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Potential risks of orthodontic therapy: a critical review and conceptual framework

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ABSTRACT

This review examines some of the potential risks of orthodontic therapy along with their evidence base. The risks of orthodontic treatment include periodontal damage, pain, root resorption, tooth devitalization, temporomandibular disorder, caries, speech problems and enamel damage. These risks can be understood to arise from a synergy between treatment and patient factors. In general terms, treatment factors that can influence risk include appliance type, force vectors and duration of treatment whilst relevant patient factors are both biological and behavioural. Hence, the natural variation between orthodontic treatment plans and patients gives rise to variations in risk. A good understanding of these risks is required for clinicians to obtain informed consent before starting treatment as well as to reduce the potential for harm during treatment. After considering each of these risks, a conceptual framework is presented to help clinicians better understand how orthodontic risks arise and may therefore be mitigated.

Keywords: Informed consent, orthodontics, risks, root resorption, temporomandibular disorder.

Abbreviations and acronyms: NiTi = nickel titanium; OIRR = orthodontic-induced root resorption; PDL = periodontal ligament; TMD = temporomandibular disorder; WSL = white spot lesions.

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INTRODUCTION

Like any medical therapy, orthodontic treatment exposes the patient to certain risks. From an ethical standpoint, the clinician must understand how these risks relate to each patient to ensure that they will receive a net benefit from treatment.[1] Failure to properly identify and manage the risks of orthodontic treatment cannot only give rise to patient dissatisfaction but also to litigation.[2] The risks of orthodontic treatment include periodontal damage, pain, root resorption, temporomandibular disorder, caries, speech problems and enamel damage. Because no two patients or orthodontic treatments can ever be identical in every respect, the question of whether a net benefit will accrue from treatment must be assessed on a case-by-case basis.

The purpose of this review is to provide a critical overview of the literature relating to the risks of orthodontic therapy. Special consideration is given to how different treatment and patient factors interact to modify the risk. A conceptual framework is presented at the end of the review to help clinicians better appreciate how orthodontic risks eventuate and how a thorough knowledge of both their patient and their appliance is needed to control these risks.

METHODS

Electronic databases including PubMed, Cochrane and Google Scholar were searched for keywords relating to the risks of orthodontic therapy. After reviewing abstracts, the full text of all relevant articles was accessed. Where articles referenced relevant studies that had not been found in the search strategy, these were also followed up. Only English-language articles from peer-reviewed journals were used. Although this strategy might have overlooked some relevant articles, it is consistent with the objective of providing an overview of what is essentially a very broad topic.

PERIODONTAL PROBLEMS

Orthodontic treatment can impact on the periodontium by promoting gingivitis, gingival recession and open gingival embrasures.

It is well-established that orthodontic appliances can impair plaque control leading to gingivitis.[3]

Whilst the associated gingival hyperplasia can be an aesthetic concern, the broader implications of gingivitis in an otherwise healthy periodontium are for, the most part, limited. However, gingivitis during orthodontics may result in periodontal breakdown in adults with active periodontal disease[4] and where a thin gingival biotype overlies an alveolar dehiscence.[5]

A number of retrospective studies have demonstrated that patients who have had orthodontic treatment are more likely to have gingival recession than individuals who have not.[6] Whilst this finding is not universal,[7] a systematic review of the evidence in this respect found that on average there is a small but significant detriment on the periodontium as a result of orthodontic therapy: 0.03 mm of recession, 0.23 mm increased pocket depth and 0.13 mm of alveolar bone loss.[8] The risk of periodontal breakdown may be higher in adult orthodontic patients. A radiographic follow-up study of 343 adult orthodontic patients reported that although mean bone loss on anterior teeth was 0.54 mm, over one-third of patients had bone loss exceeding 2 mm and this positively correlated with age.[9]

Orthodontics can challenge periodontal stability by moving the roots of teeth outside of their alveolar housing and thinning the attached gingiva.[10] The labial aspect of the lower incisors is particularly vulnerable to recession.[6] Hence, procedures which advance lower incisors (such as alignment of crowding and class II mechanics) need to be undertaken with this in mind. Despite some manufacturers' claims to the contrary, self-ligating brackets do not promote alveolar bone formation as teeth are moved facially.[11]

The modern trend towards non-extraction treatments[12] has made this topic particularly relevant. Whilst anecdotal evidence suggests that incisor advancement can predispose to gingival recession, population-based studies have failed to consistently demonstrate a relationship between the two.[13] Furthermore, a large follow-up study comparing extraction and non-extraction patients failed to show any differences in their experience of recession.[14] Despite the potential for fixed lingual retainers to impede oral hygiene, the evidence would suggest they cause minimal detriment to the periodontium over the long term.[15]

Ultimately, the multifactorial nature of periodontal attachment loss makes it difficult to quantify the contribution of orthodontic therapy. Known risk factors include age, gingival biotype, smoking, oral hygiene habits, oral piercings, frenal attachment and plaque control.[10,16] Furthermore, any comparisons that can be drawn between studies are confounded by variations in the starting malocclusion, treatment mechanics and the observation period.[13] Therefore, it is likely that there is a subset of patients with enough of these risk factors to experience clinically significant attachment loss as an outcome of orthodontic therapy. With this in mind, an individual assessment of recession risk should inform the treatment planning of the orthodontic patient and proper oral hygiene instruction should be given.[17]

Open gingival embrasures

Open gingival embrasures (or 'black triangles') occur when the interdental papilla is lost in the aesthetic zone. Whilst they can be a result of periodontal disease, they are often aetiologically distinct from gingival recession. The presence of the papilla has been linked to age, tooth morphology, proximal contact length, proximal bone height and interproximal gingival thickness.[18] Tarnow *et al.* demonstrated that open gingival embrasures are more likely to occur when the distance between the alveolar bone and the tooth contact point exceeds 5 mm.[19] Hence, orthodontic tooth movement may cause an open gingival embrasure by diverging roots.[20] Other risk factors include triangular crown morphology, teeth being in a pretreatment position where the papilla has not completely formed and the embrasure morphology itself.[21] Apart from being unaesthetic, open embrasures promote food impaction. Management strategies include bracket repositioning, reshaping of crowns and restorative treatments.

PAIN

Virtually any stage of orthodontic treatment has the potential to cause pain. Fear of pain can be a deterrent to individuals starting orthodontic treatment.[22] During treatment, pain has been shown to lessen patient compliance[23] and has also been cited as a common reason for stopping early.[24] In broad terms, two common types of pain can arise in the course of orthodontic therapy: mucosal pain from appliance trauma to the oral soft tissues and periodontal/pulpal pain from the application of orthodontic forces to the teeth.

