Chapter 3 Periodontal Risk Factors and Modification

INTRODUCTION

As a common chronic disease of the oral cavity, periodontal disease is a set of inflammatory conditions affecting the supporting structures of the dentition (Armitage, 1999). After its initiation, the disease progresses with the loss of collagen attachment to the root surface, the apical migration of the pocket epithelium, the formation of deepened periodontal pockets, and the resorption of alveolar bone. Untreated periodontal disease continues with progressive alveolar bone destruction, leading to increased tooth mobility and potential tooth loss (Page and Kornman, 1997).

Reports from epidemiological studies, analysis of tissue histology, clinical trials, and animal experiments consistently demonstrate a multi-factorial aetiology of periodontal disease. Chronic periodontitis is the most prevalent form of destructive periodontal disease (Albandar et al., 1999). Furthermore, cross-sectional and longitudinal data from epidemiological research in periodontology suggest that risk factors can be identified, and that some of these factors could be controlled to prevent the development and progression of the disease. Risk factors are part of the causal chain of a particular disease or can lead to the exposure of the host to a disease (Consensus Report, Annals of Periodontology, 1996). The presence of a risk factor implies a direct increase in the probability of a disease occurring, and if absent or modified, a reduction in that probability should occur. Risk factors are generally classified as modifiable and non-modifiable. While gender, age, and ethnicity are non-modifiable, insufficient oral hygiene or tobacco use are identified as modifiable risk factors for periodontal disease.

A risk factor may be modified by interventions, thereby reducing the probability that a particular disease will occur. However, the susceptibility to a specific disease may vary among different individuals exposed to a given risk factor over time. Additionally, cumulative interactions between both modifiable and non-modifiable risk factors, described as "complex risk factors," have been suggested (Stolk et al., 2008).

A variety of interrelated risk factors may influence both the onset of periodontal disease and its progression (Figure 3.1). The detection of periodontal disease progression remains challenging since it typically relies on the comparison of measurements made with a calibrated periodontal probe and non-standardized periapical radiographs over time. Some emphasis should be placed on the early identification of periodontal risk factors to assess the likelihood of periodontal disease progression in susceptible individuals since both methods detect periodontal breakdown only after it has occurred. The goal of this chapter is to discuss known non-modifiable and modifiable risk factors as well as their management in the dental practice to provide prevention and a careful maintenance program for the periodontal patient while following the best available evidence today.

NON-MODIFIABLE RISK FACTORS

Genetic and Hereditary Factors

Periodontal diseases are shown to be affected by genetic factors (Page and Kornman, 1997). A number of genetic disorders, such as Down syndrome, leukocyte adhesion deficiency syndrome (LADS), Papillon Lefevre syndrome, Chediak Higashi syndrome, chronic neutrophil defects, or cyclic neutropenia are associated with more or less severe periodontal conditions.

The hereditary aggregation was demonstrated in a twin study for chronic periodontitis (Michalowicz et al., 1991) and an epidemiological trial on aggressive periodontitis in a Dutch population (van der Velden et al., 1993). Following the adjustment for environmental factors such as tobacco use, it was estimated that 50% of the variance in disease may be attributed to a genetic background (Michalowicz et al., 2000).

Practical Application of Genetic Susceptibility Testing

During periodontal inflammation, inflammatory cytokines, including interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α), activate catabolic enzymes such as matrix metalloproteinases, subsequently leading to the breakdown of connective tissue. Any gene polymorphism of such proteins potentially alters the susceptibility of the host to periodontal diseases. A single nucleotide polymorphism (SNP) is a mutation that occurs when a single nucleotide is altered within its genome due to changes in the base pair sequence.

Past periodontal diagnostic research has focused on evaluating several selected candidate gene SNPs including variations of IL-1β or TNF-α. The evidence-based perspective: Evidence from a review suggests that some polymorphisms in the genes encoding interleukins (IL)-1, Fc gamma

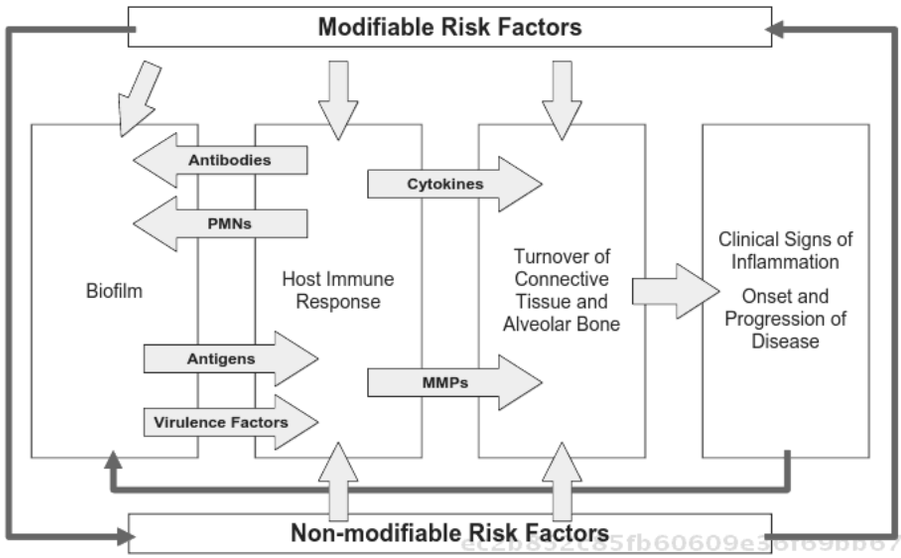

Figure 3.1. Interplay of modifiable and non-modifiable risk factors with the pathogenesis of periodontal diseases.

receptors (FcgR), IL-10, and the vitamin D receptor may be associated with periodontitis in certain ethnic groups (Loos et al., 2005). In addition, the association of the composite IL-1 genotype with periodontitis progression and/or treatment outcomes was analyzed using a systematic review approach (Huynh-Ba et al., 2007). There is limited evidence for such an association. Thus far, no specific genetic risk factor for periodontitis has been identified (Loos et al., 2005). Therefore, the clinical relevance of different commercially available genetic tests is limited.

