

Iatrogenic possibilities of orthodontic treatment and modalities of prevention

Nazeer Ahmed Meeran

ABSTRACT

The benefits of orthodontic treatment are numerous and in most cases, the benefits outweigh the possible disadvantages. Orthodontic treatment can play an important role in enhancing esthetics, function, and self-esteem in patients. However, it carries with it the risks of enamel demineralization, tissue damage, root resorption, open gingival embrasures in the form of triangular spaces, allergic reactions to nickel, and treatment failure in the form of relapse. These potential complications are easily avoidable by undertaking certain precautions and timely interventions by both the orthodontist and the patient. The orthodontist must ensure that the patient is aware of the associated risks and stress the importance of the patient's role in preventing these untoward outcomes. The decision whether to proceed with the orthodontic treatment is essentially a risk-benefit analysis, where the perceived benefits of commencing treatment outweigh the potential risks. This article provides an overview of the iatrogenic possibilities of orthodontic treatment and the role of the patient as well as the orthodontist in preventing the associated risks.

Key words: Decalcification, iatrogenic effects, orthodontic treatment

INTRODUCTION

Dental aesthetics are a key factor in overall physical attractiveness, which also contributes to self-esteem.^[1,2] This is one of the main reasons for patients to consider or undergo orthodontic treatment. Although orthodontic treatment has many recognized benefits, including improvement in dental health, function, appearance, and self-esteem, nevertheless orthodontic appliances can cause unwanted complications if adequate care is not taken during the treatment. It is important that the patients are aware of these potential risks, so that they can know their responsibilities and the expectations placed on them during the treatment. This ensures in achieving successful results without any adverse effects after the completion of orthodontic treatment. In the following some of these risks are briefly discussed.

Decalcification

Shannon^[3] recognized orthodontic patients to be at a higher risk of decalcification or caries. An orthodontic appliance could not, within itself, be a cause of caries. However, oral hygiene problems do occur when fixed appliances are worn. Meticulous

attention to oral hygiene is mandatory during the entire treatment period to avoid the risk of enamel decalcification. Banded or bonded teeth, exhibited significantly more white spot lesions compared to the controls without braces.^[4] Ogaard^[5] noticed that even 5 years after completing the treatment, orthodontic patients had a significantly higher incidence of enamel opacities than untreated controls.

Patients undergoing orthodontic treatment have significant changes in the oral environment, including an increase in *Streptococcus mutans* counts, low salivary pH, and increased retention of food particles on the appliance as well as teeth. This may lead to post-orthodontic treatment decalcification in certain patients in the absence of adequate oral hygiene. Fluoride is an anticariogenic agent and various studies^[6,7] showed fluoride to be highly effective in remineralization of incipient lesions and preventing white spot lesions. Fluoride controls plaque activity by blocking bacterial enzyme systems.^[8] Daily administration of topical fluoride and the use of fluoridated toothpaste and mouth rinses is an effective protection against white spot formation. Mouth rinses containing 0.05% sodium fluoride and stannous

Department of Orthodontics and Dentofacial Orthopedics,
Priyadarshini Dental College and Hospital, Thiruvallur,
Tamil Nadu, India

Address for correspondence: Dr. Nazeer Ahmed Meeran,
Priyadarshini Dental College and Hospital,
Pandur, Thiruvallur, Tamil Nadu, India.
E-mail: nazeerortho@yahoo.co.in

Access this article online

Quick Response Code:



Website:

www.jorthodsci.org

DOI:

10.4103/2278-0203.119678

fluoride can be used as a daily regimen during the treatment period. Fluoride varnishes and fluoride gels are also highly effective in preventing enamel demineralization.^[9,10]

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 adhesives have not been found to be effective at reducing demineralization,^[11,12] but both compomers^[13] and glass-ionomer cements^[14,15] are highly efficient in preventing it. However, glass-ionomer cements are weaker than composite resin and consequently there are a higher number of bracket failures when used for orthodontic bonding.^[16] The fluoride-releasing elastomeric ligatures may reduce the prevalence of demineralization^[17] although the addition of fluoride to elastics may affect their physical properties making them deteriorate faster in the mouth.^[18,19] Various slow- release intraoral fluoride release devices^[20] (IFRD) including copolymer membrane device, glass device containing fluoride, Hydroxyapatite- Eudragit RS 100 Diffusion controlled fluoride system and slow release tablets for intra-buccal use have been developed recently, which are capable of releasing small amounts of fluoride over a sustained period of time, possibly up to 6 months, before having to be replaced.^[20,21]

Xylitol, a natural carbohydrate sweetener, is a caries preventive agent. Xylitol is not fermented by most dental plaque bacteria and interferes with the *in-vitro* growth of *Streptococcus mutans*.^[22] Sengun found that Xylitol lozenges can significantly reduce the acidity of dental plaque in fixed appliance patients.^[22] The Xylitol lozenges helped in neutralizing the acidity of dental plaque after the administration of sucrose in orthodontic patients with fixed appliances.

Argon laser irradiation of enamel has been found to reduce the amount of demineralization by 30-50%. Fox^[23] reported that, in addition to decreasing enamel demineralization, laser treatment reduced the threshold pH at which dissolution occurred. Laser irradiation altered the surface morphology while maintaining an intact enamel surface. Several mechanisms for the enhanced caries resistance of enamel after laser irradiation have been proposed, but the exact mechanism is not known. The most likely mechanism for caries resistance is through the creation of micro-spaces within the enamel after being subjected to laser treatment. The micro-spaces created act to trap the released ions and act as sites for mineral reprecipitation within the enamel surface. The use of argon lasers (488 nm) resulted in a significantly lower mean lesion depth when compared with visible light control,^[24] augmenting the fact that argon laser irradiation might prevent white spot lesions during treatment.

ENAMEL FRACTURES DURING DEBONDING

Debonding metal brackets is a relatively simple procedure, where the forces are applied to peel the bracket base away from the enamel surface, leading to bond failure at the

bracket-adhesive interface in most of the patients. The ideal method for debonding metal brackets is to apply a force that peels the bracket base from the enamel surface without damaging the enamel.^[25] A good debonding technique is to squeeze the bracket at the base so that the bracket comes off leaving some residual composite at the enamel surface, which can be cleaned up later with a carbide bur.^[25] The cleanup procedure may be time consuming, but it is better than encountering the risk of enamel cracks and fractures.

Debonding ceramic brackets is comparatively more problematic, due to the higher bond strength as a result of both mechanical and chemical retention of the bracket base to the tooth.^[26] Moreover, shearing forces used for debonding ceramic brackets is likely to cause enamel fractures.^[27] Hence, 4 debonding techniques including mechanical debonding, electrothermal debonding, laser debonding and ultrasonic debonding have been developed specifically for ceramic brackets.^[26,27]

Mechanical debonding involves gripping the enamel – adhesive interface with sharp edged plier blades and applying a controlled slow force until the bracket is removed. This method depends on either the deformation of the bracket or stressing the adhesive to cause adhesive failure. Electro- thermal and laser debonding attempt to achieve bracket debonding by dissolving the bonding cement through heat generation and thus facilitating easy bracket removal.^[28] However, electro-thermal debonding has the risk of mucosal burns and pulpal damage due to the high heat. The main drawback of laser debonding is the cost factor involved. Ultrasonic debonding aims to facilitate debonding using ultrasonic vibrations to break the adhesive interlocking.^[28] Water spray is required during ultrasonic debonding to prevent heat buildup and to avoid pulpal damage. However, the debonding time is prolonged, ranging between 30 to 60 seconds per bracket, making the procedure highly time consuming. Safe debonding technique tries to break the bond at the bracket adhesive interface rather than the enamel-adhesive interface.^[29]

Improper debonding of orthodontic brackets, particularly ceramic brackets, can result in enamel surface cracks.^[25] They can provide stagnation areas for the development of caries, cause partial tooth fracture, or may cause unaesthetic discoloration. Zachrisson^[25] found higher prevalence of cracks in debonded teeth compared to untreated teeth. There were appreciably more cracks with chemically bonded ceramic brackets.^[26] This can be avoided to a large extent by adhering to a proper debonding technique. Identification of enamel cracks and heavily restored dentition may prompt the orthodontist to avoid using ceramic brackets in certain patients.^[28]

Restorative procedures can be carried out to manage the tooth fracture. Enamel fracture on debonding is distressing to both the patient and the orthodontist and is best prevented by taking adequate care during debonding. Applying debonding forces lower than 13 MPa and adhering to

proper debonding techniques can help avoid the incidence of accidental tooth fracture.^[25,28] Diamond burs are not advisable for cleanup because they usually leave a rough surface, which is not easily removed by polishing, resulting in increased plaque retention.^[26]

POTENTIAL ADVERSE EFFECTS TO THE PERIODONTAL TISSUES

Gingival Inflammation

Plaque is the major etiologic factor in the development of gingivitis.^[30] Experimental animal studies have shown that in the absence of plaque, orthodontic forces and tooth movements do not induce gingivitis.^[28-31] In the presence of plaque, however, similar forces are capable of inducing angular bone defects and with tipping and intruding orthodontic tooth movements, attachment loss can occur.^[31] In healthy, reduced periodontal tissue support regions, orthodontic forces kept within the biological limits do not cause gingival inflammation.^[32] The most important factor in the initiation, progression and recurrence of periodontal disease in reduced periodontium is the presence of microbial plaque.^[30-34]

Following placement of a fixed appliance, some amount of gingival inflammation is noticeable in most patients^[35] that is usually transient and does not lead to attachment loss.^[35] Gingival hyperplasia can be a potential problem around orthodontic bands, leading to pseudo-pocketing. However, this usually resolves within weeks of debanding. The importance of plaque control and good oral hygiene must be stressed to the patient before starting the fixed appliance treatment and adequate patient compliance must be ensured throughout treatment to prevent gingival inflammation.