Oral mucosal pain

Most patients undergoing treatment with fixed appliances experience oral mucosal pain at some point and for some people this can rate as the most annoying part of treatment.[25] However, this topic has not been well studied.[26] Baricevic *et al.* reported that orthodontic brackets tended to cause mucosal erosions and desquamations whereas archwires caused ulcerations.[26] It is unsurprising that the pattern of mucosal ulceration reflects the location of the appliance: lingual appliances tend to ulcerate the tongue whilst

buccal appliances tend to ulcerate the cheeks.[27] The location of the ulceration greatly impacts morbidity; the constant activity of the tongue makes lingual ulcerations more debilitating than buccal ones.[28] Mucosal irritation and discomfort can also arise during treatment with clear aligners (e.g. Invisalign) although this does not seem to rate as a significant concern to patients.[29]

Periodontal/pulp pain

Orthodontic forces against teeth can cause pain by compressing the vasculature in the periodontal ligament (PDL) resulting in inflammation of both the pulp and periodontal tissues.[30] In this respect, fixed appliances produce more pain than removable or functional appliances.[31]

After the placement of the initial archwire in a fixed appliance, the majority of patients will experience pain starting at 4 h which then peaks at 24 h and declines over the next 3 days.[32] After an adjustment visit, pain increases then tapers off over 2–3 days, giving rise to a cyclic pattern of pain throughout the course of treatment.[33]

Whilst the archwire sequence may not significantly contribute to the overall pain experience,[34] stiffer wires can result in a higher peak pain level.[35] Heat-activated nickel titanium (NiTi) wires may also cause less pain than regular NiTi wires.[36] Whilst some evidence supports passive self-ligating brackets being less painful than conventional brackets during initial alignment,[37] a recent meta-analysis concluded there was a lack of a clinically significant difference between the two bracket types in this respect.[38] However, active self-ligating brackets have been shown to be more painful than conventional brackets when engaging rectangular archwires.[39] The overall pain experience with lingual and labial appliances seems to be comparable.[40]

Patients can often experience pain at orthodontic debands and this appears to be related to the level of tooth mobility, as well as the direction of the forces used to remove the fixed appliance.[41] Ceramic brackets may be more painful to remove than metal brackets as the latter are more ductile and require less force to remove.[42]

Patients undergoing Invisalign treatment experience pain over a similar timescale to fixed appliance patients, although the evidence suggests that the overall intensity of pain in Invisalign patients is significantly less.[29] Indeed a proportion of Invisalign patients may report no pain during treatment[43] which has been attributed to the small incremental tooth movements made by consecutive aligners.[43] It is important to bear in mind that the Invisalign material and treatment protocols have evolved since these studies were published, which may alter the relevance of these findings.

Two possible therapeutic targets exist to ease periodontal/pulpal pain during orthodontic therapy: reducing inflammation and increasing blood flow within the PDL. Randomized clinical trials have demonstrated that anti-inflammatory analgesics perform better than placebo drugs at reducing pain following the initiation of orthodontic forces.[44,45] A meta-analysis by Xioting *et al.* concluded that ibuprofen, aspirin and paracetamol were equally effective in this respect, with the latter being preferred due to a better safety profile and less potential impact on tooth movement.[46]

Chewing wafers or gum can provide pain relief to orthodontic patients by disrupting compressive forces from appliances and allowing an intermittent resumption of blood flow to the PDL. Hence, both have been shown to be effective in reducing pain and may be as equally as effective as analgesics.[47] Similarly, PDL blood flow can be increased by applying low-intensity laser light to gingival areas around the teeth and this has been shown to outperform placebo for analgesia.[48] Although vibration could theoretically provide analgesia by increasing PDL blood flow as well, the evidence that vibration appliances reduce orthodontic pain is conflicting.[33,49]

A discussion of orthodontic pain would not be complete without considering the psychosocial dimension of the pain experience, which probably outweighs orthodontic factors in explaining patients' pain experiences.[50] Whilst gender and age differences are not consistently related to orthodontic pain,[41] there are clear 'non-linear' relationships between age, gender and pain: adolescents tend to report more pain than pre-adolescents and adults, and females more than males.[31] Furthermore, emotional states (such as anxiety)[51] and social interactions (such as experiencing empathy)[52] also influence how individuals experience pain during orthodontic treatment.

ROOT RESORPTION

As teeth undergo orthodontic movement, resorption of cementum and dentine may also take place. This process has been termed 'orthodontic-induced root resorption' (OIRR).[53] Whilst it has been shown that resorptive craters can heal by cementum deposition,[54] roots will become permanently shortened if the resorption separates an apical region from the rest of the root.[30]

Because OIRR is asymptomatic, it can only be diagnosed radiographically or histologically. Due to the potential for distortions, panoramic and periapical radiographs taken with the bisecting angle technique are considered to be less accurate than periapical films

taken with a paralleling technique.[55] Cone-beam studies have demonstrated good potential in this respect;[56] however, the degree of radiation exposure makes their routine use for diagnosing OIRR questionable.

Radiographic studies suggest that, overall, 48–66% of orthodontically treated teeth experience OIRR in the order of 2 mm or less.[53] Anterior teeth are more susceptible to OIRR and 1–5% will experience more than 4 mm of root shortening.[57] Histological studies, on the other hand, suggest that greater than 90% of teeth undergo some extent of OIRR.[53] The discrepancy between radiographic and histological data reflects the fact that not all resorptive lesions progress to root shortening, which is the outcome measurable on a two-dimensional radiograph.

It has been observed that susceptibility to OIRR varies between individuals.[58] Whilst genetics accounts for over half of this variation,[59] a diverse array of other patient-related factors have been associated with OIRR including age, tooth morphology, certain drugs, hormone deficiencies, hypothyroidism, hypopituitarism, alveolar bone density, root morphology, chronic alcoholism, gender, root proximity to the cortical bone, gender and the severity and type of malocclusion.[53]

The nature of the orthodontic treatment itself can also influence the experience of OIRR, although the evidence only supports 'light forces' as being protective against OIRR.[53] A recent systematic review of orthodontic forces and OIRR was unable to make any recommendation about an appropriate force level in this respect.[60]

A temporal association also exists between orthodontic forces and OIRR because intermittent forces are associated with less OIRR than continuous ones.[60] This could explain the observations that removable appliances have been shown to cause less OIRR than fixed appliances[61] and that clear aligner therapy causes similar amounts of OIRR to fixed appliances that use a 'controlled light force'.[62] There also appears to be a positive correlation between overall treatment time and OIRR[60] and pauses in treatment can reduce OIRR.[63] On the other hand, teeth treated with self-ligating appliances do not show less OIRR,[64] which is consistent with data that self-ligating appliances do not reduce overall treatment time compared with conventional ligation,[38] and also suggests that the forces of the two systems are comparable.

