REVIEW

Endodontic-orthodontic relationships: a review of integrated treatment planning challenges

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Abstract

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Literature review There is a paucity of information on the concise relationship between endodontics and orthodontics during treatment planning decisions. This relationship ranges from effects on the pulp from orthodontic treatment and the potential for resorption during tooth movement, to the clinical management of teeth requiring integrated endodontic and orthodontic treatment. This paper reviews the literature based on the definition of endodontics and the scope of endodontic practice as they relate to common orthodontic-endodontic treatment planning challenges. Literature data bases were accessed with a focus on orthodontic tooth movement and its impact on the viability of the dental pulp; its impact on root resorption in teeth with vital pulps and teeth with previous root canal treatment; the ability to move orthodontically teeth that were endodontically treated versus nonendodontically treated; the role of previous tooth trauma; the ability to move teeth orthodontically that have been subjected to endodontic surgery; the role of orthodontic treatment in the provision for and prognosis of endodontic treatment; and, the integrated role of orthodontics and endodontics in treatment planning tooth retention.

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Orthodontic tooth movement can cause degenerative and/or inflammatory responses in the dental pulp of teeth with completed apical formation. The impact of the tooth movement on the pulp is focused primarily on the neurovascular system, in which the release of specific neurotransmitters (neuropeptides) can influence both blood flow and cellular metabolism. The responses induced in these pulps may impact on the initiation and perpetuation of apical root remodelling or resorption during tooth movement. The incidence and severity of these changes may be influenced by previous or ongoing insults to the dental pulp, such as trauma or caries. Pulps in teeth with incomplete apical foramen, whilst not immune to adverse sequelae during tooth movement, have a reduced risk for these responses. Teeth with previous root canal treatment exhibit less propensity for apical root resorption during orthodontic tooth movement. Minimal resorptive/remodelling changes occur apically in teeth that are being moved orthodontically and that are well cleaned, shaped, and three-dimensionally obturated. This outcome would depend on the absence of coronal leakage or other avenues for bacterial ingress.

A traumatized tooth can be moved orthodontically with minimal risk of resorption, provided the pulp has not been severely compromised (infected or necrotic). If there is evidence of pulpal demise, appropriate endodontic management is necessary prior to orthodontic treatment. If a previously traumatized tooth exhibits resorption, there is a greater chance that orthodontic tooth movement will enhance the resorptive process. If a tooth has been severely traumatized (intrusive luxation/avulsion) there may be a greater incidence of resorption with tooth movement. This can occur with or without previous endodontic treatment. Very little is known about the ability to

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move successfully teeth that have undergone periradicular surgical procedures. Likewise, little is known about the potential risks or sequelae involved in moving teeth that have had previous surgical intervention. Especially absent is the long-term prognosis of this type of treatment.

During orthodontic tooth movement, the provision of endodontic treatment may be influenced by a number of factors, including but not limited to radiographic interpretation, accuracy of pulp testing, patient signs and symptoms, tooth isolation, access to the root canal, working length determination, and apical position of the canal obturation. Adjunctive orthodontic root extrusion and root separation are essential clinical procedures that will enhance the integrated treatment planning process of tooth retention in endodontic-orthodontic related cases.

Keywords: endodontics, extrusion, orthodontics, pulp, resorption.

Introduction

Detailed information is sparse on the overall relationships between endodontics and orthodontics during treatment planning decisions. Articles that have explored the possibility of integrated relationships have focused primarily or cursorily on individual topical questions, such as 'Does orthodontic tooth movement impact on the viability of the dental pulp'; 'Is apical root resorption, that may occur during orthodontic treatment, the same on teeth with vital pulps as teeth with previous root canal treatment?'; 'Can endodontically treated teeth be moved orthodontically as readily as non-endodontically treated teeth?'; 'What role does previous tooth trauma play in the orthodontic tooth movement of teeth with vital pulps or previous root canal treatment?'; 'Can teeth that have been managed with surgical endodontic procedures be moved orthodontically?'; 'Will ongoing orthodontic treatment affect the provision and outcome of endodontic treatment?'; and, 'How can orthodontic procedures be used in conjunction with endodontics to enhance treatment planning for tooth retention?' The purpose of this literature review paper is to provide a meaningful assessment of the literature with regard to these questions, and to determine how this information can be used in the challenges that are often encountered in the clinical treatment planning of cases in which the integration of endodontic and orthodontic principles plays an important role in treatment outcomes.

Does orthodontic tooth movement impact on the viability of the dental pulp?

During rapid tooth movement pulpal injury may occur (Seltzer & Bender 1984). This is primarily due to an alteration in the blood vessels in the apical periodontium and those entering the pulp. Clinically the teeth may have altered sensations to stimuli (Burnside *et al.* 1974). Effects of this nature may have a direct impact on the metabolism of the pulp tissue, in particular the odontoblasts in fully formed teeth, and Hertwigs epithelial root sheath in incompletely formed teeth. The pulpal changes and their consequences appear to be proportionally more severe with greater orthodontic forces.

Oppenheim (1936, 1937) showed some signs of severe pulpal degeneration in all human cases using a labiolingual expansion appliance. The movement afforded by this technique resulted in a tipping motion in the apical third of the root. His findings focused on the lack of collateral circulation to the pulp during tooth movement as being the major aetiological factor for pulpal degeneration. As a result, he recommended the use of light intermittent forces to reduce damage to the dental tissues and provide time for possible repair. Tschamer (1974) noted that some of the odontoblasts will degenerate whilst other pulpal cells will undergo atrophy during appliance activation in late adolescent patients. Prior and more recent studies evaluating the pulpal response to tooth movement have supported these findings (Skillen & Reitan 1940, Oppenheim 1942, Aisenberg 1948, Stenvik & Mjör 1970, Guevara *et al.* 1977).

Taintor & Shalla (1978) found that under normal conditions the respiratory rate of the pulp cells corresponds to the degree of dentinogenic activity. Hence the greater the activity, the greater the rate of tissue respiration. Hamersky *et al.* (1980), using radiorespirometric methods, demonstrated a significant 27.4% mean depression in the pulpal respiratory rate when the tooth is undergoing orthodontic movement. Additionally, as the age of the subject increased, the relative amount of depression in the pulpal respiratory rate increased also. These results would seem to indicate a relationship between the biologic effect of an orthodontic force and the maturity of the tooth, particularly the dentinogenic activity of the pulp. This would imply that a greater dentinogenic activity coupled with a larger apical foramen would result in a

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reduction of detrimental effects from orthodontic forces. In support of these concepts, Labart *et al.* (1980) reported an increased pulpal respiration as a result of orthodontic forces being applied to the continuously erupting rat incisor. Further support for this concept is available from the previous work by Ooshita (1975). He noted that the surrounding tissues of bone and periodontal ligament, that are linked to the tooth with an open or large foramen, demonstrate accelerated activity during tooth movement. To further clarify these issues, and to address the ability of the pulp to recover following insult, Unterseher *et al.* (1987) assessed the pulpal respiration response after a 7-day rest period. The mean respiratory rates remained depressed approximately 32.2% after the rest period. However, two subgroups were identified in the experimental pulps, one that had returned to normal respiratory rates and one that did not. Age and apical opening size correlated with the return to normal respiratory rates in 1 week. Age was negatively correlated with the respiration rate, whilst apical opening size was positively correlated with the respiration rate. Clinically, the occurrence of apical root resorption appears to be greater when orthodontic treatment is started after 11 years of age, with fixed appliances causing more resorption than removable appliances (Linge & Linge 1991). These findings would tend to correlate with a decreased size in the apical foramen of the tooth involved in orthodontic tooth movement.