Gender

Hormonal changes in women during menstruation, pregnancy, menopause, or therapy with pharmaceutical supplements have an impact on periodontal health. Disease susceptibility may be increased due to hormone-related alterations of the gingival blood flow (Kovar et al., 1985), the composition and flow rate of saliva (Laine, 2002), or the bone metabolism (Lerner, 2006). Additionally, data from epidemiological studies interestingly reveal that men may be at greater risk for periodontal diseases: in most clinical trials men are often found with worse periodontal health (Albandar, 2002; Meisel et al., 2007). Most often, however, these deteriorations may be explained by an increased prevalence of male tobacco use and mens' increased tendency to neglect oral hygiene (Meisel et al., 2007).

Gender-specific Practical Applications

Periodontal diseases have been associated with gender-specific complications such as an increased susceptibility for gingivitis during pregnancy (Russell and Mayberry, 2008), pre-term delivery, or low birth weight (Madianos et al., 2002). Therefore, gender-specific periodontal disease risk factors should be assessed in women by all oral health professionals (Krejci and Bissada, 2002). In pregnant women, a rigid recall interval including oral hygiene motivation is recommended. Consequently, it is suggested that existing periodontal inflammation should be treated before pregnancy.

Factors that are increasingly investigated in recent studies include gender-specific diseases such as osteoporosis or metastatic bone disease in relation with hormone substitute therapy or bisphosphonate medication in post-menopausal women (Diel et al., 2007; Payne et al., 1999). Bisphosphonates affect osteoclast functions, leading to the inhibition of physiological bone remodeling. With both surgical and non-surgical periodontal treatment, a bisphosphonate-associated osteonecrosis of the jaws should be considered as a complication. In one prospective cohort study of osteoporotic women in early menopause, it was found that a supplementation of estrogen may be associated with reduced gingival inflammation and impaired clinical attachment loss (Reinhardt et al., 1999).

Age

The aging process itself is suggested to be an independent risk factor for periodontal diseases (Papapanou et al., 1989). In contrast, a longitudinal study involving an elderly Scandinavian population (age 75 or older) demonstrated stable periodontal conditions for five years, suggesting a limited impact of the aging process itself in otherwise relatively healthy individuals (Ajwani and Ainamo, 2001). However, the extent and severity of periodontal diseases are shown to increase with age (Albandar, 2002) as a consequence of the cumulative burden from various risk factors such as tobacco use or plaque accumulation (Albandar, 2002; Albandar et al., 1999). Additionally, metabolic disorders, including diabetes mellitus, osteoporosis, rheumatoid arthritis, or vascular diseases, are more likely to develop in the elderly and thus affect periodontal conditions (Persson, 2006).

Age-specific Practical Applications

Life expectancy has increased significantly over the past few years in industrialized countries (Holm-Pedersen et al., 2005). As compared to previous populations, the elderly population is retaining its natural dentition, potentially leading to more periodontal problems. The presence of various chronic diseases, such as diabetes mellitus or specific medications (e.g. Vitamin K antagonists), may also interact with the periodontal condition or the treatment. Thus, there is a need for multidisciplinary treatment in many cases, due to the increased likelihood of co-morbidity in the elderly (Persson, 2006). Aging is often associated with the individual's impaired mobility, probably leading to an incapacity for regular supportive periodontal treatment. A close interplay with nursing homes or public oral health care providers may be advisable. Physical or mental disorders may affect the effectiveness of supragingival plaque control. The suggestion of simple interventions, including the weekly use of chlorhexidine-containing mouth rinses in conjunction with cognitive behavioral interventions, might be useful (Hujoel et al., 1997).

MODIFIABLE RISK FACTORS

Insufficient Oral Hygiene

Today, accumulated plaque is considered to be a dental biofilm briefly defined as a complex bacterial structure adherent to wet surfaces (Socransky and Haffajee, 2002). For the therapy of periodontal diseases, it is important to consider that biofilms can protect their microorganisms, either from the host's immune response or antimicrobial agents, and thus become difficult therapeutic targets (Socransky and Haffajee, 2002). So far, only mechanical debridement was shown to be a predictable approach to successfully destruct the dental biofilm. Therefore, mechanical plaque control should be performed supragingivally by the individual on a regular basis and subgingivally, if needed, by the oral health professional.

Any factors that facilitate biofilm formation, such as plaque retention or insufficient supragingival plaque control, are common risk factors for periodontal breakdown due to their causality with gingival inflammation and possibly the onset of periodontitis. This includes several anatomic conditions, such as enamel pearls, tongues, grooves, root furcations, and concavities, as well as root proximities (Roussa, 1998; Vermylen et al., 2005). Calculus and acquired iatrogenic factors, such as insufficient restorations, additionally contribute to plaque accumulation (Lang et al., 1983; Oliver et al., 1998).

It was proven some 40 years ago by classical experiments conducted by the work group Lüe and Theilade that oral micro-organisms are relevant for the development of inflammable periodontal diseases (Theilade et al., 1966). In a longitudinal study of more than 26 years, a further research group examined the influence of plaque-induced gingival inflammation on the subsequent loss of clinical attachment in a periodontally well-maintained Scandinavian population (Schatzle et al., 2003). The supragingival plaque accumulation correlated with the degree of gingival inflammation. However, sites with bleeding on probing at every visit demonstrated about 70% more attachment loss than sites without inflammation for the duration of the study. Moreover, it was recently shown that the susceptibility of gingivitis seems to be higher among males suffering from periodontitis (Dietrich et al., 2006).

Practical Application of Microbiological Testing

A multitude of different microbiological tests, based on morphological, enzymatic, cultural, genetic, or antigenetic bacterial properties, are available for both qualitative and quantitative microbiologic risk assessment of periodontitis. In many clinical situations, however, these tests fail to provide evidence-based recommendations for therapy (Sanz et al., 2004). Nevertheless, in a few cases, microbiologic tests can support treatment planning, including cases resistant to combined mechanical-antibiotic therapies, e.g. scaling and root planing, and the prescription of metronidazole and amoxicillin.