Attachment Loss

In many orthodontic patients, the principal reason for the associated gingival and periodontal inflammation involves mechanical irritation caused by the band or cement, in addition to trapped plaque.^[36,37] The risk of attachment loss can be anticipated when such iatrogenic irritations are present.^[38] Results of a histological study on human periodontal tissues confirm that the orthodontic banding have to be performed with great care along with excellent oral hygiene in order to avoid permanent periodontal destruction.^[38]

Sanders^[39] performed an extensive a review of the evidence-based literature in the fields of periodontics and orthodontics to clarify the relationship between orthodontic tooth movement and various types of common periodontal disorders. It was found that orthodontic treatment using optimum forces, in patients with excellent oral hygiene and in the absence of pre-existing periodontal disorders, does not pose any significant periodontal risk to the patient.^[39] However, in the presence of poor oral hygiene and preexisting untreated periodontal disorders, fixed orthodontic appliances and tooth movement can contribute to significant and permanent periodontal damage.^[40]

Adult patients may be at a higher risk of periodontal problems, particularly patients having some pre-existing periodontal disease.^[40,41] Orthodontic treatment is not contraindicated in this group, provided the disease is well-controlled and the patient is motivated enough to maintain excellent oral hygiene throughout the treatment duration.^[41] The assessment of periodontal status prior to fixed appliance treatment is of utmost importance and any pre-existing problems must be treated before initiating the treatment. Regular periodontal checks and routine scaling and polishing are highly advisable to prevent the aggravation of periodontal problems.^[41,42]

Patients with pre existing periodontal problems and bone loss, must be referred to and treated by the periodontist before initiating the orthodontic treatment.^[43] Moreover, in such patients, there is a slight modification in the biomechanics with the application of minimal and optimum orthodontic forces, bearing in mind the shortened root support.^[43-45]

Gingival Recession

An adequate amount of attached gingiva is necessary for healthy gingival tissue and to deliver orthodontic treatment without adverse periodontal complications.^[46,47] The incisors showed apical displacement of the gingival margin with labial bodily movement.^[48] Loss of connective tissue occurred in the presence of preexisting untreated gingival inflammation was present.^[49] Therefore, if the tooth movement is expected to result in a reduction of soft tissue thickness and an alveolar bone dehiscence may have occurred in the presence of inflammation, gingival recession is a risk.^[49]

Experimental studies^[50-52] indicate that as long as the tooth is moved within the envelope of the alveolar process, the risk of harmful side-effects on the marginal soft tissue is minimal. It has been found that thin, delicate tissues are more prone to exhibit recession during orthodontic treatment than in normal or thick tissue.^[51,52] If the patient exhibits a minimal zone of attached gingiva or thin tissue, a free gingival graft can be performed to enhance the type of tissue around the tooth. This helps in controlling the inflammation and should be done before initiating any orthodontic movement.^[52]

Tooth extraction is usually indicated in patients with tooth size-arch length discrepancy.^[53] Gingival invaginations present as superficial changes in the shape of gingiva, which can occur sometimes in the extraction sites after orthodontic space closure.^[54] The gingival invaginations occurred in 35% of cases after orthodontic space closure procedures.^[55] They vary from mild fissures located in the keratinized gingiva to deep clefts crossing the interdental papilla buccally or lingually through the alveolar bone deeply.^[56,57]

Histological and histo-chemical specimens taken from sites of gingival invagination demonstrated hypertrophy in the epithelial as well as the connective tissues and occasionally, loss of gingival collagen.^[58] The reason for the occurrence of gingival

invaginations is still unknown and requires further investigation. It could be due to the break-up of the continuity of the fiber models within the gingiva, and also the movement of the root.^[59] It has also been proposed that gingival peeling could be the reason for such changes.^[60] Since gingival invaginations could serve as sites for dental plaque accumulation, it has been considered as a potential risk factor for the initiation of periodontal tissue disorders during the course of orthodontic treatment.^[61]

Gingival recession has been known to occur as an adverse effect during the orthodontic treatment or after treatment completion and has been noted more frequently during buccal orthodontic tooth movements.^[62] If teeth having thin tissue are going to be moved lingually, there is a potential for the tissue to move coronally and become thicker.^[63] It is generally advisable to monitor areas of thin gingival tissues periodically as the width of the attached gingiva generally increases with normal growth from the mixed to the permanent dentition.^[64]

It has been found that most cases of gingival recession which occur during an orthodontic treatment occurred in the regions of the upper and lower anterior teeth.^[65-67] The relationship between orthodontic movements and gingival recession has been controversial in relation to tipping movements. Batenhorst *et al.*^[68] found an association between gingival recessions and orthodontic tipping tooth movements of the lower incisors in monkeys. However, other studies revealed no association between gingival recession or mucogingival defects after orthodontic tipping of the incisors.^[69,70]

During surgical decompensation in skeletal class III patients, the lower incisors are often deliberately proclined, which may lead to gingival recession or even gingival clefts.^[71,72] This possibility must be addressed during treatment planning and by undertaking sufficient care when executing the orthodontic treatment.

Black Triangles

Gingival embrasures are defined as the embrasure existing cervical to the interproximal contact.^[73] Open gingival embrasures exist when the embrasure space is not completely filled by the gingival tissue and can contribute to retention of food debris, thus adversely affect the health of the periodontium and are more common in adult patients with bone loss.^[74]

Black triangle or open gingival embrasure can occur as potential complication in about than 1/3 of all adult orthodontic patients and should be discussed with patients prior to initiating orthodontic treatment.^[73,75] The key considerations in restorative and orthodontic treatment involve preserving the interdental papilla and avoiding black triangles in the gingival embrasures of the esthetic zone as open gingival embrasures are visibly unesthetic and adversely affect a person's smile.

In a survey conducted by Kokich *et al.*,^[76] orthodontists perceived a 2 mm open gingival embrasure as noticeably less

attractive when compared with a patient with an ideal smile and normal gingival embrasure. Open gingival embrasures more than 3 mm were considered less attractive by both general dentists and the general population.

Orthodontic Correction of Open Gingival Embrasures

Root divergency of adjacent teeth is highly associated with open gingival embrasures. This either occurs naturally or is caused by improper bracket positioning during the orthodontic treatment. Kurth *et al.*^[75] noticed that a mean root angulation of 3.65° in normal gingival embrasures and an increase in root divergence by 1° increased the probability of occurrence of an open gingival embrasure from 14 to 21%. Bracket repositioning can be performed to converge maxillary incisor roots to reduce or eliminate the open gingival embrasures as paralleling divergent roots decreases the severity of a black triangle.

Care must be taken to ensure that the bracket slots are perpendicular to the long axis of the tooth and not parallel to the incisal edges during bracket placement, especially in adults with attrition of incisal edges. It is important to evaluate the periapical radiograph prior to bracket placement, especially in patients with attrition.^[77] If brackets placement is done based on incisal edges, greater root divergence may cause an open gingival embrasure, which is esthetically unappealing. Bonding brackets with the slots perpendicular to the long axis of the teeth will allow the adjacent roots to converge, and might require the worn incisal edges to be restored or contoured during the course of the treatment or at the end of treatment completion. As roots become more parallel, the contact point lengthen and move apically toward the papilla, thus reducing the incidence of open gingival embrasures.^[77]

Patients with triangular crown morphology are more susceptible to open gingival embrasures as the crowns of the central incisors are much wider incisally than cervically, resulting in a high contact point.^[77] Interproximal reduction (IPR) of enamel between the triangular crowns will broaden the contact area which will reduce open gingival embrasures. Typically, 0.5-0.75 mm of enamel is removed with IPR for correction of black triangles.^[77]

After orthodontic treatment, the direction of tooth movement and the labiolingual thickness of the supporting bone and soft tissue determines whether gingival embrasures will be present at the treatment completion stage.^[78] Maxillary incisor imbrication and rotation might have an association with open gingival embrasure spaces and it would be wise to inform patients with severely imbricated maxillary incisors that they may be predisposed to an open gingival embrasure after the orthodontic treatment.^[78]

Ko-Kimura *et al.*^[73] reported that the severity of crowding does not influence the occurrence of open embrasures as they were found to occur in a similar percentage between patients with incisor crowding of less than 4 mm and those

with 4-8 mm of incisor crowding. It was found that when the crowding was more than 8 mm, the occurrence of black triangles increased by 7%. However, these results were not statistically significant. It was also found that the treatment duration did not have any significant effect on occurrence of open gingival embrasures.^[73]

The height of the alveolar bone relative to the interproximal contact is a significant factor in determining whether a papilla will fill the gingival embrasure. In summary, the incidence of black triangles can be reduced by careful pretreatment evaluation, judgment and treatment planning. The contact position can be changed by removing interproximal enamel, esthetic restorations, or altering the root angulation, depending on the situation.^[73]

ROOT RESORPTION

Root resorption is an unwanted side effect seen with certain type of tooth movements, particularly intrusion. Fortunately, this is usually minimal, affecting the apical 1-2 mm only.^[79,80] It starts initially as either surface resorption or transient inflammatory resorption and if left unchecked, it further proceeds to progressive inflammatory resorption. Such resorption should not compromise the long-term health of the teeth.^[81] More severe resorption, where more than a quarter of the root length is lost, is rare and occurs in only 3% of patients.^[82]

The root apex is affected more than cervical or middle thirds of root because of the following reasons.^[81]

- The applied forces are concentrated on apex, particularly during intrusion.
- It is made of cellular cementum, which is less mineralized and easily capable of being resorbed.
- It depends on patent vascularization and is easily injured in faces of heavy forces with concomitant vascular stasis.