Historically, specific angiogenic changes in the human dental pulp associated with orthodontic movement have received limited study. Angiogenesis is the formation of new capillary structures ultimately leading to the organization of larger structures by a process of neovascularization (Polverini 1995). Kvinnsland *et al.* (1989), using fluorescent microspheres, showed a substantial increase in blood flow in the dental pulp of mesially tipped rat molars. Increases in force application resulted in an increase in blood flow. Nixon *et al.* (1993), using the rat model, also found that there was a significant vascular change with an increased number of functional pulpal vessels as related to the specific forces applied. Initially a hyperaemic response was visible following the force applications. These findings contradict those of Anstendig & Kronman (1972) who observed fewer blood vessels in tooth pulps subjected to orthodontic forces.

McDonald & Pitt Ford (1994) identified that blood flow changes within the pulp during tooth movement are not a mere reduction with no further response, but have dynamic changes that overcome potentially poor perfusion of the tissues. Using laser Doppler flowmetry, pulpal blood flow of permanent maxillary canines was assessed before, during, and after an application of a 50-gram force. During tooth movement there was a phase of pulpal reactive hyperaemia where perfusion of the tissues improves. The blood flow returned to normal within 72 h. This time frame has been considered insignificant regarding long-term damage to the pulp tissue (Popp *et al.* 1992), as narrowing of the pulpal space that followed orthodontic forces was considered to be the result of normal ageing. Barwick & Ramsay (1996) also used laser Doppler flowmetry during a 4-min application of an intrusive orthodontic force. Intrusive forces from 75 to 4498 g were recorded; however, pulpal blood flow was not altered during the brief application of these specified forces.

Recently Derringer *et al.* (1996), moved human teeth orthodontically, extracted the teeth, and harvested the pulps. The pulps were embedded in collagen and cultured in growth media for up to 4 weeks. New microvessels were observed within 5 days and their identification was confirmed with both light microscopy and electron microscopy. There were significantly greater numbers of microvessels at day 5 and day 10 of culture in pulp explants from orthodontically moved teeth than in the control teeth. These findings would support not only the presence of significant angiogenesis in the pulp, but also the presence of the necessary angiogenic growth factors. The factors that have been implicated and described in this process consist of PDGF (platelet-derived growth factor), EGF (epidermal growth factor), TGF-b ransforming growth factor beta) (Schultz & Grant 1991). These growth factors have been identified also in periodontal ligament wound healing (Terranova *et al.* 1987), in pulp following endodontic injury (Shirakawa *et al.* 1994), during tooth development and eruption (Klein *et al.* 1994) and during orthodontic tooth movement in cats (Davidovitch 1995),

Alterations in the pulpal vasculature with subsequent alterations in the metabolism of the pulpal cells, will usually result in an increased deposition of reparative dentine in both the coronal and radicular portions of the pulp, along with a concurrent increase in dystrophic mineralization (Seltzer & Bender 1984). This response has been reported to result, in some cases, in complete obliteration of the pulpal space (Dougherty 1968). The incidence of this occurrence, however, does not appear to be clinically significant,

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although a resultant pulpal necrosis can occur (Stuteville 1937, Oppenheim 1942, Delivanis & Sauer 1982). Most of the time changes that occur in the pulp are considered reversible, unless the pulp has undergone previous insult or challenge. Most recently, Nixon *et al.* (1993) identified a force dependent increase in predentine width that was measured at the peak of the tooth movement cycle in the rat model. Pulps were considered to be normal prior to tooth movement. No differences were found with regard to location in the pulpal space that would indicate that the pulp cannot distinguish the specific location of the applied force. What is of clinical importance appears to be the history of trauma to the tooth prior to the orthodontic tooth movement (Rotstein & Engel 1991) and the radiographic observation that the pulpal space may have narrowed prior to or during the active tooth movement.

In teeth that have undergone varying degrees of pulp space calcification, responses or lack thereof to pulp testing with electrical devices may be of no value. Burnside *et al.* (1974) showed that orthodontic forces clearly had some effect on the pulpal nerves in a study that evaluated electric pulp tester stimulation on an experimental group of teeth undergoing orthodontic treatment and a control group not receiving treatment. All the experimental groups except mandibular canines showed higher electrical thresholds than did the non-treatment controls. The clinical significance of this finding, however, was uncertain. Others have attributed the altered findings to oedema or hyperaemia following damage to the local vascular system (Nordh 1955).

Neural responses and evidence for the release of specific neural transmitters have also been assessed during orthodontic tooth movement. Intrapulpal axon response to orthodontic movement was explored by Bunner & Johnson (1982). Human teeth with open apices were subjected to short-term movement and long-term movement, and compared with teeth with closed apices subjected to short- and long-term movement. Untreated teeth served as controls. Subsequently the teeth were extracted and evaluated under both the light and electron microscopy. Although unmyelinated axons outnumbered myelinated axons, no significant differences in myelinated or unmyelinated axon numbers were observed between the experimental (orthodontic movement) and the control (no orthodontic movement) teeth. Altered myelin fibers, possibly degenerating, were observed in only a small percentage of axons in teeth moved for a short period, and no alterations were observed in teeth moved for long periods. It was concluded that intrapulpal axon alterations are minimal and not progressive with conservative tooth movement. This study infers that no irreversible insult is inflicted on healthy teeth having conservative orthodontic treatment. This does not, however, account for the fact that the changes detected generally do not produce symptoms. Rather, symptoms of reversible or irreversible pulpitis may still be present and may be masked by the discomfort felt from changes in force that are made during appliance modifications.

Using the orthodontic tooth movement model, recent studies have attempted to discover the precise metabolic events involved in neural transmission of nociceptive information. Studies focused on the peptidergic pathways, that purportedly inhibit the firing of pain-conducting fibers (Walker *et al.* 1987, Robinson *et al.* 1989, Parris *et al.* 1989), the presence of calcitonin gene-related peptide immunoreactive nerve fibers (CGRP IR) (Kimberly & Byers 1988, Kvinnsland & Kvinnsland 1990), and the presence of substance P (SP) (Parris *et al.* 1989, Nicolay *et al.* 1990).