Although an individual's inherent susceptibility to OIRR may not be modifiable by the clinician, steps can still be taken to identify those individuals who may be at a higher risk. In light of the evidence for a genetic basis,[65] consideration of the orthodontic experiences of family members may help to identify individuals who are particularly prone to OIRR.[66] Furthermore, tooth-related factors that predispose individuals to OIRR should be recognized, such as a history of trauma[67] or abnormal root shape.[68]

Radiographic screening at 6 months from the beginning of treatment has been recommended to identify those patients who are prone to OIRR.[58] Those individuals who show signs of OIRR should have biannual radiographs thereafter for the duration of treatment.[58] Where OIRR is a concern, the orthodontic force should be discontinued and this will effectively halt the process.[69]

Despite the prevalence of OIRR, there is little data in the literature on the long-term prognosis of teeth which have shortened roots and the implications of OIRR mentioned in the literature are hypothetical. Long-term follow-up studies have demonstrated that hypermobility in these teeth is rare[69] and is only observed in extreme cases.[70] This is explained by the relatively small contribution of the apical area of teeth to the overall periodontal support.[71] Kalkwarf *et al.* estimated apical root loss of 3 mm in a maxillary incisor was equivalent to a loss of 1 mm of alveolar periodontal support.[72] It has been suggested that teeth with OIRR may make poorer abutments in fixed prosthodontics[72] and may be more compromised if periodontal disease develops.[70]

TOOTH DEVITALIZATION

Orthodontic forces can causes changes in pulpal blood flow by compressing the neurovascular bundle. Whilst a number of studies have shown histological and inflammatory changes in the pulp as a result of orthodontic forces,[73] a recent review of the effects of orthodontic force on the pulp concluded that this area remains poorly understood.[74]

Early research flagged possible concerns that heavy orthodontic forces could cause pulpal necrosis by strangulation.[75] Although it is clear that the orthodontic force can cause pulpal ischaemia and degenerative changes, modern evidence suggests that the pulp is remarkably robust in resisting heavy forces[76] and that necrosis of otherwise healthy teeth during orthodontic therapy is a rare phenomenon.[74] However, teeth with a history of trauma may have a compromised vascular supply which places them at a higher risk of devitalization during orthodontic therapy.[77]

Vascular supply may also be lost in instances where root apices are moved outside of the alveolar bone[78] although reports also exist of teeth preserving vitality where bonded lingual retainers inadvertently torqued root apices beyond the cortex.[79] This suggests that slower tooth movements are less likely to sever the neurovascular bundle.

Because increased overjet is a known risk factor for incisor trauma,[80] a large proportion of orthodontic patients may have a history of incisor trauma.[81] Therefore, it is necessary to screen potential orthodontic patients for a history of dental trauma and to warn of potential loss of vitality before starting treatment. Teeth with a history of trauma, particularly those showing signs of pulp canal obliteration, should be moved with caution.[82]

Caution should also be exercised when assessing pulpal vitality of teeth undergoing orthodontic movement due to the chance of false negatives. Indeed, during orthodontic movement, vital teeth may have an increased response threshold or transient negative response when being pulp tested.[76] It has been suggested that radiographic changes in the apical region may be a more reliable marker of pulpal necrosis than vitality testing;[77] however, if necrosis occurs in the absence of infection then apical changes may not be evident.[83] Furthermore, radiographic changes around a root apex can appear for reasons other than pulpal infection. Therefore, clinicians should look for multiple signs and symptoms of pulpal necrosis before making a diagnosis of pulpal necrosis.

TEMPOROMANDIBULAR DISORDER

The diagnosis 'temporomandibular disorder' (TMD) lacks a universally accepted definition.[84] Okeson has described TMD as the signs and symptoms associated with masticatory dysfunction arising from the temporomandibular joint, the dentition and muscles.[85]

The possible relationship between orthodontics and TMD is closely related to the question of how occlusion may influence TMD in general. There is a perception that the contribution of occlusion to TMD has been historically overstated by the dental profession,[86] and that it may be more appropriately managed with a medical rather than a dental paradigm.[87]

The incidence of TMD is known to increase with age and the increasing number of adults seeking orthodontic treatment has made this topic particularly relevant.[87] Furthermore, adults may be at a theoretically higher risk of TMD from orthodontic therapy if the adaptability of their stomatognathic system is less than that of a younger person.[88]

Up until the late 1980s, comparatively little research had been undertaken to explore the relationship between orthodontics and TMD. This changed in 1987 when a US court awarded substantial damages to a patient who sued their orthodontist for 'causing' TMD.[89] Since this time, successive investigations have demonstrated that there is insufficient evidence to suggest that orthodontic therapy prevents, causes or treats TMD.[84,89–96] However, as Michelotti and Iodice pointed out in their review of this topic, the absence of evidence is not the same as the evidence of absence.[95]

Designing and executing a study capable of demonstrating a causal link between orthodontic therapy and TMD would be exceptionally challenging. In addition to controlling for the starting malocclusion and the type of orthodontic therapy, a proper evaluation would need to control for a large number TMD-related associations. These include age, social class, ethnicity, psychosocial status, gender, trauma history, ethnicity, parafunctional activities, third molar removal, co-existing pain conditions and genetics.[65,84,90] Furthermore, the challenges of defining and diagnosing TMD would need to be overcome as well as the potential for the placebo effect and inter-observer variability. Hence, Luther's observation that 'to date there has never been a satisfactory evaluation of orthodontics with respect to TMD'.[94]

Longitudinal, retrospective and cross sectional studies comparing the incidence of TMD in patients with and without a history of orthodontic treatment cannot demonstrate causality, but may highlight possible associations. Where such studies have taken place, they have failed to show an association between orthodontic therapy and TMD.[97,98] However, evidence is emerging that there could be a subset of patients who have a genetic predisposition to TMD from orthodontics.[65] Hence, it may be an oversimplification to state that there is no relationship between orthodontic treatment and TMD.

Despite the equivocal relationship between orthodontics and TMD, clinicians still have a medicolegal duty to screen potential patients for TMD and refer them appropriately when a positive finding is made.[2] Furthermore, it may be prudent to avoid starting treatment in cases where TMD already exists.[95] Patients with a history of TMD should understand that their condition could potentially stabilize, get worse or improve as a result of orthodontic therapy.[2,87]

ENAMEL DECALCIFICATION

Orthodontic appliances increase caries risk by promoting plaque accumulation and inhibiting oral hygiene.[99] White spot lesions (WSL) are one of the most common adverse effects of orthodontic therapy.[100] They arise from the refractive index change that accompanies enamel decalcification and may progress to cavitation.[101] Between 1975 and 2011, Gorelick *et al.*'s landmark study[102] on the incidence of WSL in orthodontic patients was the fourth most cited publication in the orthodontic literature.[103] Most WSL studies have been concerned with fixed appliances because the risk of their formation with removable appliances is low.[104]

During orthodontic treatment, WSL can be difficult to identify because complete plaque removal and enamel desiccation is needed for proper visualization.[105] Clinical evidence of WSL can occur as early as 4 weeks after fixed appliance placement,[106] which is comparable with non-orthodontic patients who do not brush their teeth.[107]

