



Review Article

Orthodontic iatrogenics: Balancing benefits and risks

Shubham Nagrath^{1*}, Sankalp Sood¹, Dimple Chainta¹, Nishant Negi¹, K.S Negi¹, Monika Mahajan¹,
Susheel Negi¹

¹Dept. of Orthodontics and Dentofacial Orthopaedics, HP Government Dental College & Hospital, Shimla, Himachal Pradesh, India

Abstract

Orthodontic treatment improves function, esthetics, and confidence but may cause adverse effects. Common complications include enamel demineralization, root resorption, periodontal changes, pain, and less frequent pulpal or TMJ alterations. White spot lesions are best prevented with fluoride and strict hygiene, while controlled forces and radiographic monitoring reduce resorption risk. Pain is universal, with NSAIDs most effective for relief. Evidence on other outcomes remains limited, underscoring the need for preventive strategies and individualized care.

Orthodontic complications are real but largely preventable with evidence-based practice. High-fluoride regimens, light and biologically controlled forces, and NSAID-based pain management are strongly supported by current evidence, while periodontal changes, pulpal effects, and relapse require further investigation. Careful case selection, patient education, and long-term retention remain essential to maximize benefits and minimize risks.

Keywords: Orthodontic risks, Braces complications, Root resorption, Gingival recession, Braces pain, Nickel allergy, Fluoride treatment, Light forces, Orthodontic TMD, Invisalign risks.

Received: 21-09-2025; **Accepted:** 01-10-2025; **Available Online:** 14-10-2025

This is an Open Access (OA) journal, and articles are distributed under the terms of the [Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License](https://creativecommons.org/licenses/by-nc-sa/4.0/), which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprint@ipinnovative.com

1. Introduction

The appearance of the teeth contributes significantly to an individual's perceived attractiveness and psychological well-being.¹ For this reason, orthodontic treatment is frequently sought to refine the smile and support self-esteem. Test faces exhibiting incisal crowding and median diastema are rated considerably lower in intelligence, attractiveness, and social status compared to the same faces with ideal occlusion.¹ "Behrents defined iatrogenic as unintended harm during treatment, often linked to non-compliance.² Orthodontic treatment goals primarily focus on achieving optimal oral health, aesthetics, proper stomatognathic function, and long-term stability. Compromised treatment is only justified when dictated by patient cooperation or genetic limitations. Both local and systemic risks may arise during orthodontic care. Insights from clinical cases, imaging, and histological work suggest several issues that require consideration.

1.1. Demineralisation

Enamel may be affected in different ways, such as through etching, interproximal reduction, loss of minerals around brackets, frictional wear, debonding procedures, or the removal of residual composite.

1.2. Periodontal changes

Patients may experience attachment loss that extends beyond the CEJ, apical extension of the epithelial lining, and a decrease in alveolar bone height.

1.3. Gingival response

Moderate hyperplastic gingivitis often develops within the first one to two months after fixed appliance placement.

*Corresponding author: Shubham Nagrath
Email: shubhamnagrath273@gmail.com

1.4. Loss of attachment and alveolar bone loss

Orthodontic patients show slightly greater bone loss than untreated individuals, involving all interproximal surfaces and linked to apical root resorption.³

1.5. Systemic risks

Though uncommon, issues may include allergic reactions, psychological stress, and infective endocarditis. Nickel in wires can also cause allergic dermatitis.⁴ Oral hygiene and bacteremia links with periodontal disease and dental procedures are recognised but remain debated.⁵

Risk Management includes regular monitoring (e.g., radiographs), light forces, and biocompatible materials are advised. Heavy forces risk PDL ischemia, cell death, and bone loss.⁶ Informed Consent, along with a Clear discussion of risks, benefits, and the use of biocompatible materials, helps patients balance aesthetics with potential complications.

2. Discussion

2.1. White spot lesions (WSLs) in orthodontics

White spot lesions are prevalent in cases of poor oral hygiene, these decalcifications reduce treatment quality and cause dissatisfaction.⁷ Their characteristic opaque, white, and chalky appearance results from an optical phenomenon caused by mineral loss in the enamel's surface and subsurface layers, as shown in **Figure 1**, which becomes more pronounced when the enamel is dried. It was found that teeth subjected to banding or bonding exhibited a significantly higher incidence of white spot formation compared to control group.

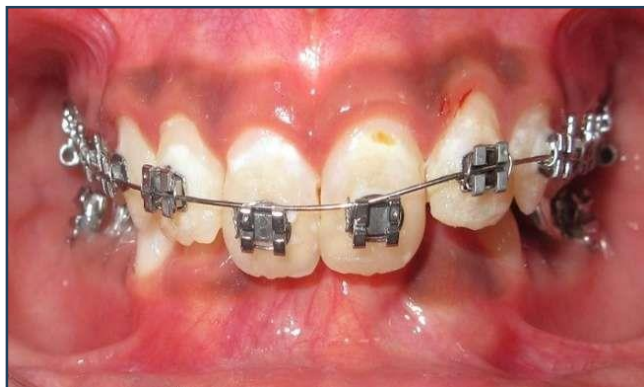


Figure 1: Teeth with white spot lesions

The analysis showed the highest prevalence in maxillary incisors and the lowest in the posterior segment. Notably, no spots appeared on the lingual surfaces of the mandibular anterior teeth with bonded retainers, suggesting that salivary flow aids resistance.⁸ Teeth banded or bonded for 12–16 months showed the exact incidence of white spots as those treated for up to 36 months. Lesions improved after appliance removal; however, overall regression was minimal.⁹

Prevention strategies focus on the use of fluoride-based products, antimicrobial agents, and patient education.

2.1.1. Prevention emphasises that factors which play a crucial role in restoring enamel integrity are:

1. Fluoride toothpaste: After 3 and 6 months, patients using 5,000 ppm fluoride toothpaste showed significantly lower plaque index than those using 1,100 ppm, confirming superior remineralisation with higher fluoride concentration.¹⁰
2. Antimicrobial rinses such as chlorhexidine.
3. Remineralisation agents like casein phosphopeptides-amorphous calcium phosphate (ACP), bioactive glass, and nano-hydroxyapatite.
4. Early risk assessment, strict oral hygiene, and patient education with informed consent are vital to minimise white spot lesions and ensure aesthetic and functional orthodontic success.

2.1.2. Critical appraisal

Evidence for WSL prevention is moderately strong, with RCTs supporting high-fluoride regimens. Variability in diagnostic methods and scarce long-term data limit comparability. Clinically, high-fluoride use and strict hygiene remain the best-supported strategies.

