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## Abstract

This review looks at some of the possible risks associated with orthodontic treatment. It is feasible to understand that these risks result from a combination of patient and treatment-related factors. The potential risks of orthodontic treatment involve pain, root resorption, pulp degeneration, periodontal damage, TMJ disfunction, dental decay, speech difficulties, and white spot lesion of the enamel. Generally, treatment factors include material and design of the appliance, the amount of force, and the length of the treatment, whereas patient factors include behaviour and compliance of the patients. As a result, the orthodontist should have a thorough grasp of these risks in order to get informed consent prior to orthodontic treatment.

Keywords: Orthodontic Treatment; Root Resorption; Pulp Degeneration; Periodontal Damage; TMJ Disfunction

## Introduction

Orthodontic treatment subjects the patient to some risks just like any other medical therapy. To guarantee that each patient will have a net benefit from treatment, the orthodontist is ethically required to understand how these risks pertain to each individual patient [1]. Incorrectly identifying and managing orthodontic treatment risks might result in patient disappointment as well as legal action [2]. Pain, root resorption, pulp degeneration, periodontal damage, TMJ disfunction, dental decay, speech difficulties, and enamel damage are among the potential risks of orthodontic treatment [3]. It is necessary to evaluate each individual case carefully to determine whether there will be a net benefit from treatment.

The aim of this study is to give an in-depth investigation of the literature on potential risks of orthodontic treatment. The interactions between various patient and treatment variables that affect risks are given special consideration.

## Pain

Almost every phase of orthodontic therapy carries the risk of pain. The concern of pain may prevent some patient from beginning orthodontic treatment [4]. Pain has been linked to worse patient compliance throughout treatment and has been mentioned as a common

cause for discontinuing treatment [4]. In general, there are two forms of pain that frequently occur during orthodontic treatment: mucosal pain from injury to the oral soft tissues caused by the appliance and periodontal/pulpal pain from orthodontic forces that applied to the teeth.

Oral mucosal pain is a common side effect of orthodontic therapy with fixed appliances, and for some patients, it might rank as the most irritating part of orthodontic therapy [5]. According to Baricevic., *et al.* orthodontic archwires tend to induce ulcerations whereas brackets cause mucosal erosions and desquamations [6]. It is not unexpected that the position of the appliance affects the location of mucosal ulceration; lingual appliances tend to affect the tongue, whereas buccal appliances affect the cheeks [7]. Wearing clear aligners (like Invisalign) could also cause mucosal irritation, however patients are unlikely to find this to be a major problem [8].

When orthodontic forces are applied to teeth, the periodontal ligament (PDL)'s vasculature is compressed, causing inflammation of the pulp and periodontal tissues. In this regard, fixed appliances hurt more than removable or functional ones [9].

The majority of patients will suffer pain following the initial archwire placement beginning at 4 hours, peaking at 24 hours, and decreasing during the next 3 days. Generally, harder wires can cause a greater pain [10]. Compared to regular nickel titanium (NiTi) wires, heat-activated NiTi wires may also be less painful [11]. some evidence supports conventional brackets being more painful than passive self-ligating brackets during initial orthodontic alignment [12]. In this respect, a recent meta-analysis founded no clinically significant difference between the two bracket types [13]. Nevertheless. conventional brackets have been found to cause more pain than active self-ligating brackets when engaging rectangular wire. Both lingual and labial appliances seem to cause the same amount of pain [14].

Orthodontic debands may frequently cause pain for patients, and this seems to be correlated with both the degree of tooth mobility and the direction of the forces applied to remove the fixed appliance. Because metal brackets are more ductile and need less force to remove, they may be easier to remove than ceramic ones [15].

Although the research shows that overall levels of pain in Invisalign patients is considerably decreased, people receiving Invisalign treatment suffer pain over a similar timeline to individuals using fixed appliances [9]. In fact, some Invisalign patients may experience no pain at all during their treatment, which has been linked to the minor, incremental tooth shifts caused by the use of subsequent aligners [16].

Reducing inflammation and improving blood flow inside the PDL are two potential therapeutic targets to relieve periodontal/pulpal pain during orthodontic treatment. Randomized clinical trials have shown that anti-inflammatory analgesics reduce pain after the application of orthodontic forces more effectively than a placebo [17]. Ibuprofen, aspirin, and paracetamol were all shown to be similarly beneficial in this regard, according to a meta-analysis by Xioting., *et al.* The latter was favoured due to its greater safety profile and less possible effects on tooth movement [2].