Tobacco Use

Earlier publications confirmed tobacco consumption as a risk factor for periodontal diseases. Over the past few years, oral health research has significantly contributed to the understanding of the mechanisms leading to the deterioration of the hard and soft tissues supporting the teeth. With the recording of the number of cigarettes smoked per day, the number of years tobacco was used, and the amount of time since tobacco use cessation, a dose response relationship was established using the Comprehensive Smoking Index (Dietrich and Hoffmann, 2004).

With increased use of tobacco, patients show higher periodontal probing depths, increased clinical attachment loss, more alveolar bone resorption, a higher prevalence of gingival recessions, and a higher risk for tooth loss (Tonetti, 1998). In contrast to this, with smokers, the clinical characteristics of gingival inflammation or bleeding on periodontal probing are less established (Dietrich et al., 2004). Smokers show less positive results after conventional, surgical, and regenerative periodontal therapy. The benefits of mucogingval surgery are reduced and less successful in smokers (Erley et al., 2006). Moreover, smoking impairs the osseointegration of oral implants and is at least partly responsible for a majority of biological complications in implant dentistry, such as periimplantitis (Strietzel et al., 2007). Based on the present understanding of periodontal diseases, the clinical findings, and the specific therapeutic outcomes with smokers, it appears to be reasonable, next to the current classification

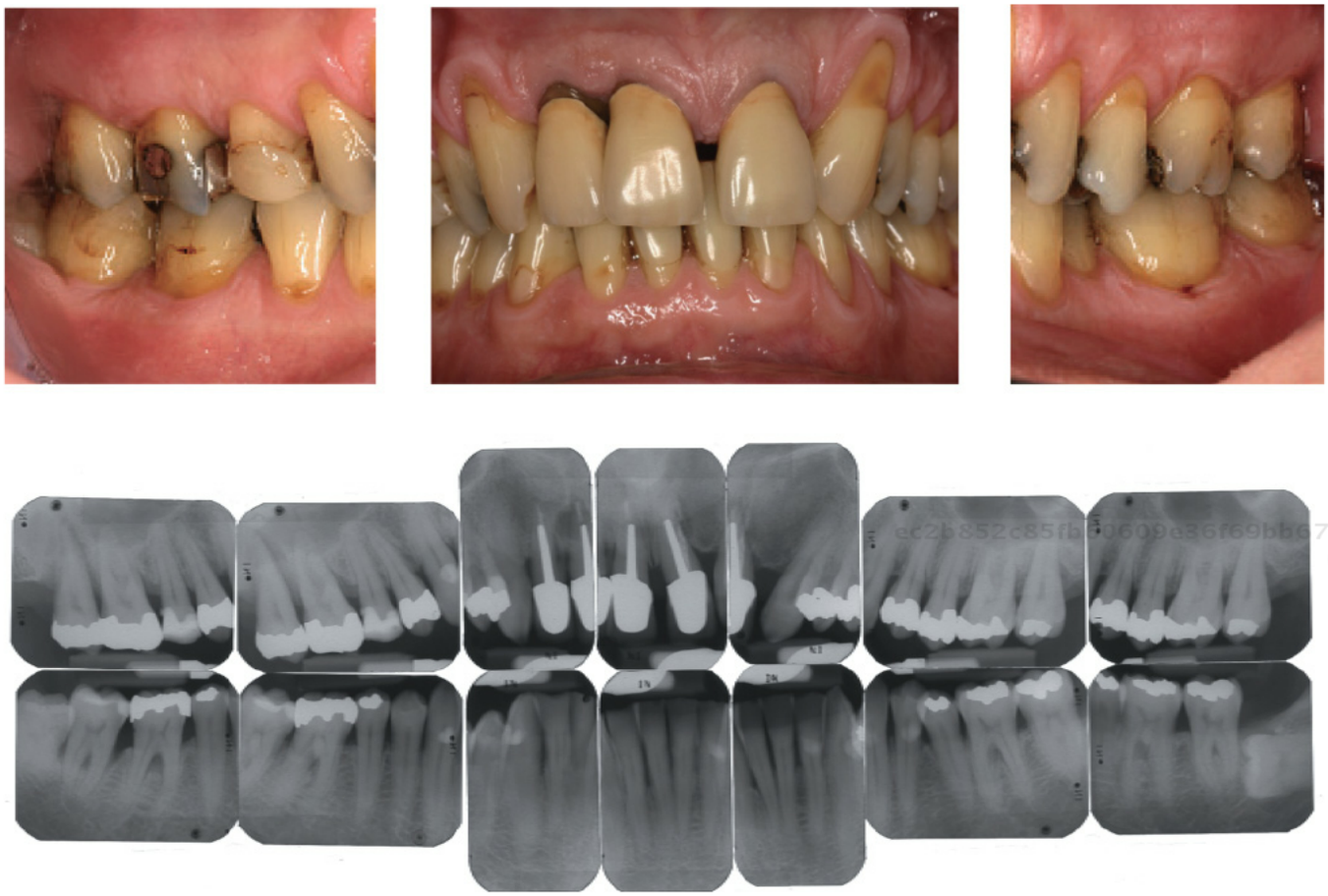

Figure 3.2. Clinical and radiographic images of a 45-year-old male smoker. Typical signs of smoker's periodontitis (20 pack years) including attachment loos, gingival recessions, and radiographic alveolar bone loss, particularly in the maxillary and mandibular front area.

of periodontal diseases, to use the term "smokers periodontitis" (Figure 3.2).

A common clinical observation is delayed wound healing after therapeutic interventions (Silverstein, 1992) (Figure 3.3).