A genetic predisposition to root resorption has been recognized recently.^[83] Decreased IL-1 production in the case of IL-1B allele 1 may result in relatively less catabolic bone modeling (resorption) at the cortical bone interface with the PDL, which may result in prolonged stress concentrated in the root of the tooth, triggering a cascade of fatigue-related events subsequently leading to root resorption.^[83]

Risk factors associated with an increased incidence and severity of root resorption includes the pre-treatment root form or length, previous dental trauma, and the type of mechanics used.^[84,85] Hyperthyroidism has also been proposed as a risk factor for root resorption, but has to be conclusively proved by systematic studies.^[86] Teeth with blunted, pipette-shaped, or short roots are at increased risk of resorption.^[81] Endodontically treated teeth can be safely moved, if the teeth are clinically symptomless and radiographically satisfactory. It is advisable to undertake the orthodontic procedure 6 months after the completion of the root canal treatment.^[87,88]

It is also highly advisable to take progress radiographs during treatment. The orthodontist should employ adequate measures to minimize the risk of severe root resorption by good pretreatment assessment of root shape and length. Cone-beam computed tomography is considered as a better imaging modality for early detection of root resorption.^[89]

If any root resorption is found, an inactive phase of 4 to 6 months before the resumption of orthodontic treatment is currently advocated.^[90] In extreme cases, treatment must be halted; appliances removed, and a surgical or prosthetic treatment plan must be adopted. If root resorption continues after appliance removal or during retention, sequential root canal therapy with calcium hydroxide is advisable. The gutta-percha filling is the definitive therapy only after root resorption ceases.^[90,91] Appropriate counseling and follow-up are necessary should severe resorption be encountered. Fortunately, root resorption rarely results in significant morbidity after orthodontic therapy and the resorptive process usually ceases with the cessation of active forces.^[92]

PULP DAMAGE AND LOSS OF TOOTH VITALITY

Various studies of changes in pulp tissue vascularity during orthodontic treatment, suggest that blood flow to the dental pulp decreases initially after orthodontic force application. However, it increases thereafter until it reaches a peak 7 days after the application of orthodontic force.^[93-96] These processes, depending on the degree of their disturbances, may cause changes in the metabolic cell activity, cell damage, or defense reactions. Orthodontic forces affect the dental pulp inducing vascular changes that are inflammatory in nature.^[97,98] As demonstrated in the rat model, the inflammatory vascular reactions subside within 3 weeks in all tissues.^[93,96]

Orthodontic patients may suffer from transient pulp ischemia, causing pain, and discomfort in the first few days after activation of an appliance. This usually settles within a week although pulp death following orthodontic treatment is occasionally reported.^[99]

This can be minimized by using a smaller sized archwire, preferably round copper Niti, during the initial stages of treatment.^[100,101] The forces used during the alignment phase must be kept as minimal as possible. Higher forces using rectangular copper Niti, does not necessarily mean faster alignment or reduction in the treatment duration. Instead, it just adds to the patient discomfort due to the pain arising from pulpal ischemia, which if continued for a longer duration, may result in permanent pulpal damage.^[102,103] If appropriate biomechanics and forces are used, pulp damage is unlikely to be a significant problem during the treatment.

With the currently available literature,^[101-107] the following recommendations can be summarized for orthodontic patients:

1. A complete and detailed history of the dentition should be taken, with specific attention to history of dental trauma.
2. Radiographic examination of the teeth for evidence of pulpal obliteration is highly important, as these teeth are at a higher risk of irreversible pulpal changes during orthodontic treatment.
3. Patients who have risk factors for pulpal necrosis with orthodontic treatment (impacted teeth, teeth with a history of trauma, caries or restorations, teeth with evidence of pulpal obliteration) should be informed about the risk of pulpal damage during treatment and informed consent taken before the treatment.
4. Light continuous orthodontic forces must be applied to move teeth, respecting physiologic boundaries.
5. Extreme care should be taken to ensure that the intended orthodontic tooth movement does not challenge the apical blood supply (e.g., compressing the root apex against the cortical plate).
6. Pulpal symptoms that arise during orthodontic treatment should be recognized early and treated appropriately without delay.^[106]

DENTIN HYPERSENSITIVITY AFTER INTERPROXIMAL ENAMEL REDUCTION

Interproximal tooth stripping is an alternative to tooth extraction or expansion of alveolar arches and might be necessary in some situations during orthodontic treatment.^[108-110] Interproximal enamel reduction (IER) is used to adjust disproportioned tooth widths and for the correction of mild to moderate crowding. Enamel reduction may lead to complications such as hypersensitivity of interproximal surfaces of stripped teeth. The sensitivity is usually not severe and mostly transient and the symptoms are known to subside over time.^[111,112]

Adequate polishing of the treated enamel surfaces is essential for ensuring good long-term prognosis of the stripped teeth because the surface roughness facilitates plaque accumulation and thereby promoting demineralization or the development of carious lesions.^[113-115]

According to a study done by Danesh *et al.*,^[116] loss of enamel, as measured by subtraction radiography, was significantly lower for the group where the enamel stripping was done with Ortho-Strips compared to those done with diamond burs. Profilometric analysis of enamel roughness showed that the use of Ortho-Strips, O-Drive D30, and New Metal Strips in the grinding mode produced equally rough surfaces. The Air Rotor in the grinding mode produced the significantly roughest surfaces and the authors recommend avoiding them as much as possible. However, there was a significant reduction of the mean roughness values in all groups when the enamel stripping was followed by polishing. It is prudent to ensure good polishing of the stripped surfaces to prevent future problems.

Tooth hypersensitivity could be one of the adverse effects of excessive proximal stripping. Professional interventions for dentinal hypersensitivity include application of fluoride varnish to promote remineralization.^[117,118] Desensitizing tooth pastes are also effective in a majority of patients.^[119] Careful interproximal reduction of enamel within recognized limits, respecting the available enamel thickness using flexible diamond strips or extra fine diamond disks with adequate cooling followed by polishing is recommended to prevent undesirable effects of proximal stripping.

DAMAGE TO THE INTRA-ORAL TISSUES

Mucosal trauma is fairly common during the orthodontic treatment and can be caused by many factors including ulceration by the brackets and the protruding archwires near the molar region, chemical burns from the acid-etchant and clumsy instrumentation.^[120]

Prevention techniques include:

- Careful instrumentation.
- Cutting distal ends short.
- Using bumper sleeve on long spans of wire.
- Providing patients with wax to avoid mucosal irritation from the brackets.
- Using high volume suction and flushing with copious water after the acid etching procedure.

Direct Damage by Removable or Fixed Components

Removable appliances

Removable appliances may be used as active appliances during the treatment for the management of minor orthodontic problems which require a simple tipping or in the form of retainers at the end of fixed orthodontic treatment.^[120,121] They carry with them the risk of tissue impingement by the wire components (retentive clasps, springs, canine retractors etc.). Undercuts should be carefully evaluated in the plaster model and blocked out prior to acrylisation and care taken to avoid any sharp edges in the appliance to avoid trauma during the insertion and removal of the appliance. Patients should be recalled a few days after appliance delivery to check for any tissue impingement or trauma.^[121]

Fixed appliance and its components parts

Lacerations and trauma to the gingiva and oral mucosa may often occur during the course of orthodontic treatment due to rubbing of the lips and cheeks on the archwire, brackets, bands and hooks, especially where long unsupported stretches of wire rest against the lips.^[120] The oral mucosa quickly keratinize and get accustomed to the new appliance relatively fast and the use of dental wax over the bracket and rubber tubing on the unsupported archwire may serve to reduce the initial trauma and discomfort.^[121]

Biomechanics involving arch wire loops and utility arches are often required during orthodontic treatment for space

closure, space maintenance or intrusion. Utmost care must be taken during their fabrication to avoid their extension into the vestibular area, which may cause tissue impingement, ulcerations and other types of tissue damage.^[120] Even minor amounts of continuous tissue impingement, if disregarded, may lead to more serious problems like ulceration or tissue hyperplasia around the loop. In extreme situations, the loop may become completely embedded in the hyperplastic tissue requiring surgical excision for removal of the hyperplastic tissue. Thus, careful fabrication and monitoring of such wire components are essential to avoid such problems.^[120,121]

Occasionally, palatal or lingual arches may cause trauma to the palate or tongue. This can be avoided by taking proper care during their fabrication, ensuring that there are no sharp margins in the fabricated appliance. Care must be taken to ensure that the distal ends of the archwire are cut off flush with the molar tube or cinched toward the tooth to avoid mucosal trauma.^[120,121]

Soft Tissue Complications Related to Micro Implants

Micro-implants are highly useful in orthodontics for skeletal anchorage in critical anchorage situations.^[122] Their simple design and ease in implantation and removal makes them a good option for the some patients requiring skeletal anchorage. However, potential problems and soft tissue complications are common with their use. Impingements and trauma to soft tissue overlying the implant is fairly common causing soft tissue damage to the buccal mucosa and attached gingiva related to the implant site. Peri-implantitis or inflammation of the gingiva around the implant can occur as a result of improper oral hygiene maintenance.^[123] Adequate oral hygiene must be ensured to prevent tissue inflammation around the implant site.

