Restorative complications of orthodontic treatment

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In brief

Patients need to be made aware,in advance of having treatment , of the possibilities of them having having some complication(s) with elective orthodontics.

Orthodontics is not a risk free option and adverse outcomes include failure to achieve their "perfect smile", relapse, resorption, recession or caries.

Issues about patient understanding of the information given to them for their valid consent have changed following the 2015 ruling of Montgomery *versus* Lancashire.

The complications of elective orthodontic treatment are numerous. Patients need to be aware, in advance, of possible problems including resorption, instability, caries, recession and failure to deliver optimal tooth position. The investment of time and resources by all concerned is considerable and if there are adverse outcomes these can be biologically costly in the longer term. A frank and full discussion of the possible problems is necessary following the findings of Montgomery *vs*. Lanarkshire in 2015.

Introduction

Orthodontic movement of teeth to achieve improved dental and facial appearance is a well-established treatment modality. Where crowding or moderately severe skeletal discrepancies are present, most patients are likely to derive at least some benefit.[1]

During orthodontic treatment teeth usually tolerate light forces transmitted to the surrounding bone by way of tension and compression in the periodontal ligaments, thereby producing the desired tooth positional changes.

However, as in other areas of elective dentistry, the various risks of any orthodontic treatment plan need to be balanced carefully against the anticipated benefits and the longevity of any treatment result. When adolescents, (or increasingly, adults) undergo orthodontic treatment the repercussions, or the development of various complications, have the potential to affect them for the remainder of their lives (Fig. 1).[2–4]

These complications may be due to biofilm-related (plaque-induced diseases) or by the exacerbation of various physiologic phenomena and/or issues related to attempting long-term retention of the teeth in their new more desirable, but not necessarily stable, positions.

In some cases, where the predicted risks of some complications are high, a pragmatic approach might well be to reconsider the perceived need for orthodontics and to do no treatment, or to do something different, thereby avoiding predictable, or unintended, harm to the patient and frustration for the well-intentioned clinician, the patient and/or their family, or significant other person.

The need for communication of any such pertinent risks to the patient or their guardian in the early discussions has been emphasised under 'duty of candour' and by a recent Supreme Court judgement in the Montgomery *versus* Lanarkshire case.[5,6]

Potential problems

Enamel demineralisation

Orthodontic brackets and associated attachments increase plaque retention due to their irregular shapes and the inherent high free surface energy materials involved in cementing them into position.[7] This is not at all surprising as brackets are likely to create physically difficult-to-clean ledges or overhangs. Orthodontic bands placed close to gingival margins due to immature gingiva, or which subsequently become sub-gingival as a result of gingival hyperplasia, make effective plaque removal more difficult to achieve. Plaque accumulation has been reported as being up to three times higher with fixed orthodontic appliances than without them.[8]

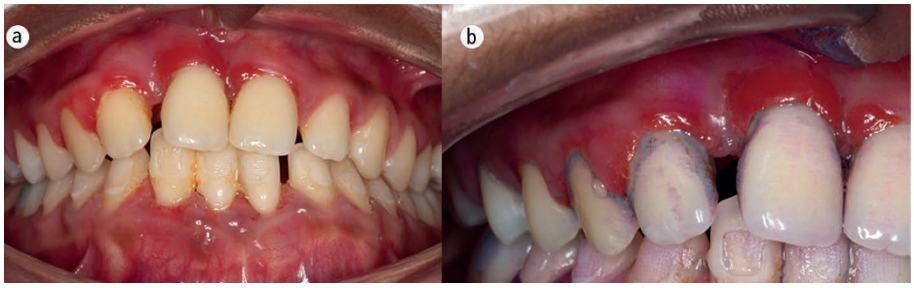

Fig. 1 (a) This patient attended secondary care complaining that her teeth had moved and were loose on completion of orthodontics one year previously; (b) The patient was disclosed to reveal masses of plaque accumulation and residual cement from previous brackets. The patient was not made aware that orthodontics could increase plaque accumulation, exacerbate periodontal disease and the final position of her teeth may not be retained 'forever'. A number of teeth had poor to hopeless prognosis

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There has been a documented decrease in salivary pH and an increased level of *Streptococcus mutans* and *Lactobacillus acidophilus* noted in orthodontic patients.[9] Despite these well recognised physical and chemical risks affecting the ecology of the plaque local to the electively placed orthodontic brackets and bands, the incidence of frank caries during orthodontic treatment has not been extensively investigated. In contrast, the enamel demineralisation in early caries – often given the more euphemistically named 'white spot lesions' – seems to have been better researched.

The incidence of 'white spot lesions', which actually involves significant demineralisation and is often a precursor to frank cavitation if the risk factors continue, has been examined by a Tufekci and colleagues.[10] In their cohort of patients the incidence was shown to be as high as 38% of the individuals at 6 months with a rise to 46% at 12 months. The control group in the study had an incidence of 11%. In other words, there was roughly a three-fold increase in risk relative to the controls. These findings confirmed earlier cross sectional study findings that showed that 50% of patients had demineralisation after orthodontic debond which was twice as great as the control group.[11] Al Maaitah and colleagues reported a 70% prevalence of white spot lesions (WSL) and 5% prevalence of caries in their cohort of 230 patients (Fig. 2).[12]

In a more recent study by Akin and colleagues, the prevalence of demineralisation was 20% before orthodontic treatment, but, rather worryingly, this increased to over 50% on completion of orthodontic treatment. Bracket type, age and dental hygiene care were significantly associated with demineralisation during orthodontic treatment.[13] The highest risk of developing demineralisation has been shown to be associated with pre-existing demineralisation. That is not particularly surprising, given the physical difficulties producing predictable declines in effectiveness of oral hygiene during treatment.[14]

Boersma and colleagues showed that demineralisation had a positive correlation with caries prevalence and with bleeding on probing, whereas *Streptococcus mutans* counts, age, treatment duration, socioeconomic status and dietary habits did not show any obvious correlation.[15]

Chapman and colleagues examined the risk factors associated with the development of white spot lesions.[16] Predictably, they identified that patients who were pre-adolescent at the start of treatment and who presented with poor oral hygiene before, or subsequently showed this during treatment, had an increased chance of developing white spot lesions. Unsurprisingly, those patients who ended up with an unfavourable clinical outcome score were also more likely to have white spot lesions. Lack of patient compliance with advice about their cleaning and diet would be the plausible, if not scientifically provable, link.[2,17]

Fig. 2 This patient presented to a general dental practitioner after debond. Due to the plaque retention associated with the orthodontic brackets and poor oral hygiene caries was diagnosed on the majority of upper anterior teeth. Unfortunately this was so severe that the upper left lateral incisor spontaneously decoronated and the upper right lateral incisor developed pulpal necrosis

In the same study, the incidence of white spot lesions relating to individual tooth positions was investigated. The most common maxillary tooth to be affected by a white spot lesion was the lateral incisor (34%), followed by the canine (31%), premolar (28%), and central incisor (17%).[16] These figures should be viewed with caution due to the lack of appropriately powered evidence. This could well be an underestimation (or less likely an over-estimation) of the true scale of the problem particularly when orthodontic treatment is undertaken in situations where reporting of adverse outcomes is not particularly common, for understandable reasons, such as in some general or in some specialist practices.

Orthodontic brackets and banding are plaque retentive in the same manner as difficult-to-clean, over-contoured restorations. Tooth decay occurring in a patient who has already had teeth electively removed for the purposes of space creation could therefore result in a significant further reduction of functional teeth. Premolar units are often removed for the purposes of space creation, but during what is often a long orthodontic plan in poorly compliant patients, decay may result in further sound tooth tissue loss.

Because of these very real risks, it is imperative to ensure that sugar intake frequency is drastically curtailed before the start of treatment and that good plaque control can be demonstrated consistently over a decent period of time before orthodontic treatment starts. Patients with active caries or poor oral hygiene have been identified as being inappropriate for orthodontics by the British Orthodontic Society.[18] Information transfer to patients is the easy bit. Patient compliance with the preventive advice is the real problem, as most experienced specialists and dentists will readily attest.

