The potential association between smoking and endodontic disease

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Abstract

Duncan HF, Pitt Ford TR. The potential association between smoking and endodontic disease. *International Endodontic Journal*, **39**, 843–854, 2006.

The aim of this review was to analyse the literature to assess the possibility of an association between smoking and endodontic disease and the prognosis of endodontically treated teeth. The review of the prognosis of endodontically treated teeth involved taking account of any potential associations with smoking and endodontic disease and marginal periodontitis, and smoking and prosthodontic outcomes. In addition, the role of smoking in implant failure and surgical wound healing was analysed with a view to drawing parallels regarding the possible implications of smoking on the outcome of surgical

Introduction

'Prognosis' is defined as a prediction of the course of a disease and comes from the Greek word πρόγνωση (prognostikos) which combines pro (before) and gnosis (knowing). In endodontics accurate prediction of prognosis requires not only correct assessment and treatment but also an understanding of the potential effects of extrinsic factors on the eventual outcome.

Smoking appears to be one of the most significant prognostic factors in the progression of marginal periodontitis (Ismail *et al.* 1990, Grossi *et al.* 1994, Beck *et al.* 1995), and is also implicated as a risk factor for oral cancer (Warnakulasuriya *et al.* 2005), oral endodontics. A MEDLINE and Cochrane library search including smoking and various endodontic keyword searches identified three papers which discussed the variables, and did not just mention them separately in the text. The literature demonstrates a paucity of evidence relating smoking with endodontic disease and prognosis, but nevertheless presents evidence of a possible influence on the prognosis of endodontically treated teeth in smokers and a likely increase in surgical complications. The possible merits of a smoking cessation protocol prior to surgical endodontics are also discussed.

Keywords: apical periodontitis, endodontics, prognosis, smoking, surgical endodontics.

Received 9 November 2005; accepted 6 March 2006

mucosal lesions, coronal and root caries (Winn 2001), as well as affecting dental implant integration (Bain & Moy 1993, Lambert *et al.* 2000, Wallace 2000) and surgical healing (Jones & Triplett 1992, Scabbia *et al.* 2001). There is a paucity of evidence linking smoking with apical periodontitis (Kirkevang & Wenzel 2003, Bergström *et al.* 2004).

Approximately 25% of the British adult population smoke cigarettes, and although this figure has declined since the 1970s smoking is now comparatively more common in young adults than any other age group (General Household Survey 2004). Men are more lightly to smoke than females and in addition are likely to be heavy smokers. There has been a sharp increase in the use of hand-rolled cigarettes in the UK in recent years. When considering any effect that smoking may have on prognosis, data should provide sensible and coherent answers to the questions commonly asked by the profession and patients.

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Therefore, the aim of this review was to assess any potential association between smoking and endodontic disease and the prognosis of endodontically treated teeth by conducting a database search using MEDLINE and the Cochrane Library. In addition, a potential association between smoking and the prognosis of endodontically treated teeth was studied by investigating and extrapolating evidence from other areas of dentistry. The review analysed the literature with relation to smoking and oral health and noted the variability of methods in some of the studies quoted. However, as there was a lack of evidence in some areas a systematic review of the literature (in its accepted sense) was not possible.

Review

Endodontic considerations

There is scant reference in the literature to any possible association between smoking and apical periodontitis or any direct interaction with endodontically treated teeth. In fact when medical databases (MEDLINE and the Cochrane Library) were used with numerous keyword searches including smoking or tobacco (Table 1) there is only a link to a small number of recently published papers (Kirkevang & Wenzel 2003, Bergström *et al.* 2004, Marending *et al.* 2005). When completing this search, studies were excluded that made no attempt to link smoking and endodontics or were only mentioned separately in the paper. Searches were carried out in English. Due to the paucity of information on the subject any study that discussed the search variables was included for analysis.

Current evidence would indicate that smoking is a significant risk factor in inflammation of the marginal periodontium (Johnson & Hill 2004, Labriola *et al.* 2005) and therefore it may be hypothesized that it would have a similar effect on the apical periodontium.