A recent literature review reported that that the prevalence of WSL ranged 2–97% of orthodontic patients.[100] This wide range has been attributed to differences in WSL measurement techniques[100] and failure to differentiate between pre-existing and new WSL during orthodontic treatment.[108] It may also reflect the multifactorial nature of caries risk. Younger patients are at greater risk due to lack of enamel maturity and a tendency to have poorer oral hygiene habits.[109] There is some evidence that male orthodontic patients are more affected by WSL than females,[110] although other studies have found the prevalence of WSL across genders to be no different.[102]

Saliva has a protective effect against WSL formation. Hence, maxillary teeth are generally more susceptible to WSL than mandibular teeth[111] and lingual appliances seem to be at low risk of promoting WSL development.[112]

After fixed appliances are removed, saliva starts to remineralize WSL.[113] This process is initially rapid but slows after several weeks.[114] Whether a Hawley or Essex type retainer is worn does not appear to influence the natural remineralization process.[114] A residual WSL may persist due to superficial remineralization,[108] particularly if a high concentration of fluoride is applied soon after deband.[115] The benefits of products containing casein phosphopeptide-amorphous calcium phosphate for WSL remineralization are not clear.[116] Other methods reported in the literature to manage WSL include resin infiltration[117] and enamel micro-abrasion.[118]

It is a truism that prevention of WSL is preferable to their post-orthodontic management. Because caries cannot form in the absence of plaque and fermentable carbohydrates,[119] a good diet and proper oral hygiene in combination with a regular fluoride regimen should be at the forefront of any strategy to manage WSL. Because patients may have an inherently higher susceptibility to caries due to salivary dysfunction, enamel solubility, medications and genetics,[100] an individualized caries prevention plan should be instituted for each orthodontic patient. Furthermore, clinicians should be wary of starting orthodontic treatment in patients with low motivation because this is associated with poor oral hygiene in orthodontic patients.[120]

SPEECH PROBLEMS

Orthodontic appliances may affect speech directly by impeding the articulation of sounds or indirectly by affecting the physical and mental health of a person.[109] Although the potential for orthodontic appliances to hinder speech has been of research interest for over 60 years,[121] professional orthodontic associations do not always address this topic in their public education campaigns regarding the risks of orthodontic treatment.[122]

The effect of orthodontic appliances on speech is primarily an issue when the lingual space is encroached upon. Hence, patients report that removable appliances affect speech more than fixed labial appliances.[123] The speech recovery time for bonded palatal expanders[124] and Hawley retainers[109] tends to be approximately 1 week. This is similar to the adaptation time for full upper dentures,[125] which may suggest that age is not a significant factor for speech adaptation in this respect. Indeed, one study involving bonded palatal expanders found no relationship between patient age and the time for speech adaptation.[124] Speech adaptation may be quicker if the thickness and amount of palatal coverage of an appliance is minimized.[126] This observation may explain the seemingly mild and short-lived impact of Invisalign on speech for many patients.[28,43] It should be noted however that these studies did not consider the impact of newer aligner features that place auxiliary features on the palatal surface of the upper incisors (e.g. bite ramps) which may affect speech.

The effect of fixed lingual appliances on speech is also well recognized. Apart from encroaching on surfaces required for phonation, lingual appliances can interfere with speech by causing ulceration to the tongue.[127] Shalish *et al.* compared the impact of Invisalign, labial and lingual appliances on quality of life and found that patients reported difficulty speaking for an average 2, 4 and 6 days, respectively.[28] Other studies have put the speech recovery time for lingual appliances between 1[128] and 3 months.[129]

ENAMEL DAMAGE

Removal of fixed orthodontic appliances (debanding) involves applying a force to disrupt bonding between the tooth surface and the appliance. The result of this force application will either be cohesive failure within the orthodontic resin itself or an adhesive failure at the interface of the tooth or the bonding surface of the appliance.[130] Enamel may be damaged in the debanding process or during cleanup of residual orthodontic cement.

Adhesive bracket failure can remove enamel if the strength of the micromechanical bond between enamel and the bonding resin exceeds the cohesive strength of the enamel itself. Ceramic brackets using a chemical bonding interface (rather than micromechanical) are at higher risk of damaging enamel due to the strength of the bond.[131] The incidence of enamel fracture from ceramic bracket removal is reported in the literature to range 10–35%.[132] It is clear that not all ceramic brackets are equal in this respect and newer generations of ceramic brackets may be less likely to fracture enamel upon debonding.[133]

At present, there are no methods to remove residual orthodontic resin that are completely atraumatic to the tooth surface.[134] Depending on the technique employed, approximately 20–50 μm of enamel is lost during resin cleanup,[135] and scratches and gouges will inevitably be left on the enamel surface.[136] Any removal of surface enamel exposes prism rods and can theoretically increase susceptibility to acid dissolution.[137] The inevitable scratching and gouging of the enamel surface from resin removal with rotary instruments has been postulated to increase susceptibility to caries and staining,[136] although this risk has been downplayed by others.[138] In the absence of clinical investigations in this respect, it is worthwhile considering that long-term follow-up studies of teeth which have had enamel ground for other purposes (such as interproximal reduction[139] or aesthetic reshaping)[140] have shown no detriment where the surface has been left smooth.

RISKS OF ORTHODONTIC TREATMENT AS A CONCEPTUAL FRAMEWORK

As the foregoing discussion demonstrates, orthodontic therapy inevitably produces a biological challenge to the stomatognathic system. The outcome of this challenge is dependent upon both the nature of the treatment that is performed and patient-related factors. Whilst some aspects of patient susceptibility to the risks are essentially fixed (e.g. genetics), others are modifiable (e.g. oral hygiene). Fig. 1 presents a conceptual framework to illustrate, in general terms, how the risk of adverse outcomes in orthodontic therapy materializes through a synergy between the treatment and the patient.

In this framework, an adverse outcome will be the result of the treatment challenge exceeding the patient's resistance and adaptability in some respect. Although this framework has natural limitations, it is hoped that it will help clinicians better appreciate the importance of having a sound understanding of the orthodontic appliances they use as well as those patient characteristics that can impact upon treatment.

Finally, clinicians must also carefully manage patients' expectations as part of their overall risk management strategy. From a medicolegal perspective, a very real risk of orthodontic treatment is patient disappointment with an intended or accidental treatment outcome. Treatment goals should represent an agreement between the patient and the clinician, and