There are various potentially significant pathogenic effects of tobacco-related substances on the periodontal tissues, immune response system, or composition of the oral flora. Periodontal destruction associated with tobacco use is caused by a wide multidimensional range of effects on different functions in cells, tissues, and organ systems. Some of these effects are diametric in nature, due to the effects of different tobacco constituents. However, when summarizing the properties of the tobacco-induced alterations in the metabolism of vasculature, connective-tissue, and bone, as well as on cell-mediated and humoral immunity, it is more than likely that tobacco use shifts the physiological balance between anabolic and catabolic mechanisms in a more destructive direction, due to an alteration of protective immune and tissue mechanisms (Johnson and Guthmiller, 2007; Palmer, 2005; Ryder, 2007). Moreover, there is evidence that tobacco consumption may change the genetically determined susceptibility for periodontal diseases (Meisel et al., 2004).

The evidence-based perspective: There is robust evidence from a systematic review (Bergstrom, 2006) that smoking is a strong risk factor for periodontal diseases. On the basis of 70 cross-sectional studies, 14 case-control studies, and 21 cohort studies, it is concluded that smoking negatively interferes with a healthy periodontal condition.

Diabetes Mellitus

Diabetes mellitus is a metabolic disorder categorized by a hyperglycemia due to impaired insulin production or insulin resistance. Insulin is a pancreatic-hormone-maintaining glucose metabolism. At least two major groups of diabetes mellitus (type 1 and type 2) are differentiated based on their pathogenesis. In addition, some diseases such as hormone-secreting tumors, conditions such as pregnancy (gestational

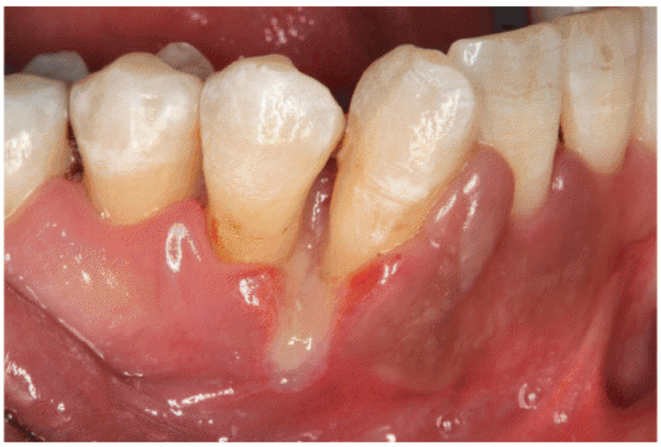

Figure 3.3. Impaired wound healing in a female smoker (age 44, 45 pack years) seven days following periodontal non-surgical debridement.

diabetes), or drugs such as corticosteroids can lead to diabetes mellitus. Treatment of diabetes mellitus primarily aims to keep blood sugar levels within a normal range. Treatment may include an interview for behavioral change for dietary adjustment, physical activity, or several drugs. They usually include oral antihyperglycemic drugs or insulin replacement therapy, as well as drugs for prevention and/or treatment of diabetes complications such as hypertension. If left untreated, serious long-term complications may occur, affecting small and large blood vessels, eyes, kidneys, nerves, or the immune system.

Diabetes mellitus has been associated with increased prevalence and severity of periodontal disease (Figure 3.4) (Emrich et al., 1991; Shlossman et al., 1990). The majority of studies demonstrate a more severe periodontal condition in diabetic adults than in adults without diabetes (Papapanou, 1996; Verma and Bhat, 2004). The type of diabetes does not affect the extent of periodontitis when the duration of diabetes is similar. However, type I diabetics develop the disease at an earlier age, and, hence, have it for longer periods, and may therefore develop a greater extent and severity of periodontitis (Oliver and Tervonen, 1994; Thorstensson and Hugoson, 1993). Well-controlled diabetics are more likely to be similar to non-diabetics in their periodontal status (Westfelt et al., 1996).

A common complication of diabetes mellitus is the increased susceptibility for microbial infections due to an impaired function of the host immune response. Diabetes mellitus may contribute to periodontal inflammation via specific mechanisms. The hyperglycemia may promote the formation of advanced glycation end products (AGE), i.e., glycated body proteins (Wautier and Guillausseau, 1998). Accumulation of AGE may have an impact on periodontal micro-vascularisation or may lead to an increased number of monocytes within the site of inflammation (Katz et al., 2005). A modification of physiologic cell functions of certain subtypes of granulocytes is also reported (Manouchehr-Pour et al., 1981a; Manouchehr-Pour et al., 1981b). Moreover, some studies suggest an alteration of pro-inflammatory mediators in gingival crevicular fluid, including tumor necrosis factor-α, prostaglandin-E2, and interleukin-1β (Engebretson et al., 2004; Salvi et al., 1997a; Salvi et al., 1997b). A further research group reported a decreased gene expression of anti-inflammatory and anti-bone-resorptive molecules such as interleukin-10 and osteoprotegerin (Duarte et al., 2007). Collagen is produced by fibroblasts and is an important molecule of the periodontium. In-vitro findings indicate a reduction of collagen synthesis in a dose-dependent fashion of glucose concentration (Willershausen-Zonnchen et al., 1991).

Interestingly, there is some evidence for periodontitis as a contributing factor in the pathogenesis of diabetes mellitus (Taylor et al., 1998). Inflammatory markers, including tumor necrosis factor-α, increase with periodontal severity and thus affect the insulin metabolism in diabetics (Engebretson et al., 2007). In contrast, their reduction occurs following antimicrobial periodontal therapy, leading to an improvement of glycemic control (Iwamoto et al., 2001).

The evidence based perspective: Evidence from a systematic review, including meta-analysis, suggests a significantly higher severity but the same extent of periodontal disease in diabetics compared with non-diabetics (Khader et al., 2006).

Stress

Stress may be caused by acute or chronic stressors. A stressor can be intrinsic or extrinsic in origin and is frequently defined as anything that causes an adaptive and non-specific neurological and physiological response in an individual. Chronic stressors are of relatively longer duration and include several “life events” such as the loss of a family member, splitting of a relationship, long-term illness, miscarriage, or “daily hassles.” Events of a relative short duration, such as traffic jams, surgical interventions, dental visits, or unpleasant questions in a medical exam, on the other hand, may act as acute stressors to an individual. The physiologic response is mediated by several immune-to-brain-to-immune regulatory pathways (Breivik et al., 2006). The individual stress coping behavior depends on genetic susceptibility and environmental and developmental factors as well as gathered experiences during the course of life.