Screw fracture during the micro implant removal can occur rarely in some patients when applying lateral forces during implant removal.^[124] It is better to avoid lateral forces on the micro implants during their removal. If the micro-implants are left in place for a very long time, this could also lead to fracture on removal as a result of partial or full osseointegration.^[125] It is preferable to remove the micro implants as soon as their need is fulfilled rather than waiting for the completion of the entire orthodontic treatment and their removal along with the arch wires and brackets during debonding procedure. This minimizes the risk of implant fracture during removal as a result of partial or complete osseointegration of the micro implants to the surrounding bone.^[124,125]

ENAMEL ABRASION

Stainless steel brackets tend to induce less enamel abrasion than ceramic brackets.^[126] It was also noticed that single crystal ceramic causes more enamel abrasion than polycrystalline ones (needs a reference). Ideally, cross bites must be corrected before placing ceramic brackets and ceramic brackets used on mandibular teeth must be kept out of occlusion to prevent

enamel abrasion. In patients with deep bite, use of bite planes is advocated to minimize interference and the subsequent risk of enamel abrasion.^[127]

HEADGEAR INJURIES DUE TO CARELESS HANDLING

Headgear can cause injury if it is displaced during sleep or rough play. The headgear bow is not only sharp but also covered in oral bacteria.^[128] There is a potential risk of a bilateral injury to the eyes because the inner arms of the face-bow are of the same width as the eyes. A penetrating eye injury might not cause immediate pain, but the oral bacteria multiply and the eye can be lost due to overwhelming infection.^[129] The face bow can also get dislocated during sleep and cause injury to the soft tissues. However, currently available headgear comes with special safety features that stop it being accidentally displaced or recoiling back into the face or eyes. Patients must be instructed regarding the safe use of headgear and if possible written instructions must be given to the parents after fitting the headgear.^[128,129]

Ideally, it would be good if the parents are present to monitor the patient when they wear and remove the headgear. The patient should first remove the head strap before proceeding to remove the face bow, as directly pulling the face bow without loosening the head strap might result in eye injury due to the recoil. Patients should be advised not to wear their headgear while playing. Where a locking face-bow has been fitted, patients should check to make sure it is seated correctly and then confirm the "lock" by trying to pull it anteriorly. The patient and parent should also be advised that in the event of eye injury suspected to be caused by any part of the orthodontic appliance, however minor, requires an immediate ophthalmologic examination.^[128,129]

ACCIDENTAL SWALLOWING OF APPLIANCE COMPONENTS

The accidental swallowing of appliance components, though very rare, is a potential hazard that cannot be taken lightly and every possible effort must be taken in preventing such an untoward incidence from happening. Accidental swallowing of appliances and retainer wires has been documented in the literature.^[130-132] It is not only the smaller components like brackets, bands, and bonded buttons which are swallowed, but even comparatively larger appliances like quad helix^[132] and RME appliances^[133] have been swallowed accidentally. The activation key of RME is one of the components which can be easily ingested by accident. A dental floss can be threaded to the key to minimize the risk. Keyes^[134] has devised a keyless RME appliance which eliminates the risk of accidental ingestion of the activation key. Sometimes the brackets might get accidentally ingested while debonding the appliance. To minimize the risk, some advocate the use of rubber dam for

safer debonding.^[135] It is always safer to debond the brackets with the archwires in the slot and as a unit to prevent loose brackets from flying into the trachea or esophagus accidentally.

The most prevalent emergency diagnostic and management method for lodged esophageal and tracheal objects is endoscopy.^[136] Forward viewing flexible pan endoscopy can be performed under local anesthesia and is best suited for intra-thoracic objects and is available in most tertiary medical centers.^[136]

Another established procedure that is relatively safe and more cost effective is the use of Foley's catheter. This involves passing a balloon distal to the ingested object, usually under fluoroscopic guidance, inflating the balloon, and then withdrawing the catheter along with the object proximally.

Symptoms of trachea-bronchial obstruction such as dyspnea, coughing, and choking indicate an emergency requiring immediate removal of the appliance component; usually by surgery.^[137,138]

Antero-posterior and lateral radiographs will reveal whether the object is lodged in the trachea or the esophagus. If the appliance is in the gastrointestinal tract, the probability is better than 90% that it will pass uneventfully in the stools. However, impaction of large objects or those with sharp objects can lead to ulcerations and perforations and therefore require immediate surgical removal.^[138,139]

Based on the currently available literature,^[137-141] the following recommendations can be made to prevent and manage the incidence of accidental swallowing of the appliance components:

1. All orthodontic practices should take into account the possibility of such an emergency in their standard operating procedures and must be well prepared to handle any contingencies. Staff must be trained to recognize emergencies and individual responsibilities must be delegated to offset any confusion in the event of such an unforeseen emergency.
2. Every orthodontist must undergo a basic life support and first-aid skills course. Updating and honing these skills at least once every 2 years is recommended as cardio pulmonary resuscitation (CPR) recommendations are updated every 5 years.
3. The use of bondable tubes and attachments carries the risk of these attachments being inhaled or swallowed if they are dislodged and hence it is advisable to cinch the archwire whenever possible.
4. Using a gauze pad as protection distal to the archwire or appliance will prevent injury to the mucosa as well as trap any stray wires that fail the "safety hold" of the pliers.
5. High-volume suction should be used while attempting procedures such as banding and bonding of teeth to minimize the risk of accidental swallowing.
6. Cutting tips of instruments must be periodically checked for any sign of wear and tear and the damaged tips or the instruments must be replaced immediately.
7. Removable quadhelix appliance and transpalatal arches must be tethered by a floss during appliance placement and additionally reinforced at the point of attachment (in palatal sheaths), using stainless steel ligatures.
8. During appliance debonding, it may be less risky if the brackets removed are allowed to remain attached to the base archwire so that they are removed as a complete unit.
9. A visual check of the cutting ends of distal cutters for trapped wires and wiping with sterile gauze is highly recommended after cutting the arch wires.

Precautions with Removable Appliances

1. All metal retentive components should be visually inspected at every appointment visit for any sign of fracture due to repeated wear. Refabrication of the appliance is indicated if this is observed.
2. The acrylic plate should be inspected for cracks due to crazing or stress, especially on load-bearing surfaces to preclude accidental damage and swallowing of the broken appliance part during use.
3. It is currently recommended that the acrylic used to fabricate the appliance be preferably radio-opaque as it will facilitate easier localization in the event of accidental ingestion.^[142]
4. The use of textured latex gloves ensures better grip on the instruments and orthodontic appliance during the treatment procedure.^[143,144]

ALLERGIC REACTIONS FROM ORTHODONTIC APPLIANCES

Orthodontic materials have the potential to induce allergic reactions in some patients.^[145,146] Nickel present in orthodontic appliances like brackets, bands, and archwires is responsible for causing allergic reactions in some patients. Latex present in gloves, elastics, and elastomeric ligatures may also cause reactions in a few patients allergic to the protein antigen present in rubber. Methyl methacrylate found in bonding agents and composite is also responsible for allergic reactions in rarely. The reactions might range from ulceration, erythematous lesions, or even anaphylactic shock, depending on the patient.^[145-148]

Nickel

The amount of Nickel in oral mucosa cells has been found to be higher, compared to untreated individuals.^[148] Nickel hypersensitivity affects three in ten of the general population, but clinically noticeable adverse reactions are documented less as the symptoms are very mild and unnoticed. However, severe reactions in the form of eczema and urticaria may sometimes occur in a few individuals.^[145,146] Nickel-induced contact dermatitis is a Type IV delayed hypersensitivity immune response, occurring 24 hours after exposure.^[147,148]

The diagnosis of nickel allergy is based on patient history, clinical findings, and patch testing. Patients become nickel sensitive due to previous contact with ornaments, glasses, and watches^[149] and may develop dermatitis in response to direct contact with headgear. It has been found that females are the most susceptible to nickel allergy. 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.^[150]

Kim^[151] found that titanium wires and brackets were the most inert and can be used intra-orally in a corrosive environment. It contains no nickel and is an excellent alternative for orthodontic patients with nickel allergy. If nickel titanium wires have to be used, then epoxy coating of the wire is recommended. This would reduce the corrosive potential and the subsequent release of nickel. If the epoxy coatings can be maintained during orthodontic procedures, corrosion of the wire and the subsequent release of metal ions into the oral environment are minimized.^[151]

Kusy^[152] evaluated the qualities and advantages of titanium brackets and found that the biocompatibility of titanium brackets was maintained by preserving the integrated base made of a single piece of pure titanium. Lesser stiffness of titanium compared to stainless steel allows torque to be fully expressed without deforming the bracket wings. Ceramic brackets or clear aligners can be used as an alternative in patients with nickel allergy.^[152]

Latex Allergy

Latex sensitivity may occur in some patients, in response to contact with latex gloves or elastomeric ligatures and intra- and extra-oral elastics.^[153] 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 with elastics.^[153]

Allergy to Bonding Agents

It was found that composite and acrylic can cause allergic reactions in some orthodontic patients. Toxicity is due to unpolymerized material (methyl methacrylate) and is greatest immediately after polymerization although cytotoxicity is still evident 2 years after polymerization.^[154] Epoxy resins have been described as the strongest industrial skin allergen produced in the last few decades. Bisphenol A, which has two epoxy groups in its carbon skeletons, belongs to this group of chemicals. Occasional mucosal reactions related to resin restorations in teeth are also reported in patients.