Walker *et al.* (1987) were the first to identify the presence of a methionine enkephalin (ME) in the pulp that was mobilized during the application of orthodontic forces. The ME significantly decreased in the pulp during the force application in an inverse log-linear relationship to the amount of applied force. Robinson *et al.* (1989) measured β-endorphin-like immunoreactivity (BE-LI) in the human tooth pulp following acute mechanical stress. β-endorphin (BE) is an active peptide derived from the precursor protein pro-opiomelanocortin and has been shown to possess profound antinociceptive qualities. Thirty patients, ranging from 11 to 30 years. participated in the study. A monotonic decrease in the BE-LI concentrations was evident according to a four premolar extraction order (extraction of all first or second premolars as part of an orthodontic treatment plan). BE is capable of modulating SP, suggesting that BE may play a role in the regulation of noxious impulses. Further studies on the action of the neuropeptides in the dental pulp during applied orthodontic forces, in particular ME and SP, were conducted by Parris *et al.* (1989). Females with teeth subjected to orthodontic forces were found to have significantly greater concentrations of ME in their pulps than males. Substance P concentration decreased significantly from the first to the third tooth extracted, then increased from the third to the fourth

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tooth. There was a positive correlation in the pulp between the concentrations of SP and ME, whilst the concentrations of both substances correlated negatively with the magnitude of orthodontic force. This correlation was enhanced when the value of the force was log-transformed.

Further assessment of SP during tooth movement was performed by Nicolay *et al.* (1990). During tooth movement, there was evidence of increased density of neuronal elements exhibiting positive staining for SP. This response was seen rapidly in the dental pulp and delayed in the periodontal ligament (PDL). The early response in the pulp was consistent with the findings of Parris *et al.* (1989) and suggests a role in pain perception. The latter response in the PDL suggests a role in cellular recruitment and alveolar bone remodelling. Studies by Kimberly & Byers (1988) and Kvinnsland & Kvinnsland (1990) have shown an increased number of CGRP IR nerves in the pulp and periradicular tissues during tooth movement. These studies, taken as a whole, indicated that peptidergic substances and neural elements take an active part in the tissue responses in pulp and supporting tissues during tooth movement. Most recently, Bender *et al.* (1997) offered a hypothetical explanation as to the role of the dental pulp in the aetiology of the resorptive process after orthodontic treatment in both vital and endodontically treated teeth. Both the somatosensory α-delta and C-fibers are sympathetic neurons that release various peptides by means of intra-axonal transport at the terminal nerve endings. The neuropeptides are Substance P, calcitonin gene-related peptide (CGRP), neurokinin A, vasoactive intestinal polypeptide, and neuropeptide Y. When released they act as neurogenic vasodilators and vasoconstrictors. Therefore, these neuropeptides play an important role in the regulation of the blood flow to the pulp and the periodontium. In particular, there is a greater CGRP-IR fibre response in the pulp and periradicular tissues during tooth movement that is accentuated around the vascular system. The resultant increase in the blood flow to these tissues during tooth movement impacts on the availability of cells of haematopoetic origin (osteoclast precursors), that are capable under local stimulatory factors to differentiate into osteoclasts and influence the resorptive remodelling process of the teeth (Rygh *et al.* 1986, Vandevska-Radunovic *et al.* 1997).

Whilst radiographic changes of pulp space obliteration during or following tooth movement are usually obvious, the lack of further dentine elaboration may not impress the clinician until other clinical or radiographic findings surface. This can occur following the introduction of orthodontic forces that are beyond the physiological tolerance of both the periodontal and pulpal vessels. Subsequent pulpal necrosis may result and may not be detected until clinically there is a darkening of the crown of the tooth, that indicates a liberation of haemoglobin that breaks down into haemosiderin (a dark yellow, iron-containing pigment), which ultimately penetrates the dentinal tubules; the presence of patient signs or symptoms; the radiographic appearance of a radiolucency; or the failure of the pulp space to close with irritational dentine in a manner compatible with adjacent teeth (Seltzer & Bender 1984). Histological findings in the pulp that may support these possibilities have been described by Mostafa *et al.* (1991) in response to orthodontic extrusion of teeth. Specific pulp responses noted included circulatory disturbances with congested and dilated vessels, odontoblastic degeneration, vacuolization and oedema of the tissues and eventual fibrotic changes. All pulps had a mean age of 18 years with a range of 16–21. Therefore, it was assumed that there were similarly sized apical foramina. Historically the duration of these types of pulpal changes has been inconclusive (Oppenheim 1942, Butcher & Taylor 1952). However, it would seem reasonable that teeth with complete apical formation and teeth with pulps that have had previous compromises such as trauma, caries, and restorations or periodontal disease (Årtun & Urbye 1988) may be more susceptible to irreversible pulpal changes or necrosis under this type of orthodontic movement.

Conclusions

The literature reviewed supports the fact that orthodontic tooth movement can cause degenerative and/or inflammatory responses in the dental pulp of teeth with completed apical formation. The impact of the tooth movement on the pulp is focused primarily on the neurovascular system, in which the release of specific neurotransmitters (neuropeptides) can influence both blood flow and cellular metabolism. The responses induced in these pulps may impact on the initiation and perpetuation of apical root remodelling or resorption during tooth movement. The incidence and severity of these changes may be influenced by previous or ongoing insults to the dental pulp, such as trauma or caries. Pulps in teeth with an incomplete apical foramen, whilst not immune to adverse sequelae during tooth movement, have a reduced risk for these responses.

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Is apical root resorption, that may occur during orthodontic treatment, the same on teeth with vital pulps as on teeth with previous root canal treatment?

According to the Glossary-Contemporary Terminology For Endodontics (1998), resorption is defined as 'a condition associated with either a physiologic or a pathologic process resulting in a loss of dentine, cementum, and/or bone.' Andreasen & Andreasen (1994) define the process further as being of three types; *surface resorption,* that is a self-limiting process, usually involving small areas followed by spontaneous repair from adjacent parts of the periodontal ligament in the form of new cementum; *inflammatory resorption,* where the initial root resorption has reached the dentinal tubules of an infected necrotic pulp or an infected leukocytic zone; and *replacement resorption,* where bone replaces the resorbed tooth material that leads to ankylosis. Ottolengui (1914) related root resorption directly to orthodontic treatment, and cited without reference, that Schwarzkopf had demonstrated resorbed roots in extracted permanent teeth in 1887. Ketcham (1927, 1929) demonstrated with radiographic evidence the differences between root shape before and after orthodontic treatment. Further population samples have verified that the pre-eminent cause of external apical root resorption is orthodontic treatment (Rudolph 1940, Massler & Malone 1954, Phillips 1955, Woods *et al.* 1992, Harris *et al.* 1993). As referenced by Tronstad (1988a), Cwyk *et al.* (1984) found that 5–10 years after completion of orthodontic treatment, 42.3% of the maxillary central incisors, 38.5% of the maxillary lateral incisors, and 17.4% of the mandibular incisors had undergone apical resorption. The overall incidence of resorption was 28.8% for the orthodontically treated incisors compared to 3.4% for the controls. Apical root resorption has been reported to be seen four times more often than lateral resorption (Tronstad 1988a). Root resorption following orthodontic treatment is considered as surface resorption or transient inflammatory resorption, because replacement resorption is rarely seen subsequent to tooth movement only (Andreasen & Andreasen 1994). Morphologically and radiographically it may present as a slightly blunted or round apex to a grossly resorbed apex.