Caries prevention and white spot lesions (WSL)

Simple methods such as the topical application of fluoride (mouthwash) have shown some success in reducing the incidence of white spot lesions, although lack of compliance with such a regimen has been shown to reduce the potential benefit especially for those in the higher risk groups.[19]

The use of sodium fluoride mouthwash has been examined in systematic reviews. There was some evidence showing that mouthwash reduces the severity of enamel decay around orthodontic brackets and that glass-ionomer cement utilised to bond the bracket reduced the prevalence of carious lesions and the severity of these lesions when compared to cementation with composite resin.[20–23] This is likely to be a function of differences in free surface energy, with the higher free surface energy of composite attracting more plaque. The obvious other factor is fluoride release because both composite and resin-modified glass-ionomer cement release minimal amounts of theoretically available fluoride. This is mainly because, in spite of some manufacturers' claims, any fluoride incorporated in the adhesive material is firmly bound in by the resin, which is

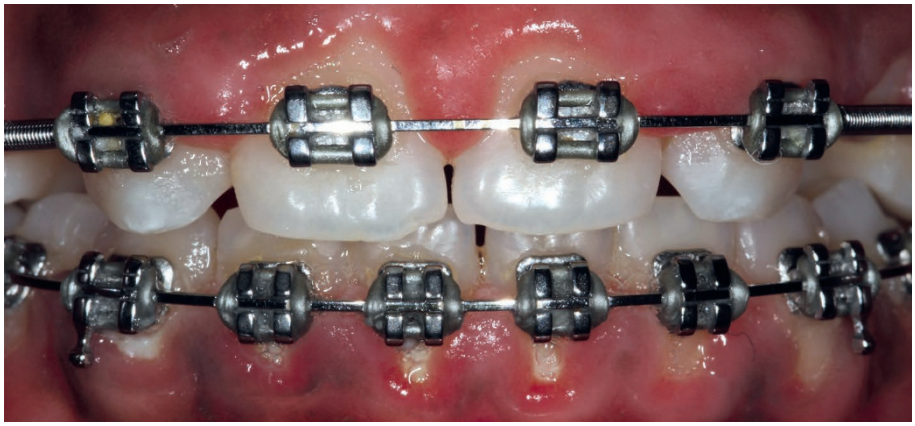

Fig. 3 This patient underwent a comprehensive course of orthodontics. Despite her attempts at maintaining a low level of plaque she developed gingival inflammation resulting in overgrowth in the vicinity of the brackets

not the case with conventional acid-base setting glass-ionomer cements.

In the follow up review, Benson *et al.* found that the patients who had fluoride varnish applications at 6 weekly intervals showed a 70% reduction in WSL although the quality of evidence is moderate due to these findings being based on a lone study.[24]

A randomised controlled trial by van der Kaaij and colleagues showed the development of WSL in 31% of patients using a fluoride mouthwash and in 47% of those using the placebo.[25] Although fluoride mouthwash reduced the development of WSL, this was still a significant problem in nearly one third of patients.

Despite the concerted efforts of a number of studies, a recent Cochrane systematic review found limited evidence, based on just three randomised controlled trials with questionable risks of bias, for fluoride application, in a variety of modes, to be beneficial in reducing the incidence of white spot lesions.[4]

It may well be that in real life clinical practice demineralisation around orthodontic brackets is an unfortunate and unpredictable complication of fixed orthodontic provision. Patients and the treating clinicians need to be more realistic about this. Awareness of these real risk factors and ensuring consistent compliance with preventive advice may be more the issue than altruistic patient education. 'Tough love' in case selection may well be the only way to reduce the incidence of these lesions, which are sometimes white and sometimes not. At the moderate level these can be unsightly white or brown or yellow, but when more severe the problems can result in the need for invasive procedures such as a restoration. In the presence of ongoing plaque accumulation that often means a continuing downward reparative spiral for the patient's lifetime.

Other sources of colour changes

The high free surface energy of bonding materials, food pigments, dyes, and corrosion products from the brackets can result in yellow colour changes to the teeth.[26] Furthermore, some intrinsic sources of discolouration which may include changes in pulp vascularisation, can result in significantly more yellow phosphoprotein being present in the reparative dentine and thereby produce an appearance of discoloured ageing of any affected teeth.[27–36]

Periodontal implications

The provision of fixed appliance therapy in healthy and well cared for mouths can result in gingival inflammation and some inflammation probably occurs in the majority of patients.[37] This inflammation does not necessarily lead to frank periodontal attachment loss, but it can give the impression of false pocketing especially in the vicinity of orthodontics bands and brackets (Fig. 3). This could be attributed to increased plaque retentive factors around an orthodontic band, in a similar way to an overhanging margin on a crown. One important periodontal consideration is in the changes induced in the periodontal flora, which are found on application of fixed appliances.[38] Mere placement of fixed braces can cause the subgingival biofilm to develop into a more perio-pathogenic flora that makes the progression from gingivitis to periodontitis more likely.[39–42]

However, when optimal hygiene levels are maintained in non-susceptible patients, gingival inflammation or frank attachment loss can be prevented.[39–42] Despite these observations other research has shown that when inflammation is ongoing with modification of the associated risk factors, periodontal tissue loss is still likely to occur, thereby leading to periodontal pocketing and possible attachment loss.[43,44] These observations provide significant reason to ensure a thorough periodontal examination is undertaken early on, especially to identify a thin biotype in both adolescents and adults before the start of any elective orthodontic treatment due to the inherent higher risk for recession in this patient group (Fig. 4). Those adolescents presenting with aggressive periodontitis may be especially at risk

Fig. 4 (a) This patient presented complaining of spaces between their teeth and progressive movement of his upper anteriors. A diagnosis of generalised moderate to severe periodontitis was made and the patient was encouraged to have periodontal treatment as opposed to orthodontic treatment, which they had requested; (b) Six years later the patient was re-referred for chronic mobility of teeth and periodontal abscesses. Despite attempts by the referring practitioner, closure of the space between the 13 and 12 had not been achieved. There was a marked increase in probing depths and delayed bleeding; (c)The upper first molar had developed a severe periodontal-endodontic lesion resulting in a hopeless prognosis

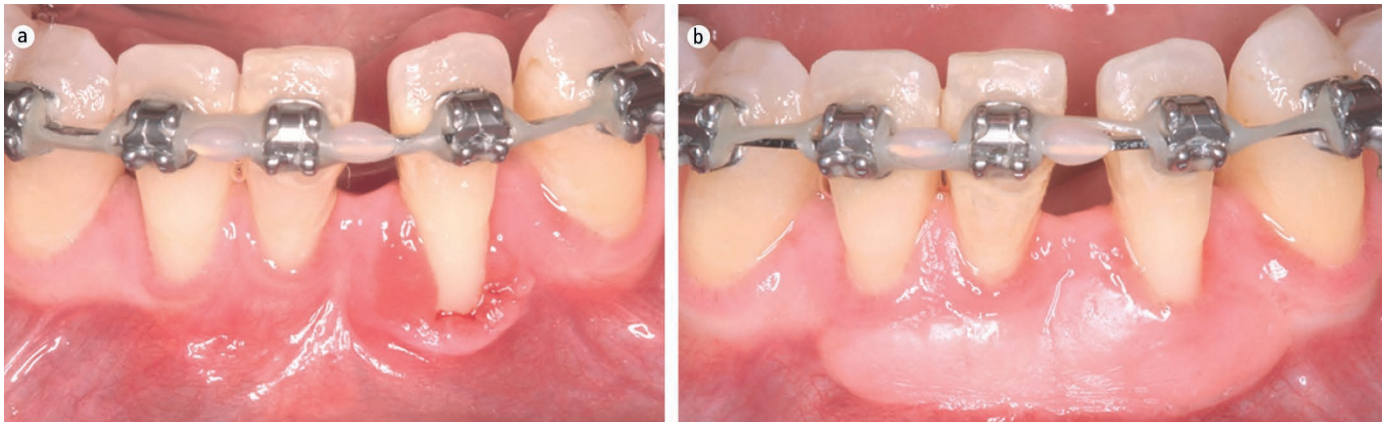

Fig. 5 (a) This patient developed labial recession on the lower left lateral incisor during orthodontic treatment; (b) This was treated with a connective tissue graft which was maintained during the completion of orthodontics. Treatment carried out by Jamie Amir, Specialist Periodontist, Ocala, Florida

if the underlying condition is not diagnosed early and treated effectively. These patients may present to an orthodontist complaining of rapid tooth movement localised to the incisors and be largely unaware of their underlying periodontal disease, or the severity of their bone or attachment loss.[45] In patients with presenting features of aggressive periodontitis the dental panoramic tomograph is unlikely to give accurate and clear information of localised bone loss around the incisors. Long cone periapical examinations are better views for periodontal tissue assessment in these situations. Conventional or vertical bitewings are likely to be more appropriate for assessing bone levels around posterior teeth.

Teeth with orthodontic bands seem to be at a greater risk of periodontal problems than those with bonded brackets, although there are conflicting reports on the reasons for this and whether this always results in irreversible tissue damage.[46] After placing a band, pockets depths can increase by approximately 0.5 mm and this may be attributed to either frank attachment loss, or false pocketing due to gingival inflammation. This is far too small not be accounted for by the error of the probing methods involved.[47]

Increased plaque retention, hormonal changes, and/or cement excess help to induce inflammation and are difficult to quantify reproducibly. Changes in the periodontal flora itself could be the possible cause of these observations. In these situations meticulous removal of excess adhesive materials, targeted oral hygiene instruction and ultrasonic instrumentation, if deemed necessary, may reduce the presence of localised inflammation.

Due to the issues listed above there is a real need to diagnose periodontal disease or identify susceptible patients early on and instigate treatment in good time before consideration for elective orthodontic treatment. Subsequently the periodontal tissues should be monitored throughout treatment, reinforcing oral hygiene when necessary, with a plan for ongoing monitoring once treatment is completed and continued into the retention phase.