2.2. Dentin hypersensitivity (DHS)

Dentin hypersensitivity (DHS) is characterised by a short, sharp pain that occurs when exposed dentin responds to thermal, tactile, osmotic, evaporative, or chemical stimuli, in the absence of any other dental pathology. Dentin hypersensitivity (DHS) shows a documented prevalence of ~15% (range 3–57%). It can occur at any age but peaks at 20–40 years, affecting women more often and at younger ages. Hygienists report DHS about twice as frequently as dentists¹¹ (Canadian Advisory Board on Dentine Hypersensitivity 2003).

The reported prevalence of dentin hypersensitivity (DHS) differs internationally. Reports on dentin hypersensitivity (DHS) shows wide variation across countries. In Indonesia, almost one in three people experience it, while in the United States the figure is closer to one in five. Prevalence in Japan has been estimated at 16%, in France about 14% during the winter but only 9% in spring, and around 13% in both Germany and Australia.¹²

The hydrodynamic theory, proposed by Brännström¹³ explains the mechanism of DHS: Dentin sensitivity is puzzling, despite dentin's insulating nature; minor temperature changes can trigger pain, explained by rapid outward fluid flow in dentinal tubules. Accurate DHS diagnosis requires excluding mimicking conditions, as shown in **Table 1**. A sharp, transient response during testing confirms DHS, while thermal tests help distinguish it from pulpitis. If pain is localised and triggered by occlusion or

percussion, alternative causes—occlusal trauma, periodontal issues, or cracked teeth—should be considered.¹⁴

Table 1: Diseases or conditions to be excluded for the diagnosis of dentin hypersensitivity

Condition	Key Clinical Features
Dental Caries	Sensitivity after demineralization reaches dentin; worsens if pulp is involved.
Cracked Tooth Syndrome	Sharp, brief pain on chewing that stops when force is removed; confirmed with bite tests.
Fractured/Traumatized Tooth	Rough enamel edges with little discomfort; enamel–dentin fractures cause thermal/mechanical sensitivity.
Pulpitis	<i>Reversible</i> : sharp, short pain to stimuli, resolves on removal. <i>Irreversible</i> : throbbing, persistent pain disturbing sleep.
Periodontal Abscess	Constant dull pain, worse with chewing; linked to deep pockets and bone loss.
Periapical Periodontitis	Constant pain on biting or percussion; due to pulp necrosis.
Pericoronitis	Localized pain/swelling around partially erupted tooth; aggravated by occlusion.
Bleaching Sensitivity	Transient pulp-like pain from bleaching agents, similar to reversible pulpitis.
Bruxism (Grinding/Clenching)	Thermal hypersensitivity, attrition, enamel microfractures, TMJ discomfort, headaches.

Adapted from Liu et al.¹⁴ Re-drawn and modified by the authors.

The management of dentin hypersensitivity (DHS) relies on both preventive and therapeutic measures. A key aspect is patient education, with emphasis on adopting careful, non-traumatic brushing habits. Such practices help limit enamel loss and lower the likelihood of gingival recession.

Additionally, behavioural changes—such as avoiding acidic diets, aggressive brushing, and parafunctional habits—help reduce dentin exposure. Non-invasive treatments, as shown in **Table 2**, relieve pain by occluding tubules and blocking nociception, while restorative or surgical options, as shown in **Table 3**, are required for structural defects to ensure lasting relief and dental preservation.¹⁵

Table 2: Showing the indications and limitations for non-invasive desensitization treatment for dentin hypersensitivity (DHS)

Category	Key Clinical Considerations
Indications – Restorative Treatment	Used when hard tissue loss threatens pulp–dentin complex, or when conservative methods fail. Aim: protect pulp with minimal enamel/dentin removal.
Indications – Mucogingival Surgery	For persistent hypersensitivity from gingival recession unresponsive to non-surgical care; also for esthetic concerns (root exposure). Goal: restore soft tissue coverage and reduce sensitivity.
Risks & Limitations	Restorations may fail (wear, caries, bond breakdown) and need repair/replacement. Surgery can be unpredictable, with variable healing and long-term stability. Success depends on technique, compliance, and patient factors.

Adapted from Liu et al.¹⁴ Re-drawn and modified by the authors.

Table 3: Indications and limitations for restorations and mucogingival surgeries for DHS treatment

Category	Key Points
Indications	Suitable when no major hard tissue loss. Best for shallow/minor cervical lesions that are stable and esthetically acceptable. Also used for mild gingival recession without added risk factors. Not for patients with contraindications to topical agents.
Risks & Limitations	Ineffective if hard tissue loss or progressing gingival recession present. Conditions like caries, microfractures, or pulpitis may mimic DHS, risking misdiagnosis. Results can be variable; repeated treatments may be needed.

Adapted from Liu et al.¹⁴ Re-drawn and modified by the authors.

2.2.1. Critical appraisal

Epidemiological data confirm DHS is common, but most studies rely on self-reporting with inconsistent criteria. Few RCTs assess prevention in orthodontic patients. Current guidance is largely consensus-based, emphasizing hygiene and desensitizing agents.

2.3. Effects of orthodontic mechanics on dental pulp

Orthodontic tooth movement relies on remodeling of paradental tissues. Force application causes tissue strain, altering vascularity and reorganizing cellular and extracellular matrices. This stimulates release of neurotransmitters, cytokines, growth factors, colony-stimulating factors, and arachidonic acid metabolites. Movement occurs in three phases, beginning with an initial rapid phase immediately after force application.¹⁵ In the

second phase, distorted PDL fibers create compression areas, disrupting blood flow and forming hyalinized zones, temporarily halting tooth movement for 4–20 days.¹⁵ The third (acceleration) and fourth (linear) phases start ~40 days after initial force application, during which pressure-side collagen fibers remain disoriented.

Inflammation in human pulp fibroblasts is influenced by neuropeptides and cytokines (IL-1, IL-3, IL-6, TNF). Extracellular aspartate aminotransferase, released during cell death, rises significantly after orthodontic force application.¹⁵ Orthodontic force affects pulpal blood flow (PBF). McDonald et al.¹⁶ found that retraction of maxillary canines with a 50 g continuous light tipping force transiently reduced PBF for ~32 minutes, followed by a prolonged increase lasting up to 48 hours.¹⁶ Furthermore, animal studies utilizing fluorescent microspheres suggest that substantial increases in PBF occur after continuous light tipping forces are applied over a 5-day period.

Understanding how orthodontic forces affect the dental pulp is essentially crucial, as orthodontic treatment has been associated with altered pulpal respiration rates, disruption of the odontoblastic layer, pulpal obliteration due to secondary dentin formation, root resorption, and, in severe cases, pulpal necrosis.¹⁶ Regular monitoring, early intervention, and customised biomechanical approaches help mitigate these risks, ensuring the preservation of periodontal and pulpal integrity.

2.3.1. Critical appraisal

Biological plausibility is well established through experimental and animal studies, but high-quality human trials are scarce. Most pulpal effects appear transient and reversible. Clinical recommendations favor light forces and careful monitoring, though evidence remains limited.

