Potential Hazards of Orthodontic Treatment – What Your Patient Should Know

PAMELA E. ELLIS AND PHILIP E. BENSON

Abstract: **Orthodontic treatment carries with it the risks of tissue damage, treatment failure and an increased predisposition to dental disorders. The dentist must be aware of these risks in order to help the patient make a fully informed choice whether to proceed with orthodontic treatment. This paper outlines the potential hazards and suggests how they may be avoided or minimized.**

***Dent Update* 2002; 29: 492–496**

Clinical Relevance: **A high proportion of adolescent patients are considering or undergoing orthodontic treatment. It is important that they understand the potential risks of wearing an orthodontic appliance.**

Although orthodontic treatment has recognized benefits, including improvements in dental health, function, appearance and self-esteem, orthodontic appliances can cause harm. The decision whether to proceed with orthodontics requires comparison of the potential risks with the potential benefits.

It is important that general dental practitioners (GDPs), even if they do not undertake orthodontic treatment themselves, are aware of these risks. The GDP usually initiates the orthodontic referral and a patient will often seek their reassurance, after the consultation with an orthodontist, about whether to go ahead with treatment. Only when the patient is informed about the reason for treatment and the risks involved can he or she make a fully informed choice and consent to go ahead.

Some patients are more at risk than others; they need to be identified early and managed appropriately to avoid adverse sequelae. The GDP's contribution is crucial, even if he or she does not fit orthodontic appliances, in helping to ensure that braces are properly maintained by reinforcing oral hygiene and preventive measures. The GDP may also help in an emergency if a wire or bracket is causing soft-tissue damage.

The potential hazards of orthodontic treatment are three-fold:

- tissue damage;
- treatment failure;
- greater predisposition to dental disorders.

***Pamela E. Ellis**, BDS, MSc, FDS, MOrth, Specialist Registrar in Orthodontics, and **Philip E Benson**, PhD, FDS (Orth), Senior Lecturer/Honorary Consultant in Orthodontics, Orthodontic Department, Charles Clifford Dental Hospital, Sheffield.*

TISSUE DAMAGE

Both intra-oral and extra-oral tissues are at risk of damage during orthodontic treatment.

Enamel Damage

Reports of the prevalence of enamel damage after orthodontic treatment have varied (Figure 1). In one cross-sectional study, 50% of individuals undergoing orthodontics had a non-developmental enamel opacity, compared with 25% of controls.[1] Another study found that, even 5 years after treatment, orthodontic patients had a significantly higher incidence of enamel opacities than untreated controls.[2]

The most important means of preventing demineralization is to ensure that the patient's oral hygiene is of a high standard throughout treatment. Fluoride is a well established anti-cariogenic agent and several methods of applying fluoride have been used during orthodontic treatment to minimize the risk of demineralization.

Topical Application

Daily use of 0.05% sodium fluoride mouthrinse has been shown to be effective,[3] although only about 50% of patients complied with daily rinsing. The worst compliers are often those patients with poor oral hygiene who are most in

***Figure 1.** Generalized enamel demineralization following orthodontic treatment.*

Figure 2. *Appearance of a fluoride-releasing elastomeric ligature (upper right lateral incisor) after 6 weeks in the mouth.*

need of mouthrinse.

Other topical applications, including stannous fluoride mouthrinse,[4] stannous fluoride gel[5] and fluoride varnish,[6] have been employed but each requires adequate compliance from the patient to work.

Fluoride-releasing Materials

Given the poor compliance with patient-applied measures, attempts have been made to use materials that release fluoride over a period of time. Fluoride-containing composite resins have not been found to be effective at reducing demineralization,[7–9] but both compomer[10] and glass-ionomer cements[11] have. However, glass-ionomers are weaker than composite resin and consequently there is a higher number of bracket failures with such materials.[12] This problem may be solved with the development of stronger resin-reinforced glass-ionomer materials.