Complications associated with gingival changes

Localised gingival recession is a recognised complication of orthodontic treatment.[48–50] Risk factors associated with increased chance of recession during orthodontics include the presence of a thin biotype, previous recession and proclination of teeth when associated with plaque induced gingival inflammation (Fig. 5).[51] In a review by Wennstrom, the presence of a thin biotype in conjunction with poor oral hygiene and movement outside of the alveolus results in a high risk of the development of recession.[52]

Adolescent patients undergoing orthodontic treatment are over four-times more likely to develop labial recession than their non-orthodontic counterparts.[53] The same group found that the prevalence of recession in a cohort of 300 patients rose from 7% to 20% at 2 years after orthodontic treatment was completed. More interestingly, these patients were followed up for 5 years and showed a gradual increase in recession to 38%.[53]

Risk factors such as the presence of a thin biotype around different teeth are relatively easy to record. This is in contrast to the ability to record the 'alveolar envelope' identified by an orthodontic specialist working group.[51] Movement of teeth outwith the 'alveolar envelope' may result in increased incidence of recession.[54] This may be difficult to assess clinically and advanced radiological imaging, if practical and reproducible, might be the only means of appreciating the true dimension of this 'alveolar envelope', preferably well before the start of treatment. Other factors associated with orthodontically induced recession are presented in Table 1.

It seems as though orthodontically-induced labial gingival recession is common. This aesthetic problem may result in patient dissatisfaction as a result of gingival margin asymmetry particularly in those patients with a high lip line who show their exposed gingival margins during normal facial movements.

Depending on the severity of the recession the patient may present with a variety of different symptoms. Common complaints include hypersensitivity, aesthetic concerns, or bleeding from the area as a result of difficulty in cleaning effectively, particularly where the recession defect is close to the a high frenal attachment or affecting the mucogingival junction.

Treatment of recession defects is controversial and varies among clinicians in different countries with different dental cultures and remuneration systems. In the majority of cases customising hygiene measures to accommodate for the recession defect may suffice. Hypersensitivity can usually be managed with desensitising toothpaste containing 5% potassium nitrate and no n-lauryl sulphate held for a number of weeks in a clear, carefully contoured, thermoplastic retainer which does not damage the vulnerable thin periodontal tissues.

It is only when patients are scrupulous with their hygiene, compliant with instructions and still really keen on correcting the gingival margin discrepancy that treatment with periodontal

plastic surgery procedures should be considered. This is likely to require a donor site from their palate depending on the method of root coverage intended. This modality is likely to result in two areas of pain and has a number of co-morbidities associated with it. In a systematic review by Chambrone root coverage procedures resulted in significant reduction in recession depth and clinical attachment gain with greater success in those with patients with the more minor defects of Miller Class I and II. Although successful in these minor defects this did not mean that the exposed root surface was covered in its entirety.[55] Furthermore, sub-epithelial connective tissue graft-based procedures seemed to provide the best outcomes due to superior percentages of mean root coverage, as well as significant increase in keratinised tissue.[55]

Table 1 Other factors associated with orthodontically induced recession. Adapted from Johal *et al*,[51] under https://creativecommons.org/licenses/by/3.0/

Risk factors	
Anatomical risk factors	Alveolar bone dehiscence
	Thin gingival biotype
	Previous recession
	Alveolar envelope in relation to the tooth
	Ectopic tooth eruption
Patient based risk factors	Poor oral hygiene
	Smoking
	Traumatic tooth brushing
	Age
	Parafunction
	Intra-oral piercing
Orthodontic advice is to:	Reinforce oral hygiene during treatment
	Avoid uncontrolled dento-alveolar expansion and maintain arch form
	Customise bonding and mechanics
	Modify tooth anatomy whenever indicated
	Consider segment arch mechanics
	Create space before using it and use it wisely
	Consider atypical extractions, for example, compromised teeth
	Avoid jiggling because it may cause periodontal and resorption problems
	Treat early (interceptive procedures and treatment in mixed dentition)
Root morphology risk factors	Long narrow roots
	Naturally pipette shaped
	Deviation in the root apex
Risks of habits	Nail biting
	Digit Sucking
Treatment risk factors	Long periods of treatment
	Application of high forces
	Intrusion of teeth
	Long distances of tooth movement
	Torqueing movements especially in the region of the palatal cortical wall
	Intermaxillary traction.
	2-3 Month pause in treatment with passive wires may decrease amount of root resorption.110

Pulp health, endodontics and orthodontic movement

The pulp is a complex neurovascular system that has abilities to react to the various stimuli applied to it. These include physiological and pathological challenges such as caries threatening a vital pulp thereby resulting in reparative dentine being laid down to protect it. Orthodontic tooth movement can cause some degenerative inflammatory reaction of the dental pulp of teeth with mature root apices and the consequences are related to the magnitude, direction and duration of these forces (Fig. 6).[56–59]

In contrast, teeth with immature apices, due to their richer innervation and blood supply, are less likely to undergo these changes.[27] Inappropriate or uncontrolled forces involved in orthodontic movement can result in increased dentine being laid down resulting in 'artificially' ageing the pulp system, sometimes resulting in canal obliteration.[27]

Orthodontic movement results in the release of specific neurotransmitters (neuropeptides), which regulate pulpal blood flow and cellular activity.[60] This self-regulatory activity of the

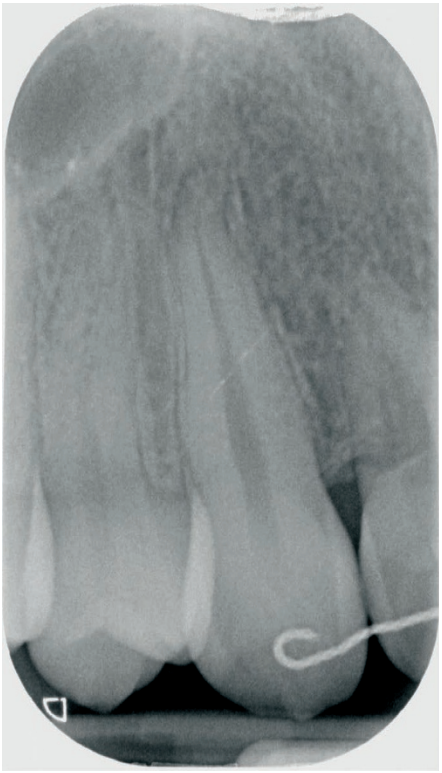

Fig. 6 This canine presented with tenderness to percussion and progressive discolouration towards the end of orthodontic treatment. Root canal treatment required removal of the direct retainer and a temporary retainer

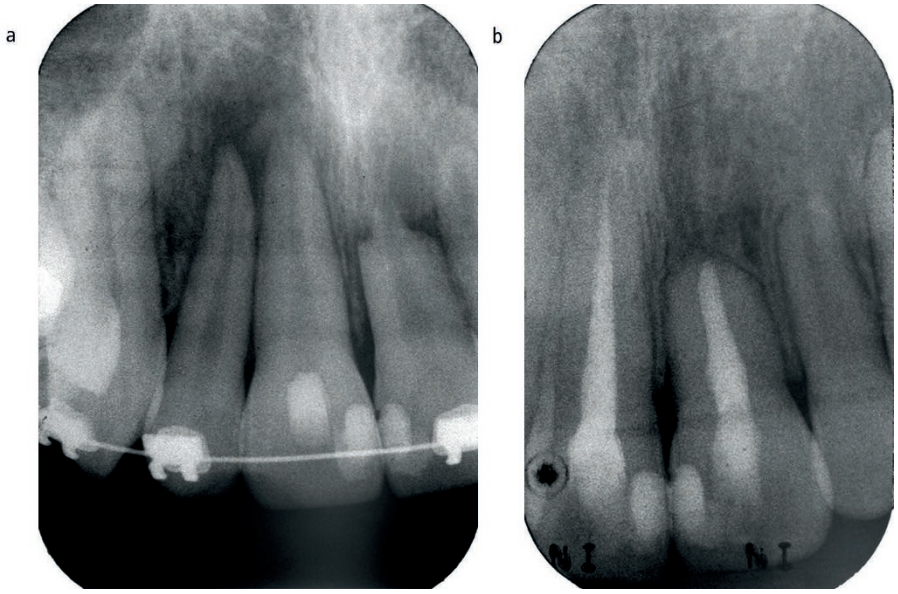

Fig. 7 (a) This patient who had no history of trauma presented with pain and swelling associated with the upper anteriors. He had teeth extirpated by the practitioner who had provided the orthodontics and was then referred to secondary care; (b) Root canal treatment was completed. Due to the extent of apical resorption and the wide-open apex the 21 required MTA apexification