Several studies indicated an association of negative stress, depression, anxiety, or poor coping behavior with periodontal diseases (Genco et al., 1999; Hugoson et al., 2002; Wimmer et al., 2002). Negative stress may lead to an increased susceptibility to periodontitis mediated through different pathways.

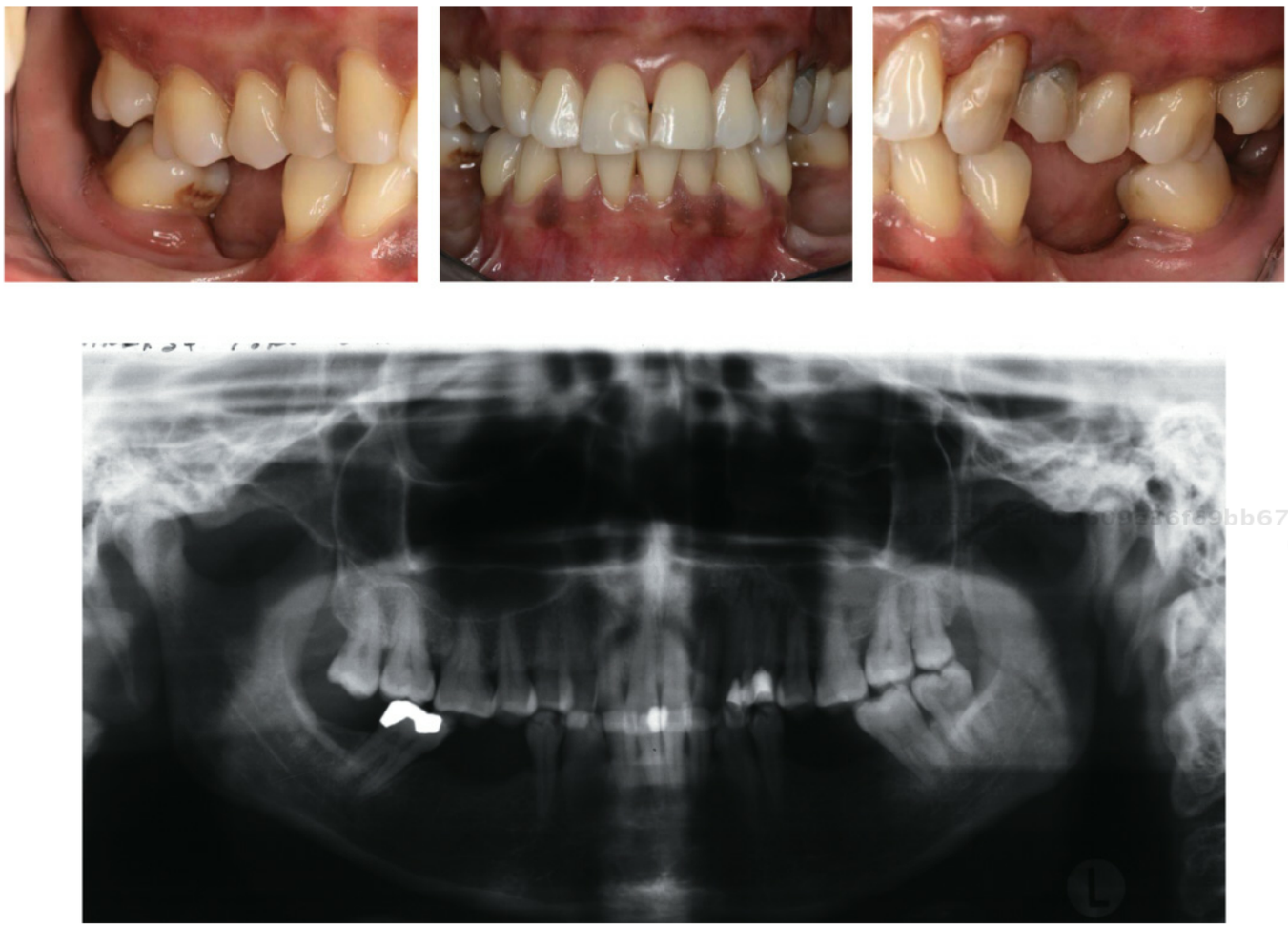

Figure 3.4. Clinical and radiographic images of a 46-year-old female patient with type II diabetes mellitus and chronic periodontitis. The metabolic disease was diagnosed in 1993 and is well controlled (level of blood sugar glucose 6, 3 mmol/l) by the physician. The patient receives oral antidiabetic drugs.

As one mechanism, it was suggested that due to stress, the oral hygiene may be limited (Deinzer et al., 2001). Additionally, academic stress was shown to cause an enhancement of interleukin-1 secretion detected in gingival crevicular fluid in a study by Deinzer and co-workers (Deinzer et al., 1999). This cytokine is a strong stimulator of osteoclasts leading to destructive bone metabolism. Interleukin-6, another inflammatory cytokine, was found to be elevated in the gingival fluid of depressed women (Johannsen et al., 2006). In addition, a prolonged reduction of the secretion of immunoglobulin A, an important salivary antibody, was observed in students participating in a major medical exam (Deinzer et al., 2000).

Cortisol and the catecholamins adrenaline and noradrenaline are the major stress hormones produced by the cortex of the suprarenal gland in response to stimulation by hypothalamus-releasing hormones. Increased levels of stress-mediated cortisol were found in the gingival crevicular fluid and in saliva (Hugo et al., 2006; Ishisaka et al., 2007; Nakajima et al., 2006). Additionally, stress-induced hypercortisolemia was linked to elevated levels of plaque and gingivitis (Hugo et al., 2006). Moreover, evidence from animal experiments reveals changes in the periodontal tissues following stress exposure (Nakajima et al., 2006). Restraint stress was able to enhance attachment loss after challenge with the putative periodontal pathogen *Porphyromonas gingivalis*. Findings from in vitro experiments suggest an effect of catecholamines on the growth of certain oral bacteria (Roberts et al., 2002). Thus, a stress-induced increase of catecholamine levels in the gingival crevicular fluid may be able to mediate the composition of the subgingival biofilm.