By reducing the compressive stresses of orthodontic appliances and enabling a temporary restore of blood flow to the PDL, chewing wafers or gum can help orthodontic patients feel less pain. As a result, both have been demonstrated to be useful for lowering pain and may be comparable as analgesics [18].

Although age and gender differences are not always associated with orthodontic pain, [19] there are definite "non-linear" associations between age, gender, and pain: preadolescent tend to report less pain than adolescents and adults, and male less than females [8].

#### **Root resorption**

Cementum and dentine may resorb as teeth are moved during orthodontic treatment. 'Orthodontic-induced root resorption' (OIRR) is the term given to this process [20]. Although it has been demonstrated that resorptive craters may be repaired by cementum deposition, roots will have irreversible shortening if resorption divides an apical area from the root [10].

48 - 66% of teeth treated orthodontically are affected by OIRR by 2 mm or less as indicated by radiographic studies. Anterior teeth are more prone to OIRR, and 1 - 5% will exhibit more than 4 mm of root shortening. Histological studies, on contrary, indicate that more than 90% of teeth experience some extent of OIRR [21]. This demonstrates the truth that not every resorptive lesions manifested as shortening of root.

OIRR has been linked to a wide range of patient-related factors, including age, tooth morphology, certain drugs, hormone deficiencies, hypothyroidism, hypopituitarism, alveolar bone density, root morphology, chronic alcoholism, root proximity to the cortical bone, trauma, gender, and the severity and type of malocclusion, even though genetics account for more than half of this variation [22].

Although the research emphasizes 'low forces' as being protective against OIRR, the orthodontic therapy can also have an impact on how OIRR is experienced [23].

Because intermittent forces are linked to lower OIRR than continuous ones, orthodontic forces and OIRR also have a temporal relationship [24]. Pauses in treatment can lower OIRR and there appears to be a positive link between total treatment time and OIRR [25]. However, teeth treated using self-ligation do not exhibit a decrease in OIRR, which is consistent with research showing that the use of such appliances does not shorten the duration of treatment when compared to conventional ligation [19] and also implies that the forces applied by the two methods are similar.

It has been advised to do radiographic screening six months after the start of treatment to identify individuals who are more likely to develop OIRR [21]. Those who have OIRR signs should continue to undergo periodic radiographs throughout their therapy [24]. In cases where OIRR is a problem, orthodontic force should be stopped, which will efficiently cease the process [26].

#### **Pulp degeneration**

By compressing the neurovascular bundle, orthodontic forces can alter pulpal blood flow. Despite the fact that several studies have demonstrated histological and inflammatory alterations in the pulp as a consequence of orthodontic forces, a recent assessment of the impacts of orthodontic force on the pulp came to the conclusion that this field is still not well understood [27].

Early studies raised the possibility that extreme orthodontic forces might strangulate the pulp, resulting in necrosis [28]. Modern research indicates that the pulp is extremely resistant in sustaining strong forces and that necrosis of otherwise healthy teeth during orthodontic therapy is an uncommon incidence, even if it is obvious that the orthodontic force can produce pulpal ischaemia and degenerative alterations [27]. The risk of pulp degeneration during orthodontic treatment is increased for teeth with a history of trauma because they may have an impaired vascular supply [29].

Orthodontic movement of the root apices beyond of the alveolar bone may cause loss of the vascular supply, however some studies showed that teeth maintaining their vitality when bonded lingual retainers unintentionally torqued root apices outside the cortex [27]. Therefore, the neurovascular bundle may be less likely to be severed when tooth moved slowly.

A considerable percentage of orthodontic patients may have a history of incisor trauma since greater overjet is a reported risk factor for incisor trauma [28]. As a result, it's essential to examine prospective orthodontic patients for a history of dental trauma and to inform them to the possibility of losing vitality prior to treatment. Moving traumatized teeth should be done carefully, especially if they exhibit sign of obliteration of the pulp [30].

Actually, when pulp being tested, teeth may temporarily respond negatively or with a higher response threshold during orthodontic movement [28]. However, if pulpal necrosis develops in the absence of infection, apical alterations may not be visible on radiographs,

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which have been proposed to be a more reliable indicator of pulp necrosis than vitality test [29]. In addition, conditions other than pulp infection may cause radiographic abnormalities at a root apex. Therefore, before diagnosing pulp necrosis, professionals should check for a variety of signs and symptoms.

