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Review Article

The influence of endodontic treatment upon periodontal wound healing

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Abstract. The interrelationship between periodontal and endodontic disease has aroused much speculation, confusion, and controversy. Pulpal and periodontal problems are responsible for more than 50% of tooth mortality today. Diagnosis is often difficult since these diseases have been studied primarily as separate entities. The toxic substances of the pulp may initiate periodontal defects through canal ramifications and patent dentinal tubules, thus impairing wound healing in regenerative procedures. Although no studies exist addressing the direct effect of pulpal infection on the outcome of guided tissue regeneration (GTR) procedures, several studies do indicate that pulpal status may play a significant rôle toward the end results of GTR. This review article discusses the potential influence of endodontic treatment on the long-term outcomes of GTR. Potential pathways between the pulp and periodontal ligament, which may be responsible for the failure of the regeneration of new periodontal attachment apparatus, are explored. Examination and review of the clinical and research findings in the literature relating to perio-endo lesions are made to demonstrate that a negative influence may exist between GTR outcomes and the status of the pulp.

Key words: endodontic infection; canal ramifications; patent dentinal tubules; guided tissue regeneration/GTR

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Regeneration of lost periodontium is one of the primary goals of periodontal therapy. Much debate, speculation, and research has focused on various aspects of wound healing so that this goal can be achieved. Guided tissue regeneration (GTR) takes advantage of the healing capabilities of the periodontium. Within the last decade, regenerative procedures have become an important treatment mode in periodontics. However, the interaction between the pulp and the periodontal apparatus and their effects on wound healing has not been clearly elucidated. In a classic study on animals with healthy periodontium (Diem et al. 1974), it was reported that regeneration of lost supporting tissues could occur on root surfaces of pulpless teeth denuded of cementum with surgically created facial defects. However, definitive conclusions could not be made regarding the regenerative potential of the attachment apparatus of endodontically-treated teeth and whether or not this potential is affected by prolonged exposure of the root surface to the oral environment. In this study, gutta-percha and Kerr sealer™ were used as filling materials. In another animal study, Andreasen (1981a) studied the effect of pulp extirpation or root canal treatment on periodontal healing after replantation of permanent incisors. Gutta-percha/Kerr sealer™ root filling consistently increased the frequency of surface and replacement resorption compared with nonextirpation or just pulp extirpation. Furthermore, replacement resorption was located closer to the apical area in gutta-percha root-filled teeth compared with teeth that were not extirpated. It was suggested that possibly the eugenol used in Kerr sealer™ may damage the periodontal ligament (PDL) and increase the frequency of surface and replacement resorption. This damage has been corroborated in experiments on rats in which root fillings with zinc oxide-eugenol elicited cementum, PDL necrosis, and small areas of ankylosis in the apical zone (Erausquin et al. 1966, Erausquin & Muruzabal. 1967, Erausquin & Muruzabal 1968, Erausquin & Devoto 1970). Other studies reported that healing did not occur against the exposed dentin of a denuded root surface following periodontal treatment of non-vital teeth. A tooth was considered non-vital if it had a root canal filling, an old non-bleeding pulp exposure with a periapical radiolucency, or a periapical ra-

diolucency based on negative results with the electric pulp tester (Morris 1957).

Furthermore, it has been suggested that periodontal healing will be influenced by endodontic pathosis. By determining the influence of endodontically-treated teeth on GTR, one may better predict long-term clinical results of regenerative procedures on pulpless teeth. The purposes of this paper are: (1) to discuss the influence of endodontic pathosis on periodontal healing; (2) to explain the differences in outcomes between surgical and non-surgical treatment; and (3) to explore the role of endodontic therapy in GTR procedures.

Association between Pulp and Periodontal Tissues

The communication between pulp and periodontal tissues has been long since demonstrated and several possible channels have been suggested (Simon 1984). It has been speculated that pathological changes may be transmitted between the pulp and the periodontal tissues. Bergenholtz & Lindhe (1978) pointed out that 57% of periodontally-involved teeth exhibited pathological alterations of the pulp. In another study, Bender & Seltzer (1972) reviewed 178 teeth histologically to determine the effects of periodontal disease on the pulp. Among the group, consisting of 57 teeth with periodontal disease but without caries or fillings, it was reported that 79% of teeth showed pathological changes within the pulp. Contrary to these reports, several researchers have stated that there is no definite relationship between periodontal disease and the pulpal status (Fish & MacLean 1936, Sauerwein 1956, Mazur & Massler 1964, Czarnecki & Schilder 1979, Dongari & Lambrianidis 1988).