Evidence suggests that fluoride-releasing elastomeric ligatures may reduce the prevalence of demineralization,[13,14] although the addition of fluoride to elastics may affect their physical properties so that they deteriorate rapidly in the mouth[15] (Figure 2). Other devices have been developed that release small amounts of fluoride over a sustained period of time, possibly up to 6 months, before having to be replaced.[16]

Enamel Fractures

Occasionally small cracks in the enamel surface are seen following removal of orthodontic brackets. Such cracks provide stagnation areas for the development of caries, cause partial tooth fracture, or may discolour.[17]

Zachrisson *et al*.[17] found that the prevalence of pronounced cracks in relation to the total number of cracks was 6% for debonded/banded teeth and 4% for untreated teeth. There were appreciably more cracks with chemically bonded ceramic brackets.[18]

Periodontium

Following placement of a fixed appliance there is gingival inflammation in almost all orthodontic patients (Figure 3). Fortunately, this inflammation is usually transient and does not lead to attachment loss.[19–21] Gingival hyperplasia can be a problem around orthodontic bands, leading to pseudo-pocketing and giving the illusion of attachment loss; however, this usually resolves within weeks of debanding.[22]

Adult patients may be at risk of periodontal problems, particularly patients who seek orthodontic treatment because of pre-existing periodontal disease (for example drifting incisors; Figure 4). Orthodontic treatment is not contraindicated in this group, provided the disease is controlled and the patient is sufficiently motivated and dextrous to maintain excellent oral hygiene during treatment.[23] Three-monthly periodontal checks and routine scaling and polishing are advisable. The orthodontist will often modify the mechanics for these patients by keeping the forces light in view of the shortened root support. Other patients who require particular attention are those with systemic diseases such as diabetes or epilepsy, particularly poorly controlled diabetics and the epileptics whose seizures are controlled by phenytoin-based drugs, which can cause gingival hyperplasia.

Particular periodontal problems can occur with certain types of treatment – for example, in the Class III patient who has appliances prior to orthognathic

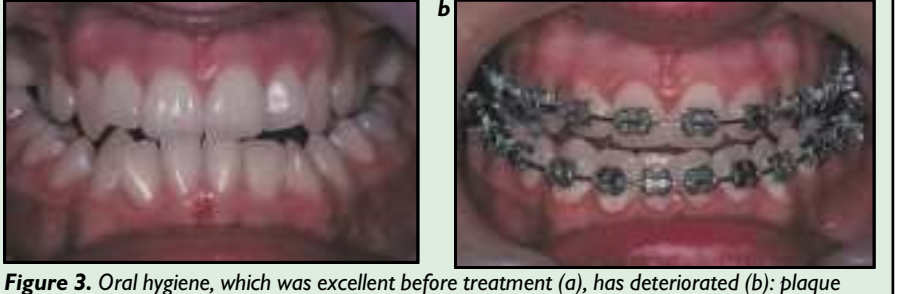

Figure 3. *Oral hygiene, which was excellent before treatment (a), has deteriorated (b): plaque accumulation and marginal gingivitis can be seen.*

Figure 4. *A patient with previous periodontal disease seeking orthodontic treatment to correct the drifted incisors. The periodontal disease is now under control and oral hygiene is excellent.*

Figure 5. *Radiograph of anterior teeth during orthodontic treatment showing blunting of the lateral incisor apex, which is characteristic of orthodontic-induced root resorption.*

Figure 6. *Mucosal trauma caused by a removable appliance component.*

surgery, the lower incisors are often deliberately proclined, which may lead to gingival recession or even gingival clefts.[24] Previously it was feared that closure of extraction spaces, particularly when the lower first premolars are lost, may lead to bunching of the gingival tissues and hence long-term periodontal problems[25] but this is not usually the case.