The specific causes of external apical root resorption (EARR) (Harris *et al.* 1993), referred to as PARR (periapical replacement resorption) by Bender *et al.* (1997) during orthodontic treatment are not well understood, but heavy forces, especially intrusive or tipping forces, are commonly implicated (Reitan 1974, Vardimon *et al.* 1991, Kaley & Phillips 1991). Levander & Malmgren (1988) indicated that teeth with blunt or pipette shaped roots of maxillary central incisors were significantly at greater risk for EARR than teeth with normal root form. Deviating root forms are also more susceptible (Oppenheim 1942, Newman 1975) as are teeth with invaginations (Kjr 1995). Harris *et al.* (1993) identified that loss of stability from adjacent teeth, increased use of fewer remaining teeth, and the loss of the root's anchorage in the bone are significant predictors of EARR. The nature of their findings also suggested that inflammatory resorption caused by infection is not an important factor in EARR under these circumstances. Brudvik & Rygh (1995) hypothesized that the determinants of the continued cycle of resorption and repair at the root apex during intrusive tooth movements seemed to be associated with the persistence and removal of necrotic cemental tissue. This may very well be accurate on teeth with vital pulps, whereas teeth with previous root canal treatment may present with different aetiological factors, such as, but not limited to, an unclean canal system, contaminated apical ramifications, and occlusal leakage. Recently Alatli *et al.* (1996) determined that acellular cementum was more readily resorbed during orthodontic tooth movement than cellular cementum. This would imply that the specific apical cementum may be more resistant due to cementocyte viability as opposed to the lateral and mid-root cementum that is acellular in nature.

With a multitude of studies identifying the role of orthodontics and other speculated causes for EARR on teeth with vital pulps, the issue of pulpless teeth or teeth with previous root canal treatment and apical root resorption comes into focus. Will these teeth exhibit greater or lesser amounts of apical root resorption during and following root canal treatment? Secondly, if these teeth are subject to the resorptive phenomenon, what will happen to the root canal filling material? Thirdly, will the apical seal in the root canal system be altered, resulting in failure of the root canal treatment?

Initially, what is the status of resorption in teeth with vital pulps versus teeth with root canal treatment?

In a literature review by Steadman, (1942) root canal treatment was criticized in that it was claimed that the devitalized root acts as a foreign body causing chronic

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irritation and root resorption. Histological sections of such resorptions showed cellular pictures typical of a foreign-body reaction. He considered that the resorption could not be controlled and therefore the prognosis for these teeth was unfavourable. Steadman even went to the point of suggesting, based on the literature, that because of the resorptions, the roots of these teeth would become ankylosed, thereby eliminating the possibility of orthodontic movement. Huettner & Young (1955) challenged Steadman's theory and evaluated the root structure of monkey teeth with both vital and nonvital pulps (root canal treatment) following orthodontic movement. Maxillary teeth were treated and obturated with gutta-percha and Kerr's root canal sealer. The mandibular teeth were treated and obturated with silver cones and sealer. All teeth initially had vital pulps, were treated in an aseptic environment, and were allowed to 'rest' for 3 weeks following root canal treatment to give the apical periodontal ligament time to heal. The edgewise fixed appliance technique of orthodontic tooth movement was used and the experiment was carried from 6 to 8 weeks prior to animal sacrifice. Histological examination showed no foreign-body reactions and the root resorption that was observed was similar in both the vital and devitalized teeth. The authors felt that careful monitoring of the orthodontic forces, endodontic aseptic treatment, and an intact periodontal membrane all contributed significantly to their findings. Similar findings of no difference in the amount of resorption with the two experimental groups was also reported also by Weiss in 1969.

Wickwire *et al.* (1974) reviewed 45 orthodontic patient case histories that contained 53 endodontically treated teeth from six practices that included the following orthodontic techniques; edgewise, Begg, and partial banding mechanotherapy. Historical data, lateral cephalograms, and appropriate radiographs were used to evaluate the teeth. Data revealed those teeth with root canal treatment moved as readily as teeth with vital pulps, but there appeared to be greater radiographic evidence of root resorption in the endodontically treated teeth when compared to the controls.

In a single case report, Anthony (1986) indicated that a tooth undergoing apexification was orthodontically moved with the concomitant deposition of a hard tissue barrier as opposed to apical resorption. Further anecdotal support for this occurrence has been provided recently by Steiner & West (1997). These clinical findings would tend to support the theories of Ooshita (1975) and Hamersky *et al.* (1980) in which the accelerated biological activity of the periradicular tissues during tooth movement may have a beneficial effect on a tooth with an open or large apical foramen.

In an *in vivo* study on cats, Mattison *et al.* (1984) showed no significant difference between external root resorption of endodontically treated and teeth with vital pulps when both were subjected to orthodontic forces. Evaluations were made histologically with the mean resorption lacunae for all teeth that were endodontically treated being 2.14 compared with the mean resorption lacunae for teeth with vital pulps being 2.24. The severity of EARR on teeth with vital pulps versus endodontically treated teeth was determined by Spurrier *et al.* (1990). Forty-three patients who had one or more endodontically treated teeth before orthodontic treatment and who exhibited signs of apical root resorption after treatment were studied. Vital contralateral incisors served as controls. Incisors with vital pulps resorbed to a significantly greater degree than incisors that had been endodontically treated. Control teeth in males exhibited a statistically significant increase in resorption over control teeth in females; however, no differences were noted between genders with the endodontically treated teeth. Similar findings were recorded by Remington *et al.* (1989), although differences were not found in the incidence of resorption between teeth with vital pulps and endodontically treated teeth in a targeted population of British school children (Hunter *et al.* 1990).