2.4. Periodontal risks in orthodontic treatment

Orthodontic treatment is very effective in achieving proper dental alignment; however, it also carries significant risks to periodontal health that need careful management. The periodontium,¹⁷ which encompasses the gingival unit (soft tissue) and the periodontal attachment apparatus (including cementum, ligament, and alveolar bone), encounters various challenges during tooth movement. Fixed orthodontic appliances can promote plaque accumulation, favouring the increase in anaerobic bacteria, particularly *Porphyromonas gingivalis*, which is a concern. The increase in anaerobic bacteria, particularly *Porphyromonas gingivalis*, is a concern. Combined with poor oral hygiene, this increases the risk of gingivitis, gingival enlargement (GE), and periodontitis. GE often appears within 1–2 months, hindering hygiene and treatment, sometimes requiring surgical removal. Management follows a two-phase approach.¹⁸ The first phase, known as cause-related therapy, aims to modify etiological factors. This is followed by a review period, after which a second surgical phase may be considered if the condition persists.

Table 4: Gingival recession (%) at T2 and T3. At T2, >85% of incisors and premolars showed no recession. By T3, mild recession (0.1–1.0 mm) increased, notably in mandibular incisors and maxillary molars. Advanced recession (>1.0 mm) remained uncommon but rose in mandibular central incisors and molars. Overall, recession progressed gradually, with anterior teeth more affected than premolars

Tooth	Recession Depth (mm)	T2 (%)	T3 (%)
Mandibular Central Incisor	0	85.3	47.8
	0.1–1.0	13.2	42.0
	>1.0	1.5	10.2
Mandibular Lateral Incisor	0	87.3	66.5
	0.1–1.0	12.2	29.1
	>1.0	0.5	4.4
Maxillary First Premolar	0	96.0	59.6
	0.1–1.0	3.5	—
	>1.0	0.5	—
Maxillary Second Premolar	0	92.8	—
	0.1–1.0	3.0	—
	>1.0	—	—
Maxillary First Molar	0	93.4	46.8
	0.1–1.0	5.5	37.0
	>1.0	0.0	3.4
Maxillary Second Molar	0	42.9	45.7
	0.1–1.0	10.3	46.7
	>1.0	1.1	7.6

Adapted from Morris et al.¹⁹ Re-drawn and modified by the authors.

Gingival recession, an apical shift of the gingival margin exposing the root, can cause aesthetic issues, hypersensitivity, loss of periodontal support, hygiene challenges, and higher caries risk. Periodontal disease and mechanical trauma are primary contributors. Morris et al found that only minimal gingival recession was present immediately after orthodontic treatment. Although gingival recession increased between the post-treatment (T2) and follow-up periods (T3), as shown in **Table 4**, the extent of recession was not severe.¹⁹ No correlation was observed between the degree of mandibular incisor proclination and gingival recession during or after treatment, as shown in **Figure 4**. However, a weak association was noted between the amount of maxillary expansion during treatment and subsequent gingival recession observed after treatment. Orthodontic forces can also lead to gingival recession by moving tooth roots too close to or through the alveolar cortical plates, resulting in bone dehiscences. Since recession typically occurs in areas with underlying bone dehiscences, it is reasonable to assume that gingival tissue lacking proper alveolar bone support may recede further.

Gingival recession (GR) management includes restorative, orthodontic, and surgical options. Restorations can mask localised defects but must avoid plaque-retentive margins. Orthodontic repositioning may aid in bone growth and gingival migration, although surgery may be necessary in some cases. Frenectomy is advised for high frenal attachments that worsen recession and hinder hygiene.²⁰ Additionally, orthodontic treatment can sometimes result in black triangles, which occur due to root divergence or papillary loss during incisor alignment. Several clinical approaches can be employed to correct the aesthetic and functional challenges. Interproximal reduction or restorative camouflage.²¹ Another significant risk associated with orthodontic mechanics is root resorption, which often affects the maxillary incisors and occurs when excessive intrusive forces exceed the cementum repair capacity. Risk reduction involves maintaining strict oral hygiene—using electric toothbrushes, chlorhexidine rinses, and floss threaders—to limit plaque and inflammation. Surgical options include gingivectomy for enlargement and connective tissue grafts for recession.

Additionally, biomechanical caution is essential—avoiding excessive orthodontic forces and carefully monitoring high-risk movements, such as intrusion and space closure, can help prevent irreversible damage. While inflammation typically subsides after orthodontic treatment, some residual defects, including gingival recession and root resorption, may persist. Successful orthodontic outcomes rely on preemptive periodontal assessment, patient education, and interdisciplinary collaboration to minimise irreversible periodontal damage.

2.4.1. Critical appraisal

Cohort studies show mild gingival recession and inflammation are possible, but causality with orthodontics

alone is uncertain. Outcomes are heavily influenced by hygiene and biotype. Preventive periodontal assessment and hygiene instruction are justified, though evidence strength is low to moderate.

2.5. External apical root resorption (EARR)

Root resorption occurs when the pressure exerted on cementum surpasses its ability to repair, leading to dentin exposure and subsequent degradation by multinucleated odontoclasts. Feiglin et al²² (1984) stated that Orthodontic tooth movement can lead to apical root resorption; however, affected teeth generally remain vital, eliminating the need for endodontic treatment. This resorption is attributed to the forces applied during orthodontic movement and the formation of a hyaline zone, a localised sterile necrotic area within the periodontal ligament.²² Several radiographic studies have demonstrated an increase in both the severity and incidence of root shortening after orthodontic treatment, with various contributing factors, including hormonal and nutritional influences, genetic predisposition, treatment duration, previous trauma, patient age, and the stage of root formation at the onset of treatment.²³ McFadden et al found that root shortening was more significant in maxillary than mandibular incisors with an average reduction of 1.84 mm in maxillary incisors and 0.61 mm in mandibular incisors, as shown in **Table 5**.²³ A comparative study on the incidence and extent of root resorption, changes in marginal bone support, and clinical crown length between treated upper jaw and untreated lower jaw demonstrated that 50% of maxillary teeth exhibited apical root resorption, with 88% of these cases being less than 2mm.²⁴ Furthermore, a statistically significant reduction in marginal bone support was observed in the upper jaw, but not in the lower jaw.²⁴

Table 5: Root length changes in maxillary (A) and mandibular (B) incisors among 38 patients. Most showed mild–moderate elongation (0–4 mm). Root shortening was rare (7 maxillary, 3 mandibular). Overall, elongation was more common, especially mild gains (0–2 mm) in mandibular incisors

Tooth Group	Root Length Category (mm)	Patient Count
Maxillary Incisors (A)	Gain of 0–2 mm	15
	Gain of 2–4 mm	12
	Reduction of 4–6 mm	7
Mandibular Incisors (B)	Gain of 0–1 mm	17
	Gain of 1–2 mm	15
	Gain of 2–4 mm	7
	Reduction of 0–2 mm	3

Adapted from McFadden et al.²³ Redrawn by the authors.