Root Damage

Root shortening is almost inevitable in patients with fixed appliances (Figure 5). Fortunately this is usually minimal, affecting the apical 1–2 mm only. Such resorption should not compromise the long-term health of the teeth.[26] More severe resorption, where more than a quarter of the root length is lost, occurs in only 3% of patients.[27]

Risk factors associated with an increased incidence and severity of root resorption include the pre-treatment root form or length, previous dental trauma and the type of mechanics used. Teeth with blunted, pipette-shaped, or short roots are at increased risk of resorption.[28,29] Root-filled teeth are not necessarily at greater risk of root resorption and may safely be moved using orthodontic appliances, providing:

- teeth are clinically symptomless and radiographically satisfactory;
- it is 6 months after a new root filling;
- a radiograph is taken 6 months after the start of active treatment.[30]

The orthodontist should employ sensible measures to minimize the risk of severe root resorption by good pretreatment assessment of root shape and length. For at-risk individuals, precautions can be taken either before treatment to modify the plan or during treatment to change the mechanics used.

Pulp Damage

Orthodontic patients may suffer from transient pulp ischaemia, causing pain and discomfort in the first few days after adjustment of an appliance. This usually settles within a week, although pulp death following orthodontic treatment is occasionally reported.[31] If appropriate treatment mechanics and forces are used, pulp damage is unlikely to be a significant problem.

Soft-tissue damage

Intra-oral and extra-oral soft tissues can be damaged in two ways:

- direct damage by removable or fixed components (Figure 6);
- indirect damage by allergic reactions to nickel and latex.

Patients may suffer from mouth ulcers, due to rubbing of the lips and cheeks on brackets, bands or cleats, as they become accustomed to fixed appliances. Fortunately, the oral tissues quickly toughen up to a new appliance, but whilst this is occurring vegetable wax can be used to give temporary relief. Occasionally, palatal or lingual arches may cause trauma to the palate or tongue.

Some individuals continually damage their appliances leading to extra, unscheduled appointments and prolonged treatment times. It helps to recognize these patients early, counsel them about diet and habits and take extra precautions, such as placing bands rather than bonds.

The Use Of Headgear

Headgear can cause injury if it is displaced either during sleep or rough play. The headgear bow is not only sharp but also covered in oral bacteria. A penetrating eye injury may not cause immediate pain, but the oral bacteria multiply and the eye can be lost due to overwhelming infection.[32] To minimize the risk of injury, headgear now has safety features that stop it being accidentally displaced or recoiling back into the face or eyes (Figure 7). Patients should be given both verbal and written safety instructions after fitting headgear.[33]

Damage from Orthodontic Materials

Orthodontic materials can induce allergic reactions.

Nickel

Nickel hypersensitivity affects three in ten of the general population,[34] and nickel is found in stainless steel wires, bands, brackets and headgear. Patients become nickel sensitive due to previous contact with jewellery, glasses and watches[34] and may develop dermatitis in response to direct contact with headgear. Females are most susceptible, perhaps due to ear piercing.

For sensitive patients, exposed metalwork should be covered with tape or plasters or headgear use discontinued. Intra-oral signs and symptoms of nickel hypersensitivity are rare because the concentrations of nickel necessary to provoke a reaction in the mouth are higher than those needed on the skin.[35] Intra-oral signs are highly variable and difficult to diagnose, for example erythematous areas[36] or severe gingivitis in the absence of plaque.[37] Because such signs and

Figure 7. *NiTom safety headgear bow (Ortho Kinetics Corp, Vista, CA, USA). This has an additional arm that clips over the headgear bow distal to the molar tube.*

Figure 8. *Poor oral hygiene and demineralization has forced early discontinuation of treatment. There is residual spacing, cross-bite, increased overbite and overjet.*

symptoms are difficult to spot, nickel allergy in response to orthodontic appliances may be under-diagnosed.

Latex

Latex sensitivity may occur in response to contact with latex gloves or elastomeric ligatures (modules) and intra- and extra-oral elastics. In the latex-sensitive patient, steel ligatures or self-ligating brackets may be preferred. The treatment plan might need to be modified, avoiding Class II or Class III traction.