Pulpotomy

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Vital pulp therapy, including pulpotomy is the treatment of choice for primary teeth, immature permanent teeth and in traumatic exposures (Schröder 1972, Pitt Ford & Shabahang 2002). The importance of smoking on pulpotomy is open to conjecture as it is only legal to smoke over 16 years of age in the UK, and therefore the majority of pulpotomies will be carried out in nonsmoking patients. However, traumatic injuries can occur at any age and although this may not be a large group in terms of global endodontics, it is prudent to Table 1 Endodontic MEDLINE search strategy (three articles)

	Search history in	Results
Keyword	combination with	(no. articles)
Smoking	Endodontically treated teeth	4
Tobacco		1
Smoking	Endodontics	6
Tobacco		3
Smoking	Root canal treatment	3
Tobacco		1
Smoking	Root canal therapy	8
Tobacco		2
Smoking	Prognosis of endodontically	0
Tobacco	treated teeth	0
Smoking	Prognosis of root canal treated teeth	1
Tobacco		0
Smoking	Apical periodontitis	6
Tobacco		2
Smoking	Dental periapical condition	1
Tobacco		1
Smoking	Periapical disease	8
Tobacco		2
Smoking	Periapical pathology	8
Tobacco		2
Smoking	Periapical pathosis	0
Tobacco		0
Smoking	Apicectomy	0
Tobacco		0
Smoking	Apicoectomy	1
Tobacco		0
Smoking	Surgical endodontics	1
Tobacco		2
Smoking	Root end surgery	3
Tobacco		0
Smoking	Root end resection	0
Tobacco		0
Smoking	Endodontic surgery	0
Tobacco		0
Smoking	Periradicular surgery	1
Tobacco		0

consider if there is a potential influence of smoking on pulp tissue.

In a preliminary study (Awawdeh *et al.* 2002), 46 samples of pulp tissue were removed from extracted or endodontically treated painful teeth and 20 from clinically healthy teeth that were extracted for orthodontic reasons. These pulps were examined for three neuropeptides, substance P (SP), neurokinin A (NKA) and calcitonin-gene related-peptide (CRGP). This group of neuropeptides are linked to significant changes in blood flow inflammation and pulpal pain. It was found that NKA, SP and CGRP were present in much higher concentrations in painful teeth than nonpainful teeth and that CGRP was significantly higher in the pulps of smokers compared with nonsmokers. Cigarette-induced bronchospasm has already been shown to occur as a result of nicotine stimulating CGRP release by C fibres in the lungs (Hong et al. 1995). The increase in pulpal CRGP concentration in smokers suggests that this neuropeptide may play an important role in the understanding of pulpal inflammation in smokers. It was concluded it seemed probable that nicotine causes release of CGRP from afferent nerves in human pulp tissue (Awawdeh et al. 2002). It should be noted that the control group was of a more defined age range than the experimental group and was likely to be more dentally motivated (orthodontic extractions) than the experimental group (emergency patients). The study did however limit other possible confounding factors such as previous treatment, centrally acting drugs or relevant medical conditions. This research is preliminary and therefore the clinical implications of this laboratory-based work are unknown; further work in this area is awaited before any substantive comments on prognosis can be made.