Mah *et al.* (1996) evaluated the effectiveness of orthodontic forces in moving root-filled teeth and the degree of EARR that may occur in the ferret animal model. Three months after root canal treatment and tooth movement with an orthodontic spring, tooth movement was assessed from pre- and post-treatment mandibular casts and by fluorescence microscopy from labelled (procion red dye) bone deposition. Root-filled teeth and those with vital pulps moved similar distances when subjected to the same forces. Root-filled teeth showed greater loss of cementum after tooth movement than teeth with pulps, but without significant differences in radiographic root length. The root-filled teeth also showed more resorption lacunae than teeth with vital pulps, but the small difference in incidence between active (orthodontically) root-filled teeth and inactive root- filled teeth was not statistically significant. This suggests that the incidence of resorption lacunae may be related to nonvitality and probably the presence of periradicular lesions rather than orthodontic forces. These findings are in agreement with those of Mattison *et al.* (1984).

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To the contrary, Bender *et al.* (1997) have suggested that the loss of the release of neuropeptides from a pulp that has been removed, would result in a decrease of the CGRP-IR fibers and a reduction in the amount of resorption seen in endodontically treated teeth. Their suggestions have been supported by Parlange & Sims (1993) and Bondemark *et al.* (1997), especially when using attractive magnetics for controlled tooth extrusion following deep coronal fractures. In the former study, the periodontal vascular network and axonal characteristics were within normal limits with no significant changes in blood flow or neuronal densities. In the latter study, no resorption was noted. Finally, in a sample of 39 pairs of contralateral teeth with and without endodontic treatment in 36 patients, Mirabella & Årtun (1995) found that there was significantly less resorption in endodontically treated teeth.

If root filled teeth are subject to the resorptive phenomenon, what will happen to the root canal filling material?

The literature is lacking regarding this occurrence and its ultimate sequelae. Many possibilities exist.

1. The tooth may resorb, exfoliate, and the filling material may be removed with the tooth.
2. The tooth may resorb, exfoliate, and the filling material may be left in the bone. In these cases if the material is gutta-percha, a fibrous capsule will probably surround it. It is also possible that a sinus tract may form and the material will require removal.
3. In cases of both gutta-percha and silver cones, the extended material may undergo resorption itself after the tooth has undergone resorption and exfoliation.
4. In some cases the root may begin resorption, exposing the filling material, and subsequently the resorption ceases with the filling material protruding beyond the new apical foramen. In this situation the root is often seen to develop a new periodontal ligament space and lamina dura around the root apex in close approximation to the filling material.
5. In other cases, once the apical resorption begins, a radiolucency develops around the root apex and the filling material. A sinus tract may develop or there may be incidences of localized swelling. Likewise the tooth may remain symptom free and function normally.

Will the apical seal in the root canal system be altered, resulting in failure of the root canal treatment?

This issue also has been poorly addressed in the literature and is subject to empirical speculation. It seems reasonable and logical that if a root canal has been properly cleaned, shaped, and three-dimensionally obturated, that the apical seal would be sustained no matter what the extent of the apical resorption. Here too, however, multiple possibilities exist.

1. Even though the apical seal remains intact during the resorptive process, the complete seal of the canal may be challenged by coronal leakage (Saunders & Saunders 1994).
2. Exposure of the dentinal tubules that may harbour necrotic tissue, bacterial endotoxins and bacteria may serve as sources that provide sufficient apical irritation to stimulate an extended or deleterious inflammatory resorptive process (Peters *et al.* 1995, Nissan *et al.* 1995).
3. There is clinical evidence of apical root resorption on teeth with previous root canal treatment in which a portion of the root is missing, the filling material is visible in the surrounding periradicular tissues, there is no radiographic evidence of pathosis, and a normal periodontal ligament space and lamina dura are present (Rönnerman 1973).

Conclusions

The literature supports, but not conclusively, that teeth with previous root canal treatment exhibit less propensity for apical root resorption during orthodontic tooth movement. Clinical observations and recent literature that has addressed the role of neuropeptides in tooth movement, support the concept that minimal resorptive/remodelling changes occur apically in teeth that are well-cleaned, shaped, and three-dimensionally obturated. This outcome would depend on the absence of coronal leakage or other avenues for bacterial ingress. Other factors, such as specific root anatomical forms, may predispose to a greater incidence of resorption during movement.

Can endodontically treated teeth be moved orthodontically as readily as nonendodontically treated teeth?

Based on the previous discussion, endodontically treated teeth can be moved as readily and for the same

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distances as teeth with vital pulps (Huettner & Young 1955, Wickwire *et al.* 1974, Mattison *et al.* 1983, Remington *et al.* 1989, Spurrier *et al.* 1990, Hunter *et al.* 1990, Mah *et al.* 1996). This presumes that there would be no other factors that may prevent tooth movement, such as the presence of replacement resorption (ankylosis) that may occur following certain traumatic incidences or be the result of injury to the apical periodontal ligament by the root canal filling material (Andreasen 1981, Kristerson & Andreasen 1984, Andreasen & Andreasen 1994). Because there is a risk of EARR during the movement of any teeth, however, it is recommended that teeth requiring root canal treatment during orthodontic movement be initially cleaned and shaped followed by the interim placement of calcium hydroxide (Andreasen & Andreasen 1994). This should be maintained during the active phases of tooth movement, with the final canal obturation occurring upon completion of orthodontic treatment. This approach is not recommended when an already successful gutta-percha filling is in place prior to tooth movement.

Conclusions

Endodontically treated teeth can be moved orthodontically as readily as teeth with vital pulps. If teeth require root canal treatment during orthodontic movement, it is recommended that the root canals be cleaned, shaped and an interim dressing of calcium hydroxide be placed. The tooth should be sealed occlusally to prevent bacterial leakage. Canal obturation is accomplished upon the completion of orthodontic tooth movement.

What role does previous tooth trauma play in the orthodontic tooth movement of teeth with vital pulps or previous root canal treatment?

The risk, incidence and type of root resorption that can occur following all types of tooth trauma have been clearly delineated (Andreasen & Andreasen 1994). The assessment of the effects of orthodontic tooth movement on previously traumatized teeth however, has received little attention in the literature.

Rönnerman (1973) provided a report of two cases in which there were coronal fractures due to trauma. Root canal treatments were performed and both cases were treated identically during orthodontic tooth movement. However, there was a long time period in both cases between the initial trauma and the orthodontic intervention. In one case there was no evidence of root resorption, whilst in the other case the apical 3 mm of the root was resorbed, exposing the gutta-percha filling to the bone. Reformation of a normal periodontal ligament was visible on the radiograph without evidence of periradicular pathosis. It was concluded that orthodontic intervention can be considered on teeth that have been traumatized.