Other Materials

Other orthodontic materials that may cause allergic reactions are composite and acrylic. Toxicity is due to unpolymerized material and is greatest immediately following polymerization, although cytotoxicity is still evident 2 years after polymerization.[38] No-mix adhesives are more toxic than two-paste adhesives.[39]

TREATMENT FAILURE

Failure to complete a course of orthodontic treatment is frustratingly common (4–23%).[40] Its sequelae include residual spacing and malalignment, traumatic overbite, residual overjet, cross-bite and relapse (Figure 8).

Treatment may fail through:

- patient non-compliance;
- incorrect diagnosis;
- incorrect management.

It is essential to talk to all orthodontic patients to establish whether they perceive a need for a treatment and fully appreciate their commitment – treatment times of approximately 2 years, followed by a lengthy period of retention. They must demonstrate good oral hygiene and be free from active dental disease at the start.

A patient's motivation to maintain good oral hygiene throughout treatment can decline. This may lead to early removal of appliances to avoid damage to the teeth and supporting structures. When patients request their appliances to be removed early for personal reasons treatment goals cannot be met. Sometimes patients have difficulty in tolerating the appliance most appropriate for correction of their malocclusion. In such cases often a compromised plan can be formulated, but not always.

Treatment may also fail because the diagnosis and treatment plan were incorrectly formulated, for example in a Class III patient where simple treatment fails due to continued growth. We can minimize the number of occasions when treatment goals are not met through good record taking and recognition of our own limitations.

Relapse

Teeth placed in an unstable position during orthodontic treatment have a high potential for relapse. Furthermore, certain occlusal traits, such as rotated teeth and midline diastemas, have a high probability of relapse. Several long-term reviews of patients 10 or 20 years after orthodontic treatment demonstrate that, even with orthodontic treatment of a

TISSUE DAMAGE		
Tissue	**Problem**	**Treatment**
Enamel	Demineralization	Oral hygiene instruction; daily fluoride mouthrinses; fluoridated elastomeric ligatures
	Fractures	Mechanical not chemical bonding (ceramic brackets); careful debonding (especially ceramic brackets)
Periodontium	Gingivitis Bone loss	Good oral hygiene throughout treatment Regular periodontal checks and 3-monthly scaling and polishing in adult patients
Root	Resorption	Identification of 'at risk' individuals; careful use of treatment mechanics
Pulp	Ischaemia Death	Avoidance of excessive forces; pre-warn the patient Caution with heavily restored teeth
Soft tissues	Iatrogenic damage	Careful use of instruments; careful fitting and adjusting of appliances to avoid sharp edges

TREATMENT FAILURE	
Problem	**Treatment**
Incorrect diagnosis	Carefully collect full records and documentation at the start
Incorrect management	Keep up-to-date with latest treatment techniques
Patient non-compliance	Fully inform patient about treatment times and expectations

INCREASING PREDISPOSITION TO OTHER DISORDERS	
Disorder	**Management**
Temporomandibular joint disorder	Record signs and symptoms before treatment; advise patients seeking treatment for such disorder that there may not be an improvement with orthodontics
Periodontal	Maintain good levels of oral hygiene; professional prophylaxis where required

Table 1. *Problems that may occur during orthodontic treatment.*

high standard, with the teeth placed in a seemingly stable position, teeth will still move.[41] It is important that patients understand that teeth move throughout life; this is physiological and not necessarily due to relapse. For teeth to remain straight, some form of indefinite retention will be required.

GREATER PREDISPOSITION TO DENTAL DISORDERS

It has been suggested that orthodontics may increase the predisposition to certain disorders, including temporomandibular disorders and periodontal disease. Studies investigating the relationship between temporomandibular disorders and orthodontic treatment have found no association between the two.[42,43,44] Patients who have undergone orthodontic treatment do not have an increased predisposition to periodontal disease.[20]

Table 1 outlines problems that may occur during orthodontics and lists some suggestions to prevent them. Before contemplating orthodontics, the referring practitioner, patient and orthodontist should reflect on the risks and the benefits of treatment. With vigilant selection, diagnosis, treatment planning, monitoring and timely intervention we can ensure that the majority of our patients benefit by improved facial and dental aesthetics and function.

