *J Med Life*. 2012;*5*:145–148.
169. Darcey J, Qualtrough A. Resorption: part 1. Pathology, classification and aetiology. *Br Dent J*. 2013;*214*:439–451.
170. Haney JM, Leknes KN, Lie T, Selvig KA, Wikesjo UM. Cemental tear related to rapid periodontal breakdown – A case report. *J Periodontol*. 1992;*63*:220–224.
171. Ishikawa I, Oda S, Hayashi J, Arakawa S. Cervical cemental tears in older patients with adult periodontitis. Case reports. *J Periodontol*. 1996;*67*:15–20.
172. al-Jasser N, Hashim H. Periodontal findings in cases of incisor cross-bite. *Clin Pediatr Dent*. 1995;*19*:285–287.
173. Behlfelt K, Eriksson L, Jacobson L, Linder-Aronson S. The occurrence of plaque and gingivitis and its relationship to tooth alignment within the dental arches. *J Clin Periodontol*. 1981;*8*:329–337.
174. Ainamo J. Relationship between malalignment of the teeth and periodontal disease. *Scand J Dent Res*. 1972;*80*:104–110.

175. Helm S, Petersen PE. Causal relation between malocclusion and periodontal health. *Acta Odontol Scand*. 1989;*47*:223–228.
176. El-Mangoury NH, Gaafar S, Mostafa YA. Mandibular anterior crowding and periodontal disease. *Angle Orthod*. 1987;*57*:33–38.
177. Jensen BL, Solow B. Alveolar bone loss and crowding in adult periodontal patients. *Community Dent Oral Epidemiol*. 1989;*17*:47–51.
178. Årtun J, Osterberg SK. Periodontal status of secondary crowded mandibular incisors. *J Clin Periodontol*. 1987;*14*:261–266.
179. Geiger AM, Wasserman BH, Turgeon LR. Relationship of occlusion and periodontal disease Part VIII — Relationship of crowding and spacing to periodontal destruction and gingival inflammation. *J Periodontol*. 1974;*45*:43–49.
180. Jacobson L, Linder-Aronson S. Crowding and gingivitis: a comparison between mouth breathers and nose breathers. *Scand J Dent Res*. 1972;*80*:500–504.
181. Ingervall B, Jacobson U, Nyman S. A clinical study of the relationship between crowding of teeth, plaque and gingival condition. *J Clin Periodontol*. 1977;*4*:214–222.
182. Zawawi KH, Al-Zahrani MS. Gingival biotype in relation to incisors' inclination and position. *Saudi Med J*. 2014;*35*:1378–1383.
183. Cook DR, Mealey BL, Verrett RG, et al. Relationship between clinical periodontal biotype and labial plate thickness: an in vivo study. *Int J Periodontics Restorative Dent*. 2011;*31*:345–354.
184. Joshipura KJ, Kent RL, DePaola PF. Gingival recession – Intra-oral distribution and associated factors. *J Periodontol*. 1994;*65*:864–871.
185. Khocht A, Simon G, Person P, Denepitiya JL. Gingival recession in relation to history of hard toothbrush use. *J Periodontol*. 1993;*64*:900–905.
186. Paloheimo L, Ainamo J, Niemi M-L, Viikinkoski M. Prevalence of and factors related to gingival recession in Finnish 15- to 20-year old subjects. *Community Dent Health*. 1987;*4*:425–436.
187. Gorman WJ. Prevalence and etiology of gingival recession. *J Periodontol*. 1967;*38*:316–322.
188. Richman C. Is gingival recession a consequence of an orthodontic tooth size and/or tooth position discrepancy? "A paradigm shift". *Compend Contin Educ Dent*. 2011;*32*:62–69.
189. Kim YK, Kwon EY, Cho YJ, Lee JY, Kim SJ, Choi J. Changes in the vertical position of interdental papillae and interseptal bone following the approximation of anterior teeth. *Int J Periodontics Restorative Dent*. 2014;*34*:219–224.
190. Vermylen K, De Quincey GN, van 't Hof MA, Wolffe GN, Renggli HH. Classification, reproducibility and prevalence of root proximity in periodontal patients. *J Clin Periodontol*. 2005;*32*:254–259.
191. Vermylen K, De Quincey GN, Wolffe GN, van 't Hof MA, Renggli HH. Root proximity as a risk marker for periodontal disease: a case-control study. *J Clin Periodontol*. 2005;*32*:260–265.
192. Trossello VK, Gianelly AA. Orthodontic treatment and periodontal status. *J Periodontol*. 1979;*50*:665–671.
193. Årtun J, Kokich VG, Osterberg SK. Long-term effect of root proximity on periodontal health after orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1987;*91*:125–130.
194. Kim T, Miyamoto T, Nunn ME, Garcia RI, Dietrich T. Root proximity as a risk factor for progression of alveolar bone loss: the Veterans Affairs dental longitudinal study. *J Periodontol*. 2008;*79*:654–659.
195. Hirschfeld I. Food impaction. *J Am Dent Assoc*. 1930;*17*:1504–1528.
196. Hancock EB, Mayo CV, Schwab RR, Wirthlin MR. Influence of interdental contacts on periodontal status. *J Periodontol*. 1980;*51*:445–449.
197. Jernberg GR, Bakdash MB, Keenan KM. Relationship between proximal tooth open contacts and periodontal disease. *J Periodontol*. 1983;*54*:529–533.

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