One of the first comprehensive studies was provided by Wickwire *et al.* (1974). Forty-five patients with 53 endodontically treated teeth, the majority of which had received traumatic injuries (crown fractures, intrusions, luxations, and avulsions) prior to orthodontic treatment, were evaluated. Approximately 50% of the teeth had been fractured also and four had been subjected to root-end resections. Orthodontic treatment times ranged from one to 36 months. Responses to movement of the traumatized teeth were considered equivalent to the teeth with vital pulps. The incidence of untoward sequelae was unremarkable, although there was some indication that the more severe the traumatic injury, the poorer the prognosis for the endodontically treated tooth in orthodontic therapy. The radiographic findings indicated that the incidence of root resorption was greater in the endodontically treated teeth when compared with an adjacent nontraumatized tooth with a vital pulp. These findings were further corroborated by Hines (1979) during the evaluation of previously avulsed or partially avulsed teeth to orthodontic movement (n = 81). All teeth were replanted following trauma with 10 having root canal treatment and 71 without the benefit of endodontic intervention. Subsequently 28 teeth required root canal treatment during orthodontic movement. Resorption of previously avulsed or partially avulsed teeth occurred more readily during and after orthodontic treatment. Likewise it was suggested that the increased susceptibility to root resorption is due to the reduced vitality of the traumatized tooth.

Zachrisson & Jacobsen (1974) evaluated the response of anterior teeth with root fractures, in varying degrees of malocclusion, to orthodontic movement. The fractures were transverse and located in the middle or apical third of the root. Clinical and radiographic assessments at the time of the accident and before and after orthodontic treatment were made. Although the sample was small (n = 4), the following conclusions were drawn. Orthodontic movement of teeth with repaired root fractures is possible, even if the fractures at the time of the accident are extensive

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with marked fragment dislocation. In cases where the repair occurs without separation of the fragments, the apical fragment may remain attached to the coronal portion throughout and following orthodontic treatment, but separation of the segments may be enhanced by orthodontic movement. It also was considered advisable that teeth with these types of fractures be observed at least 2 years before initiating orthodontic movement. A similar corroborating case report was published by Hovland *et al.* (1983) in which a maxillary central incisor with a transverse fracture at the junction of the apical and middle third of the root was evaluated 2 years post-trauma. The tooth was responsive to sensitivity testing and mobility was within normal limits. Orthodontic anterior high-pull headgear was used to correct a deep overbite and a Class II Division I malocclusion for approximately 1 year. As a follow-up, a maintenance programme with a Hawley appliance was advocated. The traumatized tooth was moved palatally and intruded during treatment. An 8-year recall shows the patient to be symptom free and in normal occlusion. Clinically, mobility was within normal limits and there was no discomfort to percussion or palpation on the central incisor. The crown was not discoloured. Radiographically the horizontal fracture line was present without evidence of resorption, but the root canal space in the apical third appeared obliterated. Cephalometric tracings revealed movement of the entire tooth as a single unit.

Guyman *et al.* (1980) created an animal model for the predictable development of ankylosis without complications, such as periradicular abscess formation. Monkey teeth were extracted, root canal treatment was performed, and the teeth were purposefully left out of the mouth for an extended period to achieve drying of the periodontal ligament. Subsequently the teeth were replanted and splinted. Clinically ankylosis was verified by lack of mobility and the presence of a percussion sound consistent with ankylosis. Radiographic findings also verified the loss of the periodontal ligament space in the presence of bone in intimate contact with dentine. The teeth were then used as orthodontic abutments for intermaxillary and premaxillary suture expansion. The teeth did not exhibit clinical or histological signs of movement through bone when the orthodontic force was applied. The presence of resorption of cementum and dentine was verified histologically, with osteoid tissue deposited over the resorbed tooth structures. These findings would imply that teeth that have been traumatized, endodontically treated, and provide evidence of clinical and radiographic ankylosis cannot be moved orthodontically.

Malmgren *et al.* (1982) addressed the frequency and degree of root resorption in traumatized incisors (complicated and uncomplicated crown fractures, concussions, subluxations and luxations) that had been moved orthodontically. Twenty-seven orthodontic patients with 55 traumatized incisors were compared to 55 consecutive patients without traumatized teeth. All controls were treated with either an edgewise appliance (n = 33) or a Begg appliance (n = 22). The degree of root resorption was scored and compared both internally in the traumatized group and with the nontraumatized group. Neither the intraindividual nor the interindividual comparisons supported the hypothesis that traumatized teeth have a greater tendency toward root resorption than nontraumatized teeth. Traumatized teeth with signs of root resorption prior to orthodontic treatment, however, may be more prone to root resorption during orthodontic tooth movement.

One of the most damaging injuries to a mature tooth and its supporting structures is an intrusion luxation. These injuries are often accompanied by comminution or fracture of the alveolar socket (Andreasen 1970, Andreasen & Andreasen 1994). Pulpal death usually occurs and the possibility of replacement resorption (ankylosis) and loss of marginal bone support is quite high. The management of these types of cases may be controversial, and therefore, it has been suggested that a decreased incidence of ankylosis may be obtained by using orthodontic extrusion (Andreasen & Andreasen 1994). Turley *et al.* (1984) examined the differences between orthodontic extrusion and observation and re-eruption following intrusive luxation injuries. Dog teeth were intentionally intruded and then subjected to one of the two previously mentioned protocols. Observations included radiographic measurements of tooth movement, clinical estimates of tooth mobility, and radiographic and histological assessments of root resorption, ankylosis, and periradicular pathosis. Root resorption, ankylosis, and pulp necrosis were common findings. If the pulp did remain vital, calcification usually occurred. Following 11–13 weeks of force activation, 10 of 12 traumatized teeth showed clinical, radiographic and histological evidence of ankylosis, irrespective of orthodontic treatment. The ankylosed teeth did not move during treatment, but the teeth used for force application actually intruded from 1.7 to 6.5 mm. The ability of the traumatized tooth to be repositioned following intrusive luxation apparently

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depended on the degree of trauma sustained, with the least traumatized being repositioned through extrusion.

The prognosis for pulp survival following an intrusive luxation injury is enhanced with teeth having immature root development (Andreasen & Andreasen 1994, Jacobs 1995). Whilst this type of injury may lend itself to normal re-eruption, there is a high risk of root resorption (58%; Andreasen & Andreasen 1991). This is due to damage to the periodontal ligament attachment and root surface. The apical area of the tooth may become rapidly moth-eaten due to the crushing injury of the intrusive force. Also susceptible is the cervical area of the tooth. If the pulp becomes infected, there may be an additional aetiological factor to enhance the resorptive process, although the contribution of a necrotic, infected pulp to a cervical resorptive response has been questioned (Tronstad 1988b, Trope *et al.* 1998). Therefore in these circumstances it may be advisable to extrude the tooth over a period of 3 to 4 weeks, keeping pace with the repair of the marginal bone (Andreasen & Andreasen 1994). If external inflammatory root resorption is observed, the pulp must be removed immediately and calcium hydroxide is placed. Likewise, it may be advisable to slow or halt the mechanical eruptive process until the resorptive process is forestalled. Drysdale *et al.* (1994) recommend that a permanent root filling be placed prior to orthodontic tooth movement where practical. Following an intrusive injury to the tooth with an immature root, this guideline may not be appropriate.