To effectively detect and monitor root resorption, monitoring requires a periapical radiograph at 6 months, followed by three-month radiographic follow-ups,

particularly for anterior teeth in high-risk patients. A panoramic radiograph taken at 6–12 months helps evaluate resorption and bracket positioning at the end of alignment.²⁵ Prevention of root resorption involves the use of light forces (<70 g), intermittent treatment, and 3D imaging, which have been proven to be effective. Anti-inflammatory agents (such as triamcinolone, dexamethasone, and indomethacin) may suppress resorbing cells. Long-term calcium hydroxide therapy is favoured for its antibacterial action, enzyme inhibition, and promotion of hard tissue repair.

Treatment is typically discontinued once radiographic evidence of a continuous periodontal ligament space is observed (6–12 months), followed by permanent obturation. The orthodontist plays a crucial role in preventing EARR during treatment, as shown in **Figure 2** and implementing appropriate interventions when resorption occurs.

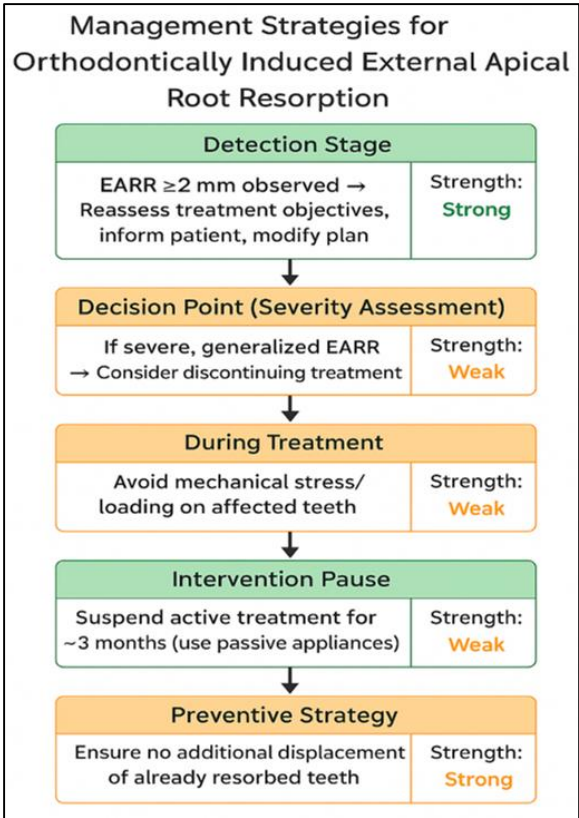


Figure 2: Recommended management strategies for orthodontically induced external apical root resorption (EARR)²⁵

Ultimately, after-treatment care as shown in **Table 6** and managing the consequences of EARR remain primary concerns for both patients and their dental practitioners.²⁶

2.5.1. Critical appraisal

Evidence consistently links intrusive forces to EARR, with several prospective studies supporting this association. However, risk thresholds and predictive markers remain unclear. Clinical consensus supports light forces and radiographic monitoring as the safest approach.

Table 6: After treatment care requirements

Item	Requirement	Strength
1	Follow standard retention protocol post-orthodontics to ensure tooth stability and monitor root/periodontal health.	Strong
2	Inform patients: root resorption halts after appliance removal, is painless, but may cause mobility if root <10 mm; risk of early tooth loss rises with periodontal disease.	Strong

2.6. Pain and discomfort after orthodontic appointments

Orthodontic pain typically peaks 24 hours post-procedure and is primarily attributed to periodontal ligament inflammation and ischemic changes, with psychological factors such as anxiety and fear further lowering pain thresholds. Erdinç AM, Dinçer B²⁶ found that pain following archwire insertion begins within 2 hours, peaks at 24 hours, and gradually decreases by the third day.

Their study also indicated no significant differences in pain perception between genders or wire sizes, except for greater use of pain relief medication in the 0.014-inch wire group at 24 hours. Pain is a common experience in orthodontic treatment, with approximately 90% to 95% of patients reporting discomfort. Pain intensity peaks within the first 24 hours after the application of orthodontic forces, and most patients continue to experience discomfort throughout the first week, as shown in **Figure 3**. As many as 25% to 42% still report some degree of pain after 7 days, and while pain is generally manageable, up to 10% of patients discontinue treatment due to early pain experiences.²⁷

Furthermore, the thought of having a painful experience discourages some patients from seeking orthodontic treatment. Moreover, the anticipation of pain discourages some individuals from seeking orthodontic treatment, even when it is objectively needed.²⁷ Statistical evaluation using repeated-measures analysis of variance followed by post-hoc studentized range testing showed that, at every time point assessed, patients given a placebo reported significantly higher levels of discomfort compared with those who received ibuprofen or aspirin.²⁸ Furthermore, ibuprofen was found to be more effective than aspirin, particularly at 6 and 24 hours and on the second day after separator placement, as well as at 2 and 6 hours and on days 2, 3, and 7 following archwire placement. These findings support the recommendation of ibuprofen as the preferred analgesic for managing post-orthodontic discomfort. Beyond pharmacological interventions, non-pharmacological pain management strategies have also proven effective. Vibratory stimulation, chewing wafers, and Transcutaneous Electrical Nerve Stimulation (TENS) have been explored as methods to alleviate orthodontic pain.

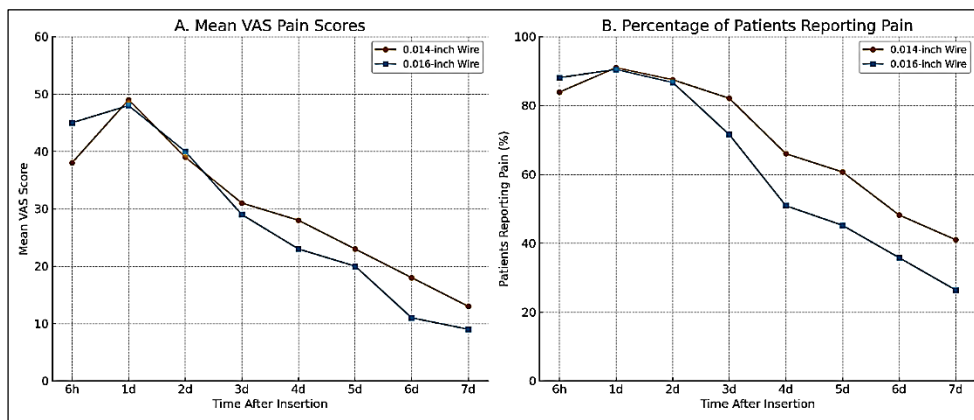


Figure 3: Pain response following insertion of 0.014-inch and 0.016 inch orthodontic wires. (A) Mean visual analogue scale (VAS) pain scores recorded at 6 hours, and daily up to 7 days after wire placement. (B) Percentage of patients reporting pain at corresponding time intervals. Both wire types demonstrated peak discomfort within the first 24 hours, followed by a gradual reduction over the subsequent week. Pain intensity and frequency were consistently greater with the 0.016 inch wire compared to the 0.014-inch wire²⁷

Roth et al. investigated the use of transcutaneous electrical nerve stimulation (TENS) for pain relief following tooth separation.²⁹ The study reported that discomfort was noticeably lower at 24, 36, and 48 hours in the treatment group in relation to the placebo and control groups. Reducing pain during the initial stages of therapy helps patients adapt more easily, encourages cooperation, and makes orthodontic care more comfortable overall.