Apical periodontitis

Until recently there had been no suggestion of a possible link between smoking and apical periodontitis or endodontics in general. The first mention of a link was published in 2003 in a radiological study from Denmark in which 613 random individuals aged 20-60 years were contacted and asked to attend for radiographs in order to assess risk factors associated with apical periodontitis (Kirkevang & Wenzel 2003). The periapical radiographs were assessed using the periapical index (Ørstavik et al. 1986) and it was noted whether apical periodontitis was present or not. The most important indicator of any tooth having apical periodontitis was radiographic evidence of a root canal filling. Lack of dental attendance, inadequate coronal fillings and in addition smoking were shown to be statistically associated with apical periodontitis which was the first time that the link with smoking had been made in the dental literature. However, the association was weak and the attendance rate in the study was only 51% a factor that may introduce selection bias in the results. In addition, there was no clinical examination carried out and only radiographs were used to assess the presence of apical periodontitis. It is recognized that radiographic evaluation is a rather insensitive method of assessment as apical inflammation is often present in the absence of radiological signs. This has been demonstrated in histological evaluation of upper maxillary teeth (Brynolf 1967). Another potentially important factor to consider is that the study was cross-sectional, and there remained the possibility that any significance could be attributed to the delayed bony healing in smokers compared with nonsmokers. In several orthopaedic studies bony healing was slower in smokers than in nonsmokers (Kwiatkowski et al. 1996, Haverstock & Mandracchia 1998, Castillo et al. 2005). Kirkevang & Wenzel (2003) suggested this delay in healing may result in an over-representation of disease in the smoking group in their study. Confidence in assuming a positive association never mind a causeeffect relationship from a cross-sectional study is weakest followed by case-control and observational cohort studies. Smoking studies are in addition prone to various types of bias that arise from the fact that subjects essentially 'self-select' their own exposure groups (Caplan 2004). Randomized prospective studies are difficult to organize as it is not possible to assign subjects to a smoking or nonsmoking group. The link between apical periodontitis and smoking was further studied by Bergström et al. (2004) who in another cross-sectional study retrospectively examined 247 intra-oral radiographs of smokers, nonsmokers and former smokers and compared for incidence of apical disease. Although the mean number of periapical lesions was 6% in smokers. 4% in former smokers and 3% in nonsmokers, the association between smoking and periapical lesions was not significant after controlling for age. It was concluded that their study offered no evidence of a link between apical periodontitis and smoking. The above studies were similar in design in that they were both radiographic studies and did not involve a clinical examination. The second study (Bergström et al. 2004) did not examine a random sample of a general population as had the initial study (Kirkevang & Wenzel 2003), but rather examined a subpopulation of Swedish musicians, who may differ from the general population. It is difficult to control for confounding factors in cross-sectional studies particularly when any influence on apical periodontitis is likely to be multifactorial. Confounding factors such as caries, socio-economic class and regularity of dental care are likely to be better controlled in the subpopulation than the general population and this may account for the differing results between the studies. However, due to the paucity of literature on the subject all the evidence should still be presented. As the authors themselves stated (Bergström et al. 2004) the investigation was cross-sectional in design and the conclusion should be regarded as temporary until confirmed by long-term observations.

This limitation of cross-sectional studies was addressed in a recent study (Marending *et al.* 2005) which related the impact of many patient-related factors with the outcome of root canal treatment.

A subpopulation of dental hospital patients in which 84 teeth were root filled and 66 had radiological data from more than 30 months were studied. After stepwise logistic regression was applied to the data it was found that although immune status of the individual and quality of the root canal treatment were significant predictors for treatment outcome, smoking had negligible impact. In order to reduce the variability of multiple operators the root canal treatment was carried out by one operator, however, there was no attempt to control other possible confounding factors such as the type of treatment performed (de novo or retreatment) or whether the tooth had preoperative radiographic evidence of a lesion or not. Type of treatment has been demonstrated to be a significant factor on healing of apical periodontitis (Sjögren et al. 1990). In addition, any conclusions from this study are limited owing to the small number of patients in each group, 17 smokers and 31 nonsmokers, and the fact that participating patients had more than one experimental tooth included in the study. This is acknowledged by the authors who summarized that large patient numbers are necessary to reveal if there is such any correlation between smoking and apical periodontitis.

Therefore, the current studies do not show a significant effect of smoking on either the incidence or healing of periapical lesions. Longitudinal studies are required to make firm conclusions.

Surgical endodontics

Smoking has local and systemic effects on the microcirculation (Lehr 2000), wound healing (Silverstein 1992) and the immune system (Kinane & Chestnutt 2000, Sørensen *et al.* 2004). As well as impairing hard tissue healing (W-Dahl & Toksvig-Larsen 2004, Castillo *et al.* 2005, Gullihorn *et al.* 2005) and soft tissue healing (Webster *et al.* 1986, Chang *et al.* 1996), smoking is associated with wound infection after soft tissue incision procedures (Sørensen *et al.* 2003). Smoking is likely to affect healing in surgical endodontic cases that involve bony and soft tissue healing.

Many factors influence the healing after surgical endodontics in the short-term, including surgical trauma during reflection and lack of compression of the tissues after suture placement (Gutmann & Harrison 1994). It is often assumed that healing is uneventful after endodontic surgery but little is known concerning the influence of external factors on the healing process. Factors which may influence healing in the long term include residual infection (Chong *et al.* 1997), angle of apical bevel (Gilheany *et al.* 1994) or type of material used for an apical seal (Dorn & Gartner 1990). It is likely that these factors are more important than smoking in failure of apical surgery but all factors which may have an association with healing should be considered.