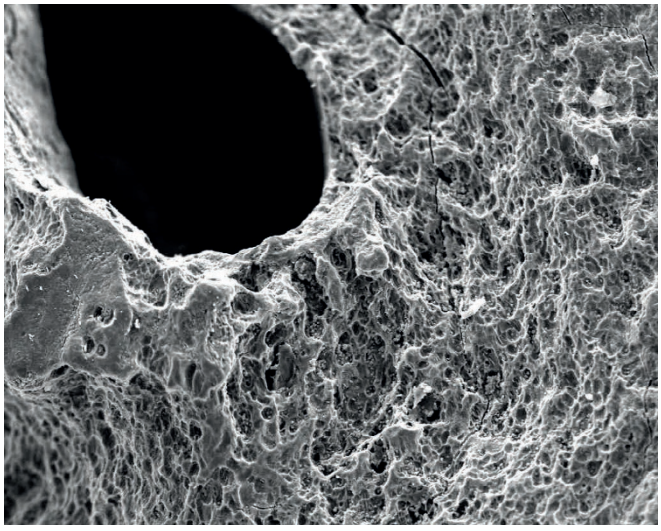

Fig. 8 A SEM view of the apex of an extracted upper lateral incisor. This was extracted one month after orthodontic treatment had been completed. Attempts were made to save the tooth but unfortunately these were unsuccessful and the tooth required extraction and prosthetic replacement with a resin-bonded bridge. Note the numerous resorptive lacunae present providing an excellent niche for bacteria

pulp modifies the ability of the root apices to resorb or remodel when an orthodontic force is applied to the tooth.[61,62] As might be expected, those teeth with healthy pulps react more favourably to orthodontic movement than those with periapical pathology.[27,63] On the other hand, there is some conflicting evidence that teeth with intact pulps are actually more susceptible to resorption than necrotic ones.[64–68]

Necrotic teeth with concurrent periapical pathology usually present with root resorption due to inflammatory mediator release triggered by the presence of a biofilm. This resorption is exacerbated by orthodontic movement resulting in root shortening and resorption.[27,69] Although it may seem trivial, the need to provide root canal treatment before orthodontic treatment as opposed to after orthodontics is important to ensure long-term retention of the tooth (Fig. 7). Some foresight is required to identify heavily restored or traumatised teeth, to conduct appropriate radiographic and other appropriate examination of them and potentially seek advice on the need for further endodontic or restorative treatment before starting elective orthodontics.

Continued resorption over a prolonged orthodontic treatment plan is likely to result in a more difficult endodontic procedure due to a wider apical foramen, thinner dentine walls, a larger periapical lesion and a more mature biofilm making the tooth more difficult to treat successfully (Fig. 8).[27] In cases where teeth have been optimally chemo-mechanically debrided and obturated, the likelihood of resorption or significant remodeling is reduced.[27] Where teeth have been badly traumatised, which is more likely to occur in younger patients, especially those with Class II malocclusions, the need for root canal treatment is usually more acute. The prognosis depends a lot on the severity of the trauma, its direction, how and when it was managed as well as the proposed tooth movements. In mild traumatic injuries such as concussion where vitality has not been compromised, or where bacterial invasion of the root canal system has not developed, there is a decreased risk of resorption. In those cases where more severe injuries have occurred, or when the treatment was suboptimal at the time, the risk of complications are higher with increased chances of endodontic problems, ankylosis or root resorption occurring.

These risk assessment issues highlight the need to assess appropriately the peri-apical health of teeth before the provision of orthodontic treatment. A dental panoramic view may well be adequate when assessing the development and eruption of teeth and their relative root morphology but are not particularly helpful when assessing the presence of periapical pathology, especially in the anterior regions, before or during treatment. A long cone periapical radiograph is to be preferred in these circumstances. Periapical health of those teeth already known to be at risk or indeed those that reveal themselves to be dead during treatment need ongoing monitoring while treatment is being provided. If pulpal necrosis becomes apparent during orthodontics, treatment should be suspended and endodontics instigated before re-starting tooth movement.

Fig. 9 This patient underwent a course of orthodontics that resulted in chronic mobility of her anterior teeth

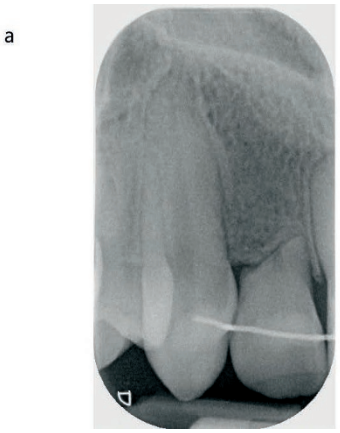

Fig. 10 This patient presented with chronic mobility of the upper right lateral incisor after prolonged orthodontic treatment. Somewhat ironically the treatment was promoted as being 'rapid' and 'easy'. On detachment of the retainer this increased markedly. The patient and her mother stated they had been unaware of any serious risks being associated with orthodontic treatment

Root resorption

Some apical root resorption is almost unavoidable in the provision of orthodontics (Figs 8 and 9).[2] This might seem to be insignificant due to the benefits to the patient of improvements in the positions of the crowns of the teeth. Nonetheless, some caution is advised, particularly when considering the possible repercussions of treatment with pre-existing short teeth or where orthodontics is being done again. In a study by Linge & Linge examining apical root resorption of maxillary anterior teeth a significant number lost 2.5 mm of root length.[70] Approximately a fifth of patients had greater than 2.5 mm of root loss.[70]

In one systematic review the mean amount of root shortening ranged between 1–2 mm.[72] Janson and colleagues detected mild resorption (described as the apex having an irregular contour) in 43% of patients with moderate (described as apical blunting with an almost flat surface) an increased incidence of 53%.[72] Lopateine and Dumbravaite found root resorption was 2 mm or greater in up to 18% of cases and over 4 mm in 5% of cases.[73]

Unfortunately, the reporting methods that were used failed to recognise that certain teeth would be more compromised than others due to differing pre-existing root lengths as well as not really appreciating the three dimensional nature of resorption. For example, a lateral incisor (usually less than 20 mm long) with moderate root resorption is likely to be more compromised than a canine with the same proportion of root resorption where the root is often 25 mm or longer.

The amount of resorption and the tooth's prognosis has been investigated. Kalkwarf and colleagues showed that 4 mm of root shortening was equal to roughly 20% of periodontal attachment loss.[74] If orthodontics is undertaken again at some stage in the future then resorption is even more likely to recur, thereby further compromising root length.

When one considers periodontitis-susceptible patients, any serious previously orthodontically-induced root length loss is likely to accentuate the effective severity of horizontal bone loss and thereby possibly expedite the loss of teeth. Where resorption is severe, mobility or drifting of the teeth may well result, and the final outcome of this is unpredictable.

Jonson and colleagues examined a cohort of patients up to 25 years after the provision of orthodontic treatment. They found that effective root lengths of less than 10 mm were at a higher risk of chronic mobility. Although mobility did not seem to affect tooth retention in a predictable way, any such potential later complications really requires adequate explanation at the discussion stages for consent to be valid.[6,75]

The factors influencing root resorption include those associated with biological ageing of the pulp as well as the magnitude and duration of the forces being applied to the teeth. It appears that where teeth are intruded, or the apex is moved labially, or palatally, that the incidence of apical root resorption increases.[71,76]

A longer treatment period seems to be associated with increased amounts of resorption.[77,78] Maxillary anterior teeth seem to be at a greater risk than other teeth with the maxillary lateral incisor being at the greatest risk (Fig. 10).[79,80] Root apices that are pipette shaped or curved have also been shown to be susceptible.[79,80] An increased overjet, as opposed to an increased overbite, has been shown to be significantly associated with greater root resorption.[79,80]

Basic orthodontic principles suggest that light and intermittent forces are less likely to result in resorption. That basic principle needs to be considered, and caution is advised in promising swift or stable outcomes or a supposed 'perfect smile', especially when many patients perceive or expect, partly as a result of some heavily advertised treatments, that all 'ideal' tooth movement – rather than some anterior alignment – is going to happen predictably within a relatively short period.

Alveolar bone damage

The majority of patients who undergo orthodontic treatment will experience loss of up to 1 mm of alveolar bone height.[81,82] Superficially, given the potential improvements in appearance and probable patient satisfaction, this might not seem to be significant for an adolescent patient with good oral hygiene when considered in isolation from the other complications noted above. If some patients lose 1 mm loss of alveolar bone height but also develop more virulent changes in their periodontal flora and/or get some apical root resorption then the multiple effects of orthodontics will be compounded and such an unfortunate combination can present a very real and tangible risk to the patient's dentition later in their life.

Nelson and colleagues examined alveolar bone loss in a cohort of adult patients ranging from 20 to 70 years of age.[83] Thirty-six percent of patients had one or more surfaces with bone

Fig. 11 This patient presented complaining movement of teeth and roughness of the retainer. Unfortunately, he had severe periodontal disease localised to these sites due to his apparent inability to maintain good plaque control in this region

loss of 2 mm or more.[83] Sample means of the most severe bone loss was documented as being 1.8 mm but with a standard deviation of over 1 mm.[83]

Retention and relapse

The success of any treatment modality can be assessed by the length of time the treatment lasts, without complication, or the need to re-treat or modify the results. Where restorations are considered, a tooth can retain a filling or crown for a 'reasonable' period but the benefit of the restoration needs to be balanced against the initial collateral damage incurred in providing it and the need for further, possibly more complicated, future treatments once the restoration fails.