A negative aspect of dealing with an intruded tooth with immature root development is that the pulpal status is not always readily apparent or readily assessible. This is due to the fact that pulp testing is reasonably unreliable in a tooth with an open apex, whether traumatized or not. It is not uncommon for a tooth with immature root development to respond negatively to any stimulus up to 1 year or more after trauma and still not evidence radiographic signs of pulpal necrosis (Andreasen & Andreasen 1994).

Conclusions

A traumatized tooth can be moved orthodontically with minimal risk of resorption, provided the pulp has not been severely compromised (infected or necrotic). If there is evidence of pulpal demise, appropriate endodontic management is necessary prior to orthodontic treatment. If a previously traumatized tooth exhibits resorption, there is a greater chance that orthodontic tooth movement will enhance the resorptive process. If a tooth has been severely traumatized (intrusive luxation/avulsion) there may be a greater incidence of resorption, with or without root canal treatment.

Can teeth that have been managed with surgical endodontic procedures be moved orthodontically?

Whilst successful movement of teeth following surgical endodontic procedures has been observed clinically, very little has been written addressing the ramifications of the approach to treatment. Considerations would include the propensity for a greater amount of apical resorption due to the exposed dentine on the resected root face, irritation and persistent inflammation that may be caused by the root-end filling material, the adequacy of the seal achieved with the root-end filling material. Other factors to consider are the quality of the nonsurgical root canal obturation at the level of resection, the potential for exposed, contaminated dentinal tubules at the point of resection, and the potential for localized marginal periodontitis in those cases where a dehiscence or fenestration may be present. In this context the major causes for failure following surgical endodontic procedures have been identified as failure to debride and obturate thoroughly the root canal system, and the superimposition of periodontal disease in the surgical site (Rud & Andreasen 1972, Rud *et al.* 1972). Low levels of success with surgical endodontics have been reported also in the absence of a sound buccal cortical plate of bone (Hirsch *et al.* 1979, Skoglund & Persson 1985). Recently this scenario has prompted the use of guided tissue regenerative procedures to enhance surgical outcomes (Pecora *et al.* 1995, Rankow & Krasner 1996, Uchin 1996).

The earliest report dealing with the orthodontic movement of teeth with previous endodontic nonsurgical and surgical treatment is attributed to Baranowskyj (1969). In this study the rate of healing of the hard and soft alveolar tissues was assessed on teeth that had root fillings and previous periradicular surgeries, and that were subject to an early application of orthodontic intrusive forces. Histological assessment of six-week specimens (dogs – experimental and control) indicated that healing was completely delayed in the teeth with root fillings and root-end resections. There was no visible attempt at bone regeneration in the surgical defect or at formation of a new periodontal ligament or cementum. The surgical defects were filled

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with degenerating blood clots and there was evidence of attempts at organization and infiltration with endothelial buds (angiogenesis). The control group showed almost complete healing of all tissues. Histological assessment of both groups at 12 weeks indicated regeneration of bone and periodontal ligament was complete in the control and approximately two-thirds of the experimental group. The apical cementum in the experimental group was also only one-third as complete and a mild resorptive response was evident. It was concluded that the early application of orthodontic forces after surgical endodontic treatment markedly delayed the healing process and the specific cause was identified as tooth mobility and its impact on the ossifying media of the periradicular tissues.

Whilst not definitive, another report on the orthodontic movement of root-end resected teeth is attributed to Wickwire *et al.* (1974). In their endodontically treated population of 53 teeth that were moved orthodontically, four had received root-end resections. Although there was a greater incidence of root resorption with the movement of endodontically treated teeth, no mention was made as to the status of the root-end resected teeth.

Conclusions

As can be seen by the paucity of published literature and lack of information in dental texts, very little is known about the ability to move successfully teeth that have undergone periradicular surgical procedures. Likewise, little is known about the potential risks or sequelae involved in moving teeth that have had previous surgical intervention. Especially absent is the long-term prognosis of this type of treatment.

Will ongoing orthodontic treatment affect the provision and outcome of endodontic treatment?

The presence of ongoing orthodontic treatment may impact on the provision or endodontic treatment, depending on a number of factors. From a diagnostic standpoint, radiographs may reflect osseous changes that may be misinterpreted as being of pulpal origin. Likewise, the radiographic changes could be viewed as being from a vertical fracture or periodontal defect. Full metallic bands may prevent an accurate response to electrical or thermal pulp testing, in addition to obscuring decay both radiographically and clinically. Patient symptoms may be due to the tooth movement or to an inflamed or degenerating pulp, thus making a differential diagnosis very difficult, especially if there has been a history of trauma. Also, the presence of pulpal calcifications may be due to both an inflamed degenerating pulp following trauma or to orthodontic tooth movement (Stuteville 1937, Oppenheim 1942, Delivanis & Sauer 1982, Seltzer & Bender 1984).

Tooth isolation for root canal treatment may also be compromised by the presence of orthodontic bands and wires. The placement of a rubber dam in these cases usually requires some creativity and the need for additional measures to block potential avenues of leakage. Often rubber dam clamps may also be modified by grinding or bending to meet each anatomical challenge.

Endodontic coronal access openings in teeth being moved orthodontically is usually not a problem in posterior teeth. Although rarely used nowadays, the presence of full metallic bands on anterior teeth may pose a challenge as the position of the access may have to be altered, the band may have to be partially destroyed, or the band may have to be removed temporarily during endodontic treatment. If acid-etched facial brackets are used there is no problem. If there are lingually or palatally placed brackets, severe alterations in the position of the access opening are required, although straight-line access in these teeth can be achieved by creating openings down the long axis of the tooth through the incisal edge (LaTurno & Zillich 1985). With the advent of rapid debonding techniques, orthodontic bracket removal has been simplified with minimal destruction to the tooth structure and minimal time commitment for removal and replacement (Bishara & Trulove 1990, Krell *et al.* 1993)

Working length determination in teeth actively undergoing tooth movement may also be challenging in the presence of apical resorption or even just root blunting in which there is no discrete apical constriction. The extent of the apical resorption can vary widely, with intrusive forces usually demonstrating a greater loss in length (mean 2.5 mm) (Dermaut & DeMunck 1986). Apical resorption usually destroys the natural constriction of the cemento-dentinal junction, resulting in a highly irregular, three-dimensionally rough, jagged, and notched root end. The periodontal ligament space is often widened and accentuated (Remington *et al.* 1989). This will create difficulty in locating a biologically acceptable position at which to establish the working length. Whilst the extent of proximal surface root-end resorption is discernible, the degree of buccal and lingual tooth loss is distinctly ambiguous. Andreasen (1986) has shown that buccal