REFERENCES

1. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. *Am J Orthod* 1982; **81:** 93–98.
2. 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 Dentofac Orthop* 1989; **96:** 423–427.
3. Geiger AM, Gorelick L, Gwinnett AJ, Benson BJ. Reducing white spot lesions in orthodontic populations with fluoride rinsing. *Am J Orthod Dentofac Orthop* 1992; **101:** 403–407.
4. Boyd RL. Comparison of three self-applied topical fluoride preparations for control of decalcification. *Angle Orthod* 1993; **63:** 25–30.
5. Boyd RL. Long-term evaluation of a SnF_2 gel for control of gingivitis and decalcification in adolescent orthodontic patients. *Int Dent J* 1994; **44:** 119–130.
6. Buyukyilmaz T, Tangugsorn V, Ogaard B, Arends J, Ruben J, Rolla G. The effect of titanium tetrafluoride (TiF_4) application around orthodontic brackets. *Am J Orthod Dentofac Orthop* 1994; **105:** 293–296.
7. Mitchell L. An investigation into the effect of a fluoride releasing adhesive on the prevalence of enamel surface changes associated with directly bonded orthodontic attachments. *Br J Orthod* 1992; **19:** 207–214.
8. Turner PJ. The clinical evaluation of a fluoride-containing orthodontic bonding material. *Br J Orthod* 1993; **20:** 307–313.
9. Banks PA, Burn A, O'Brien K. A clinical evaluation of the effectiveness of including fluoride into an orthodontic bonding adhesive. *Eur J Orthod* 1997; **19:** 391–395.
10. Millett DT, McCluskey LA, McAuley F, Creanor SL, Newell J, Love J. A comparative clinical trial of a compomer and a resin adhesive for orthodontic bonding. *Angle Orthod* 2000; **70:** 233–240.
11. Marcusson A, Norevall LI, Persson M. White spot reduction when using glass ionomer cement for bonding in orthodontics: a longitudinal and comparative study. *Eur J Orthod* 1997; **19:** 233–242.
12. Norevall LI, Marcusson A, Persson M. A clinical evaluation of a glass ionomer cement as an orthodontic bonding adhesive compared with an acrylic resin. *Eur J Orthod* 1996; **18:** 373–384.
13. Banks PA, Chadwick SM, Asher-McDade C, Wright JL. Fluoride-releasing elastomerics – a prospective controlled clinical trial. *Eur J Orthod* 2000; **22:** 401–407.
14. Mattick CR, Mitchell L, Chadwick SM, Wright J. Fluoride-releasing elastomeric modules reduce decalcification: a randomized controlled trial. *J Orthod* 2001; **28:** 217–219.
15. Miethke RR. Comment on determination of fluoride from ligature ties. *Am J Orthod Dentofac Orthop* 1997; **111:** 33A.
16. Marini I, Pelliccioni GA, Vecchiet F, Alessandri Bonetti G, Checchi L. A retentive system for intra-oral fluoride release during orthodontic treatment. *Eur J Orthod* 1999; **21:** 695–701.
17. Zachrisson BU, Skogan O, Hoymyhr S. Enamel cracks in debonded, debanded, and orthodontically untreated teeth. *Am J Orthod* 1980; **77:** 307–319.
18. Artun J. A post-treatment evaluation of multibonded ceramic brackets in orthodontics. *Eur J Orthod* 1997; **19:** 219–228.
19. Alstad S, Zachrisson BU. Longitudinal study of periodontal condition associated with orthodontic treatment in adolescents. *Am J Orthod Dentofac Orthop* 1979; **76:** 277–286.
20. Sadowsky C, BeGole EA. Long-term effects of orthodontic treatment on periodontal health. *Am J Orthod* 1981; **80:** 156–172.