This can be illustrated by looking at survival studies where a complication free period of approximately 5 years could be considered to be acceptable for most simple restorations but unfortunately this is seldom achieved with some types of restorations in the UK.[84] Where orthodontics is provided and retention is required it would seem reasonable to consider this 5-year period as one possibly helpful outcome measure of treatment. Long-term studies have illustrated that despite treatment in a specialist setting, teeth placed in an unstable position will move and because of this 'indefinite' retention is then required.[85,86] Nevertheless, 10 years after the completion of orthodontic treatment, only 30% to 50% of orthodontic patients effectively retained the tooth positions which were obtained.at the end of their active orthodontics.[85-87] After 20 years, this reduced to 10%.[85-87]

Fixed retainers, as opposed to the removable Hawley type of retainer, or vacuum formed variety, seemingly might provide some reassurance of stability for the patient and the dentist, without the need for consistent removable appliance wear at night (Fig. 11). Unfortunately, this might be linked with other potential problems including greater plaque accumulation and localised periodontal inflammation with the potential loss of bone and gingiva as a longer term result.[88,89] Potentially, fixed retainers can result in gingival recession on the lingual surface of the teeth. Recession on the buccal side can occur during orthodontic treatment, particularly when teeth are moved labially in the presence of thin labial bone and/or a thin periodontal biotype. When this occurs teeth may well become sensitive and subsequent inadequate cleaning can exacerbate an already compromised periodontal status as discussed above.[88,89]

A longitudinal study by Schneider and Ruf examined bonded palatal maxillary retainers placed from canine to canine. Over a period of 30 months, 58% (the majority) of all patients had some problems with their bonded retainer.[90] This short follow-up period might well suggest a greater frequency of problems over the expected lifetime of the patient. That somewhat alarming figure needs to be considered against how patients measure the success of their orthodontic treatment. Anterior dental spacing has been identified as significantly associated with oral health-related quality of life.[91,92]

High failure rates seem to be more common with lower anterior retainers. Over a relatively short period of 6 months, 38% of bonded retainers exhibited debond with a higher incidence being noted among the 'directly' bonded group.[93] These results were echoed in part by Bovali and colleagues.[94]

It is likely that in the future, given the apparent increase in advertising of 'short-term' orthodontic treatments and the introduction of a plethora of different devices, with very limited long-term evidence to support these, that the incidence of de-bonding of the retainers required afterwards could well increase dramatically in the future. It is reassuring to see some anterior teeth alignment systems seem to be delivering more robust training in diagnosis, case selection and technique support than some other systems, many with crassly superficial or over promising market share driven names.

The reliance on successful retainer bonding for holding of teeth in potentially unstable positions is technique sensitive and therefore risky. Other retention methods can be considered but these are more reliant on patient compliance, which is outwith of the control of the treating clinician. In a questionnaire study a significant cohort of patients reported the main reasons for their non-compliance was that they simply forgot to wear their retainers, finding them 'a hassle' to wear or found that the retainers did not fit after a period of non wear (Fig. 12).[95]

Some relapse of orthodontic tooth movements may be inevitable over time and dependent to some extent on the ongoing physiological forces applied to them by soft tissues over their lifetime. This can be a major risk in orthagnathic cases where the soft tissue forces can be particularly strong. In such cases, provision of both fixed and removable retainers has been suggested as a possible solution.[96]

One aspect that has been identified is the need for long-term follow up of retainers (Fig. 13).[96] Due to the nature of bonded retainers the detection of failure is difficult to achieve by the patient. 'Silent' debond, as seen in bridgework, is likely to occur resulting in loss of retention. Often, the first time the de-bond is diagnosed is when the tooth has already moved out of alignment, by which time the damage has already been done.

Relapse represents a significant consent issue for the long-term perception of success of orthodontic treatment. Rather worryingly a recent systematic review examining the treatment options to prevent relapse of lower front teeth after orthodontic treatment found no compelling evidence to support any particular treatment option.[87]

Fig. 12 Blanching of the soft tissue at upper right central and right canine on initial placement of the Essix retainer indicating significant risk of trauma to the vulnerable gingival tissues on these teeth unless adjusted immediately

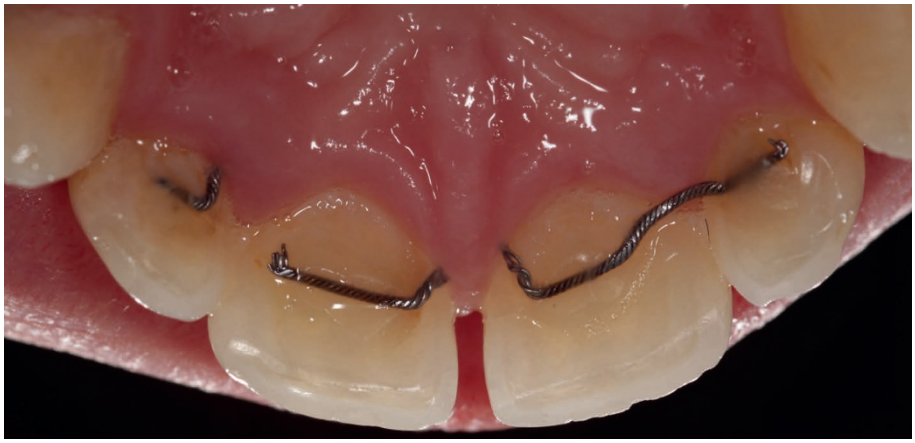

Fig. 13 Drifting and relapse of a previously closed diastema. Rather than replacing the broken and abraded wire the dentist had merely bent the broken braded wire to stop it irritating the tongue

Inability to reach treatment goals

There is limited evidence in the literature specifying precisely the reason(s) for abandoning orthodontic treatment.[97–100] This might be due to either an inability to achieve the desired tooth movements, or a lack of patient compliance, resulting in one or more of the increased risks as described above (Fig. 14). In such situations seeking a restorative opinion might be important to consider alternative approaches to manage the problems at that point.

Impacted teeth can provide a treatment-planning quandary for orthodontic, oral surgery and restorative specialties. Impacted canines can provide the orthodontist with a number of interesting diagnostic and treatment planning challenges. Until the advent of digital radiology, visualising the exact position of the impacted tooth and its effect on any adjacent teeth and other structures was not always reliable.[101]

If left unchecked impacted teeth can cause the loss of other teeth due to the effects of the processes involved in their attempted eruption.[102] Bringing the canine into the line of the arch can be difficult to achieve in some situations and requires a good level of compliance with reliable and adequate anchorage. In some cases the repositioned canine may develop unfortunate complications such as mobility or recession.[103–106] On occasions the impacted tooth can be ankylosed and any attempt to orthodontically bring the tooth into the line of the arch can result in anterior and lateral open bites (Fig. 14).

Elective removal of the impacted tooth usually requires a general anaesthetic with invasive surgery, which often results in a large bony defect in the site with an additional risk

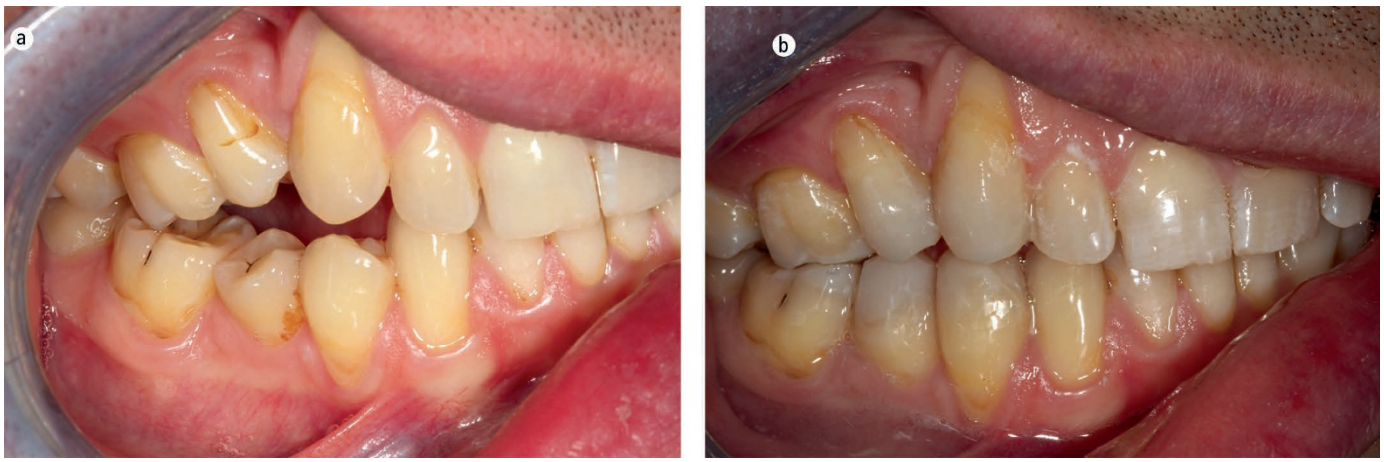

Fig. 14 (a) Presenting condition after failure of prolonged orthodontic treatment; (b) Pragmatic direct resin composite bonding of teeth to close gaps was done with a deliberately lighter material in case the patient subsequently wanted to have the teeth bleached

Fig. 15 (a) This patient had extensive orthodontics and jaw surgery to 'improve her occlusion'. The posterior teeth were firmly in occlusion immediately after the operation and one month later; (b) Unfortunately four months later the posterior teeth came out of contact and the anterior teeth splayed and became loose as a consequence; (c) Pragmatic direct bonding was undertaken to re-establish posterior contacts and reduce pressure on the anterior teeth. Note scar tissue

of surgical damage to the adjacent teeth. It can be argued that leaving impacted teeth in their position, after diagnosis and discussion and monitoring the area for any pathological changes, such as cyst formation or resorption of adjacent teeth, is a pragmatic and sometimes more sensible approach than risky surgery.