As there are no papers which discuss the variables of smoking and surgical endodontics in the literature. other research is relevant if information on wound healing is to be extrapolated to the field of surgical endodontics. Table 1 illustrates the lack of published research in this area. As a result of the paucity of direct information, the mechanism of how smoking will affect healing in the short- and long-term is open to conjecture. Smoking may have a direct effect on the marginal periodontium where an epithelial attachment protects the bone surrounding the tooth from the oral cavity. It also seems likely that smoking has a systemic effect which affects bony and soft tissue healing throughout the body. Although there are differences between periodontal and endodontic surgical healing, there are similarities. Periodontal surgery may be different in that it will involve epithelial healing in an inflammed area and healing by secondary intention, whilst this is generally not the case with surgical endodontics. In addition, surgical endodontics is unlikely to involve repositioning of the flap after removal of diseased gingival tissues. However, both procedures, surgical extractions and implant placement involve incisions in the oral cavity in the presence of plaque and are subject to the direct and systemic effects of cigarette smoke. Therefore, although care must be taken in extrapolating the results too closely from other dental disciplines they should be considered. The only way to assess postsurgical endodontics wound healing would be to compare surgical endodontics in smokers with nonsmokers in a prospective clinical trial. However, as the success of surgical endodontics using modern instruments and techniques (Rubinstein & Kim 2002, Chong et al. 2003) is reported to be in excess of 85% it would therefore need to be a large study for there to be any likelihood of significance being demonstrated in the outcome between smokers and nonsmokers. In addition, if the prospective randomized studies available on the subject of success of surgical endodontics (Mead et al. 2005) are analysed, they are both limited in number and not one study has greater than 200 patients as a sample group. Therefore, the possibility of a prospective study emerging in this area appears to be limited.

Unfortunately, when the most recent studies that evaluate outcome of surgical endodontics are analysed,

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not even one includes any reference to smoking as a risk factor (Velvart 2002, Chong *et al.* 2003, Velvart *et al.* 2004, Gagliani *et al.* 2005). This is in contrast with equivalent surgical periodontal (Scabbia *et al.* 2001, Trombelli *et al.* 2003) and implantology studies (Wallace 2000, Geurs *et al.* 2001).

What exactly is meant by prognosis in endodontics?

When advising patients on the prognosis of endodontically treated teeth, the association between smoking and apical periodontitis is likely to affect survival of the tooth adversely, and must be considered. As endodontic treatment aims to protect or cure the periapical tissues from inflammation, prognosis of root filled teeth is described in terms of absence of apical periodontitis, in other words was the treatment a success/failure in achieving this over time (Friedman 1998). A patient's signs and symptoms as well as a radiological assessment are used to assess healing of apical periodontitis, although radiological methods are insensitive and do have their limitations (Brynolf 1967). However, although long-term monitoring is essential for assessment of apical periodontitis it is not the entire picture as patients and clinicians are also interested in why endodontically treated teeth are lost. This long-term survival can be measured by analysing eventual loss of teeth. If long-term prognosis and the reason for extraction of teeth are studied in various population groups, it is found that endodontically treated teeth represent a substantial percentage of this group. Tooth extraction in 200 Swedish patients was recorded over 5-7 years and 33% of the teeth that were extracted were endodontically treated (Eckerbom et al. 1992). As only 4% of all the patients' teeth were root filled at baseline it was concluded that endodontically treated teeth were lost more often than other teeth.

Perhaps surprisingly there is limited evidence for the reason for extraction of endodontically treated teeth. Meeuwissen & Eschen (1983) studied a group of Dutch servicemen over 20 years and used the definition of extraction as failure of endodontically treated teeth. They found that in addition to apical periodontitis other factors such as marginal periodontitis or dental caries may have been the reason for extraction; however, they did not have data to verify this assumption. These findings have been corroborated in longitudinal studies (Sjögren *et al.* 1990, Vire 1991, Fuss *et al.* 1999). What can be concluded, is that when the long-term prognosis of root filled teeth is considered the evidence suggests that endodontically treated teeth are more

likely to fail for prosthodontic and marginal periodontal reasons than for endodontic reasons. How these are affected by smoking will now be explored.

Prosthodontic considerations

If a decision is made preoperatively to restore a tooth it is essential that the patient's oral hygiene and ability to care for the restoration in their mouth are properly assessed. Should smoking status be an additional consideration in deciding whether or not a tooth should be restored?