Recently, many studies have demonstrated significant microbiological similarities between infected root canals and advanced periodontitis (Tanner et al. 1982, Kipioti et al. 1984, Trope et al. 1988, Kerekes & Olsen 1990). Kobayashi et al. (1990) reported that the predominant obligate anaerobes common to both regions were *Streptococcus, Peptostreptococcus, Eubacterium, Bacteroides*, and *Fusobacterium*. Besides these microbial findings, the similarity in the composition of cellular infiltrates also suggests the existence of a communication between the pulp and periodontal tissues (Bergenholtz et al. 1983). Thus, these findings infer that cross-contamination between the pulp and the periodontal tissues is possible.

Although disease transmission from the pulp to the periodontal tissue is possible, the influence of periodontal disease on pulpal status remains controversial (Bergenholtz & Lindhe 1978, Czarnecki & Schilder 1979, Dongari & Lambrianidis 1988, Tronstad & Langeland 1971, Langeland et al. 1974, Harrington 1979). When pathologic changes occur in the pulp tissue as a result of periodontal disease, the pulp usually does not degenerate as long as the main canal is not involved (Langeland et al. 1974). Seltzer et al. (1963) and Lantelme et al. (1976) reported that root canals were narrowed by the deposition of large quantities of reparative dentin along the dentinal walls of the periodontally-involved teeth. The pulpal response appears to be reparative, involving rapid deposition of dentin with cellular inclusions, or resorptive, followed by a reparative response rather than an inflammatory response (Sinai & Soltanoff 1973). According to Bergenholtz & Lindhe (1978), the possible reasons for the absence of additional pulpal reactions to plaque formation on scaled dentin may be that the dentinal tubules in the external regions of the dentin are more widely spaced, and the diameter of the tubules is narrower in the periphery than at the pulp surface. Adriaens et al. (1988) suggested that the odontoblastic processes extend into the inner third of the dentinal tubules in mature teeth and acted as mechanical barriers for the microorganisms migrating towards the dental pulp.

Since the change in the periodontal structure is of an inflammatory nature, there is general agreement that pulpal disease can initiate and/or maintain periodontal disease (Czarnecki & Schilder 1979). Blomlöf et al. (1988) evaluated the effects of various pulpal conditions and treatment measures on cells and tissue reactions in the marginal hard/soft tissue interface. The results indicated that pulpal conditions and endodontic dressings influence marginal healing and repair as long as patent dentinal tubules are present. It was suggested that permanent endodontic treatment with an inert material should be performed before the protective cementum layer is removed. This is in agreement with Harrington (1979), who reported that periodontal reattachment to the root surface can be inhibited if definitive periodontal therapy is undertaken prior to root canal therapy.

The Pathways of Endodontic Infection to Periodontal Tissues

Pulpal and periodontal tissues are closely related and the disease transmission between these two lesions has been demonstrated. The intercommunication channels are neural (reflex) pathways, lateral canals, dentinal tubules, Sharpey's fibers, apical foramina, and common vasculolymphatic drainage pathways (Simon 1984). The most intimate and demonstratable relationship between the two tissues is via the vascular system, as anatomically demonstrated by the presence of the apical foramen, lateral (accessory) canals and dentinal tubules. These patent canals may serve as potential routes of inflammatory interchange. The possible mechanisms are as follows:

Apical foramina, lateral, and accessory canals

The blood supply is derived from the inferior and superior alveolar arteries and reaches the PDL from three sources: apical vessels, penetrating vessels from the alveolar bone, and anastomosing vessels from the gingiva (Cohen 1960). These vessels run closer to the bone than to the cementum (Carranza et al. 1966) and the blood supply increases from the incisors to the molars. In all single-rooted teeth the blood supply of the individual PDL is greatest in the gingival third and least in the middle third. In multi-rooted teeth the supply is similar in the middle and apical thirds (Birn 1966). Langeland et al. (1974) reported that the total histological disintegration of pulp occurs only when all main apical foramina are infected by bacterial plaque. Therefore, apical foramina are the major pathways of disease transmission between the infected root canals and periodontal tissues. This is in agreement with Walton & Garnick (1986), who stated that the passage of irritants from pulp was more likely from the periapex through the inter-radicular medullary bone. Despite apical foramina, lateral and accessory canals are other anatomical pathways of concern. Although these canal ramifications are most frequently found in the apical third of the root (17.0%) and least at the base of the root (1.6%),

the body of the root has the second highest percentage (8.8%) (De Deus 1975). Another important region is the molar furcation area since most regenerative procedures are performed at these locations. According to early studies in molars, 59% patent lateral or accessory canals were present in the coronal and middle thirds of the roots (Lowman et al. 1973), and more than one canal was found at the trifurcation and bifurcation areas (Burch & Hulen 1974). In addition, Gutmann (1978) reported that 28.4% of the furcation region demonstrated accessory canals. Therefore, once the pulp is infected, toxic substances could potentially impair the healing or destroy the periodontal structures via these canal ramifications. This may partially explain why the unpredictable GTR results were noted in these bi- or tri-furcation regions.

Changes in cementum

Cementum is the calcified outer covering of the anatomical root. The cervical two-thirds of the root is covered by acellular cementum. Sharpey's fibers are the major component of acellular cementum and are completely calcified except in a 10 to 50 micron wide zone near the cemento-dentinal junction (Selvig 1965). In young animals, cementum is very permeable and permits the diffusion of dyes from the pulp canal as well as from the external root surfaces. The high degree of permeability was demonstrated by Wasserman et al. (1941) who reported that devitalized teeth can still absorb one-tenth the amount of radioactive phosphorus through the cementum as compared to vital teeth. It may be suggested that intact root cementum is not an effective barrier to prevent peneration.

Cementum resorption is very common. Based on a histologic study of 15 human dentitions containing 261 teeth and their supporting tissues, a classic study by Henry & Weinmann (1951) reported that cementum resorption occurred in 90.5% of the sample teeth. Among these, 76.8% occurred in apical third, 19.2% in the middle third, and 4.0% in the gingival third. When cementum resorption occurs, the dentinal tubules may become exposed. The susceptibility of cervical resorption has been attributed to the absence of either uncalcified precementum or reduced enamel epithelium (Southman 1967). Interestingly, the uncalcified surface layer of precementum was once considered to be the natural barrier to excessive apical migration of the junctional epithelium (Gottlieb 1946). Therefore, the precementum may play a protective role against marginal inflammation.

In contrast to Wasserman et al. (1941), Stallard (1968) reported that intact root cementum acted as a barrier to an inward penetration of a dye solution (2% aqueous solution of trypan blue). The experiment was performed on periodontally healthy teeth; thus, the observation may not be valid for diseased cementum of teeth with periodontitis. In addition, different methodologies and materials were used in these two studies to evaluate molecular penetration into cementum. When periodontal disease caused exposure of the root surface, cementum alterations occurred including structural, chemical, and cytotoxic changes. As suggested by Armitage & Christie (1973a), toxic substances could enter exposed cementum through voids formed after the decomposition of Sharpey's fibers.

In the dentin and cementum adjacent to periodontal pockets and being most numerous near the dentino-cemental junction, pathologic granules were found by Bass (1951) in decalcified specimens as refractile brownish granules by transmitted light and as bright white granules by incident light at about a 45° to 50° angle with dark background. Furthermore, with electron microscopic examination, Armitage & Christie (1973b) found vacuole-like formations of 15~25 μm under the cemental surface extending 5~15 μm into the dentin. Interestingly, they also found one case in which the pathologic granules were under an unexposed root surface. In addition, Armitage et al. (1983) reported cemental granules in unexposed cementum from carious teeth with microbe-laden root canals. Noncarious teeth with intact pulpal tissue were devoid of this cemental change. It was then suggested that the microbial byproducts may denature collagen, thereby giving rise to the structural change known as a pathological granule.