HIV/AIDS

The acquired immunodeficiency syndrome (AIDS) is caused by infection with the human immunodeficiency virus (HIV), leading to a destruction of the immune system of the affected host. The $CD4^+$ T cells as a subset of T lymphocytes are

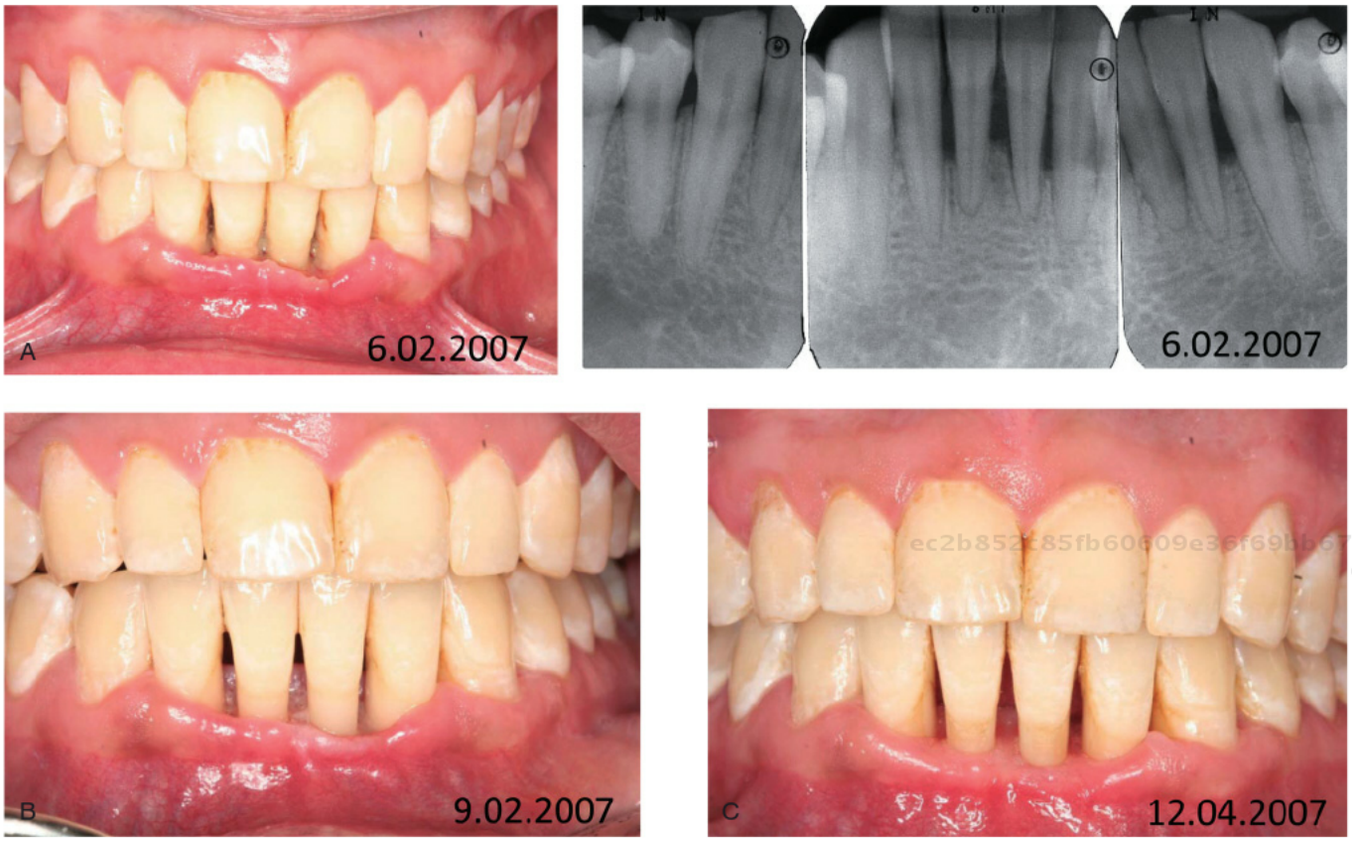

Figure 3.5. Necrotizing ulcerative periodontitis in a 26-year-old HIV-positive male (20 cigarettes per day) with medical observation for virus load and CD4 counts and no further prescription of HIV medication (HAART). A) Pre-therapeutical clinical and radiographic images, B) clinical view three days following non-surgical periodontal therapy, C) clinical view two months following therapy.

destroyed in particular. AIDS consists of various (opportunistic) infections including pulmonary and gastrointestinal infections and/or clinical manifestations including neurological and psychiatric involvement as well as tumors and other malignancies.

The current status of immunosuppression, assessed by $CD4^+$ lymphocyte levels and viral load with HIV, are predictors of AIDS as well as HIV-associated complications in the oral cavity (Kroidl et al., 2005). A multitude of oral lesions, including Karposi's sarcoma, linear gingival erythema (LGE), necrotizing ulcerative gingivitis (NUG), and necrotizing ulcerative periodontitis (NUP), were described in individuals infected with HIV (Figure 3.5). However, the likelihood of HIV-associated oral diseases has decreased in recent years, due to advanced treatment approaches in HIV/AIDS therapy, such as highly active antiretroviral therapy (HAART) (Reichart, 2006).

HAART combines the use of several drugs, affecting or inhibiting different stages of the retrovirus life cycle, including viral entry in host cells, syntheses of viral DNA, or activity of viral proteases. A number of studies dealt with the issue of occurrence of periodontal disease in HIV-seropositive subjects and AIDS patients (Lamster et al., 1994; McKaig et al., 1998). After controlling for CD41 counts, HIV-infected people taking HIV-antiretroviral medication were five times less likely to suffer from periodontitis compared with those not taking such medication (McKaig et al., 1998).