## **Periodontal damage**

The periodontium can be affected by orthodontic therapy via encouraging gingivitis, gingival recession, and open gingival embrasures. It is commonly known that orthodontic appliances can make it more difficult to control plaque, which may lead to gingivitis [3].

Retrospective researches have shown that patients who have received orthodontic therapy have more tendency to develop gingival recession than other individuals [31]. Although not always the case, a systemic review of the relevant literature revealed that, on average, orthodontic therapy can cause gingival recession of 0.03 mm, increased pocket depth measuring 0.23 mm, and alveolar bone loss measuring 0.13 mm [32]. Adult orthodontic patients may be more susceptible to periodontal disease. According to Nelson PA, average bone loss of the anterior teeth was 0.54. with more than 2 mm bone loss was reported in one-third of the patients. This was linked to the age [33].

Orthodontic treatment might jeopardize the integrity of the periodontium, by moving the roots beyond the alveolar bone [34]. Lower incisors are particularly prone to recession labially [31]. Therefore, it is important to keep this in mind during orthodontic treatments that push lower incisors forward.

A significant follow-up research reported no difference between extraction and non-extraction therapy in term of gingival recession [35]. Fixed lingual retainers may interfere with dental hygiene; however, the research indicates long-term harm to the periodontium from these appliances is limited [36].

Eventually, it is challenging to determine the exact impact of orthodontic therapy due to the complex nature of periodontal attachment loss. Age, gingival biotype, smoking, oral hygiene, frenal attachment, and control of plaque are all recognized risk factors [34].

The interdental papilla loses its position in the aesthetic zone, resulting in open gingival embrasures (also known as "black triangles"). Although they may be caused by periodontal disease, they frequently have a different etiology than gingival recession. Age, tooth shape, proximal contact length, proximal bone height, and interproximal gingival thickness have all been connected to the condition of the papilla [37]. According to Tarnow., *et al.* open gingival embrasures are more prone to develop when there is more than 5 mm distance between the alveolar bone and the tooth contact [38]. In addition to being unesthetic, open embrasures more prone to food impaction. Crown reshaping, bracket repositioning, and restorative procedures are examples of strategies for management.

#### **Temporomandibular disorder**

There is not a consensus definition for the diagnosis of "temporomandibular disorder" (TMD) [39]. TMD, according to Okeson, is characterized by the signs and symptoms of masticatory dysfunction brought on by the temporomandibular joint, the teeth, and the muscles [40].

The subject of how occlusion could affect TMD in general is closely tied to the potential connection between orthodontics and TMD. There is a belief that the dentistry profession previously overestimated the role of occlusion in TMD [41].

The study of the association between orthodontics and TMD was relatively understudied until the late 1980s. This changed in 1987 after a patient who sued their orthodontist for 'causing' TMD received a significant judgement of damages from a US court. Since that point, more researches have shown that there is not enough evidence to support any claims that orthodontic treatment causes, prevents, or treats TMD [39,42].

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It would be quite difficult to plan and carry out a study that may show a causal relationship between orthodontic treatment and TMD. A meaningful evaluation would need to account for a significant number of TMD-related associations in addition to initial malocclusion and the types of orthodontic treatment. Age, socioeconomic status, gender, trauma history, ethnicity, parafunctional activities, third molar extraction, concurrent pain disorders, and genetics are some of related factors [39]. In addition, there would be difficulties in identifying and diagnosing TMD, as well as a chance of the placebo effect and interobserver variability. As a result, some studies concluded that orthodontics has never been evaluated satisfactorily with respect to TMD to date [42].

However, there may be a minority of individuals who have a genetic susceptibility to TMD from orthodontics [39]. Therefore, saying there is no link between orthodontic therapy and TMD may be oversimplified.

Orthodontists nevertheless have a medicolegal obligation to assess patients at risk for TMD and refer them properly when a positive diagnosis is obtained, despite the ambiguous association between orthodontics and TMD [2]. Additionally, delaying therapy in cases of TMD that already present may be wise [42]. Patients with a history of TMD should be aware that orthodontic treatment has the ability to stabilize, make their condition worse, or make it better [2].

#### **Dental decay**

Orthodontic appliances elevate the risk of caries through facilitating accumulation of plaque and impeding oral hygiene [43]. The most frequent side effect of orthodontic treatment is white spot lesions (WSL) that can develop cavitation [44]. Because of the low possibility of WSL development with removable appliances, the majority of WSL investigations have focused on fixed appliances [45].