2.6.1. Critical appraisal

Strong RCT evidence shows NSAIDs, especially ibuprofen, are effective for pain control. Non-drug methods show promise but lack consistent validation. Pain is predictable, short-lived, and best managed with combined approaches.

2.7. Iatrogenic possibilities and soft tissue complications in orthodontic treatment

Orthodontic therapy can lead to unintended complications such as soft tissue trauma, enamel wear, and headgear injuries.

- 1 Brackets and wires often cause irritation or ulcers, and ceramic brackets in particular tend to produce more enamel abrasion than stainless steel ones. Viazis AD³⁰ reported that metal brackets caused minimal enamel wear—half of the samples showed no detectable damage—while all ceramic bracket groups exhibited significant abrasion, as shown in **Table 7**.

- 2 Headgear-related ocular injuries, particularly in 10–14-year-olds, are another concern. Of recorded cases, 57% were linked to molar bands and 43% to removable appliances.³¹ Most incidents (63%) occurred at night, particularly in patients with a history of prior headgear detachment. The presence of oral microorganisms on the face-bow significantly increases the risk of infection, which may be resistant to antibiotics. In rare cases, such infections have caused permanent vision loss or contralateral endophthalmitis.³¹
- 3 Micro-implants are useful for anchorage but may cause peri-implantitis, tissue irritation, or even break during removal, making hygiene and careful handling important. Corticotomy can shorten treatment time but may lead to bone loss, bleeding, or root resorption. Clear aligners are more comfortable and aesthetic, though not without their own drawbacks.

The MAUDE³² database has reported adverse events related to Invisalign, including breathing difficulties, anaphylactic reactions, and skin issues—possibly due to isocyanate exposure in polyurethane materials. Relapse from poor force control can be prevented with precise appliance design (cinched wires, radio-opaque materials), headgear safety education, and regular monitoring.

Table 7: Staged description of enamel surface changes associated with ceramic bracket contact and repeated mechanical loading

Stage	Description	Clinical / Experimental Interpretation
A	Initial bracket-to-enamel contact	At the onset of appliance placement, the ceramic bracket comes into direct contact with the enamel surface. Only subtle microscopic alterations are observed at this stage, typically in the form of fine scratches or superficial wear marks. Clinically, these changes are not visible but may represent the earliest signs of mechanical stress on the enamel.

Table 7 Continued....		
B	Repeated mechanical loading	With ongoing orthodontic forces and bracket activity, the enamel begins to show more pronounced surface alterations. Progressive abrasion results in localized enamel loss, with defects expanding beyond the initial contact points. Experimentally, this stage is characterized by measurable surface roughness, while clinically it may predispose to plaque accumulation and further wear.
C	Baseline enamel surface (pre-test condition)	Prior to mechanical testing or cyclic loading, the enamel surface appears smooth, intact, and uniform. This stage serves as the reference standard, allowing comparison with post-loading conditions. SEM or schematic evaluations typically show a continuous, unbroken surface with no evidence of pitting or irregularities.
D	Post-test enamel surface (after cyclic loading)	Following repeated bracket loading, the enamel surface becomes irregular, with visible pits, fissures, and cracks. These defects indicate loss of surface integrity and represent the cumulative effect of mechanical stress. Clinically, such changes may compromise enamel strength and esthetics, and may increase susceptibility to plaque retention and sensitivity.

Adapted from Viazis et al.³⁰ Re-drawn by the authors

Ultimately, optimising biomechanics while preserving soft tissues, ensuring informed consent, and adopting interdisciplinary care are key to safe outcomes.

2.7.4. Critical appraisal

In vitro studies confirm higher enamel wear with ceramic brackets compared to metal. Evidence is moderate but not always reflective of intraoral conditions. Clinically, caution is advised when selecting ceramic appliances.

2.8. Allergic reactions in orthodontic treatment

Orthodontic materials can cause allergic reactions, most commonly from nickel, latex, or acrylic. *Nickel allergy*, often seen in young females with prior sensitivities, may lead to nickel allergic contact stomatitis (NiACS), as shown in **Figure 4**, Oral diagnosis is challenging, as lesions can resemble trauma, autoimmune disorders, or aphthous ulcers. Nickel sensitivity is usually confirmed with patch testing or in-vitro cell-proliferation assays.³³

Acrylic resins in retainers and dentures may provoke allergic reactions. Stomatitis venenata, associated with wearing plastic dentures, has been reported, with findings indicating that the liquid monomer of methyl methacrylate can cause allergic reactions upon contact with the skin or oral mucosa.³⁴ Research suggests that residual monomer from incomplete polymerisation acts as the primary allergen responsible for contact stomatitis caused by acrylic resin. Symptoms of allergic reactions include erythema, burning sensations, and urticaria. Symptoms include erythema, burning, and urticaria. The selection of glove materials should also be considered to reduce exposure. Self-reported hand dermatoses affect 42% of dental professionals,³⁵ as shown in **Table 8**.

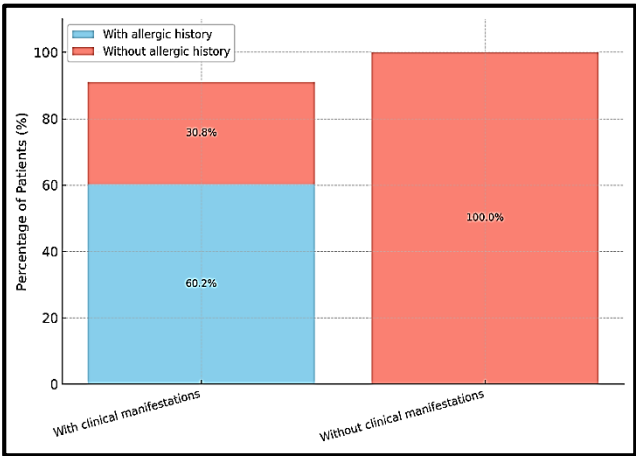


Figure 4: Distribution of allergic history among patients with and without clinical manifestations of nickel-associated contact sensitivity (NiACS).Patients with clinical manifestations of NiACS showed a higher frequency of prior allergic history (60.2%) compared with those without history (30.8%). In contrast, all patients without clinical manifestations reported no allergic history (100%)³³

Allergic reactions in orthodontics are managed with nickel-free brackets (ceramic, titanium), NRL-free products (nitrile gloves, steel ligatures), and resin substitutes (polycarbonate, clear aligners). Patient history, patch testing, and hypoallergenic materials are key for prevention and safety.