The effects of taking HAART on the outcome of periodontal therapy was assessed by Jordan and co-workers (Jordan et al., 2006). Chronic periodontitis patients with HIV can be successfully treated by non-surgical scaling and root planing, followed by supportive periodontal therapy. However, a close collaboration of oral health care providers and general practitioners should become a routine procedure in HIV patients' care.

Nutrition

Possible consequences of nutrition deficiencies on oral and periodontal health have been reviewed (Dorsky, 2001). Several nutrients have been found to have a negative impact on periodontal health when not sufficiently delivered, such as

vitamins, trace metals, antioxidants, and proteins (Eklund and Burt, 1994; Krall, 2001; Nishida et al., 2000a; Nishida et al., 2000b). So far, however, there are no reports in the current literature on the effect of nutrition counseling, either on the periodontal status or on the outcome of periodontal therapy.

RISK FACTOR MODIFICATION

Behavioral Change

Primary and secondary prevention oriented toward the change of inappropriate behavior is about to become a part of daily dental care. Traditional periodontal care includes the instruction of proper oral hygiene methods. Unfortunately, many health education approaches seem to be inefficient in accomplishing long-term change, potentially leading to frustration of both the patient and the clinician. Additionally, there is a shortcoming of evidence in both the dental and psychology literature on effective methods for behavior counseling in periodontal care, particularly regarding:

- Individual oral hygiene instructions for optimal oral hygiene
- Effective tobacco use prevention and cessation counseling to help abstain from tobacco
- Appropriate dietary counseling for a healthy diet

It may be necessary to apply different behavior change counseling methods to target individual behavior to get reliably effective outcomes in periodontal care. According to the best available evidence for oral hygiene instructions, the repeated demonstration of a cleaning device may be applied. For tobacco use cessation, in addition to pharmacotherapy, the method of the five A's (Ask, Advise, Assess, Assist, Arrange) may be used (Fiore, 2000). Additionally, type 2 diabetic patients may be referred to nutritionists for dietary counseling. From a practical point of view, however, it may be complicated and even discouraging to approach the periodontal patient with a variety of different methods targeting the same purpose: establishing appropriate behavior to improve the outcomes of both periodontal therapy and long-term supportive periodontal care.

Brief Motivational Interviewing (BMI)

Aiming for simplicity, it may be preferable to apply one single counseling method for behavior change in periodontal care that is shown to be effective in both primary and secondary prevention of oral diseases. Numerous behavioral research studies have confirmed the success achieved by state-of-the-art motivational interviewing (MI). MI is a patient-centered interviewing technique which was initially used as an auxiliary tool during counseling for smoking cessation and alcohol abuse (Miller and Rollnick, 2002). In the context of dentistry, a "short form" of MI known as "brief motivational interviewing" (BMI) appears to be suited for use during health behavior interventions in dental practices. The aim of BMI is to achieve the following objectives within a short amount of time (i.e., less than five minutes):

1. To question the patient concerning her motivation to change her behavior, or
2. To give the patient the self-confidence he may need to accomplish the envisaged change of behavior, and
3. To reach an agreement to discuss behavior change at another appointment.

BMI uses a patient-driven pathway which is reflected by the acronym **OARS**:

Open-ended questions: "Yes" or "no" answers often terminate the topic of a conversation. Using "who," "what," "where," or "why" questions further allows the patient to provide more information for the counseling.

Affirm: Acknowledging the feelings of the patient offers validation and assurance that the counselor is actively listening.

Reflection: Playing back the conversation to the patient often demonstrates empathy and may also highlight ambivalent behavior.

Summarize: Providing an overview of the conversation allows confirmation of potential key points in the process of change.

For further information on communication methods for behavioral change counseling, the reader is referred to the textbook of Miller and Rollnick (Miller and Rollnick, 2002).

Supragingival Plaque Control

Proper self-performed mechanical plaque removal and compliance with needs-related recall visits are critical components of successful prevention and therapy of periodontal diseases. Both surgical and non-surgical periodontal treatment are only shown to be successful along with supragingival plaque control (Magnusson et al., 1984; Rosling et al., 1976). Additionally, periodontal therapy in combination with adequate, self-performed supragingival plaque control has been demonstrated to be effective in maintaining periodontal health for more than 20 years (Axelsson et al., 2004). Consequently, this approach has been considered as the "gold standard" for periodontal care (Figure 3.6). It is recognized that the daily removal of the bacterial biofilm represents the most important risk factor control (Figure 3.7). For the detailed instruction of supragingival plaque control for the prevention and treatment of gingivitis and periodontitis, the reader is referred to textbooks on periodontal therapy.

The evidence-based perspective: Evidence from a review suggests that an optimal level of self-performed oral hygiene