WSL can be challenging to locate during orthodontic treatment because accurate visibility requires full plaque clearance and enamel dehydration. As early as 4 weeks following the placement of a fixed appliance, clinical evidence of WSL may be seen, which is equivalent to non-orthodontic individuals who neglect to brush their teeth [46].

According to a recent review of the literature, 2 - 97% of orthodontic patients had WSL. This considerable variation has been linked to variations in WSL assessment methods and the failure to distinguish between pre-existing and new WSL during orthodontic therapy [44]. It could also illustrate how complex the risk of cavities is. Younger individuals are more vulnerable because to immaturity of the enamel and a propensity for less stringent oral hygiene measures [47]. Although other studies have identified no difference in the prevalence of WSL across genders, there is some indication that male patients have more WSL than female orthodontic patients [47].

Saliva acts as a barrier to the development of WSL. Therefore, maxillary teeth tend to be more vulnerable to WSL than mandibular teeth, and lingual appliances have lower risk of WSL [48].

Saliva begins to remineralize WSL after removal of fixed appliances. The process starts quickly then slows down within weeks [49]. The natural remineralization process does not appear to be affected by the use of a Hawley or Essex type retainer [49]. It is unclear what advantages casein phosphopeptide-amorphous calcium phosphate derivatives have for WSL remineralization. Resin infiltration and enamel micro-abrasion are further management strategies for WSL that have been mentioned in the literature [50].

## Speech difficulties

Speech can be affected directly by orthodontic appliances by inhibiting sound articulation or indirectly by impacting a person's physical and mental well-being. Despite the possibility of orthodontic appliances to impair speech has been investigated as an area of inquiry for more than 60 years [51].

When the lingual space is invaded, orthodontic appliances might have a negative impact on speech. Patients reported that fixed labial appliances have a lower impact on speech than removable appliances [52]. For Hawley retainers and bonded palatal expanders, the speech recovery period typically lasts one week [51]. The adaptation period for complete upper dentures is comparable to this [53]. It would imply that, in this case, age is not a crucial role in speech adaptation. In fact, research on bonded palatal expanders revealed no link between patient age and duration for speech adaptation [52]. If the thickness and amount of palatal covering of an appliance are kept to a minimum, speech adaptation could go more quickly [53].

Lingual appliances can hinder speech in addition to intruding on surfaces needed for phonetics by causing ulcers on the tongue [54]. Patients reported difficulties speaking for an average of 2, 4 or 6 days, respectively, when Shalish., *et al.* examined the effects of Invisalign, labial and lingual appliances on quality of life [10]. The speech recovery period for lingual appliances has been estimated in other research to be between 1 and 3 months [55].

#### **Enamel damage**

Debanding, the process of removing fixed orthodontic appliances, includes using effort to break the binding between the appliance and the tooth surface. This force application will have one of two effects: either cohesive failure within the orthodontic resin itself, or adhesive failure at the tooth-to-adhesive contact or the bonding surface of the device [56]. Debanding procedure or the removal of remaining orthodontic cement may cause damage to the enamel.

If the cohesive strength of the enamel itself is greater than the strength of the micromechanical bond between the enamel and the bonding resin, adhesive bracket failure may result in the removal of the enamel. Due to the bond's strength, ceramic brackets that use a chemical interface (as opposed to a micromechanical one) are more likely to damage enamel [57]. According to the research, the likelihood of enamel fracture after removing ceramic brackets ranges from 10 to 35% [58]. The latest generation of ceramic brackets have less susceptibility to enamel fracture during debonding [59].

There are no entirely non-traumatic techniques for removing remaining orthodontic resin [57]. Almost 20 to 50 micrometres of enamel are lost during resin removal, and gouges and scratches will unavoidably remain on the surface of the enamel [56]. The prism rods are exposed when surface enamel is removed, which presumably increases the sensitivity to acid breakdown [60]. Although this concern has been dismissed by others, it has been hypothesized that the enamel surface's unavoidable gouging and scratching from resin removal using rotating equipment increases susceptibility to staining and caries [59].

#### Conclusion

Through a study of relevant research, this review has taken into account some of the most common risks associated with orthodontic treatment. It has been demonstrated that the risks of orthodontic therapy vary depending on the patient and their treatment plans. Professionals should evaluate the patients' susceptibility to these potential risks when developing treatment plans, and patients should be fully informed about the risks during the process of informed consent. A certain level of clinical knowledge and experience are unavoidably needed to do this. specific treatment strategy for every patient is liable to expose patients to a lower risk of adverse outcomes.

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