2.8.1 Critical appraisal

Nickel, latex, and acrylic allergies are documented but rare, with evidence mostly from small studies and case reports. Despite weak data, clinical vigilance and alternative materials are recommended for sensitive patients.

Table 8: Specific occupation-related factors reported by the dental professionals in association with skin or respiratory symptoms. Total number of subjects with symptoms (n = 49)

Exposure Category	Specific Agents / Materials	Reported Number of Complaints	Notes on Clinical Relevance
Resins and Adhesives	(Meth)acrylates, dental composites	14 (acrylates), 5 (composites)	Frequently used in restorative procedures; associated with skin and respiratory sensitivity.
Protective Equipment	Natural rubber latex gloves, face masks	13 (latex gloves), 6 (masks)	Common allergens; latex linked to contact dermatitis, masks to irritation or breathing issues.
Cleaning Agents	Workplace detergents, disinfectant sprays	5 (detergents), 1 (sprays)	Regular exposure may lead to dermatitis or respiratory irritation.
Dental Materials	Iodoform, eugenol, gypsum (plaster)	1 each	Occasionally used substances; rare but possible sensitizers.
Procedural Factors	Dental instruments, sandblasting exposure	1 each	Mechanical exposure and dust-related irritation reported.

Adapted from Kerosuo et al.³⁵ Re-drawn and modified by the authors.

2.9. Temporomandibular disorder (TMD) and orthodontic treatment

TMD, which affects the TMJ and masticatory muscles, involves pain, joint sounds, and restricted movement. The assessment of mandibular dysfunction includes;

1. Evaluating key symptoms such as pain or tenderness in the masticatory muscles or TMJs
2. Sounds during condylar movement
3. Limited or uncoordinated mandibular motion
4. Abnormal mandibular reference positions.

This comprehensive approach evaluates both pathological and subclinical dysfunction.³⁶ The aetiology of TMD is multifactorial, involving occlusal discrepancies (e.g., posterior crossbites), psychological stress, hormonal influences (notably higher prevalence in women), and joint hyperlaxity.

A study by Sadowsky & Begole evaluated TMJ function in 75 orthodontically treated patients (ages 25–55) who had undergone complete fixed appliance therapy 10–35 years earlier.³⁷ A high prevalence of mandibular shift from the retruded contact to the intercuspal position was observed in both the treated and control group. However, it was significantly greater in the control group.

Functional appliances, such as the Herbst, are effective in treating Class II malocclusions, particularly in growing patients with a brachyfacial growth pattern.³⁸ Relapse can stem from poor growth, unstable occlusion, or oral habits. Retention is often supported with removable appliances. In acute TMD, orthodontic care should be delayed, while active cases are managed conservatively with splints or physiotherapy. Long-term stability relies on a biopsychosocial approach that considers occlusion, psychology, and posture.

2.9.1. Critical appraisal

Long-term cohort studies show no clear causal link between orthodontics and TMD. Evidence is relatively strong in reassuring that orthodontics does not increase TMD risk. Conservative management remains the standard for symptomatic cases.

2.10. Relapse and inadvertent tooth movement post orthodontic treatment

Retention must be maintained until functional reorganization of the bone has occurred; however, Oppenheim noted that this rule lacked precise data and had limited clinical value.³⁹ The duration of retention is highly individualized, influenced by factors such as age, constitution, race, type of anomaly, treatment duration, and appliance type. Unlike bone and the periodontal ligament, gingival tissue does not fully return to its pretreatment state, which may contribute to post-retention relapse.⁴⁰ Age-related changes like arch narrowing and incisor crowding also play a role.⁴¹ Retention methods include removable Hawley retainers as shown in **Figure 6**, clear aligners as shown in **Figure 7**, fixed retainers (bonded lingual wires) and adjunct techniques like interproximal reduction (IPR)⁴² as shown in. A four-week period without archwires before debonding, and selective stripping of lower anterior teeth in high-risk patients, have been suggested to enhance stability. Rotated teeth are over-corrected early in treatment, with care taken to avoid excessive expansion.⁴² As Oppenheim emphasized, while many can move teeth, success lies in precise, biologically sound diagnosis and execution.

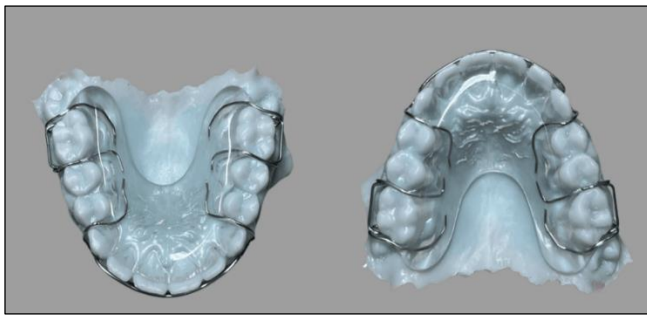


Figure 5: Maxillary removable orthodontic appliance (Hawley-style retainer) placed on a dental cast, occlusal view



Figure 6: Dental casts fitted with clear thermoplastic retainers



Figure 7: Intraoral view showing a fixed lingual retainer bonded to the anterior teeth

For orthodontic treatment to be truly effective, its benefits must significantly outweigh the risks of potential harm. Clinicians must carefully evaluate the risk–benefit ratio of treatment options before planning management of malocclusions. Preventive measures, patient-specific considerations, and careful treatment planning are essential to minimize adverse effects. Further well-designed studies and controlled clinical trials are necessary to better understand the etiology, severity, and influencing factors of these iatrogenic effects.

Managing these challenges means building treatment plans that suit the patient, clearly explaining the risks, and, when needed, working with other specialists. Basic steps such as preventive routines, clear guidance for patients, and steady monitoring should be part of everyday practice. “At a

broader level, making dental materials safer, keeping records of treatment problems, and improving training on treatment-related risks can all lower complications and improve results.”

2.11. Critical appraisal

Evidence confirms relapse is multifactorial, influenced by growth, biotype, and appliance type. Few RCTs compare retention methods, making guidelines inconsistent. Long-term individualized retention is widely accepted as necessary.