Aside from this pathologic change that may influence the integrity of cementum, the disruption of the intermediate cementum poses another important concern. Intermediate cementum is a highly calcified and ill-defined amorphous zone near the cemento-dentinal junction of certain teeth and appears to contain cellular remnants of Hertwig's root sheath embedded in calcified ground substance. This layer was seen predominately on roots of molars and premolars and rarely observed on incisors or deciduous teeth (Mostehy & Stallard 1968, Lester 1969). Based on immunohistochemical studies, the intermediate cementum is actually an enameloid layer which is produced by Hertwig's epithelial root sheath (Lindskog 1982a, Lindskog 1982b, Lindskog & Hammarström 1982). The potential functions of this layer are suggested as follows: (1) a permeability barrier between cementum and dentin; (2) a precursor for cementogenesis in root development; and (3) a precursor for cementogenesis in wound healing (Harrison & Roda 1995). Therefore, it acts as an effective barrier between an infected pulp and the PDL for the prevention of inflammatory resorption (Andreasen 1981b, Hammarström et al. 1986). When it has been damaged, the noxious contents of the infected pulp pass through the dentin and stimulate more aggressive and rapid root resorption. However, the potential function of intermediate cementum as a precursor to reparative cementogenesis is highly speculative. Further investigation is needed to elucidate the role of intermediate cementum in wound healing.

Exposure of dentinal tubules

Approximately 15,000 dentinal tubules per square millimeter exist on the external root surface in the area of the cemento-enamel junction (Harrington 1979). Furthermore, there are 9,000~24,000 /mm^2 of odontoblastic processes located at the dentino-enamel junction, and there are 40,000~70,000 / mm^2 at the pulpal border (Tronstad & Langeland 1971). Dentinal tubules can be exposed by the congenital absence of cementum in the region of the cemento-enamel junction in approximately 5~10% of the population (Noyes et al. 1938), from traumatic injuries of the root cementum, from root planing procedures (Dongari & Lambrianidis 1988), and via root resorption.

The Impact of Endodontic Infection on Periodontal Wound Healing

Root resorption

In dental trauma, at least three varieties of resorption have been recognized: surface resorption, inflammatory resorp-

tion, and replacement resorption (Andreasen & Hjørting-Hansen 1966). These types of root resorption are not specific to acute trauma and may also occur after orthodontic, periodontal, pedodontic and endodontic treatment procedures (Andreasen 1985).

It has been proven that root resorption occurs in human teeth afflicted with periodontitis (Bergenholtz & Lindhe 1978). Although deep root resorption has not been observed, some surface resorption has been noted following bone grafting procedures (Bowers et al. 1989). In some cases, the resorption extended into dentin. If the canal system contains necrotic tissue or an infected leukocytic zone which harbors bacteria, inflammatory resorption may occur through dentinal tubules. If the root canal contains normal or inflamed pulp tissue, repair of the resorptive cavity could take place with reposition of new cementum (Andreasen 1981c).

Although it is not possible to completely remove all cementum during scaling and root planing (Nishimine & O'Leary 1979, Cole et al. 1980), the dentin can still be exposed to the pocket environment in areas where cementum has been removed. Inflammation in the periodontal pocket is thought to be the primary stimulus for root resorption, not the mere exposure of the dentinal surface (Lindskog et al. 1988a).

Lövdal et al. (1961) stated that an endodontic infection alone could not initiate the development of marginal gingival lesions. It was thought to be the dental plaque that evoked an inflammatory response in the gingival pocket. Ehnevid et al. (1993b) proposed that if given enough time, an untreated root-canal infection, either by itself or in conjunction with a plaque-induced deep marginal periodontal pocket, may maintain an inflammatory response within the connective tissues of the marginal gingiva. This supports the findings of Blomlöf et al. (1992). Therefore, the major consequence of endodontic infection to the periodontal structures appears to be persistent and chronic inflammation. Even though Diem et al. (1974) did not find an apparent relationship between changes in crevicular depth and the status of the pulp, recent studies have suggested that untreated endodontic infection may influence periodontal healing, leading to a higher risk of attachment loss (Jansson et al. 1993), an increased epithelial downgrowth along denuded dentin surfaces with marginal communication (Jansson et al. 1995), a reduced mean pocket depth reduction with non-surgical periodontal treatment (Ehnevid et al. 1993b), and the formation of a wider periodontal space with healing via a long junctional epithelium (Ehnevid et al. 1994).