21. Polson AM, Subtelny JD, Meitner SW, *et al.* Long-term periodontal status after orthodontic treatment. *Am J Orthod Dentofac Orthop* 1988; **93:** 51–58.
22. Zachrisson BU. Cause and prevention of injuries to teeth and supporting structures during orthodontic treatment. *Am J Orthod* 1976; **69:** 285–300.
23. 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 Dentofac Orthop* 1989; **96:** 191–198.
24. McComb JL. Orthodontic treatment and isolated gingival recession: a review. *Br J Orthod* 1994; **21:** 151–159.
25. Robertson PB, Schultz LD, Levy BM. Occurrence and distribution of interdental gingival clefts following orthodontic movement into bicuspid extraction sites. *J Periodontol* 1977; **48:** 232–235.
26. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: Part 1. Literature review. *Am J Orthod Dentofac Orthop* 1993; **103:** 62–66.
27. Kaley J, Phillips C. Factors related to root resorption in edgewise practice. *Angle Orthod* 1991; **61:** 125–132.
28. Linge BO, Linge L. Apical root resorption in upper anterior teeth. *Eur J Orthod* 1983; **5:** 173–183.
29. Levander E, Malmgren O. Evaluation of the risk of root resorption during orthodontic treatment: a study of upper incisors. *Eur J Orthod* 1988; **10:** 30–38.
30. Drysdale C, Gibbs SL, Ford TR. Orthodontic management of root-filled teeth. *Br J Orthod* 1996; **23:** 255–260.
31. Rotstein I, Engel G. Conservative management of a combined endodontic-orthodontic lesion. *Endodont Dent Traumatol* 1991; **7:** 266–269.
32. Booth-Mason S, Birnie D. Penetrating eye injury from orthodontic headgear – a case report. *Eur J Orthod* 1988; **10:** 111–114.
33. Samuels RH, Jones ML. Orthodontic facebow injuries and safety equipment. *Eur J Orthod* 1994; **16:** 385–394.
34. Bass JK, Fine H, Cisneros GJ. Nickel hypersensitivity in the orthodontic patient. *Am J Orthod Dentofac Orthop* 1993; **103:** 280–285.
35. Magnusson B, Bergman M, Bergman B, Soremark R. Nickel allergy and nickel-containing dental alloys. *Scand J Dent Res* 1982; **90:** 163–167.
36. Dunlap CL, Vincent SK, Barker BF. Allergic reaction to orthodontic wire: report of case. *J Am Dent Assoc* 1989; **118:** 449–450.
37. Grimsdottir MR, Hensten-Pettersen A, Kullmann A. Cytotoxic effect of orthodontic appliances. *Eur J Orthod* 1992; **14:** 47–53.
38. Tell RT, Sydiskis RJ, Isaacs RD, Davidson WM. Long-term cytotoxicity of orthodontic direct-bonding adhesives. *Am J Orthod Dentofac Orthop* 1988; **93:** 419–422.
39. Terhune WF, Sydiskis RJ, Davidson WM. *In vitro* cytotoxicity of orthodontic bonding materials. *Am J Orthod* 1983; **83:** 501–506.
40. Brattstrom V, Ingelsson M, Aberg E. Treatment co-operation in orthodontic patients. *Br J Orthod* 1991; **18:** 37–42.
41. Little RM. Stability and relapse of dental arch alignment. *Br J Orthod* 1990; **17:** 235–241.
42. Kremenak CR, Kinser DD, Melcher TJ, *et al.* Orthodontics as a risk factor for temporomandibular disorders (TMD) II. *Am J Orthod Dentofac Orthop* 1992; **101:** 21–27.
43. Egermark I, Thilander B. Craniomandibular disorders with special reference to orthodontic treatment: An evaluation from childhood to adulthood. *Am J Orthod Dentofac Orthop* 1992; **101:** 28–34.
44. Sadowsky C. The risk of orthodontic treatment for producing temporomandibular disorders: A literature review. *Am J Orthod Dentofac Orthop* 1992; **101:** 79–83.