3. Conclusion

Orthodontic treatment is an effective approach to correcting malocclusion and improving patient confidence, but it is accompanied by a spectrum of iatrogenic risks. This review shows that enamel demineralization, root resorption, periodontal changes, pulpal alterations, and treatment-related pain are the most frequently reported adverse effects. The strength of supporting evidence varies: high-quality trials strongly support preventive fluoride use, strict oral hygiene, and NSAID-based pain control, while data on pulpal changes, soft tissue irritation, and relapse remain weaker and often derived from observational studies. Material-related reactions such as nickel and latex sensitivity are uncommon but clinically relevant, underscoring the importance of individualized treatment planning and allergy screening.

Key clinical recommendations:

1. Prescribe high-fluoride regimens during active treatment to reduce white spot lesions.
2. Use light, biologically compatible forces (<70 g) with regular radiographic follow-up to detect early root resorption.
3. Screen for thin periodontal biotypes and reinforce hygiene instructions before treatment.
4. Manage pain with NSAIDs such as ibuprofen, alongside non-pharmacological methods when appropriate.
5. Use nickel-free or latex-free alternatives for patients with sensitivities.
6. Apply individualized, long-term retention protocols to limit relapse.

4. Limitations

This review has limitations inherent to its narrative design. Although major databases were searched and reference lists screened, the absence of a registered protocol means there is potential for selection bias. The included evidence is heterogeneous, with many studies based on small samples, short follow-up periods, or inconsistent diagnostic criteria. These factors restrict direct comparisons across studies and reduce the strength of some conclusions.

5. Future Directions

Further progress requires high-quality, multicenter clinical trials using standardized definitions and outcome measures. Specific gaps include the long-term effects of orthodontic forces on pulpal vitality, the role of periodontal biotype in treatment stability, and the most effective retention strategies to reduce relapse. Addressing these areas will help refine evidence-based guidelines, reduce complications, and ensure that the benefits of orthodontic therapy consistently outweigh its unintended consequences.

6. Ethical Approval

None.

7. Source of Funding

None.

8. Conflict of Interest

None.

9. Acknowledgement

None.

References

1. Kerosuo H, Hausen H, Laine T, Shaw WC. The influence of incisal malocclusion on the social attractiveness of young adults in Finland. *Eur J Orthod*. 1995;17(6):505–12. <https://doi.org/10.1093/ejo/17.6.505>.
2. Behrents RG. Iatrogenics in orthodontics. *Am J Orthod Dentofacial Orthop*. 1996;110(3):235–8. [https://doi.org/10.1016/s0889-5406\(96\)80005-9](https://doi.org/10.1016/s0889-5406(96)80005-9).
3. Zachrisson BU. Cause and prevention of injuries to teeth and supporting structures during orthodontic treatment. *Am J Orthod*. 1976;69(3):285–300. [https://doi.org/10.1016/0002-9416\(76\)90077-4](https://doi.org/10.1016/0002-9416(76)90077-4).
4. Bass JK, Fine H, Cisneros GJ. Nickel hypersensitivity in the orthodontic patient. *Am J Orthod Dentofacial Orthop*. 1993;103(3):280–5. [https://doi.org/10.1016/0889-5406\(93\)70009-D](https://doi.org/10.1016/0889-5406(93)70009-D).
5. Leong JW, Kunzel C, Cangialosi TJ. Management of the American Heart Association's guidelines for orthodontic treatment of patients at risk for infective endocarditis. *Am J Orthod Dentofacial Orthop*. 2012;142(3):348–54. <https://doi.org/10.1016/j.ajodo.2012.05.002>.
6. Toms SR, Eberhardt AW. A nonlinear finite element analysis of the periodontal ligament under orthodontic tooth loading. *Am J Orthod Dentofacial Orthop*. 2003;123(6):657–65. [https://doi.org/10.1016/s0889-5406\(03\)00164-1](https://doi.org/10.1016/s0889-5406(03)00164-1).
7. Maxfield BJ, Hamdan AM, Tüfekçi E, Shroff B, Best AM, Lindauer SJ. Development of white spot lesions during orthodontic treatment: perceptions of patients, parents, orthodontists, and general dentists. *Am J Orthod Dentofacial Orthop*. 2012;141(3):337–44. <https://doi.org/10.1016/j.ajodo.2011.08.024>.
8. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. *Am J Orthod*. 1982;81(2):93–8. [https://doi.org/10.1016/0002-9416\(82\)90032-x](https://doi.org/10.1016/0002-9416(82)90032-x).
9. Heinrich-Weltzien R, Kühnisch J, van der Veen M, de Josselin de Jong E, Stösser L. Quantitative light-induced fluorescence (QLF)—a potential method for the dental practitioner. *Quintessence Int*. 2003;34(3):181–8.
10. Baysan A, Lynch E, Ellwood R, Davies R, Petersson L, Borsboom P. Reversal of primary root caries using dentifrices containing 5,000 and 1,100 ppm fluoride. *Caries Res*. 2001;35(1):41–6. <https://doi.org/10.1159/000047429>.
11. West NX. Dentine hypersensitivity: preventive and therapeutic approaches to treatment. *Periodontol*. 2008;48:31–41. <https://doi.org/10.1111/j.1600-0757.2008.00262.x>.
12. Murray LE, Roberts AJ. The prevalence of self-reported hypersensitive teeth. *Niger Postgrad Med J*. 2011;18(3):205–9.
13. Brannstrom M. The hydrodynamic theory of dentinal pain: sensation in preparations, caries, and the dentinal crack syndrome. *J Endod*. 1986;12(10):453–7. [https://doi.org/10.1016/S0099-2399\(86\)80198-4](https://doi.org/10.1016/S0099-2399(86)80198-4).
14. Liu XX, Tenenbaum HC, Wilder RS, Quock R, Hewlett ER, Ren Y-F. Pathogenesis, diagnosis and management of dentin hypersensitivity: an evidence-based overview for dental practitioners. *BMC Oral Health*. 2020;20(1):220. <https://doi.org/10.1186/s12903-020-01199-z>.
15. Krishnan V, Davidovitch Z. Cellular, molecular, and tissue-level reactions to orthodontic force. *Am J Orthod Dentofacial Orthop*. 2006;129(4):469.e1–32. <https://doi.org/10.1016/j.ajodo.2005.10.007>.
16. Barwick PJ, Ramsay DS. Effect of brief intrusive force on human pulpal blood flow. *Am J Orthod Dentofacial Orthop*. 1996;110(3):273–9. [https://doi.org/10.1016/s0889-5406\(96\)80011-4](https://doi.org/10.1016/s0889-5406(96)80011-4).
17. Fiorellini JP, Stathopoulou PG. Anatomy of the periodontium. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, editors. *Carranza's Clinical Periodontology*. 12th ed. St. Louis, MO: Elsevier Saunders; 2015. p. 9–10.
18. Chesterman J, Beaumont J, Kellett M, Durey K. Gingival overgrowth: Part 2: management strategies. *Br Dent J*. 2017;222(3):159–65. <https://doi.org/10.1038/sj.bdj.2017.111>.
19. Morris JW, Campbell PM, Tadlock LP, Boley J, Buschang PH. Prevalence of gingival recession after orthodontic tooth movements. *Am J Orthod Dentofacial Orthop*. 2017;151(5):851–9. <https://doi.org/10.1016/j.ajodo.2016.09.027>.
20. Mostafa D, Fatima N. Gingival recession and root coverage up to date, a literature review. *Dentistry review*. 2022;2(1):100008. <https://doi.org/10.1016/j.dentre.2021.100008>.
21. Al-Zarea BK, Sghaireen MG, Alomari WM, Bheran H, Taher I. Black triangles causes and management: A review of literature. *Br J Appl Sci Technol*. 2015;6(1):1. <https://doi.org/10.9734/BJAST/2015/11287>.
22. Feiglin B. Root resorption. *Aust Dent J*. 1986;31(1):12–22. <https://doi.org/10.1111/j.1834-7819.1986.tb02978.x>.
23. McFadden WM, Engstrom C, Engstrom H, Anholm JM. A study of the relationship between incisor intrusion and root shortening. *Am J Orthod Dentofacial Orthop*. 1989;96(5):390–6. [https://doi.org/10.1016/0889-5406\(89\)90323-5](https://doi.org/10.1016/0889-5406(89)90323-5).
24. Hollender L, Rönnerman A, Thilander B. Root resorption, marginal bone support and clinical crown length in orthodontically treated patients. *Eur J Orthod*. 1980;2(4):197–205. <https://doi.org/10.1093/ejo/2.4.197-a>.
25. Sondejker CFW, Lamberts AA, Beckmann SH, Kuitert RB, van Westing K, Persoon S, et al. Development of a clinical practice guideline for orthodontically induced external apical root resorption. *Eur J Orthod*. 2020;42(2):115–24. <https://doi.org/10.1093/ejo/cjz034>.
26. Erdinç AM, Dinçer B. Perception of pain during orthodontic treatment with fixed appliances. *Eur J Orthod*. 2004;26(1):79–85. <https://doi.org/10.1093/ejo/26.1.79>.
27. Bergius M, Broberg AG, Hakeberg M, Berggren U. Prediction of prolonged pain experiences during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2008;133(3):339.e1–8. <https://doi.org/10.1016/j.ajodo.2007.09.013>.
28. Ngan P, Wilson S, Shanfeld J, Amini H. The effect of ibuprofen on the level of discomfort in patients undergoing orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1994;106(1):88–95. [https://doi.org/10.1016/S0889-5406\(94\)70025-7](https://doi.org/10.1016/S0889-5406(94)70025-7).
29. Roth PM, Thrash WJ. Effect of transcutaneous electrical nerve stimulation for controlling pain associated with orthodontic tooth