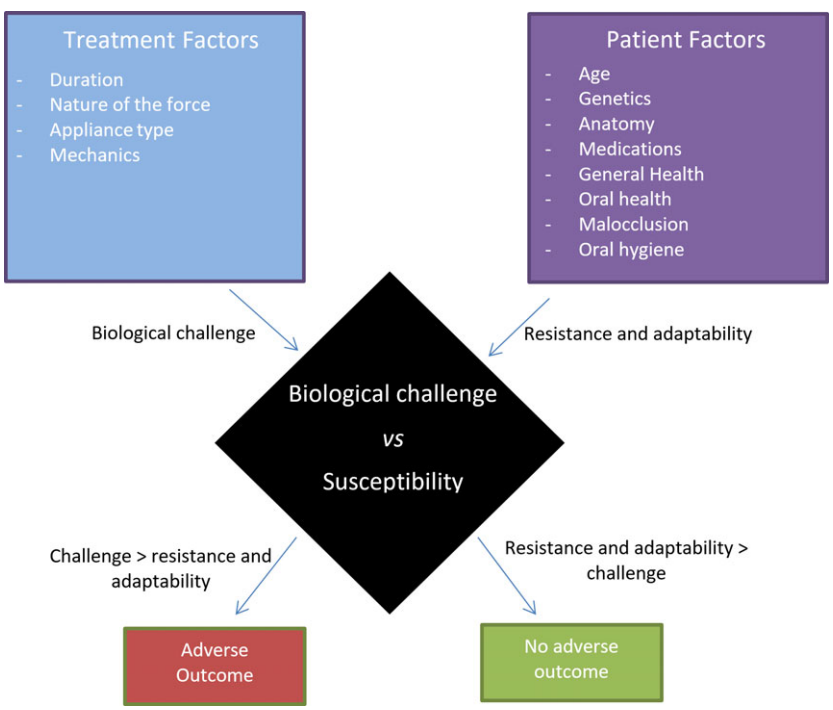

Fig. 1 A conceptual framework to explain the risks of orthodontic therapy.

clinicians must therefore be honest with themselves and patients about whether treatment objectives are realistic. Whilst an adverse outcome may not necessarily be construed as negligence, failure to properly warn about it beforehand could be.

CONCLUSIONS

This review has considered some of the main risks of orthodontic treatment by way of an overview of relevant literature. It has been shown that the risks of orthodontic treatment vary between individuals and treatment plans. Clinicians should develop treatment plans in light of an assessment of their patients' susceptibility to these risks and patients should be duly informed of these risks as part of informed consent. Doing so inevitably requires a degree of experience and skill on the part of the clinician. In light of this, a one-size-fits-all treatment philosophy is liable to expose patients to a higher risk of adverse outcomes.

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REFERENCES

1. Gillon R. Medical ethics: four principles plus attention to scope. BMJ 1994;309:184–188.
2. Abdelkarim A, Jerrold L. Risk management strategies in orthodontics. Part 1: clinical considerations. Am J Orthod Dentofac Orthop 2015;148:345–349.
3. Naranjo AA, Triviño ML, Jaramillo A, Betancourth M, Botero JE. Changes in the subgingival microbiota and periodontal parameters before and 3 months after bracket placement. Am J Orthod Dentofacial Orthop 2006;130:275.e17–275.e22.
4. Boyd RL, Leggott PJ, Quinn RS, Eakle WS, Chambers D. Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissues versus those of adolescents. Am J Orthod Dentofacial Orthop 1989;96:191–198.
5. Zachrisson BU. Tooth movements in the periodontally compromised patient. In: Lindhe J, Karring T, Lang N, eds. Clinical Periodontology and Implant Dentistry. Copenhagen; Munksgaard: Wiley-Blackwell, 2008: 1271.
6. Renkema AM, Fudalej PS, Renkema AAP, Abbas F, Bronkhorst E, Katsaros C. Gingival labial recessions in orthodontically treated and untreated individuals: a case – control study. J Clin Periodontol 2013;40:631–637.
7. Gomes SC, Varela CC, da Veiga SL, Rösing CK, Oppermann RV. Periodontal conditions in subjects following orthodontic therapy. A preliminary study. Eur J Orthod 2007;29:477–481.
8. Bollen A-M, Cunha-Cruz J, Bakko DW, Huang GJ, Hujoel PP. The effects of orthodontic therapy on periodontal health: a systematic review of controlled evidence. J Am Dent Assoc 2008;139:413–422.
9. Nelson PA, Artun J. Alveolar bone loss of maxillary anterior teeth in adult orthodontic patients. Am J Orthod Dentofac Orthop 1997;111:328–334.
10. Wennström J-L. Mucogingival considerations in orthodontic treatment. Semin Orthod 1996;2:46–54.
11. Wright N, Modarai F, Cobourne MT, Dibiase AT. Do you do Damon? What is the current evidence base underlying the philosophy of this appliance system? J Orthod 2011;38:222–230.
12. Janson G, Maria FRT, Bombonatti R. Frequency evaluation of different extraction protocols in orthodontic treatment during 35 years. Prog Orthod 2014;15:51.
13. Renkema AM, Navratilova Z, Mazurova K, Katsaros C, Fudalej PS. Gingival labial recessions and the post-treatment proclination of mandibular incisors. Eur J Orthod 2015;37:508–513.
14. Villard NM, Patcas R. Does the decision to extract influence the development of gingival recessions? J Orofac Orthop 2015;76:476–492.
15. Booth FA, Edelman JM, Proffit WR. Twenty-year follow-up of patients with permanently bonded mandibular canine-to-canine retainers. Am J Orthod Dentofac Orthop 2008;133:70–76.
16. Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. J Am Dent Assoc 2003;134:220–225.
17. Johal A, Katsaros C, Kuijpers-Jagtman AM. State of the science on controversial topics: missing maxillary lateral incisors–a report of the Angle Society of Europe 2012 meeting. Prog Orthod 2013;14:20.
18. Chow YC, Eber RM, Tsao YP, Shotwell JL, Wang HL. Factors associated with the appearance of gingival papillae. J Clin Periodontol 2010;37:719–727.
19. Tarnow DP, Magner AW, Fletcher P. The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal dental papilla. J Periodontol 1992;63:995–996.
20. Kurth JR, Kokich VG. Open gingival embrasures after orthodontic treatment in adults: prevalence and etiology. Am J Orthod Dentofac Orthop 2001;120:116–123.
21. Sharma AA, Park JH. Esthetic considerations in interdental papilla: remediation and regeneration. J Esthet Restor Dent 2010;22:18–28.
22. Oliver RG, Knapman YM. Attitudes to orthodontic treatment. Br J Orthod 1985;12:179–188.
23. Sergl HG, Klages U, Zentner A. Functional and social discomfort during orthodontic treatment–effects on compliance and prediction of patients' adaptation by personality variables. Eur J Orthod 2000;22:307–315.
24. Brown DF, Moerenhout RG. The pain experience and psychological adjustment to orthodontic treatment of preadolescents, adolescents, and adults. Am J Orthod Dentofacial Orthop 1991;100:349–356.
25. Kvam E, Gjerdet NR, Bondevik O. Traumatic ulcers and pain during orthodontic treatment. Community Dent Oral Epidemiol 1987;15:104–107.
26. Baricevic M, Mravak-Stipetic M, Majstorovic M, Baranovic M, Baricevic D, Loncar B. Oral mucosal lesions during orthodontic treatment. Int J Paediatr Dent 2011;21:96–102.
27. Caniklioglu C, Oztürk Y. Patient discomfort: a comparison between lingual and labial fixed appliances. Angle Orthod 2005;75:86–91.
28. Shalish M, Cooper-Kazaz R, Ivgi I, Canetti L, Tsur B, Bachar E, *et al.* Adult patients' adjustability to orthodontic appliances. Part I: a comparison between Labial, Lingual, and Invisalign™. Eur J Orthod 2012;34:724–730.
29. Fujiyama K, Honjo T, Suzuki M, Matsuoka S, Deguchi T. Analysis of pain level in cases treated with Invisalign aligner: comparison with fixed edgewise appliance therapy. Prog Orthod 2014;15:64.