In an epidemiological study in Sweden (Axelsson et al. 1998) comparing smokers with nonsmokers in various age groups (35-, 50-, 65- and 75-year-old patients) it was shown that although oral hygiene was similar between smokers and nonsmokers there was a difference in mean attachment loss between the two groups. It was also shown that intact tooth surfaces were fewer in 35-, 50-, 65- and 75-year-old smokers and that 35-year-old smokers exhibited a larger number of decayed and filled tooth surfaces compared with nonsmokers. It was concluded that smoking is a significant risk factor for tooth loss, probing attachment loss and dental caries. Any link between caries and smoking clearly presents a challenge to the restoration and prognosis of endodontically treated teeth, as generally these teeth are heavily restored and may well be crowned. Furthermore, the survival of endodontically treated teeth mainly depends on adequate coronal restoration (Meeuwissen & Eschen 1983. Vire 1991. Fuss et al. 1999).

There is significantly more root caries in smokers compared with nonsmokers particularly in patients over 50 years of age with marginal periodontitis (Locker & Leake 1993, Ravald *et al.* 1993, Hahn *et al.* 1999). It was suggested that this cariogenic effect was probably not due to the direct effects of smoke itself (Hahn *et al.* 1999), but other oral factors associated with smoking such as reduced flow of saliva and gingival exudate (MacGregor 1989). Dietary factors were not discussed as a possible factor in the above papers. However, perhaps the most likely reason for the increase of root caries in patients who smoke is that they are more prone to marginal periodontitis than nonsmokers (Johnson & Hill 2004).

In conclusion, smokers have an increased prevalence of root caries and are also at increased risk of marginal periodontitis. Therefore, when root filled teeth are being restored in smokers extra care must be taken to ensure margins are supragingival and maintenance regarding diet and oral hygiene is optimal.

Periodontal considerations

Smoking and marginal periodontitis

Over recent years much evidence has emerged to suggest a strong link between smoking and periodontal disease (Kinane & Chestnutt 2000, Johnson & Hill 2004). The first evidence of a link between marginal periodontitis and smoking dates back to 1940s when Pindborg (1947, 1949) discovered a correlation between smoking and ulceromembranous gingivitis and a link between smoking and calculus deposits. Although reports existed about smoking affecting wound healing (Mosely et al. 1978), it was not until the epidemiological studies at the end of 1980s that a link between smoking and destructive marginal periodontitis was shown (Bergström & Eliasson 1987, Beck et al. 1990. Ismail et al. 1990). It was considered that the increase in disease might have been due to oral hygiene practices being poorer in smokers but some studies have shown that the increased bone loss remained in smokers even after the variable of oral hygiene was minimized or adjusted (Stoltenberg et al. 1993, Linden & Mullally 1994, Kerdvongbundit & Wikesjö 2000). It has been concluded that smoking was the strongest predictor of severity of marginal periodontitis after the factors of age, plaque and calculus, race, gender and systemic disease had been controlled (Grossi et al. 1994, 1995). In a recent review it was concluded that adult smokers are approximately three times as likely as nonsmokers to have marginal periodontitis (Johnson & Hill 2004).

Response to periodontal therapy and maintenance

Do patients who smoke respond to periodontal treatment predictably? Smoking has been identified as one of the major factors influencing response to periodontal therapy. In a recent systematic review on the subject (Labriola et al. 2005) it was concluded that following nonsurgical periodontal therapy people who smoked experienced less reduction in probing depth than nonsmokers (Preber & Bergström 1986, Grossi et al. 1996, Kaldahl et al. 1996). Several studies have shown the improvement of attachment to be about two-thirds that of nonsmokers. In addition, longitudinal studies examining smokers and nonsmokers treated surgically have shown less gain in bone height (Boström et al. 1998) in smokers. There is also a recurrence of disease with increased bone and tooth loss when maintenance therapy and recall visits are irregular (Becker et al. 1984, De Vore et al. 1986). Smoking has been linked to negative healing response after flap debridement surgery (Preber & Bergström 1990, Scabbia *et al.* 2001, Trombelli *et al.* 2003) and regeneration procedures (Stavropoulos *et al.* 2004, Trombelli *et al.* 2005).

When the provision of endodontic treatment is being considered for any tooth it is important that the periodontal status of the patient is assessed not just around the individual tooth but generally around the mouth. This is of particular relevance in a smoker as teeth have a twofold greater chance of being lost in periodontally maintained patients who smoke and an even greater chance in nonperiodontally maintained patients who smoke (McGuire & Nunn 1996).

Smoking and tooth loss

To what extent is smoking considered an additional risk in the loss of teeth? General population studies have shown that the longer patients smoke the more extensive is their relative tooth loss (Österberg & Mellström 1986, Holm 1994, Axelsson *et al.* 1998, Ylöstalo *et al.* 2004).