Bacterial penetration into dentinal tubules may also occur. Adriaens et al. (1988) concluded that most bacteria were located in the outer 300 μm of the dentinal tubules. The infected dentinal tubule and lacuna could act as reservoirs from which bacterial recolonization of treated root surfaces may arise. Occasionally microorganisms can still be detected in root canals following the completion of endodontic therapy (Nair et al. 1990, Sen et al. 1995). Thus, the shaping and cleaning of a canal system followed by a permanent endodontic filling may not necessarily eliminate or prevent the bacterial recolonization of the dentinal tubuli (Lengheden et al. 1990). It has even been suggested that complete endodontic treatment may only suppress inflammatory resorption (Andreasen 1981a). In the majority of cases, a proper root canal treatment allows the remaining bacteria in the dentinal tubules to be inactived or insufficiently maintained to cause pathology. However, these bacteria may get nutrition from the periodontal environment once dentinal tubules are patent (Peters et al. 1995).

In many instances, it should be noted that the histopathological status of the pulp is not clinically detectable following conventional diagnostic testing. A total of 153 teeth in 90 subjects were studied by Hirsch et al. (1989). Seventy-seven teeth responded in the normal range to pulp testing; however, 52% of these had no recoverable tissue from their root canal systems upon endodontic opening. In a study by Petersson et al. (1986), endodontic treatment needs of 610 endodontically-treated teeth were grouped into three categories according to different diagnostic evaluations, including detection via radiographs, during operative procedures, and during treatment planning for prosthetics. It was found that 61% of teeth requiring endodontic therapy were devoid of periapical changes on radiographs. Thus the histological status of the pulp could further complicate the process of periodontal healing.

Diseased pulps and their byproducts, using the dentinal tubules as conduits to the periodontium, can initiate an inflammatory reaction on the periodontium. Clastic activity is enhanced on the osseous and dentinal surfaces of the periodontal apparatus (Sterrett 1986). Aukhil et al. (1986) reported that granulation tissue from the bone also has a high resorptive potential for inducing root resorption. The ability of gingival connective tissue to induce root resorption is either less or delayed when compared to the connective tissue derived from bone. Interestingly, the protective downgrowth of the long junctional epithelium will prevent the occurrence of resorption on the damaged root surface following various surgical and non-surgical periodontal procedures (Andreasen 1985). It is reasonable to suggest that if the downgrowth of epithelium is prevented through the use of GTR, root resorption may become a problem.

Besides epithelial downgrowth, a viable PDL may also protect against root resorption. A positive and highly significant correlation between cell damage in the cementoblast layer and the presence of surface, inflammatory, and replacement resorption was found (Andreasen 1981c). Replacement resorption was associated with causing the greatest loss of viable cementoblasts per unit root surface length. In a study, evaluating the effect of periodontal healing, Andreasen & Kristerson (1981) found that removal of the PDL before replantation of mature permanent incisors initiated a rapid osteogenesis in the alveolus, causing ankylosis. However, if the damaged area was placed next to a zone on the root surface with a viable PDL, a later resorptive process ensued and the ankylosis area was gradually resorbed. It has been suggested that the remnants of the epithelial root sheath in the PDL may also play a significant role in guarding against resorption and ankylotic fusion between alveolar bone and tooth (Orban 1952, Löe & Waerhaug 1961, Spouge 1980).

Apical migration of epithelial cells

The epithelial rests of Malassez have been the subject of interest from the earliest days of periodontal research. Based on ultrastructural studies, Orban (1953) suggested the possibility that the epithelial rests of Malassez have an endocrine function. However, in a later

study by Valderhaug & Nylen (1966), based on ultrastructural morphology and histochemistry, the epithelial rests were found to exist as groups of epithelial cells surrounded by an outer argyrophilic limiting membrane that resembled the epithelial cells observed in the epidermis and oral mucous membranes. Thus, one may conclude that the epithelial rests of Malassez have little or nothing in common with secretory cells. However, the role of epithelial cells within the PDL still remains ill-defined.