30. Proffit WR, Fields HW, Sarver DM. Contemporary Orthodontics. St Louis, Missouri: Mosby, Inc., 2013:301–302.
31. Krishnan V. Orthodontic pain: from causes to management–a review. Eur J Orthod 2007;29:170–179.
32. Erdinç AME, Dinçer B. Perception of pain during orthodontic treatment with fixed appliances. Eur J Orthod 2004;26:79–85.
33. Lobre WD, Callegari BJ, Gardner G, Marsh CM, Bush AC, Dunn WJ. Pain control in orthodontics using a micropulse vibration device: a randomized clinical trial. Angle Orthod 2015;1–6.
34. Mandall NA, Lowe C, Worthington HV, Sandler J, Derwent S, Abdi-Oskouei M, *et al*. Which orthodontic archwire sequence? A randomized clinical trial. Eur J Orthod 2006;28:561–566.
35. Sandhu SS, Sandhu J. A randomized clinical trial investigating pain associated with superelastic nickel-titanium and multistranded stainless steel archwires during the initial leveling and aligning phase of orthodontic treatment. J Orthod 2013;40:276–285.
36. Cioffi I, Piccolo A, Tagliaferri R, Paduano S, Galeotti A, Martina R. Pain perception following first orthodontic archwire placement–thermoelastic vs superelastic alloys: a randomized controlled trial. Quintessence Int 2012;43:61–69.
37. Pringle AM, Petrie A, Cunningham SJ, McKnight M. Prospective randomized clinical trial to compare pain levels associated with 2 orthodontic fixed bracket systems. Am J Orthod Dentofacial Orthop 2009;136:160–167.
38. Čelar A, Schedlberger M, Dörfler P, Bertl M. Systematic review on self-ligating vs. conventional brackets: initial pain, number of visits, treatment time. J Orofac Orthop 2013;74:40–51.
39. Bertl MH, Onodera K, Čelar AG. A prospective randomized split-mouth study on pain experience during chairside archwire manipulation in self-ligating and conventional brackets. Angle Orthod 2013;83:292–297.
40. Wu AKY, McGrath C, Wong RWK, Wiechmann D, Rabie ABM. A comparison of pain experienced by patients treated with labial and lingual orthodontic appliances. Eur J Orthod 2010;32:403–407.
41. Bergius M, Kiliaridis S, Berggren U. Pain in orthodontics: a review and discussion of the literature. J Orofac Orthop 2000;37:125–137.
42. Williams OL, Bishara SE, Ortho D. Patient discomfort levels at the time of debonding: a pilot study. Am J Orthod Dentofac Orthop 1992;101:313–317.
43. Nedwed V, Miethke R-R. Motivation, acceptance and problems of invisalign patients. J Orofac Orthop 2005;66:162–173.
44. Patel S, McGorray SP, Yezierski R, Fillingim R, Logan H, Wheeler TT. Effects of analgesics on orthodontic pain. Am J Orthod Dentofac Orthop 2011;139:e53–e58.
45. Steen Law SL, Southard KA, Alan S, Logan HL, Jakobsen JR. An evaluation of preoperative ibuprofen for treatment of pain associated with orthodontic separator placement. Am J Orthod Dentofac Orthop 2000;118:629–635.
46. Xiaoting L, Yinb T, Yangxic C. Interventions for pain during fixed orthodontic appliance therapy A systematic review. Angle Orthod 2010;80:925–932.
47. Farzanegan F, Zebarjad SM, Alizadeh S, Ahrari F. Pain reduction after initial archwire placement in orthodontic patients: a randomized clinical trial. Am J Orthod Dentofacial Orthop 2012;141:169–173.
48. Dominguez A, Velasquez SA. Effect of low level laser therapy on pain following activation of orthodontic final archwires: a randomized controlled clinical trial. Photomed Laser Surg 2012;31:36–40.
49. Miles P, Smith H, Weyant R, Rinchuse DJ. The effects of a vibrational appliance on tooth movement and patient discomfort: a prospective randomised clinical trial. Aust Orthod J 2012;28:213–218.
50. Ukra A, Bennani F, Farella M. Psychological aspects of orthodontics in clinical practice. Part one: treatment-specific variables. Prog Orthod 2011;12:143–148.
51. Mangnall LAR, Dietrich T, Scholey JM. A randomized controlled trial to assess the pain associated with the debond of orthodontic fixed appliances. J Orthod 2013;40:188–196.
52. Keith DJ, Rinchuse DJ, Kennedy M, Zullo T. Effect of text message follow-up on patient's self-reported level of pain and anxiety. Angle Orthod 2013;83:605–610.
53. Weltman B, Vig KWL, Fields HW, Shanker S, Kaizar EE. Root resorption associated with orthodontic tooth movement: a systematic review. Am J Orthod Dentofac Orthop 2010;137:462–476.
54. Brudvik P, Rygh P. Transition and determinants of orthodontic root resorption-repair sequence. Eur J Orthod 1995;17:177–188.
55. Leach HA, Ireland AJ, Whaites EJ. Radiographic diagnosis of root resorption in relation to orthodontics. Br Dent J 2001;190:16–22.
56. Dudic A, Giannopoulou C, Leuzinger M, Kiliaridis S. Detection of apical root resorption after orthodontic treatment by using panoramic radiography and cone-beam computed tomography of super-high resolution. Am J Orthod Dentofac Orthop 2009;135:434–437.
57. Walker SL, Tieu LD, Flores-Mir C. Radiographic comparison of the extent of orthodontically induced external apical root resorption in vital and root-filled teeth: a systematic review. Eur J Orthod 2013;35:796–802.
58. Årtun J, Van't Hullenaar R, Doppel D, Kuijpers-Jagtman AM. Identification of orthodontic patients at risk of severe apical root resorption. Am J Orthod Dentofac Orthop 2009;135:448–455.
59. Hartsfield JK. Pathways in external apical root resorption associated with orthodontia. Orthod Craniofacial Res 2009;12:236–242.
60. Roscoe MG, Meira JBC, Cattaneo PM. Association of orthodontic force system and root resorption: a systematic review. Am J Orthod Dentofac Orthop 2015;147:610–626.
61. Linge BO, Linge L. Apical root resorption in upper anterior teeth. Eur J Orthod 1983;5:173–183.
62. Barbagallo LJ, Jones AS, Petocz P, Darendeliler MA. Physical properties of root cementum: Part 10. Comparison of the effects of invisible removable thermoplastic appliances with light and heavy orthodontic forces on premolar cementum. A microcomputed-tomography study. Am J Orthod Dentofac Orthop 2008;133:218–227.
63. Levander E, Malmgren O, Eliasson S. Evaluation of root resorption in relation to 2 orthodontic treatment regimes – a clinical experimental-study. Eur J Orthod 1994;16:223–228.
64. Fleminga PS, Johalb A. Self-ligating brackets in orthodontics a systematic review. Angle Orthod 2010;80:575–584.
65. Slade GD, Diatchenko L, Ohrbach R, Maixner W. Orthodontic treatment, genetic factors, and risk of temporomandibular disorder. Semin Orthod 2008;14:146–156.
66. Harris EF, Kineret SE, Tolley EA. A heritable component for external apical root resorption in patients treated orthodontically. Am J Orthod Dentofacial Orthop 1997;111:301–309.
67. Malmgren O, Goldson L, Hill C, Orwin A, Petrini L, Lundberg M. Root resorption after orthodontic treatment of traumatized teeth. Am J Orthod 1982;82:487–491.
68. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part I. Diagnostic factors. Am J Orthod Dentofac Orthop 2001;119:505–510.