Tang^[154] reported that the presence of oxygen-inhibited layer renders bonding resins 33% more cytotoxic *in vitro*. Light-cured and chemically cured 2-paste materials had their mean cytotoxicities approximating their inert controls over 6 days. Chemical cured liquid-paste materials are more cytotoxic than light-cured and chemically cured 2-paste materials. No-mix

adhesives have been found to be more toxic than two-paste adhesives and must be avoided.^[155]

Terhune *et al.*^[156] tested orthodontic bonding materials for *in vitro* cytotoxicity and found that all materials showed cytotoxicity immediately after preparation. Polymerized adhesives generally showed decreased toxicity. Sealant materials showed statistically significant greater toxicity than paste resins, both initially after mixing and after 30 days. The significant finding in this study was that these materials not only were toxic immediately after mixing but remained toxic for extended periods of time. Excess material should be removed from teeth by thorough scaling and flushing with water and high-volume suction, particularly in areas adjacent to the gingiva.^[157]

PROLONGATION IN THE TREATMENT DURATION

Some individuals frequently damage or break their appliances leading to extra, unscheduled appointments and prolonged treatment times.^[158] These individuals are usually careless with their appliance and it helps to recognize these patients early during the treatment. They can be counseled regarding dietary habits and extra precautions can be taken, such as placing bands in the molar teeth rather than bonding them, to prevent frequent appliance breakages. They must be advised to keep away from contact sports, avoid sticky foods, restrict hard food items in their diet, and avoid biting foodstuff like carrots and apples with their incisors as they are responsible for majority of bond failures in orthodontic patients.^[158]

TREATMENT FAILURE AND RELAPSE

Failure to complete a course of orthodontic treatment is frustratingly common^[159] (4-23%). This may either be due to the patient's insistence on removing the appliance earlier for personal reasons like marriage or the orthodontists' opinion that further continuation of treatment may jeopardize the health of the dentition and the periodontal ligament, in the face of severe root resorption. Sometimes the patient might not be able to maintain oral hygiene in a satisfactory way as expected, resulting in worsening of the periodontal problem and the incidence of white spot lesions, requiring early appliance removal.^[159]

Usually treatment may fail due to: patient non-compliance; incorrect diagnosis and incorrect management. Patient attitude towards treatment plays a very important role in ensuring predictable and successful treatment outcomes.^[160] It is essential to communicate with all orthodontic patients in order to establish whether they perceive a need for the treatment and fully appreciate their commitment to treatment. This is because orthodontic treatment may extend up to 2 years depending on the case, followed by a lengthy period of retention, requiring

adequate patient compliance. They must demonstrate their ability to maintain good oral hygiene, and be free from active dental disease at the start of treatment.^[159,160]

Patient noncompliance in wearing the prescribed orthopedic appliances and class II elastics is also one of the reasons for treatment failure. Sometimes treatment may fail because of incorrect diagnosis and poorly formulated treatment plan.^[160] Rarely, it could be due to unforeseen circumstances like unpredicted mandibular growth in a skeletal Class III patient, beyond the expected age, which might not be under the control of the orthodontist.^[161] We can minimize the number of occasions when treatment goals are not met by taking complete and accurate patient history and diagnostic records, ensuring positive patient attitude before starting the treatment, analyzing the various treatment options available, and choosing the best among them.^[160,161]

Finally, recognizing and acknowledging our own limitations is very important in avoiding treatment failures. This is especially true when some orthodontists try to treat a purely surgical case by orthodontic camouflage, with poor results, either due to patient compulsion and his insistence to be treated non-surgically or due to improper diagnosis. This could have been avoided by patient counseling and explaining the limitations of certain treatment protocols and establishing the treatment goals before starting the treatment. Recent advances in digital imaging are a boon to the clinician, where the visualized treatment objective (VTO) can be utilized as an education and motivational tool for the patient.^[162] The patients can visualize the treatment goals and end result even before starting the treatment, with the help of these newer software programs, thus ensuring better patient compliance and motivation right at the start of treatment.

Relapse

Finishing and detailing the occlusion play an important role in maintaining the stability of the teeth in the new position, by ensuring good cusp to fossa relationship.^[163-165] Planning the anchorage at the start of treatment is essential to prevent unwanted mesial drift of the anchor molars. Teeth placed in an unstable position have a high potential for relapse.^[166] Loss of anchorage might result in the case finishing in an end on molar relationship, which might contribute to post-treatment relapse in some patients.^[167] Rotated teeth have a high chance of relapse and the extent of overcorrection has to be planned by the treating clinician depending on the severity of the initial rotation. Midline diastema too has a high probability of relapse requiring fixed retention indefinitely.^[168]

Several long-term reviews of patients 10 or 20 years after orthodontic treatment demonstrate that, even with orthodontic treatment of high standard, with the teeth placed in a seemingly stable position, teeth will still move in the absence of adequate retention.^[169-170] It is important that the patients understand that teeth move throughout life; this is physiological and not

necessarily due to relapse. The patients must be informed before the start of treatment so that the patient does not have unrealistic expectations regarding the treatment. For teeth to remain straight, some form of indefinite retention will be required. The retention required for a particular patient must be planned well before the start of treatment.

TMJ PROBLEMS

The role of occlusion in the development of temporomandibular disorders (TMD's) has been well researched in the orthodontic literature and is a subject of high controversy.^[171-175] A review of the currently available literature regarding the relationship between morphologic and functional occlusal factors relative to TMD reveals that there is a relatively low association of occlusal factors in causing TMD.^[171,172]

Severe skeletal anterior open bite, discrepancy between centric relation and centric occlusion (CR-CO discrepancy) greater than 4 mm, over-jet greater than 6 to 7 mm, unilateral lingual crossbite, and five or more missing posterior teeth are the conditions that have been associated with specific diagnostic groups of TMD conditions.^[172] It has been proposed by McNamara *et al.*^[172] that the first three factors often are associated with TMJ arthropathies and may be the result of osseous or ligamentous changes within the temporomandibular articulation.

The current literature evidence indicates that orthodontic treatment performed during adolescence generally does not increase or decrease the odds of developing TMD later in life.^[173-176] It has been found that there is no elevated risk of TMD associated with any particular type of orthodontic mechanics or with extraction protocols.^[174] Although a stable occlusion is a reasonable orthodontic treatment goal, not achieving a specific gnathologically ideal occlusion does not necessarily result in TMD signs and symptoms.^[172,176] Thus, according to the existing literature, the relationship of TMD to occlusion and orthodontic treatment is minor. Signs and symptoms of TMD occur in healthy individuals and increase with age, particularly during adolescence; thus, TMDs that originate during various types of dental treatment may not be related to the treatment but may be a naturally occurring phenomenon.^[173,174] Despite the current evidence indicating that orthodontic treatment is not a contributory factor,^[173-176] it is yet highly advisable to carry out a TMD examination for all potential orthodontic patients to detect preexisting problems.

CONCLUSION

Before proceeding with orthodontic treatment, both the patient and the orthodontist should reflect on the risks and the benefits of the proposed treatment. Most of the adverse effects of orthodontic treatment can be easily avoided by undertaking certain precautions during the treatment. Patient's attitude is also an important factor in ensuring predictable and successful

treatment outcomes. The orthodontist must counsel the patient, ascertain his or her expectations, and assess whether they are realistic. Some patients, particularly those contemplating surgery, may expect a radical change in their personal life or have expectations of restoring a failing marriage and are high liability cases to treat because of over-expectation from the proposed treatment. They must be recognized at the start and the limitations must be explained to them. With vigilant selection, diagnosis, treatment planning, monitoring, timely intervention, and good patient cooperation, we can ensure that the majority of orthodontic patients benefit by improved facial and dental aesthetics as well as function, without any unwanted side effects at the end of treatment.