Due to the cumulative effects of smoking, the risk of tooth loss is greater in the older patient, which is pertinent when analysis of the distribution of endodontically treated teeth indicates that older patients have more root fillings. Buckley & Spångberg (1995) reported on the distribution of endodontically treated teeth and found that the prevalence of both endodontically treated teeth and pulpal disease increased with age. In a large study of the general population in Sweden (Ödesjö *et al.* 1990) it was found that in patients under 50 years of age <10% of all teeth were endodontically treated whilst in the over 50-years group this figure doubled.

Wound healing

Effect of smoking on healing

The effects of smoking on wound healing were first reported in a description of impaired healing of the hand in a smoker with arteriosclerosis (Mosely & Finseth 1977). There have been reports since documenting the effect of smoking on healing in head and neck surgery (Kuri *et al.* 2005), plastic surgery (Webster *et al.* 1986, Chang *et al.* 1996), orthopaedics (Kwiatkowski *et al.* 1996, Castillo *et al.* 2005), general surgery (Sørensen *et al.* 2005), animal studies (Mosely *et al.* 1978, Lawrence *et al.* 1984) and dentistry (Bain & Moy 1993, Lindquist *et al.* 1996, Scabbia *et al.* 2001). However, although the effects of smoking on healing are well recognized in clinical practice, extensive controlled studies are limited. Most *ex vivo* studies

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have examined only the effects of nicotine, but tobacco smoke also contains a range of other toxic substances including carbon monoxide and hydrogen cyanide which are all likely to impact negatively on healing (Silverstein 1992). Although the exact mechanisms are unknown it does appear that smoking interferes with the wound healing process by affecting fibroblasts, the microvasculature and the immune system.

Fibroblast effects. Fibroblasts are important cells in repair and remodelling. In addition to their production of collagen, elastic and reticular fibres, they orchestrate these processes by proliferating, migrating and secreting proteins such as cytokines and growth factors. Smoking has been shown to delay fibroblast migration to the wound area leading to an accumulation at the periphery of the wound which may prevent healing and increase scarring (Wong et al. 2004). Nicotine has also been shown to inhibit myofibroblast differentiation in human fibroblasts in vitro (Fang & Svoboda 2005). Fibroblast function is impaired by smoking (Raulin et al. 1988) which also produces a dose-dependent inhibition of human gingival fibroblast adhesion and viability (Poggi et al. 2002).

Microvasculature effects. A link between reduced tissue perfusion and wound healing was demonstrated in laboratory experiments, where nicotine was shown to interfere with aspects of wound healing between days 6 and 10 (Moselv et al. 1978). The vascular effects of smoking have been extensively studied and were reviewed (Lehr 2000). Chronic cigarette smoking exerts far reaching deleterious effects on both the morphological and functional aspects of the microcirculation. The mechanisms for this are unclear but include compromised endothelialdependent vasorelaxation, platelet aggregation, cell dysfunction and activation of endothelial circulating leucocytes. Functional consequences of smoking included vasoconstriction and reduced tissue perfusion whilst morphological observations demonstrate thickening of arteriole walls.

The blood supply to the skin is reduced in smokers (Freiman *et al.* 2005) as is that in the finger and retina (Rojanapongpun & Drance 1993). This reduction in blood supply is important in skin flaps as their success depends on an adequate supply of blood and oxygen (Silverstein 1992). Nicotine administered centrally through the carotid artery induced vasoconstriction in the peripheral gingival circulation in animals (Clarke

humans was observed when smoke was allowed into the oral cavity but not inhaled (Shuler 1968). This area is further complicated by studies that have used laser Doppler flowmetry to assess gingival blood flow and have shown despite the vasoconstriction, there is no decrease in peripheral blood supply in patients who smoke, perhaps due to a general rise in blood pressure (Baab & Öberg 1987, Palmer et al. 1999). What is of interest is that microcirculatory changes are largely reversible after cessation of the smoking habit, which helps to explain why cardiovascular risk is reduced when smokers give up the habit even after the first myocardial infarct (Aberg et al. 1983). This normalization of the microcirculation forms the basis for smoking cessation protocols implemented around surgical procedures.