In a more recent study by Spouge (1980), the possible characteristics of the rests of Malassez in periodontal health and disease were discussed. It was concluded that: (1) the epithelial network is continuous and covers the whole root; (2) the epithelial rests of Malassez have been shown to be continuous with the apical border of the attachment epithelium (junctional epithelium); (3) the rests may play a role in the maintenance of the PDL; (4) the rests may undergo reactive proliferation in response to an adjacent focus of infection. Unlike epithelial cells, they do not migrate over the surface of a healing wound but in fact move through the wound, thus separating the clot from the underlying bed of granulation tissue (Florey 1970). These conclusions have been supported by Lindskog et al. (1988b), Berg et al. (1990), and Mackenzie (1988). Furthermore, it has been speculated that through proliferation and elongation of the marginal pocket epithelium, the epithelial rests can form cystic linings in response to inflammation.

The Rôle of Root Canal Infection in Periodontal Healing

Contrasting effects of endodontic infection on surgical and non-surgical treatment

Although it has been reported that endodontic infection may influence periodontal healing, different outcomes may occur between surgical and non-surgical treatment. Following periodontal therapy, a soft and hard tissue interface develops along with a long junctional epithelium. In a three-part human study using DFDBA (Bowers et al. 1989a, b, c), it was reported that non-submerged, non-grafted sites healed with a long junctional epithelium. The comparison of different types of periodontal treatment has also been well documented in longitudinal studies (Hill et al. 1981, Pihlstrom et al. 1983, Ramfjord et al. 1987, Becker et al. 1988). When using pocket depth and attachment level as measurements, it was suggested that there were no significant differences between surgical and non-surgical treatment in the long-term, but different outcomes may exist with short-term results.

Recently, Ehnevid et al. (1993a) suggested that following scaling and root planing, periodontal healing was significantly impaired over time in teeth with periapical pathology. However, periapical pathosis had no significant effect for surgically-treated teeth. In another study involving non-surgical treatment, Ehnevid et al. (1993b) stated that the mean pocket depth reduction was significantly reduced in teeth with periapical pathology when compared with teeth without periapical pathosis. However, no significant correlation between periapical pathology and mean pocket depth reduction in surgical treatment could be found. A possible explanation for the varying results with the respect to periapical pathosis affecting periodontal healing with surgical or non-surgical treatment is proposed as follows: (1) scaling and root planing was more difficult to perform adequately in teeth with more advanced loss of radiographic attachment; (2) osteoplasty probably resulted in less long junctional epithelium formation; and (3) mean pocket depth following periodontal surgery with osteoplasty was reduced to a level such that less root surfaces were covered by marginal soft tissue. Consequently, greater pocket depth reduction, less attachment gain, and more gingival recession following surgical treatment led to less soft tissue coverage with less patent dentinal tubules and canal ramifications exposed to the soft tissue. In turn, this results in less toxic substances migrating into the marginal soft tissues. Therefore, periodontal surgical procedures may decrease the negative influence that endodontically-treated teeth may have on periodontal healing .

Endodontic infection and guided tissue regeneration

The objective of GTR is to gain new clinical attachment, improve bone level, and minimize postoperative recession (Polson 1994). By placing subgingival barriers, GTR takes advantage of the behavior of different periodontal tissues during wound healing (Greenstein 1993). Although GTR procedures have become the standard of treatment, the results still remain equivocal. In a study using 40 intrabony defects in 23 human subjects, Cortellini et al. (1993) reported that a probing attachment level gain of 2 mm or more was detected in almost 90% of treated sites. None of the sites lost attachment and residual probing depths were 3 mm or shallower in 95% of the sites. On the contrary, others reported less favorable results. Selvig et al. (1993) observed that the overall healing pattern following barrier membrane-supported flap surgery appeared to be similar to that generally observed for conventional reconstructive flap surgery. In general, GTR results in a 1.6 to 3.5 mm gain of clinical attachment, a 2.6 to 4.0 mm probing depth reduction, and a 0.9 to 1.6 mm recession (Polson 1994).