REFERENCES

- 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;17:505-12.
- Gazit-Rappaport T, Shailish MH, Gazit E. Psychosocial reward of orthodontic treatment in adult patients. *Eur J Orthod* 2010;32:441-6.
- Shannon L. Prevention of decalcification in orthodontic patients. *J Clin Orthod* 1981;15:694-705.
- Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. *Am J Orthod* 1982;81:93-8.
- 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* 1989;96:423-7.
- Geiger AM, Gorelick L, Gwinnett AJ, Benson BJ. Reducing white spot lesions in orthodontic populations with fluoride rinsing. *Am J Orthod* 1992;101:403-7.
- Boyd RL. Comparison of three self-applied topical fluoride preparations for control of decalcification. *Angle Orthod* 1993;63:25-30.
- Oogard B, Gjermo P, Rolla G. Plaque inhibiting effect in orthodontic patients of a dentifrice containing stannous fluoride. *Am J Orthod Dentofacial Orthop* 1980;78:266-72.
- Delbem AC, Brighenti FL, Vieira AE, Cury JA. *In vitro* comparison of the cariostatic effect between topical application of fluoride gels and fluoride toothpaste. *J Appl Oral Sci* 2004;12:121-6.
- Büyükyilmaz T, Tangugsorn V, Ogaard B, Arends J, Ruben J, Rølla G. The effect of titanium tetrafluoride (TiF₄) application around orthodontic brackets. *Am J Orthod Dentofacial Orthop* 1994;105:293-6.
- Turner PJ. The clinical evaluation of a fluoride containing orthodontic bonding material. *Br J Orthod* 1993;20:307-13.
- 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;19:391-5.
- Millett DT. A comparative clinical trial of a compomer and a resin adhesive for orthodontic bonding. *Angle Orthod* 2000;70:233-40.
- 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;19:233-42.
- Vahid-Dastjerdie E, Borzabadi-Farahani A, Pourmofidi-Neistanak H, Amini N. An in-vitro assessment of weekly cumulative fluoride release from three glass ionomer cements used for orthodontic banding. *Prog Orthod* 2012;13:49-56.
- 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;18:373-84.
- Banks PA. Fluoride-releasing elastomers - a prospective controlled clinical trial. *Eur J Orthod* 2000;22:401-7.
- Mattick CR, Mitchell L, Chadwick SM, Wright J. Fluoride-releasing elastomeric modules reduce decalcification: A randomized controlled trial. *J Orthod* 2001;28:217-9.
- Miethke RR. Comment on determination of fluoride from ligature ties. *Am J Orthod Dentofacial Orthop* 1997;111:33A.
- Pessan JP, Al-Ibrahim NS, Bauzalaf MA, Taumba KJ. Slow-release fluoride devices: A literature review. *J Appl Oral Sci* 2008;16:238-44.
- Marini I. A retentive system for intraoral fluoride release during orthodontic treatment. *Eur J Orthod* 1999;21:695-701.
- Sengun A, Sari Z, Ramoglu SI, Malkoç S, Duran I. Evaluation of the dental plaque pH recovery effect of a xylitol lozenge on patients with fixed orthodontic appliances. *Angle Orthod* 2004;74:240-4.
- Fox JL, Yu D, Otsuka M, Higuchi WI, Wong J, Powell GL. Initial dissolution rate studies on dental enamel after CO₂ laser irradiation. *J Dent Res* 1992;71:1389-98.
- Noel L, Rebellato J, Sheats RD. The effect of argon laser irradiation on demineralization of human enamel adjacent to orthodontic brackets: An *In vitro* Study. *Angle Orthod* 2003;73:249-58.
- Zachrisson BU, Skogan O, Höymyhr S. Enamel cracks in debonded, debanded, and orthodontically untreated teeth. *Am J Orthod* 1980;77:307-19.
- Artun J. A post-treatment evaluation of multibonded ceramic brackets in orthodontics. *Eur J Orthod* 1997;19:219-28.
- Eslamian L, Borzabadi-Farahani A, Mousavi N, Ghasemi A. A comparative study of shear bond strength between metal and ceramic brackets and artificially aged composite restorations using different surface treatments. *Eur J Orthod* 2012;34:610-7.
- Naini FB, Gill DS. Tooth fracture associated with debonding a metal orthodontic bracket: A case report. *World J Orthod* 2008;9:268:e32-6.
- Viazis AD, Cavanaugh G, Bevis RR. Bond strength of ceramic brackets under shear stress: An *in vitro* report. *Am J Orthod* 1990;98:214-21.
- Loe H, Theilade E, Jensen SB. Experimental gingivitis in man. *J Periodontol*. 1965;36:177-87.
- Ericsson I, Thilander B. Orthodontic forces and recurrence of periodontal disease: An experimental study in the dog. *Am J Orthod* 1978;74:41-50.
- Ericsson I, Thilander B. Orthodontic relapse in dentitions with reduced periodontal support: An experimental study in dogs. *Eur J Orthod* 1980;2:51-7.
- Ericsson I, Thilander B, Lindhe J. Periodontal conditions after orthodontic tooth movements in the dog. *Angle Orthod* 1978;48:210-8.
- Ericsson I, Thilander B, Lindhe J, Okamoto H. The effect of orthodontic tilting movements on the periodontal tissues of infected and non-infected dentitions in dogs. *J Clin Periodontol* 1977;4:278-93.
- Polson AM, Subtelny JD, Meitner SW, Polson AP, Sommers EW, Iker HP, et al. Long-term periodontal status after orthodontic treatment. *Am J Orthod* 1988;93:51-8.
- Boyd RL, Baumrind S. Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissue versus those of adolescents. *Angle Orthod* 1992;62:117-26.
- Zachrisson S, Zachrisson BU. Gingival condition associated with orthodontic treatment. *Angle Orthod* 1972;42:26-34.
- Alexander SA. Effects of orthodontic attachments on the gingival health of permanent 2nd molars. *Am J Orthod Dentofac Orthop* 1991;199:337-40.
- Sanders NL. Evidence-based care in orthodontics and periodontics: A review of the literature. *J Am Dent Assoc* 1999;130:521-7.
- Eliasson LA, Hugoson A, Kuroi J, Siwe H. The effects of orthodontic treatment on periodontal tissues in patients with reduced periodontal support. *Eur J Orthod* 1982;4:1-9.
- Steffensen B, Storey AT. Orthodontic intrusive forces in the treatment of periodontally compromised incisors: A case report. *Int J Perio Rest Dent* 1993;13:433-41.
- McComb JL. Orthodontic treatment and isolated gingival recession: A review. *Br J Orthod* 1994;21:151-9.
- Mathews DP, Kokich VG. Managing treatment for the orthodontic patient with periodontal problems. *Semin Orthod* 1997;3:21-38.
- Gartrell JG, Mathews DP. Gingival recession: The condition, process and treatment. *Dental Clin North Am* 1976;1:199-213.
- Dorfman HS. Mucogingival changes resulting from the mandibular incisor tooth movement. *Am J Orthod* 1978;74:286-97.
- Dannan A. An update on periodontic - orthodontic interrelationships. *J Indian Soc Periodontol* 2010;14:66-71.
- Lang NP, Loe H. The relationship between the width of keratinized gingiva and gingival health. *J Periodontol* 1972;43:623-7.