Management of a visually missing canine tooth can be a particular challenge, sometimes depending on whether the ectopic canine is left *in situ* or removed. If left *in situ* implant-retained crowns would obviously be unachievable. Conversely, if the tooth is removed, the resultant bony site may be so heavily compromised that the situation is beyond the scope of grafting with predictable materials. The provision of a denture for a solitary space in this patient cohort is unlikely to be acceptable regardless of the age of the patient. The published high success rates with cantilevered resin bridges without preparation may well offer the sensible solution in many such cases.[107,108]

Orthagnathic treatment: risks and benefits

The provision of jaw surgery in conjunction with orthodontics is a significant commitment for both the patient and the clinical team (Fig. 15). The management often starts with orthodontics to decompensate the tooth positions followed by jaw surgery, which can be physically and psychologically traumatic, because it involves surgical movement of either the maxilla or the mandible or both. Unsurprisingly, this treatment can result in some significant and occasionally severe complications.

Mensink and colleagues cited unfavourable fractures, otherwise known as a 'bad split', as a common complication of bilateral sagittal split osteotomies.[109] They cited an incidence of 5% per split site, with this risk increasing if the patient was having both maxillary and mandibular arches treated. An unfortunate and unplanned 'bad split', depending on its severity, can jeopardise even the most carefully planned treatment plan, thereby resulting in a compromised result for the patient and possibly the need for corrective or unplanned further treatment.[109]

There may well be endodontic repercussions of orthagnathic surgery. This can become necessary particularly where segments are surgically mobilised and the blood supply to the root apex is interrupted thereby, compromising the health of the neurovascular bundle which can lead to pulp necrosis necessitating root canal treatment.

Advertising, marketing claims and the potential for future litigation

Subsequent to the Montgomery judgement and the increased litigious culture in the UK, clinicians might well consider being very wary of claims being advertised for some orthodontic systems and techniques.[6,111] Claims that a certain system is 'faster', 'quicker' or 'rapid' with fewer or associated complications, such as initial pain or root resorption, really do need to be substantiated with much more robust and clear evidence, which is free from commercial bias and published in peer review journals. These claims of supposedly risk free or 'super efficient' treatment by companies are unlikely to stem from an unbiased clinician base and are more likely to be driven by marketing departments, who are focused mainly on increasing their market share. As such some, clinicians can be attracted to supposedly more rapid treatments or those that can be attempted with lower requirements for formal prolonged training. Once convinced by this commercially driven salesmanship they are likely to promote this approach to their patients, possibly under the false impression that it is 'better' than other techniques. The repercussions of any dissatisfied patient are unlikely to affect the promoting company. It is much more likely to result in complaints or litigation against the clinician who advocated or tried the treatment. These issues were recently highlighted in a letter to the *BDJ* detailing observations by an orthodontic clinician about a company making claims that were not entirely scientifically substantiated. The Advertising Standards Agency were informed and the claims were subsequently removed.[111]

Different orthodontic systems are more likely to work if the patients are adequately assessed by experienced, properly trained and skilled clinicians. Robust consent processes outlining the realistic material risks and limitations of what is achievable and sensible should reduce the probability of later patient dissatisfaction or disappointment.[112]

Discussion

Where orthodontics is provided in patients with recognisable risk factors there is an increased chance of complications for the patient. If orthodontics is provided in a suboptimal manner, or with inappropriate systems, then complications are also more likely to arise. Tooth movement can be considered to be significantly less damaging than some aspects of supposedly 'cosmetic dentistry' involving irreversible damage for extended ceramic veneers or crowns of various types. Practitioners need to be fully aware of the various risks and inform their patients appropriately of these. The recent heavy advertising of short-term orthodontics (STO) has increased the provision of tooth movement by specialist and non-specialists significantly. This has recently been re-described in at least one system as 'anterior alignment' orthodontics. The variation in training and knowledge of individuals providing this type of orthodontic treatment

is likely to be considerable. Somewhat worryingly, training for some STO systems seems to take place over a matter of days, as opposed to years, for specialist training. There is an overarching responsibility for the dental profession overall to ensure that the public understand clearly the differences in training and competence of those providing treatment. Unfortunately, some of the complications described in this paper are more likely to occur in cases that are complex, but which may not have been identified early on, being treated by individuals with less experience and without the appropriate training in the systems they use. In the light of the 2015 judgement in Montgomery *versus* Lanarkshire and the litigious nature of UK dentistry, these issues cannot be minimised or overlooked.

The ability to recognise simple straightforward cases that can be delivered over a 'short term' requires experience and a sound overall orthodontic knowledge base. As a result of changes in the demographics of orthodontic provision, complications are likely to increase. Sadly, the incidence of complaints and claims as well as other repercussions, may only be realised in the fullness of time.[112]