According to Tonetti et al. (1993), the amount of regenerated tissue under the membrane is dependent upon the baseline depth of the intrabony component. Therefore, it has been proposed that the deeper the defect, the greater amount of tissue regeneration. When comparing probing depth reduction, gain of clinical attachment, and gingival recession between conventional surgery and GTR, GTR procedures have resulted in more soft tissue coverage than conventional surgery. With deeper baseline depth and more soft tissue coverage, it may suggest that more patent dentinal tubules and canal ramifications are exposed under subgingival barriers. In turn, more toxic substances could exist in marginal soft tissues of teeth with periapical pathosis. Therefore, it is reasonable to propose that endodontic infection may play a role in the end results of GTR procedures. Unfortunately, minimal information exists in connection with status of the root canal and its influence on GTR. Therefore, it is critical that future studies focus on the relationship between endodontically-treated teeth and GTR.

Conclusions

Based on previous studies, it seems reasonable to believe that, in non-surgical treatment, infectious materials in the canal system may influence periodontal healing through canal ramifications and patent dentinal tubules. However, in surgical treatment, due to greater pocket depth reduction, more attachment loss, and more gingival re-

cession in the short term, the infected pulp or endodontically-treated tooth may not impair the healing process. Although one may speculate that endodontic treatment may affect the outcomes of GTR procedures, there is still no clear evidence in the literature. Therefore, further carefully designed, well-documented and controlled studies are necessary to determine to what extent, if any, the influence that the pulpal and periradicular status of a tooth may have on the outcomes of GTR.

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Zusammenfassung

Der Einfluß endodontischer Behandlung auf parodontale Wundheilung

Der wechselseitige Zusammenhang zwischen den Erkrankungen des Parodonts und Endodonts hat zu vielen Vermutungen, zu Verworrenheit und umstrittenen Behauptungen Anlaß gegeben. Pulpale und parodontale Probleme sind für mehr als 50% der heutigen Zahnmortalität verantwortlich. Da diese Erkrankungen in erster Linie als gesonderte Einheiten studiert worden sind, ist die Diagnose oft schwierig. Durch Ramifikationen des Wurzelkanals und durchgängige Dentintubuli können toxische Substanzen der Pulpa parodontale Defekte einleiten und somit die Wundheilung bei reparativen Prozessen beeinträchtigen. Obwohl keine Untersuchungen vorliegen, die sich unmittelbar mit dem Einfluß der Pulpainfektion auf das Ergebnis der gesteuerten Geweberegeneration (GTR) befassen, lasse jedoch einige Studien erkennen, daß der Zustand der Pulpa eine bedeutende Rolle für die Endergebnisse der GTR-Behandlung spielen könnte. Die hier vorliegende Übersicht erörtert den denkbaren Einfluß endodontischer Behandlung auf die langfristigen Ergebnisse der GTR. Mögliche Leitwege zwischen Pulpa und Desmodont werden untersucht, die für Fehlschläge bei der Regeneration neu anzulegender parodontaler Attachmentstrukturen verantwortlich sein könnten. Sichtung und Besprechung im Schrifttum beschriebener, mit par- und endodontalen Läsionen zusammenhängender klinscher Befunde und Untersuchungsergebnisse macht deutlich, daß zwischen den Ergebnissen der GTR und dem Zustand der Pulpa eine negative Beeinflussung vorliegen könnte.

Résumé

Influence du traitement endodontique sur la guérison parodontale

La relation entre maladie parodontale et endodontique a déjà fait l'objet de beaucoup de discussions, spéculations, confusions et controverses. Les problèmes endo-parodontaux sont actuellement responsables de plus de 50% de la mortalité dentaire. Le diagnostic est souvent difficile puisque ces maladies ont été étudiées au départ en tant qu'entités séparées. Les substances toxiques de la pulpe peuvent provoquer des lésions parodontales par les ramifications canalaires et les tubules dentinaires, mettant donc en danger la guérison lors des procédés de régénération. Bien qu'aucune étude n'existe en ce qui concerne l'effet direct de l'infection pulpaire sur le résultat des procédés de régénération tissulaire guidée (GTR), différentes études indiquent que l'état pulpaire aurait une influence significative envers les résultats de GTR. Cette article de revue discute l'influence potentielle du traitement endodontique sur les résultats à long terme de GTR. Les voies potentielles entre la pulpe et le ligament parodontal qui peuvent être responsables de l'échec de la régénération d'un nouvel appareil d'attache parodontal sont explorées. L'examen et la revue des découvertes cliniques et de recherche dans la litérature à propos des lésions endoparo démontrent l'influence négative qui peut exister entre les résultats GTR et la pulpe.

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