48. Dorfman HS. Mucogingival changes resulting from mandibular incisor tooth movement. *Am J Orthod* 1978;74:286-97.
49. Coatoam GW, Behrents RG, Bissada NF. The width of keratinized gingiva during orthodontic treatment: Its significance and impact on periodontal status. *J Periodontol* 1981;52:307-13.
50. Foushee DG, Moriarty JD, Simpson DM. Effects of mandibular orthognathic treatment on mucogingival tissues. *J Periodontol* 1985;56:727-33.
51. Maynard JG. The rationale for mucogingival therapy in the child and adolescent. *Int J Periodontics Restorative Dent* 1987;7:36-51.
52. Steiner GG, Pearson JK, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol* 1981;52:314-20.
53. Bolton WA. The clinical application of a tooth size analysis. *Am J Orthod* 1962;48:504-29.
54. Edwards JG. The prevention of relapse in extraction cases. *Am J Orthod* 1971;60:128-44.
55. Kuroi J, Ronnerman A, Heyden G. Long-term gingival conditions after orthodontic closure of extraction sites: Histological and histochemical studies. *Eur J Orthod* 1982;4:87-92.
56. Rivera Circuns AL, Tulloch JF. Gingival invagination in extraction sites of orthodontic patients: Their incidence, effects on periodontal health, and orthodontic treatment. *Am J Orthod* 1983;83:469-76.
57. Wehrbein H, Bauer W, Diedrich PR. Gingival invagination area after space closure: A histologic study. *Am J Orthod Dentofacial Orthop* 1995;108:593-8.
58. Ronnerman A, Thilander B, Heyden G. Gingival tissue reactions to orthodontic closure of extraction sites: Histologic and histochemical studies. *Am J Orthod* 1980;77:620-6.
59. Atherton JD. The gingival response to orthodontic tooth movement. *Am J Orthod* 1970;58:179-86.
60. Robertson PB, Schultz LD, Levy BM. Occurrence and distribution of interdental gingival clefts following orthodontic movement into bicuspid extraction sites. *J Periodontol* 1977;48:232-5.
61. Helm S, Petersen PE. Causal relation between malocclusion and periodontal health. *Acta Odontol Scand* 1989;47:223-8.
62. Steiner GG, Pearson JK, Ainamo J. Changes of the Marginal Periodontium as a Result of Labial Tooth Movement in Monkeys. *J Periodontol* 1981;52:314-20.
63. Boyd RL. Mucogingival considerations and their relationship to orthodontics. *J Periodontol* 1978;49:67-76.
64. Sperry TP, Speidel TM, Isaacson RJ, Worms FW. The role of dental compensations in the orthodontic treatment of mandibular prognathism. *Angle Orthod* 1977;47:293-9.
65. Hall WB. The current status of mucogingival problems and their therapy. *J Periodontol* 1981;52:569-75.
66. Pearson LE. Gingival height of lower central incisors, orthodontically treated and untreated. *Angle Orthod* 1968;38:337-9.
67. Coatoam GW, Behrents RG, Bissada NF. The width of keratinized gingiva during orthodontic treatment: Its significance and impact on periodontal status. *J Periodontol* 1981;52:307-13.
68. Batenhorst KF, Bowers GM, Williams JE. Tissue changes resulting from facial tipping and extrusion of incisors in monkeys. *J Periodontol* 1974;45:660-8.
69. Allais D, Melsen B. Does labial movement of lower incisors influence the level of the gingival margin? A case-control study of adult orthodontic patients. *Eur J Orthod* 2003;25:343-52.
70. Djeu G, Hayes C, Zawaideh S. Correlation between mandibular central incisor proclination and gingival recession during fixed appliance therapy. *Angle Orthod* 2002;72:238-45.
71. Dorfman HS. Mucogingival changes resulting from mandibular incisor tooth movement. *Am J Orthod* 1978;74:286-97.
72. Pearson LE. Gingival height of lower central incisors, orthodontically treated and untreated. *Angle Orthod* 1968;38:337-9.
73. Ko-Kimura N, Kimura-Hayashi M, Yamaguchi M, Ikeda T, Meguro D, Kanekawa M, *et al.* Some factors associated with open gingival embrasures following orthodontic treatment. *Aust Orthod J* 2003;19:19-24.
74. Tarnow DP, Magner AW, Fletcher P. The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal dental papilla. *J Periodontol* 1992;63:995-6.
75. Kurth JR, Kokich VG. Open gingival embrasures after orthodontic treatment in adults: Prevalence and etiology. *Am J Orthod Dentofacial Orthop* 2001;120:116-23.
76. Kokich VO Jr, Kiyak HA, Shapiro PA. Comparing the perception of dentists and lay people to altered dental esthetics. *J Esthet Dent* 1999;11:311-24.
77. Wu YJ, Tu YK, Huang SM, Chan CP. The influence of the distance from the contact point to the crest of bone on the presence of the interproximal dental papilla. *Chang Gung Med J* 2003;26:822-8.
78. Burke S, Burch JG, Tetz JA. Incidence and size of pretreatment overlap and posttreatment gingival embrasure space between maxillary central incisors. *Am J Orthod Dentofacial Orthop* 1994;105:506-11.
79. McNab S, Battistutta D, Taverne A, Symons AL. External apical root resorption following orthodontic treatment. *Angle Orthod* 2000;70:227-32.
80. Motokawa M, Sasamoto T, Kaku M, Kawara T, Matsuda Y, Terao A, *et al.* Association between root resorption incident to orthodontic treatment and treatment factors. *Eur J Orthod* 2012;34:350-6.
81. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: Part 1. Literature review. *Am J Orthod* 1993;103:62-6.
82. Kaley J, Phillips C. Factors related to root resorption in edgewise practice. *Angle Orthod* 1991;61:125-32.
83. Hartsfield JK Jr, Everett ET, Al-Qawasmi RA. Genetic factors in EARR and orthodontic treatment. *Crit Rev Oral Biol Med* 2004;15:115-22.
84. Mirabella AD, Artun J. Risk factors for apical root resorption of maxillary anterior teeth in adult orthodontic patients. *Am J Orthod* 1995;108:48-55.
85. Levander E, Malmgren O. Evaluation of the risk of root resorption during orthodontic treatment: A study of upper incisors. *Eur J Orthod* 1988;10:30-8.
86. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. *J Clin Pediatr Dent* 1999;23:259-64.
87. Wickwire N, McNeil M, Norton L, Deull R. The effects of tooth movement upon endodontically treated teeth. *Angle orthod* 1974;44:235-42.
88. Rotstein I, Engel G. Conservative management of a combined endodontic-orthodontic lesion. *Endod Dent Traumatol* 1991;7:266-9.
89. Ponder SN, Benavides E, Kapila S, Hatch NE. Quantification of external root resorption by low- Vs high-resolution cone-beam computed tomography and periapical radiography: A volumetric and linear analysis. *Am J Orthod Dentofacial Orthop* 2013;143:77-91.
90. Linge BO, Linge L. Apical root resorption in upper anterior teeth. *Eur J Orthod* 1983;5:173-83.
91. Leonen V, Dermaut LR, Degreick J, De Pauw GA. Apical root resorption of upper incisors during the torquing stage of the tip-edge technique. *Eur J Orthod* 2007;29:583-8.
92. Remington DN, Joondeph DR, Artun J, Riedel RA, Chapko MK. Long-term evaluation of root resorption occurring during orthodontic treatment. *Am J Orthod Dentofacial Orthop* 1989;93:43-6.
93. Vandevska-Radunovic V, Kristiansen AB, Heyeraas KJ, Kvinnsland S. Changes in blood circulation in teeth and supporting tissues incident to experimental tooth movement. *Eur J Orthod* 1994;16:361-9.
94. Hamersky PA, Weimer AD, Taintor JF. The effect of orthodontic force application on the pulpal tissue respiration rate in human premolar. *Am J Orthod* 1980;77:368-78.
95. Nixon CE, Saviano JA, King GJ, Keeling SD. Histomorphometric study of dental pulp during orthodontic tooth movement. *J Endod* 1992;19:13-6.
96. Vandevska-Radunovic V, Kvinnsland S, Hals Kvinnsland I. Effect of experimental tooth movement on nerve fibers immunoreactive to calcitonin gene-related peptide, protein gene product 9.5, and blood vessel density and distribution in rats. *Eur J Orthod* 1997;19:517-29.
97. Stenvik A, Mjor IA. Pulp and dentine reactions to experimental tooth intrusion: A histological study of the initial changes. *Am J Orthod* 1970;57:370-85.
98. Guevara M, McClugage SG. Effects of intrusive forces upon the microvasculature of dental pulp. *Angle Orthod* 1980;50:129-34.
99. Hamilton RS, Gutmann JL. Endodontic-orthodontic relationships:

- A review of integrated treatment planning challenges. *Int Endod J* 1999;32:343-60.
100. Mostafa YA, Iskander KG, El-Mangoury NH. Iatrogenic pulpal reactions to orthodontic extrusion. *Am J Orthod Dentofacial Orthop* 1991;99:30-4.
 101. Yamaguchi M, Kasai K. The Effects of Orthodontic Mechanics on the Dental Pulp. *Seminars Orthod* 2007;13:272-80.
 102. Stenvik A, Mjor IA. Pulp and dentine reactions to experimental tooth intrusion: A histological study of the initial changes. *Am J Orthod* 1970;57:370-85.
 103. Cave SG, Freer TJ, Podlich HM. Pulp-test responses in orthodontic patients. *Aust Orthod J* 2002;18:27-34.
 104. Santamaria M Jr, Milagres D, Stuani AS, Ruellas AC. Initial changes in pulpal microvasculature during orthodontic tooth movement: A stereological study. *Eur J Orthod* 2006;28:217-20.
 105. Ramazanzadeh BA, Sahhafian AA, Mohtasham N, Hassanzadeh N, Jahanbin A, Shakeri MT. Histological changes in human dental pulp following application of intrusive and extrusive orthodontic forces. *J Oral Sci* 2009;51:109-15.
 106. Jacobs SG. The treatment of traumatized permanent anterior teeth: Case report and literature review. Part I-Management of intruded incisors. *Aust Orthod J* 1995;13:213-8.
 107. Unterscher RE, Nieberg LG, Weimer AD, Dyer JK. The response of human pulpal tissue after orthodontic force application. *Am J Orthod Dentofacial Orthop* 1987;92:220-4.
 108. Sheridan JJ. Air rotar stripping. *J Clin Orthod* 1985;19:43-59.
 109. Sheridan JJ. Airrotar stripping updates. *J Clin Orthod* 1987;21:81-8.
 110. Peck H, Peck S. Reproximation (enamel Stripping) as an essential orthodontic treatment ingredient. *St Louis CV Mosby*, 1975;513-27.
 111. El-Mangoury NH, Moussa MM, Mostafa YA. *In vivo* remineralization after Air-rotar stripping. *J Clin Orthod* 1991;25:75-8.
 112. Sicorsca-Bochinska J, Jamrozczek K, Lagocka R, Lipsky M, Nowicka A. Dental hypersensitivity after vertical stripping of enamel. *Ann Acad med Stetin* 2009;55:65-7.
 113. Arends J, Christoffersen J. The nature of early carious lesions in enamel. *J Dent Res* 1986;65:2-11.
 114. Radlanski RJ, Jager A, Schweska R, Bertzbach F. Plaque accumulations caused by interdental stripping. *Am J Orthod Dentofacial Orthop* 1988;94:416-20.
 115. Tuverson DL. Anterior interocclusion relations. Part I. *Am J Orthod* 1980;78:361-70.
 116. Danesh G, Hellak A, Lippold C, Ziebur T, Schafer E. Enamel surfaces following interproximal reduction with different methods. *Angle Orthod* 2007;77:1004-10.
 117. Sheridan JJ, Ledoux PM. Air-rotor stripping and proximal sealants. An SEM evaluation. *J Clin Orthod* 1989;23:790-4.
 118. Hansen EK. Dentin hypersensitivity treated with a fluoride-containing varnish or a light-cured glass-ionomer liner. *Scand J Dent Res* 1992;100:305-9.
 119. Tilliss TS, Keating JG. Understanding and managing dentin hypersensitivity. *J Dent Hyg* 2002;76:296-313.
 120. Zachrisson BU. Causes and prevention of injuries to teeth and supporting structures during orthodontic treatment. *Am J Orthod* 1976;69:285-300.
 121. Kerosuo HM, Dahl JE. Adverse patient reactions during orthodontic treatment with fixed appliances. *Am J Orthod* 2007;132:789-95.
 122. Kyung HM, Park HS, Bae SM, Sung JH, Kim IB. Development of orthodontic micro-implants for intraoral anchorage. *J Clin Orthod* 2003;37:321-8.
 123. Ellis PE, Benson PE. Potential hazards of orthodontic treatment – what your patient should know. *Dent Update* 2002;29:493-97.
 124. Casaglia A, Dominici F, Pachi F, Turlà R, Cerroni L. Morphological observations and fractological considerations on orthodontic miniscrews. *Minerva Stomatol* 2010;59:465-76.
 125. Nienkemper M, Wilmes B, Renger S, Mazaud-Schmelter M, Drescher D. Improvement of mini-implant stability in orthodontics. *Orthod Fr* 2012;83:201-7.
 126. Viazis AD, DeLong R, Bevis RR, Rudney JD, Pintado MR. Enamel abrasion from ceramim orthodontic brackets under an artificial oral environment. *Am J Orthod* 1990;98:103-9.
 127. Chen YJ, Yao CC, Chang HF. Nonsurgical correction of skeletal deep overbite and class II division 2 malocclusion in an adult patient. *Am J Orthod Dentofacial Orthop* 2004;126:371-8.
 128. Booth-Mason S, Birnie D. Penetrating eye injury from orthodontic headgear: A case report. *Eur J Orthod* 1988;10:111-4.
 129. Samuels RH, Jones ML. Orthodontic facebow injuries and safety equipment. *Eur J Orthod* 1994;16:385-94.
 130. Hinkle FG. Ingested retainer: A case report. *Am J Orthod Dentofacial Orthop* 1987;92:46-9.
 131. Dibiasi AT, Samuels RH, Ozdiler E, Akcam MO, Turkkahraman H. Hazards of orthodontics appliances and the oropharynx. *J Orthod* 2000;27:295-302.
 132. Allwork JJ, Edwards IR, Welch IM. Ingestion of a quadhelix appliance requiring surgical removal: A case report. *J Orthod* 2007;34:154-7.
 133. Sfondrini MF, Cacciafesta V, Lena A. Accidental ingestion of a rapid palatal expander. *J Clin Orthod* 2003;37:201-2.
 134. Keles A. Keles keyless expander: A new approach for rapid palatal expansion. *World J Orthod* 2008;9:407-11.
 135. Chate RA. Safer orthodontic debonding with rubber dam. *Am J Orthod Dentofacial Orthop* 1993;103:171-4.
 136. Brady PG. Esophageal foreign bodies. *Gastroenterol Clin North Am* 1991;20:691-701.
 137. Abdel-Kader HM. Broken orthodontic trans-palatal archwire stuck to the throat of orthodontic patient: Is it strange? *J Orthod* 2003;30:11.
 138. Sittitriai P, Pattarasakulchai T, Patatiwong H. Esophageal foreign bodies. *J Med Assoc Thai* 2000;83:1514-8.
 139. Milton M, Hearing SD, Ireland AJ. Ingested foreign bodies associated with orthodontic treatment: Report of three cases and review of ingestion/aspiration incident management. *Br Dent J* 2001;190:592-6.
 140. Lee BW. Case report – swallowed piece of archwire. *Aust Orthod J* 1992;12:169-70.
 141. Kuo SC, Chen YL. Accidental swallowing of an endodontic file. *Int Endo J* 2008; 41:617-2.
 142. Absi EG, Buckley JG. The location and tracking of swallowed dental appliances: The role of radiology. *Dentomaxillofac Radiol* 1995;24:139-42.
 143. Chua KL, Taylor GS, Bagg J. A clinical and laboratory evaluation of three types of operating gloves for use in orthodontic practice. *Br J Orthod* 1996;23:115-20.
 144. VanMeter BH, Aggarwal M, Thacker JG, Edlich RF. A new powder-free glove with a textured surface to improve handling of surgical instruments. *J Emerg Med* 1995;13:365-8.
 145. Noble J, Ahing SI, Karaiskos NE, Wiltshire WA. Nickel allergy and orthodontics. A review and report of 2 cases. *Br Dent J* 2008;204:297-300.
 146. Amini F, Borzabadi Farahani A, Jafari A, Rabbani M. *In vivo* study of metal content of oral mucosa cells in patients with and without fixed orthodontic appliances. *Orthod Craniofac Res* 2008;11:51-6.
 147. Kolokitha OE, Chatzistavrou E. A severe reaction to Ni – containing orthodontic appliances. *Angle Orthod* 2009;79:186-92.
 148. Al-Tawil NG, Marcusson JA, Möller E. Lymphocyte transformation test in patients with nickel sensitivity: An aid to diagnosis. *Acta Derm Venereol* 1981;61:511-5.
 149. Bass JK, Fine H, Cisneros GJ. Nickel hypersensitivity in the orthodontic patient. *Am J Orthod* 1993;103:280-5.
 150. Magnusson B, Bergman M, Bergman B, Soremark R. Nickel allergy and nickel-containing dental alloys. *Scand J Dent Res* 1982;90:163-7.
 151. Kim H. Corrosion of Stainless Steel, NiTi, coated nickel titanium and titanium orthodontic wires. *J Dent Res* 1999;69:39-44.
 152. Kusy RP, Whitley JQ, Ambrose WW, Newman JG. Evaluation of titanium brackets for orthodontic treatment: Part I. The passive configuration. *Am J Orthod* 1998;114:558-72.
 153. Hain LA, Longman LP, Field EA, Harrison JE. Natural rubber latex allergy: Implications for the orthodontist. *J Orthod* 2007;34:6-11.
 154. Tang AT, Li J, Ekstrand J, Liu Y. Cytotoxicity tests of *in situ* polymerized resins: Methodological comparisons and introduction of a tissue culture insert as a testing device. *J Biomed Mater Res* 1999;45:214-22.
 155. Gioka C, Bourauel C, Hiskia A, Kletsas D, Eliades T, Eliades G. Light-cured or chemically cured orthodontic adhesive resins? A selection based on the degree of cure, monomer leaching, and cytotoxicity. *Am J Orthod* 1999;127:413-9.

156. Terhune WF, Sydiskis RJ, Davidson WM. In-vitro cytotoxicity of orthodontic bonding materials. *Am J Orthod* 1983;83:501-6.
157. Tell RT, Sydiskis RJ, Isaacs RD, Davidson WM. Long-term cytotoxicity of direct-bonding adhesives. *Am J Orthod Dentofacial Orthop* 1983;84:344-50.
158. Brattstrom V, Ingelsson M, Aberg E. Treatment co-operation in orthodontic patients. *Br J Orthod* 1991;18:37-42.
159. Daniels AS, Seacat JD, Inglehart MR. Orthodontic treatment motivation and cooperation: A cross-sectional analysis of adolescent patients' and parents' responses. *Am J Orthod* 2009;136:780-7.
160. Nanda RS, Kearn MJ. Prediction of cooperation in orthodontic treatment. *Am J Orthod* 1992;102:15-21.
161. Albino JE, Lawrence SD, Lopes CE, Nash LB, Tedesco LA. Cooperation of adolescents in orthodontic treatment. *J Behav Med* 1991;14:53-70.
162. Jacobson A, Sadowsky LP. A visualized treatment objective. *J Clin Orthod* 1980;14:554-71.
163. Roth RH. Functional occlusion for the orthodontist. Part III. *J Clin Orthod* 1981;1:32-50.
164. Andrews LF. The six keys to normal occlusion. *Am J Orthod Dentofacial Orthop* 1972;63:296-309.
165. Casco J, Vaden J, Kokich V. American Board of orthodontics objective grading system for dental casts and panoramic radiographs. *Am J Orthod Dentofacial Orthop* 2000;114:530-2.
166. Little RM. Stability and relapse of dental arch alignment. *Br J Orthod* 1990;17:235-41.
167. Zachrisson BU. Important aspects of long-term stability. *J Clin Orthod* 1997;31:562-83.
168. Taner TU, Haydar B, Kavuklu I, Korkmaz A. Short-term effects of fiberotomy on relapse of anterior crowding. *Am J Orthod Dentofac Orthop* 2000;118:617-23.
169. Sadowsky C, Schneider BJ, BeGole EA, Tahir E. Long-term stability after orthodontic treatment: Nonextraction with prolonged retention. *Am J Orthod Dentofac Orthop*. 1994;116:243-9.
170. Bondemark L, Holm AK, Hansen K, Axelsson S, Mohlin B, Brattstrom V, *et al.* Long-term stability of orthodontic treatment and patient satisfaction. A systematic review. *Angle Orthod* 2007;77:181-91.
171. Michelotti A, Iodice G. The role of orthodontics in temporomandibular disorders. *J Oral Rehabil* 2010;37:411-29.
172. McNamara JA Jr, Seligman DA, Okesan JP. Occlusion, Orthodontic treatment, and temporomandibular disorders: A review. *J Orfac Pain* 1995;9:73-90.
173. Mohlin B, Axelsson S, Paulin G, Pietilä T, Bondemark L, Brattström V, *et al.* TMD in relation to malocclusion and orthodontic treatment. *Angle Orthod* 2007;77:542-8.
174. Kremenak CR, Kinser DD, Melcher TJ, Wright GR, Harrison SD, Ziaya RR. Orthodontics as a risk factor for temporomandibular disorders (TMD) II. *Am J Orthod Dentofacial Orthop* 1992;101:21-7.
175. Egermark I, Thilander B. Craniomandibular disorders with special reference to orthodontic treatment: An evaluation from childhood to adulthood. *Am J Orthod Dentofacial Orthop* 1992;101:28-34.
176. Sadowsky C. The risk of orthodontic treatment for producing temporomandibular disorders: A literature review. *Am J Orthod Dentofacial Orthop* 1992;101:79-83.

How to cite this article: Meeran NA. Iatrogenic possibilities of orthodontic treatment and modalities of prevention. *J Orthodont Sci* 2013;2:73-86.

Source of Support: Nil, **Conflict of Interest:** None declared.

Announcement

Android App



Download
**Android
application**

FREE

A free application to browse and search the journal's content is now available for Android based mobiles and devices. The application provides "Table of Contents" of the latest issues, which are stored on the device for future offline browsing. Internet connection is required to access the back issues and search facility. The application is compatible with all the versions of Android. The application can be downloaded from <https://market.android.com/details?id=comm.app.medknow>. For suggestions and comments do write back to us.