et al. 1981). However, a local vasoconstrictive effect in

Immune system effects. Tobacco appears to undermine the host response by affecting the immune system. The mechanism is complex and has been reviewed (Barbour et al. 1997, Kinane & Chestnutt 2000). Smoking does interfere with the innate and acquired immune response (Kinane & Chestnutt 2000) with neutrophils affected in their migration (Kenny et al. 1977), their basic function (Ryder et al. 2002, Sørensen et al. 2004) and their ability to phagocytose (MacFarlane et al. 1992). Macrophages are also affected by smoking with decreased immune function (Schwartz & Weiss 1994). Cytokine levels are reduced in smokers compared with nonsmokers (Tappia et al. 1995) and immunoglobulin production is decreased (Johnson et al. 1990). Locally gingival crevicular fluid is reduced in smokers (Kinane & Radvar 1997, Giannopoulou et al. 2003), which would reduce the volume of antibodies and other defence molecules entering the oral cavity.

Surgical dentistry

Smoking increases the incidence of localized osteitis following third molar extraction (Sweet & Butler 1978) by four to five times; and the incidence of osteitis was 10 times greater in smokers who smoked in the 4-day period immediately afterwards.

Implantology

The long-term survival of endosseous implants is good (Adell *et al.* 1981, Lindquist *et al.* 1996, Testori *et al.* 2001). Bone resorption may occur around implants in long-term studies and this appears to be influenced by several factors of which smoking and oral hygiene are the most important. In a 15-year longitudinal study of

47 edentulous patients with mandibular implants, the marginal bone loss was significantly greater in smokers than in nonsmokers (Lindquist *et al.* 1996). The results were confirmed in other studies (Bain & Moy 1993, De Bruyn & Collaert 1994, Lambert *et al.* 2000).

Smoking cessation protocols

Uneventful wound healing is desirable in surgical cases, and smoking appears to reduce the chances of this occurring. The effect of smoking at the time of surgery is critical to the long-term success of implants (De Bruyn & Collaert 1994). As a result a protocol, based on the medical literature has been devised for smokers at the time of implant placement (Kave 1986, Bain 2003, Kuri et al. 2005). Protocols in general surgery have suggested giving up cigarettes 2 weeks before and until 1 week after the operation (Kave 1986). The scientific basis for these figures is that a preoperative abstinence of more than 5 days will allow for any nicotine to clear and therefore reverse the initial vasoconstriction as well as achieve higher levels of platelet adhesion and blood viscosity. A postoperative phase of 5 days would allow for flap epithelialization; but for bone healing and regeneration the patient should abstain up to 2 months (Bain 1996).

The effect of smoking on failure in 78 patients, in whom 233 implants were placed was studied (Bain 1996). Three groups were selected: nonsmokers, smokers who followed a cessation protocol and those smokers who continued. The smoking cessation protocol chosen involved the patients abstaining from smoking for 1 week before the surgery and for 8 weeks after implant placement. There was no significant difference between failure rates in smokers who followed the protocol and nonsmokers, however, a difference between smokers who continued and the other two groups was found.

These studies are relevant when surgical endodontics is considered as this also involves soft and hard tissue healing. Although there have been no smoking cessation trials in surgical endodontics, it is reasonable to extrapolate.

Conclusions

The retention of teeth is one of the major goals of restorative dentistry. The effect of smoking on the prognosis of endodontically treated teeth has thus far been a largely ignored area although much research exists concerning the effect of smoking on surgical, periodontal and restorative procedures. To provide reasonable advice on the possible effects of smoking on prognosis much needs to be extrapolated from other areas. Despite a paucity of evidence the following conclusions can be drawn.

• Endodontically treated teeth are lost more often than nonendodontically treated teeth and smoking increases general tooth loss by a factor of 2, through a combination of periodontal and prosthodontic reasons (McGuire & Nunn 1996).

• Smoking has significant effects on the process of wound healing in soft and hard tissues. These effects have been documented in oral surgery, periodontal surgery and implant surgery. If it is considered reasonable to extrapolate these effects to surgical endodontics, it can be concluded that smoking may impair the healing response. It has not been shown that smoking is a prognostic factor, as periapical healing after surgical endodontics is assumed to be largely uneventful and long-term healing is likely to be determined by other more important factors. A smoking cessation programme could be developed for surgical endodontics.

• There is conflicting evidence as to whether smoking increases the prevalence of apical periodontitis.

• Large longitudinal prospective trials are required to examine the influence of smoking on apical periodontitis and the outcome of surgical endodontics.

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International Endodontic Journal, 39, 843-854, 2006

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