1. Kragt L, Dhamo B, Wolvius E B, Ongkosuwito E M. The impact of malocclusions on oral health-related quality of life in children-a systematic review and meta-analysis. *Clin Oral Investig* 2015. Epub ahead of print.
2. Ellis P E, Benson P E. Potential hazards of orthodontic treatment-what your patient should know. *Dent Update* 2002; **29:** 492–496.
3. Travess H, Roberts-Harry D, Sandy J. Orthodontics. Part 6: Risks in orthodontic treatment. *Br Dent J* 2004; **196:** 71–77.
4. Cheng LL. Limited evidence indicates fluoride may prevent demineralized white lesions during orthodontic treatment. *J Am Dent Assoc* 2015; **146:** 699–701.
5. Farrell A M, Brazier M. Not so new directions in the law of consent? Examining Montgomery v Lanarkshire Health Board. *J Med Ethics* 2015; DOI: 10.1136/medethics-2015-102861.
6. D'Cruz L, Kaney H. Consent - a new era begins. *Br Dent J* 2015; **219:** 57–59.
7. Cardoso Mde A, Saraiva P P, Maltagliati L Á *et al*. Alterations in plaque accumulation and gingival inflammation promoted by treatment with self-ligating and conventional orthodontic brackets. *Dental Press J Orthod* 2015; **20:** 35–41.
8. Klukowska M, Bader A, Erbe C *et al*. Plaque levels of patients with fixed orthodontic appliances measured by digital plaque image analysis. *Am J Orthod Dentofacial Orthop* 2011; **139:** e463–e470.
9. Vizitiu T C, Giuca M C, Ionescu E. Influence of orthodontic treatment on oral streptococci. *Roum Arch Microbiol Immunol* 2011; **70:** 105–108.
10. Tufekci E, Dixon J S, Gunsolley J C, Lindauer S J. Prevalence of white spot lesions during orthodontic treatment with fixed appliances. *Angle Orthod* 2011; **81:** 206–210.
11. Gorelick L, Geiger A M, Gwinnett A J. Incidence of white spot formation after bonding and banding. *Am J Orthod* 1982; **81:** 93–98.
12. Al Maaitah E F, Adeyemi A A, Higham S M, Pender N, Harrison J E. Factors affecting demineralization during orthodontic treatment: a post-hoc analysis of RCT recruits. *Am J Orthod Dentofacial Orthop* 2011; **139:** 181–191.
13. Akin M, Tezcan M, Ileri Z, Ayhan F. Incidence of white spot lesions among patients treated with self-and conventional ligation systems. *Clin Oral Investig* 2015; **19:** 1501–1506.
14. Julien K C, Buschang P H, Campbell P M. Prevalence of white spot lesion formation during orthodontic treatment. *Angle Orthod* 2013; **83:** 641–647.
15. Boersma JG, van der Veen M H, Lagerweij M D, Bokhout B, Prahl-Andersen B. Caries prevalence measured with QLF after treatment with fixed orthodontic appliances: influencing factors. *Caries Res* 2005; **39:** 41–47.
16. Chapman J A, Roberts W E, Eckert G J, Kula K S, González-Cabezas C. Risk factors for incidence and severity of white spot lesions during treatment with fixed orthodontic appliances. *Am J Orthod Dentofacial Orthop* 2010; **138:** 188–194.
17. Chambers C, Stewart S, Su B, Sandy J, Ireland A. Prevention and treatment of demineralisation during fixed appliance therapy: a review of current methods and future applications. *Br Dent J* 2013; **215:** 505–511.
18. British Orthodontic Society. When should i avoid making an orthodontic referral? Guidance available online http://www.bos.org.uk/Information-for-Dentists/Making-an-Orthodontic-Referral/When-Should-I-Avoid-Making-an-Orthodontic-Referral (accessed 21 September 2016).
19. Geiger A M, Gorelick L, Gwinnett A J, Benson B J. Reducing white spot lesions in orthodontic populations with fluoride rinsing. *Am J Orthod Dentofacial Orthop* 1992; **101:** 403–407.
20. Mitchell L. Decalcification during orthodontic treatment with fixed appliances-an overview. *Br J Orthod* 1992; **19:** 199–205.
21. Norevall L I, 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.
22. Marcusson A, Norevall L I, 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.
23. Chung C K, Millett D T, Creanor S L, Gilmour W H, Foye R H. Fluoride release and cariostatic ability of a compomer and a resin-modified glass ionomer cement used for orthodontic bonding. *J Dent* 1998; **26:** 533–538.
24. Benson P E, Parkin N, Dyer F, Millett D T, Furness S, Germain P. Fluorides for the prevention of early tooth decay (demineralised white lesions) during fixed brace treatment. *Cochrane Database Syst Rev* 2013 **12:** CD003809.
25. van der Kaaij NC, van der Veen MH, van der Kaaij MA, ten Cate JM. A prospective, randomized placebo-controlled clinical trial on the effects of a fluoride rinse on white spot lesion development and bleeding in orthodontic patients. *Eur J Oral Sci* 2015; **123:** 186–193.
26. Faltermeier A, Rosentritt M, Reicheneder C, Behr M. Discolouration of orthodontic adhesives caused by food dyes and ultraviolet light. *Eur J Orthod* 2008; **30:** 89–93.
27. Hamilton R S, Gutmann J L. Endodontic-orthodontic relationships: a review of integrated treatment planning challenges. *Int Endod J* 1999; **32:** 343–360.
28. Stenvik A. Pulp and dentine reactions to experimental tooth intrusion. (A histologic study-long-term effects). *Rep Congr Eur Orthod Soc* 1969; 449–464.
29. Delivanis H P, Sauer G J. Incidence of canal calcification in the orthodontic patient. *Am J Orthod* 1982; **82:** 58–61.
30. Küçükkeleş N, Okar I. Root resorption and pulpal changes due to intrusive force. *J Marmara Univ Dent Fac* 1994; **2:** 404–408.
31. Raiden G, Missana L, Santamaria de Torres E, Kozuszko S, Pedroso R. Pulpal response to intrusive orthodontic forces. *Acta Odontol Latinoam* 1998; **11:** 49–54.
32. Bauss O, Röhling J, Rahman A, Kiliaridis S. The effect of pulp obliteration on pulpal vitality of orthodontically intruded traumatized teeth. *J Endod* 2008; **34:** 417–420.
33. Bauss O, Röhling J, Meyer K, Kiliaridis S. Pulp vitality in teeth suffering trauma during orthodontic therapy. *Angle Orthod* 2009; **79:** 166–171.
34. Ramazanzadeh B A, Sahhafian A A, Mohtasham N, Hassanzadeh N, Jahanbin A, Shakeri M T. Histological changes in human dental pulp following application of intrusive and extrusive orthodontic forces. *J Oral Sci* 2009; **51:** 109–115.
35. Han G, Hu M, Zhang Y, Jiang H. Pulp vitality and histologic changes in human dental pulp after the application of moderate and severe intrusive orthodontic forces. *Am J Orthod Dentofacial Orthop* 2013; **144:** 518–522.
36. Lazzaretti D N, Bortoluzzi G S, Torres Fernandes L F, Rodriguez R, Grehs R A, Martins Hartmann M S. Histologic evaluation of human pulp tissue after orthodontic intrusion. *J Endod* 2014; **40:** 1537–1540.
37. Boyd R L, Leggott P J, Quinn R S, Eakle W S, Chambers D. Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissues *versus* those of adolescents. *Am J Orthod Dentofacial Orthop* 1989; **96:** 191–198.
38. Atack N E, Sandy J R, Addy M. Periodontal and microbiological changes associated with the placement of orthodontic appliances. A review. *J Periodontol* 1996; **67:** 78–85.
39. Paolantonio M, Festa F, di Placido G, D'Attilio M, Catamo G, Piccolomini R. Site-specific subgingival colonization by Actinobacillus actinomycetemcomitans in orthodontic patients. *Am J Orthod Dentofacial Orthop* 1999; **115:** 423–428.
40. van Gastel J, Quirynen M, Teughels W, Coucke W, Carels C. Longitudinal changes in microbiology and clinical periodontal variables after placement of fixed orthodontic appliances. *J Periodontol* 2008; **79:** 2078–2086.
41. Ristic M, Vlahovic Svabic M, Sasic M, Zelic O. Clinical and microbiological effects of fixed orthodontic appliances on periodontal tissues in adolescents. *Orthod Craniofac Res* 2007; **10:** 187–195.
42. Naranjo A A, Triviño M L, Jaramillo A, Betancourth M, Botero J E. Changes in the subgingival microbiota and periodontal parameters before and 3 months after bracket placement. *Am J Orthod Dentofacial Orthop* 2006; **130:** 275.e17–e22.
43. Artun J, Urbye K S. The effect of orthodontic treatment on periodontal bone support in patients with advanced loss of marginal periodontium. *Am J Orthod Dentofacial Orthop* 1988; **93:** 143–148.
44. Wennström J L, Stokland B L, Nyman S, Thilander B. Periodontal tissue response to orthodontic movement of teeth with infrabony pockets. *Am J Orthod Dentofacial Orthop* 1993; **103:** 313–319.
45. Hazan-Molina H, Levin L, Einy S, Aizenbud D. Aggressive periodontitis diagnosed during or before orthodontic treatment. *Acta Odontol Scand* 2013; **71:** 1023–1031.
46. Gkantidis N, Christou P, Topouzelis N. The orthodontic-periodontic interrelationship in integrated treatment challenges: a systematic review. *J Oral Rehabil* 2010; **37:** 377–390.
47. Al Shayeb K N, Turner W, Gillam D G. Accuracy and reproducibility of probe forces during simulated periodontal pocket depth measurements. *Saudi Dent J* 2014; **26:** 50–55.
48. Artun J, Krogstad O. Periodontal status of mandibular incisors following excessive proclination. A study in adults with surgically treated mandibular prognathism. *Am J Orthod Dentofacial Orthop* 1987; **91:** 225–232.
49. Zimmer B, Seifi-Shirvandeh N. Changes in gingival recession related to orthodontic treatment of traumatic deep bites in adults. *J Orofac Orthop* 2007; **68:** 232–244.
50. Kamak G, Kamak H, Keklik H, Gurel H G. The effect of changes in lower incisor inclination on gingival recession. *Sci World J* 2015; **2015:** 193, 206.
51. Johal A, Katsaros C, Kiliaridis S *et al*. State of the science on controversial topics: orthodontic therapy and gingival recession (a report of the Angle Society of Europe 2013 meeting). *Prog Orthod* 2013; **14:** 16.
52. Wennström J L. Mucogingival considerations in orthodontic treatment. *Semin Orthod* 1996; **2:** 46–54.
53. Renkema A M, Fudalej P S, Renkema A A, Abbas F, Bronkhorst E, Katsaros C. Gingival labial recessions in orthodontically treated and untreated individuals: a case-control study. *J Clin Periodontol* 2013; **40:** 631–637.
54. Wennström J L, Lindhe J, Sinclair F, Thilander B. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol* 1987; **14:** 121–129.