69. Remington DN, Joondeph DR, Artun J, Riedel RA, Chapko MK. Long-term evaluation of root resorption occurring during orthodontic treatment. Am J Orthod Dentofacial Orthop 1989;96:43–46.
70. Jönsson A, Malmgren O, Levander E. Long-term follow-up of tooth mobility in maxillary incisors with orthodontically induced apical root resorption. Eur J Orthod 2007;29:482–487.
71. Sjolien T, Zachrisson BU. Periodontal bone support and tooth length in orthodontically treated and untreated persons. Am J Orthod 1973;64:28–37.
72. Kalkwarf KL, Krejci RF, Pao YC. Effect of apical root resorption on periodontal support. J Prosthet Dent 1986;56:317–319.
73. von Böhl M, Ren Y, Fudalej PS, Kuijpers-Jagtman AM. Pulpal reactions to orthodontic force application in humans: a systematic review. J Endod 2012;38:1463–1469.
74. Javed F, Al-Kheraif AA, Romanos EB, Romanos GE. Influence of orthodontic forces on human dental pulp: a systematic review. Arch Oral Biol 2015;60:347–356.
75. Butcher EO, Taylor AC. The vascularity of the incisor pulp of the monkey and its alteration by tooth retraction. J Dent Res 1952;31:239–247.
76. Han G, Hu M, Zhang Y, Jiang H. Pulp vitality and histologic changes in human dental pulp after the application of moderate and severe intrusive orthodontic forces. Am J Orthod Dentofacial Orthop 2013;144:518–522.
77. Bauss O, Schäfer W, Sadat-Khonsari R, Knösel M. Influence of orthodontic extrusion on pulpal vitality of traumatized maxillary incisors. J Endod 2010;36:203–207.
78. Proffit WR, Fields HW, Sarver DM. Contemporary Orthodontics. St Louis, Missouri: Mosby, Inc., 2013:300.
79. Pazera P, Fudalej P, Katsaros C. Severe complication of a bonded mandibular lingual retainer. Am J Orthod Dentofacial Orthop 2012;142:406–409.
80. Nguyen QV, Bezemer PD, Habets L, Prahl-Andersen B. A systematic review of the relationship between overjet size and traumatic dental injuries. Eur J Orthod 1999;21:503–515.
81. Bauss O, Röhling J, Schwestka-Polly R. Prevalence of traumatic injuries to the permanent incisors in candidates for orthodontic treatment. Dent Traumatol 2004;20:61–66.
82. Bauss O, Röhling J, Rahman A, Kiliaridis S. The effect of pulp obliteration on pulpal vitality of orthodontically intruded traumatized teeth. J Endod 2008;34:417–420.
83. Möller AJ, Fabricius L, Dahlén G, Ohman AE, Heyden G. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. Scand J Dent Res 1981;89:475–484.
84. Luther F. Orthodontics and the temporomandibular joint: where are we now? Part 1. Orthodontic treatment and temporomandibular disorders. Angle Orthod 1998;68:295–304.
85. Okeson JP. Management of Temporomandibular Disorders and Occlusion. St Louis, Missouri: Mosby, Inc., 2013:164.
86. Manfredini D, Bucci MB, Montagna F, Guarda-Nardini L. Temporomandibular disorders assessment: medicolegal considerations in the evidence-based era. J Oral Rehabil 2011;38:101–119.
87. Christensen L, Luther F. Adults seeking orthodontic treatment: expectations, periodontal and TMD issues. Br Dent J 2015;218:111–117.
88. Okeson JP. Evolution of occlusion and temporomandibular disorder in orthodontics: past, present, and future. Am J Orthod Dentofac Orthop 2015;147:S216–S223.
89. McNamara JA Jr. Orthodontic treatment and temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:107–117.
90. Reynders RM. Orthodontics and temporomandibular disorders: a review of the literature (1966-1988). Am J Orthod Dentofac Orthop 1990;97:463–471.
91. McNamara J, Seligman D, Okeson J. Occlusion, orthodontic treatment, and temporomandibular disorders: a review. J Orofac Pain 1995;9:73–90.
92. Kim M-RM, Graber TTM, Viana MA. Orthodontics and temporomandibular disorder: a meta-analysis. Am J Orthod Dentofac Orthop 2002;121:438–446.
93. Luther F. TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics. Br Dent J 2007;202:38–39.
94. Luther F, Layton S, McDonald F. Orthodontics for treating temporomandibular joint (TMJ) disorders (Review). Cochrane Database Syst Rev 2010;7:CD006541.
95. Michelotti A, Iodice G. The role of orthodontics in temporomandibular disorders. J Oral Rehabil 2010;37:411–429.
96. Leite RA, Rodrigues JF, Sakima MT, Sakima T. Relationship between temporomandibular disorders and orthodontic treatment: a literature review. Dental Press J Orthod 2013;18:150–157.
97. Luther F. Orthodontics and the temporomandibular joint: where are we now? Part 2. Functional occlusion, malocclusion, and TMD. Angle Orthod 1998;68:305–318.
98. Manfredini D, Stellini E, Gracco A, Lombardo L, Nardini LG, Siciliani G. Orthodontics is temporomandibular disorder-neutral. Angle Orthod 2016;86:649–654.
99. Chatterjee R, Kleinberg I. Effect of orthodontic band placement on the chemical composition of human incisor tooth plaque. Arch Oral Biol 1979;24:97–100.
100. Heymann GC, Grauer D. A contemporary review of white spot lesions in orthodontics. J Esthet Restor Dent 2013;25:85–95.
101. Kidd E. Essentials of Dental Caries. Oxford: Oxford University Press, 2005:22.
102. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. Am J Orthod 1982;81:93–98.
103. Hui J, Han Z, Geng G, Yan W, Shao P. The 100 top-cited articles in orthodontics from 1975 to 2011. Angle Orthod 2013;83:491–499.
104. Alexander SA. The effect of fixed and functional appliances on enamel decalcifications in early Class II treatment. Am J Orthod Dentofac Orthop 1993;103:45–47.
105. Tufekci E, Dixon JS, Gunsolley JC, Lindauer SJ. Prevalence of white spot lesions during orthodontic treatment with fixed appliances. Angle Orthod 2011;81:206–210.
106. Ogaard B, Rølla G, Arends J. Orthodontic appliances and enamel demineralization. Part 1. Lesion development. Am J Orthod Dentofacial Orthop 1988;94:68–73.
107. Von der Fehr FR, Löe H, Theilade E. Experimental caries in man. Caries Res 1970;4:131–148.
108. Shungin D, Olsson AI, Persson M. Orthodontic treatment-related white spot lesions: a 14-year prospective quantitative follow-up, including bonding material assessment. Am J Orthod Dentofac Orthop 2010;138:136–137.
109. Haydar B, Karabulut G, Ozkan S, Aksoy AU, Ciğer S. Effects of retainers on the articulation of speech. Am J Orthod Dentofac Orthop 1996;110:534–540.
110. Al Maaitah EF, Adeyemi AA, Higham SM, Pender N, Harrison JE. Factors affecting demineralization during orthodontic treatment: a post-hoc analysis of RCT recruits. Am J Orthod Dentofac Orthop 2011;139:181–191.
111. Ogaard B. Prevalence of white spot lesions in 19-year-olds: a study on untreated and orthodontically treated persons 5 years