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or lingual resorption cannot be discerned until 20 to 40% of the root structure has been demineralized and ankylosis has occurred. Therefore, if apical resorption presents with a scalloped or uneven proximal margin radiographically, significant three-dimensional resorption has occurred, further complicating working length determination (Gutmann & Leonard 1995). Creation of an apical stop in these situations must rely on the clinician's judgement, drawing on experience, tactile sensation, and reliable diagnostic radiographic techniques. If the root end is wide open from the resorptive destruction, electronic apex locators are unreliable and of little clinical value. Therefore, the coronal-most point on the root above the resorbed apex which exhibits sound radiodensity must be identified. This position is used as the new radiographic apex and the working length is established 1.0–2.0 mm coronal to that point (Gutmann & Leonard 1995, Hovland & Dumsha 1997). In cases of extensive, irregular apical resorption the new working length can conceivably be 5.0 mm or more coronal from the original root apex. Paper points may be helpful in determination of the canal exit, if the canal can be dried of the periradicular fluid. Inflamed periradicular tissues will moisten the tip of the paper point at the level of the canal exit. Using radiographic assessment, paper point testing, and the experience of tactile sensation, a reasonable estimate of the working length can be ascertained.

Finally, canal obturation of teeth being orthodontically moved may result in fills that are beyond the confines of the tooth (Weine 1996). This is especially true when using thermally softened gutta-percha and vertical compaction techniques. It may also occur with lateral compaction because the canal walls may be quite parallel owing to the resorption of the normally narrowed and constricted root apex. In these cases, techniques of creating an apical matrix or custom fitting of a master cone may be appropriate (Pitts *et al.* 1984, Hovland & Dumsha 1997).

Conclusions

During orthodontic tooth movement, the provision of endodontic treatment may be influenced by a number of factors, including but not limited to radiographic interpretation, accuracy of pulp testing, patient signs and symptoms, tooth isolation, access to the root canal, working length determination, and apical position of the canal obturation.

How can orthodontic procedures be used in conjunction with endodontics to enhance treatment planning for tooth retention?

The prime use of orthodontic tooth movement to enhance endodontic procedures and tooth retention is in the realm of root or tooth extrusion (Ingber 1974, 1976, Simon *et al.* 1978, 1980, Delivanis *et al.* 1978, Ivey *et al.* 1980, Stern & Becker 1980, Garret 1985, Biggerstaff *et al.* 1986, Weine 1996, Lovdahl & Wade 1997). (Authors note: there are a significant number of published articles that address this subject and it would be redundant to reference all just for the sake of completeness. The reader is referred to the last two references for a more thorough treatise on this subject as it refers to endodontic relationships and the practical aspects of tooth extrusion). Common indications for this procedure include fractured tooth margins below crestal bone, deep carious margins in teeth requiring root canal treatment, resorptive perforations, postspace preparation perforations, aberrant coronal access openings, and some isolated infrabony defects.

The prime objective of tooth extrusion or forced eruption is to provide both a sound tissue margin for ultimate restoration and to create a periodontal environment (biologic width) that will be easy for the patient to maintain. The use of root extrusion, in conjunction with periodontal crown lengthening, has saved many good teeth from extraction. It is not the purpose of this paper to detail the nature of these techniques and the readers are referred to more descriptive and thorough sources (Lovdahl & Wade 1997). However, for purposes of an overview regarding this relationship, a few papers will be addressed.

Delivanis *et al.* (1978) detailed a case report where the fracture of the crown of the tooth extended 2 mm below the alveolar crest and the tooth was saved through an endodontic-orthodontic approach. Following a pulpotomy, orthodontic attachments were directly bonded to the two teeth on either side of the fractured tooth. The fractured crown received a direct bonded button placed as high gingivally as possible. A sectional archwire was fitted to the adjacent teeth and an elastic force was used to extrude the fractured tooth. Simon *et al.* (1978) indicated that orthodontic extrusion should become a routine procedure in dentistry. They also stressed that the orthodontically extruded tooth must be stabilized for 8–12 weeks prior to fabrication of a permanent post and core. Stern &

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Becker (1980) discussed orthodontic extrusion as an aesthetic alternative to surgical crown lengthening and the lowering of the alveolar crest 2–3 mm. They indicated that with an extrusive force, there was additional bone deposition lining the socket. Unlike other orthodontic procedures, in extrusion, bone resorption does not occur. Bundle bone is replaced by lamellar bone. If excessive forces are used however, significant pulpal changes or necrosis may easily result (Mostafa *et al.* 1991). They also indicated that Begg brackets and a multistrand wire allowed for three times the interbracket length whilst allowing a decrease in eruptive force of 27 times, thereby reducing concerns over necrosis or resorption. Rapid extrusion may produce limited amounts of resorption over a short time span (Malmgren *et al.* 1991), but with long-term assessments are unavailable. Following a histological assessment, Simon *et al.* (1980) indicated that extrusion of endodontically treated teeth did not present any apparent problems. They reported that the alveolar housing moves occlusally as the tooth is extruded followed by bone deposition at the alveolar crest and throughout the interradicular area. These findings corroborated those of Ingber (1974, 1976). Subsequently, additional contouring of the gingiva or osseous recontouring (crown lengthening) is usually required to optimize aesthetic results and the biologic width. Apical radiolucencies noted from the extrusion were normal by the fourth week and the PDL was normal after 7 weeks. New bone was evident at the alveolar crest, interradicular, and apical areas.

Biggerstaff *et al.* (1986) also discussed extrusion of teeth previously thought to be nonrestorable due to fractures, deep carious lesions, internal resorption, or endodontic perforations using a multidisciplinary approach of endodontics, orthodontic extrusion, repositioning of the biologic width and a definitive restoration. Using 20–30 g of eruptive force resulted in eruption with alveolar crestal new bone which, coupled with a biologic width realignment procedure, afforded superior aesthetics to crown lengthening procedures only. Furthermore, crestal bone apposition may reclaim 1-, 2-, and 3-walled periodontal defects. Similarly, periodontal implications of orthodontic tooth movement were studied by Polson *et al.* (1984) by creating intrabony periodontal angular pockets on the mesial and distal areas of incisors in rhesus monkeys. Teeth were moved through these defects, ultimately eliminating the angular defects.

Since the ultimate success of any endodontic procedure relies significantly on how well the tooth is ultimately restored, orthodontic tooth movement can be used to enhance embrasure spaces in teeth that are endodontically treated (Casullo & Matarazzo 1980, Lovdahl & Wade 1997). For example, second molars that have drifted into a distally decayed first molar can be uprighted. Molars that are resected (hemisected or root-amputated) can often benefit from enhanced embrasure spaces through the use of orthodontic movement (Gutmann & Harrison 1994).

Conclusions

Adjunctive orthodontic root extrusion and root separation are essential clinical procedures that will enhance the integrated treatment planning process of tooth retention in endodontic-orthodontic related cases.

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