55. Tatakis D N, Chambrone L, Allen E P *et al*. Periodontal soft tissue root coverage procedures: a consensus report from the AAP Regeneration Workshop. *J Periodontol* 2015; **86:** S52–55.
56. Hamersky P A, Weimer A D, Taintor J F. The effect of orthodontic force application on the pulpal tissue respiration rate in the human premolar. *Am J Orthod* 1980; **77:** 368–378.
57. Holmstedt J O, McClugage SG Jr, Clark J S, Guevara M J. Osteodentin formation in continuously erupting teeth of guinea pigs. *J Dent Res* 1977; **56:** 1569–1576.
58. Taintor J F, Shalla C L. Comparison of respiration rates in different zones of rat incisor pulp. *J Dent* 1978; **6:** 63–70.
59. Stenvik A, Mjör I A. Pulp and dentine reactions to experimental tooth intrusion. A histologic study of the initial changes. *Am J Orthod* 1970; **57:** 370–385.
60. Derringer K A, Jaggers D C, Linden R W. Angiogenesis in human dental pulp following orthodontic tooth movement. J *Dent Res* 1996; **75:** 1761–1766.
61. Kimberly C L, Byers M R. Inflammation of rat molar pulp and periodontium causes increased calcitonin gene-related peptide and axonal sprouting. *Anat Rec* 1988; **222:** 289–300.
62. Kvinnsland I, Kvinnsland S. Changes in CGRP-immunoreactive nerve fibres during experimental tooth movement in rats. *Eur J Orthod* 1990; **12:** 320–329.
63. Artun J, Urbye K S. The effect of orthodontic treatment on periodontal bone support in patients with advanced loss of marginal periodontium. *Am J Orthod Dentofacial Orthop* 1988; **93:** 143–148.
64. Spurrier S W, Hall S H, Joondeph D R, Shapiro P A, Riedel RA. A comparison of apical root resorption during orthodontic treatment in endodontically treated and vital teeth. *Am J Orthod Dentofacial Orthop* 1990; **97:** 130–134.
65. Mirabella A D, Artun J. Risk factors for apical root resorption of maxillary anterior teeth in adult orthodontic patients. *Am J Orthod Dentofacial Orthop* 1995; **108:** 48–55.
66. Mirabella A D, Artun J. Prevalence and severity of apical root resorption of maxillary anterior teeth in adult orthodontic patients. *Eur J Orthod* 1995; **17:** 93–99.
67. Parlange L M, Sims MR. A T E M. stereological analysis of blood vessels and nerves in marmoset periodontal ligament following endodontics and magnetic incisor extrusion. *Eur J Orthod* 1993; **15:** 33–44.
68. Bender I B, Byers M R, Mori K. Periapical replacement resorption of permanent, vital, endodontically treated incisors after orthodontic movement: report of two cases. *J Endod* 1997; **23:** 768–773.
69. Wickwire N A, Mc Neil M H, Norton L A, Duell R C. The effects of tooth movement upon endodontically treated teeth. *Angle Orthod* 1974; **44:** 235–242.
70. Linge L, Linge B O. Patient characteristics and treatment variables associated with apical root resorption during orthodontic treatment. *Am J Orthod Dentofacial Orthop* 1991; **99:** 35–43.
71. Segal G R, Schiffman P H, Tuncay O C. Meta analysis of the treatment-related factors of external apical root resorption. *Orthod Craniofac Res* 2004; **7:** 71–78.
72. Janson G R, De Luca Canto G, Martins D R, Henriques J F, De Freitas MR. A radiographic comparison of apical root resorption after orthodontic treatment with 3 different fixed appliance techniques. *Am J Orthod Dentofacial Orthop* 2000; **118:** 262–273.
73. Lopatiene K, Dumbravaite A. Risk factors of root resorption after orthodontic treatment. *Stomatologija* 2008; **10:** 89–95.
74. Kalkwarf K L, Krejci R F, Pao Y C. Effect of apical root resorption on periodontal support. *J Prosthet Dent* 1986; **56:** 317–319.
75. Jönsson A, Malmgren O, Levander E. Long-term follow-up of tooth mobility in maxillary incisors with orthodontically induced apical root resorption. *Eur J Orthod* 2007; **29:** 482–487.
76. Fox N. Longer orthodontic treatment may result in greater external apical root resorption. *Evid Based Dent* 2005; **6:** 21.
77. Snelgrove RA. Generalized idiopathic apical root resorption as an incidental finding in an adolescent: a case history. *Dent Update* 1995; **22:** 276–278.
78. Baumrind S, Korn E L, Boyd R L. Apical root resorption in orthodontically treated adults. *Am J Orthod Dentofacial Orthop* 1996; **110:** 311–320.
79. Sameshima G T, Sinclair P M. Predicting and preventing root resorption: Part II. Treatment factors. *Am J Orthod Dentofacial Orthop* 2001; **119:** 511–515.
80. Sameshima G T, Sinclair P M. Predicting and preventing root resorption: Part I. Diagnostic factors. *Am J Orthod Dentofacial Orthop* 2001; **119:** 505–510.
81. Alstad S, Zachrisson B U. Longitudinal study of periodontal condition associated with orthodontic treatment in adolescents. *Am J Orthod* 1979; **76:** 277–286.
82. Harris E F, Baker W C. Loss of root length and crestal bone height before and during treatment in adolescent and adult orthodontic patients. *Am J Orthod Dentofacial Orthop* 1990; **98:** 463–469.
83. Nelson P A, Artun J. Alveolar bone loss of maxillary anterior teeth in adult orthodontic patients. *Am J Orthod Dentofacial Orthop* 1997; **111:** 328–334.
84. Burke F J, Lucarotti P S. Ten-year outcome of crowns placed within the General Dental Services in England and Wales. *J Dent* 2009; **37:** 12–24.
85. Little RM. Stability and relapse of dental arch alignment. *Br J Orthod* 1990; **17:** 235–241.
86. Little R M, Riedel R A, Stein A. Mandibular arch length increase during the mixed dentition: postretention evaluation of stability and relapse. *Am J Orthod Dentofacial Orthop* 1990; **97:** 393–404.
87. O'Neill J. No randomised trials of interventions to manage relapse of lower incisors after orthodontic treatment. *Evid Based Dent* 2013; **14:** 116–117.
88. Levin L, Samorodnitzky-Naveh G R, Machtei E E. The association of orthodontic treatment and fixed retainers with gingival health. *J Periodontol* 2008; **79:** 2087–2092.
89. Pandis N, Vlahopoulos K, Madianos P, Eliades T. Long-term periodontal status of patients with mandibular lingual fixed retention. *Eur J Orthod* 2007; **29:** 471–476.
90. Schneider E, Ruf S. Upper bonded retainers. *Angle Orthod* 2011; **81:** 1050–1056.
91. Bernabé E, Flores-Mir C. Influence of anterior occlusal characteristics on self-perceived dental appearance in young adults. *Angle Orthod* 2007; **77:** 831–836.
92. Sardenberg F, Martins M T, Bendo C B, Pordeus I A, Paiva S M, Auad S M, Vale M P. Malocclusion and oral health-related quality of life in Brazilian school children. *Angle Orthod* 2013; **83:** 83–89.
93. Taner T, Aksu M. A prospective clinical evaluation of mandibular lingual retainer survival. *Eur J Orthod* 2012; **34:** 470–474.
94. Bovali E, Kiliaridis S, Cornelis M A. Indirect vs direct bonding of mandibular fixed retainers in orthodontic patients: a single-centre randomized controlled trial comparing placement time and failure over a 6-month period. *Am J Orthod Dentofacial Orthop* 2014; **146:** 701–708.
95. Pratt M C, Kluemper G T, Lindstrom A F. Patient compliance with orthodontic retainers in the postretention phase. *Am J Orthod Dentofacial Orthop* 2011; **140:** 196–201.
96. Fleming P S, Springate S D, Chate R A. Myths and realities in orthodontics. *Br Dent J* 2015; **218:** 105–110.
97. Fleury J E, Deboets P, Assaad C, Maffre N, Voisin D, Viou F. [The abandonment of treatment in orthodontics]. *Rev Stomatol Chir Maxillofac 1989*; **90:** 30–38.
98. Cardonne C R, Pacheco A H, García O A, Coales M C, Zaldívar E R, Ravinal L P. [Dropouts from orthodontic treatment. Factors which cause it]. *Rev Cubana Estomatol* 1989; **26:** 97–104.
99. Brattström V, Ingelsson M, Aberg E. Treatment cooperation in orthodontic patients. *Br J Orthod* 1991; **18:** 37–42.
100. Murray AM. Discontinuation of orthodontic treatment: a study of the contributing factors. *Br J Orthod* 1989; **16:** 1–7.
101. Alqerban A, Jacobs R, Fieuws S, Willems G. Radiographic predictors for maxillary canine impaction. *Am J Orthod Dentofacial Orthop* 2015; **147:** 345–354.
102. Doğramaci E J, Sherriff M, Rossi-Fedele G, McDonald F. Location and severity of root resorption related to impacted maxillary canines: a cone beam computed tomography (CBCT) evaluation. *Aust Orthod J* 2015; **31:** 49–58.
103. Becker A, Chaushu S. Long-term follow-up of severely resorbed maxillary incisors after resolution of an etiologically associated impacted canine. *Am J Orthod Dentofacial Orthop* 2005; **127:** 650–654.
104. Chaushu S, Becker A, Zeltser R, Branski S, Vasker N, Chaushu G. Patients perception of recovery after exposure of impacted teeth: a comparison of closed-*versus* open-eruption techniques. *J Oral Maxillofac Surg* 2005; **63:** 323–329.
105. Crescini A, Nieri M, Buti J, Baccetti T, Pini Prato G P. Orthodontic and periodontal outcomes of treated impacted maxillary canines. *Angle Orthod* 2007; **77:** 571–577.
106. Crescini A, Nieri M, Buti J, Baccetti T, Pini Prato G P. Pre-treatment radiographic features for the periodontal prognosis of treated impacted canines. *J Clin Periodontol* 2007; **34:** 581–587.
107. Djemal S, Setchell D, King P, Wickens J. Long-term survival characteristics of 832 resin-retained bridges and splints provided in a post-graduate teaching hospital between 1978 and 1993. *J Oral Rehabil* 1999; **26:** 302–320.
108. King P A, Foster L V, Yates R J, Newcombe R G, Garrett M J. Survival characteristics of 771 resin-retained bridges provided at a UK dental teaching hospital. *Br Dent J* 2015; **218:** 423–428.
109. Mensink G, Verweij J P, Frank M D, Eelco Bergsma J, Richard van Merkesteyn J P. Bad split during bilateral sagittal split osteotomy of the mandible with separators: a retrospective study of 427 patients. *Br J Oral Maxillofac Surg* 2013; **51:** 525–529.
110. Weltman B, Vig K W, Fields H W, Shanker S, Kaizar E E. Root resorption associated with orthodontic tooth movement: a systematic review. *Am J Orthod Dentofacial Orthop* 2010; **137:** 462–476.
111. Stanford N. Orthodontics: Fast removal of claims. *Br Dent J* 2016; **220:** 220.
112. Alani A, Kelleher M, Haemmings K *et al*. Balancing the risks and benefits associated with cosmetic dentistry- a joint statement by UK specialist dental societies. *Br Dent J* 2015; **218:** 543–548.