after treatment. Am J Orthod Dentofacial Orthop 1989;96:423–427.

112. van der Veen MH, Attin R, Schwestka-Polly R, Wiechmann D. Caries outcomes after orthodontic treatment with fixed appliances: do lingual brackets make a difference? Eur J Oral Sci 2010;118:298–303.
113. Willmot DR. White lesions after orthodontic treatment: does low fluoride make a difference? J Orthod 2004;31:235–242; discussion 202.
114. Kim S, Katchooi M, Bayiri B, Sarikaya M, Korpak AM, Huang GJ. Predicting improvement of postorthodontic white spot lesions. Am J Orthod Dentofac Orthop 2016;149:625–633.
115. Robertson MA, Kau CH, English JD, Lee RP, Powers J, Nguyen JT. MI Paste Plus to prevent demineralization in orthodontic patients: a prospective randomized controlled trial. Am J Orthod Dentofac Orthop 2011;140:660–668.
116. Chen H, Liu X, Dai J, Jiang Z, Guo T, Ding Y. Effect of remineralizing agents on white spot lesions after orthodontic treatment: a systematic review. Am J Orthod Dentofac Orthop 2013;143(376–382):e3.
117. Neuhaus KW, Graf M, Lussi A, Katsaros C. Late infiltration of post-orthodontic white spot lesions. J Orofac Orthop 2010;71:442–447.
118. Akin M, Basciftci FA. Can white spot lesions be treated effectively? Angle Orthod 2012;82:770–775.
119. Kidd E. Essential of Dental Caries. Oxford: Oxford University Press, 2005:7.
120. Bukhari OM, Sohrabi K, Tavares M. Factors affecting patients' adherence to orthodontic appointments. Am J Orthod Dentofac Orthop 2016;149:319–324.
121. Feldman EW. Speech articulation problems associated with placement of orthodontic appliances. J Speech Hear Disord 1956;21:34–38.
122. Orthodontists AS of Risks of Orthodontic Treatment [Internet]. 2016. Available at: http://aob.aso.org.au/Docs/Orthodontics/Risks.htm. Accessed 28 Aug 2016.
123. Stewart FN, Kerr WJS, Taylor PJS. Appliance wear: the patient's point of view. Eur J Orthod 1997;19:377–382.
124. De Felippe NLO, Da Silveira AC, Viana G, Smith B. Influence of palatal expanders on oral comfort, speech, and mastication. Am J Orthod Dentofac Orthop 2010;137:48–53.
125. Tanaka H. Speech patterns of edentulous patients and morphology of the palate in relation to phonetics. J Prosthet Dent 1973;29:16–28.
126. Stratton CS, Burkland GA. The effect of maxillary retainers on the clarity of speech. J Clin Orthod 1993;27:338–340.
127. Slater RD. Speech and discomfort during lingual orthodontic treatment. J Orthod 2013;40(Suppl 1):S34–S37.
128. Fillion D. Improving patient comfort with lingual brackets. J Clin Orthod 1997;31:689–694.
129. Hohoff A, Seifert E, Fillion D, Stamm T, Heinecke A, Ehmer U. Speech performance in lingual orthodontic patients measured by sonagraphy and auditive analysis. Am J Orthod Dentofac Orthop 2003;123:146–152.
130. Alessandri Bonetti G, Zanarini M, Incerti Parenti S, Lattuca M, Marchionni S, Gatto MR. Evaluation of enamel surfaces after bracket debonding: an in-vivo study with scanning electron microscopy. Am J Orthod Dentofac Orthop 2011;140:696–702.
131. Joseph VPRE. The shear bond strengths of stainless steel and ceramic brackets used with chemically and light activated composite resins. Am J Orthod Dentofac Orthop 1990;97:121–125.
132. Azzeh E, Feldon PJ. Laser debonding of ceramic brackets: a comprehensive review. Am J Orthod Dentofac Orthop 2003;123:79–83.
133. Habibi M, Nik TH, Hooshmand T. Comparison of debonding characteristics of metal and ceramic orthodontic brackets to enamel: an in-vitro study. Am J Orthod Dentofac Orthop 2007;132:675–679.
134. Janiszewska-Olszowska J, Szatkiewicz T, Tomkowski R, Tandecka K, Grocholewicz K. Effect of orthodontic debonding and adhesive removal on the enamel - current knowledge and future perspectives - a systematic review. Med Sci Monit 2014;20:1991–2001.
135. Baumann DF, Brauchli L, van Waes H. The influence of dental loupes on the quality of adhesive removal in orthodontic debonding. J Orofac Orthop 2011;72:125–132.
136. Gwinnett AJ, Gorelick L. Microscopic evaluation of enamel after debonding: clinical application. Am J Orthod 1977;71:651–665.
137. Ozer T, Başaran G, Kama JD. Surface roughness of the restored enamel after orthodontic treatment. Am J Orthod Dentofacial Orthop 2010;137:368–374.
138. Zachrisson BU, Arthun J. Enamel surface appearance after various debonding techniques. Am J Orthod 1979;75:121–127.
139. Thordarson A, Zachrisson BU, Mjör IA. Remodeling of canines to the shape of lateral by grinding: a long-term clinical and radiographic evaluation incisors. Am J Orthod Dentofaciaöl Orthop 1991;100:123–132.
140. Zachrisson BU, Nyøygaard L, Mobarak K. Dental health assessed more than 10 years after interproximal enamel reduction of mandibular anterior teeth. Am J Orthod Dentofac Orthop 2007;131:162–169.

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