- movement. *Am J Orthod Dentofacial Orthop.* 1986;90(2):132–8. [https://doi.org/10.1016/0889-5406\(86\)90045-4](https://doi.org/10.1016/0889-5406(86)90045-4).
30. Viazis AD, DeLong R, Bevis RR, Rudney JD, Pintado MR. Enamel abrasion from ceramic orthodontic brackets under an artificial oral environment. *Am J Orthod Dentofacial Orthop.* 1990;98(2):103–9. [https://doi.org/10.1016/0889-5406\(90\)70003-U](https://doi.org/10.1016/0889-5406(90)70003-U).
 31. Samuels RH, Orth D, Orth M. A review of orthodontic face-bow injuries and safety equipment. *Am J Orthod Dentofacial Orthop.* 1996;110(3):269–72.
 32. Allareddy V, Nalliah R, Lee MK, Rampa S, Allareddy V. Adverse clinical events reported during Invisalign treatment: Analysis of the MAUDE database. *Am J Orthod Dentofacial Orthop.* 2017;152(5):706–10. <https://doi.org/10.1016/j.ajodo.2017.06.014>.
 33. Genelhu MCLS, Marigo M, Alves-Oliveira LF, Malaquias LCC, Gomez RS. Characterization of nickel-induced allergic contact stomatitis associated with fixed orthodontic appliances. *Am J Orthod Dentofacial Orthop.* 2005;128(3):378–81. <https://doi.org/10.1016/j.ajodo.2005.03.002>.
 34. Nealey ET, Del Rio CE. Stomatitis venenata: reaction of a patient to acrylic resin. *J Prosthet Dent.* 1969;21(5):480–4. [https://doi.org/10.1016/0022-3913\(69\)90068-7](https://doi.org/10.1016/0022-3913(69)90068-7).
 35. Kerosuo E, Kerosuo H, Kanerva L. Self-reported health complaints among general dental practitioners, orthodontists, and office employees. *Acta Odontol Scand.* 2000;58(5):207–12. <https://doi.org/10.1080/000163500750051755>.
 36. Solberg WK, Woo MW, Houston JB. Prevalence of mandibular dysfunction in young adults. *J Am Dent Assoc.* 1979;98(1):25–34. <https://doi.org/10.14219/jada.archive.1979.0008>.
 37. Sadowsky C, BeGole EA. Long-term status of temporomandibular joint function and functional occlusion after orthodontic treatment. *Am J Orthod.* 1980;78(2):201–12. [https://doi.org/10.1016/0002-9416\(80\)90060-3](https://doi.org/10.1016/0002-9416(80)90060-3).
 38. Pancherz H. The Herbst appliance—its biologic effects and clinical use. *Am J Orthod.* 1985;87(1):1–20. [https://doi.org/10.1016/0002-9416\(85\)90169-1](https://doi.org/10.1016/0002-9416(85)90169-1).
 39. Oppenheim A. The crisis in orthodontia. *Int J Orthod Dent Child.* 1934;20(3):250–8. [https://doi.org/10.1016/S0097-0522\(33\)90477-3](https://doi.org/10.1016/S0097-0522(33)90477-3).
 40. Redlich M, Shoshan S, Palmon A. Gingival response to orthodontic force. *Am J Orthod Dentofacial Orthop.* 1999;116(2):152–8. [https://doi.org/10.1016/s0889-5406\(99\)70212-x](https://doi.org/10.1016/s0889-5406(99)70212-x).
 41. Littlewood SJ, Kandasamy S, Huang G. Retention and relapse in clinical practice. *Aust Dent J.* 2017;62 Suppl 1:51–7. <https://doi.org/10.1111/adj.12475>.
 42. Aasen TO, Espeland L. An approach to maintain orthodontic alignment of lower incisors without the use of retainers. *Eur J Orthod.* 2005;27(3):209–14. <https://doi.org/10.1093/ejo/cji012>.

Cite this article: Nagrath S, Sood S, Chainta D, Negi N, Negi KS, Mahajan M, Negi S. Orthodontic iatrogenics: Balancing benefits and risks. *Int J Oral Health Dent.* 2025;11(3):195–206.