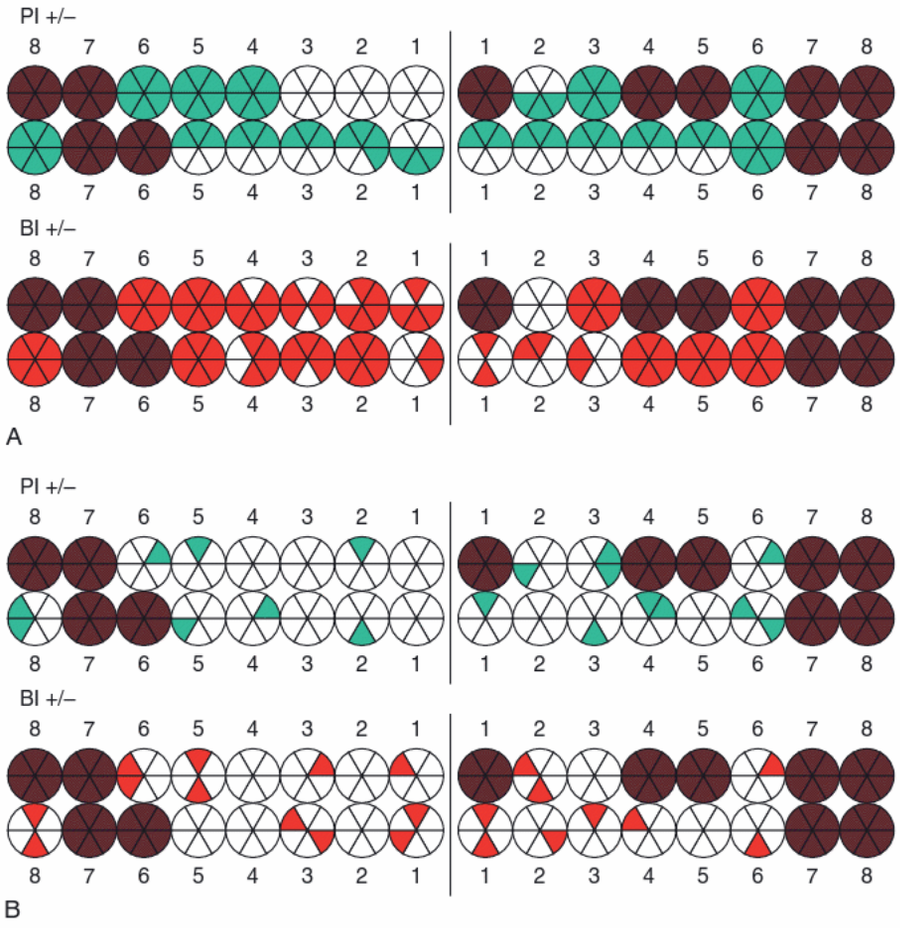

Figure 3.6. Oral hygiene motivation. Oral hygiene assessment: Visibility of supragingival plaque (PI, green) and ginigival bleeding (BI, red) are analyzed on six sites per tooth; missing teeth are colored black. The initial scores (A) indicate insufficient supragingival plaque control as well as gingival inflammation. Following oral hygiene instruction (toothbrush and interdental brushes) the plaque control improved (B).

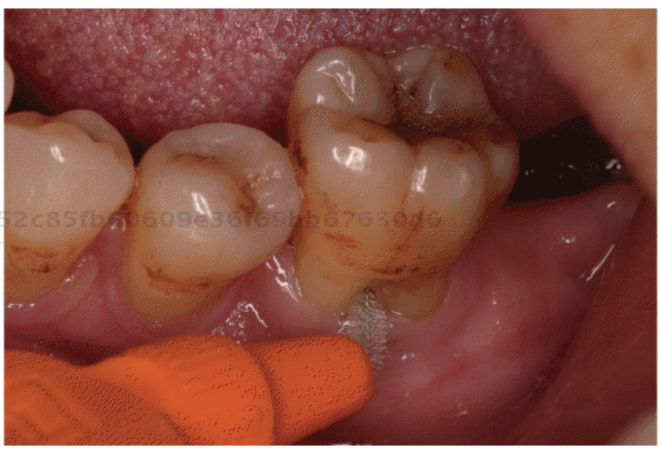

Figure 3.7. Oral hygiene aids (interdental brushes).

can have major effects on the subgingival biofilm composition and thus lead to significant therapeutic implications for the treatment of periodontal diseases (Ower, 2003).

Tobacco Use Cessation

Recent reports reveal short-term effects after quitting smoking as well as long-term results of smoking cessation on the periodontal and peri-implant status (Bergstrom, 2004) (Bain, 1996; Heasman et al., 2006). Generally, the periodontal status of former smokers is found to be intermediate between that of people who never smoked and current smokers (Bergstrom et al., 2000). Furthermore, smoking cessation has been shown to be beneficial for periodontal conditions: former smokers show less alveolar bone loss (Bergstrom et al., 2000; Bolin et al., 1993; Paulander et al., 2004) and reduced tooth loss (Krall et al., 1997) and present better outcomes after periodontal therapy (Kaldahl et al., 1996; Preshaw et al., 2005).

The potential benefit of smoking cessation is likely to be mediated through a number of different pathways. They may include shifts toward a less pathogenic subgingival flora; recovery of the gingival microcirculation; restoration of neutrophil function, metabolism, and viability; damping of the enhanced immune response; and re-establishment of any imbalance in the local or systemic production of cytokines (Heasman et al., 2006). The duration of the recovery of periodontal and peri-implant tissues following tobacco use cessation has not been determined yet. However, with the National Health and Nutrition Examination Survey (NHANES) of 12,623 patients in the USA, periodontal recovery was shown to be influenced by intensity, duration, and recency

of smoking. Additionally, with this data, a half-time (50% risk reduction) of one and a half years has been computed (Dietrich and Hoffmann, 2004).

The evidence-based perspective: A review summarizing data from epidemiological, cross-sectional, and case control studies strongly suggests that smoking cessation is beneficial to patients following periodontal treatments (Heasman et al., 2006). The periodontal status of former-smokers following treatment demonstrates that quitting smoking is beneficial. However, there are only limited data from long-term longitudinal clinical trials to demonstrate unequivocally the periodontal benefit of quitting smoking. Despite the lack of data from intervention studies, these findings suggest that smoking cessation may generally result in a long-term benefit to the periodontal condition. In addition, there is no need for randomized, controlled trials of the effectiveness of smoking cessation on oral health outcomes because such trials would not be feasible and would be too costly. However, well-designed observational studies are needed to fill the knowledge gaps.

Based on this body of evidence, tobacco use cessation becomes an important factor in daily dental care. Every member of the dental practice team plays an important role in the teamwork of smoking cessation counseling. With an appropriate assignment of tasks for every team member, patients are welcomed professionally, asked regularly about their smoking status, and continuously monitored.

A comprehensive model for smoking prevention and cessation applicable for both dental and dental hygiene education has been presented by Ramseier (2003) (Figure 3.8). This tobacco use cessation strategy is based on: (1) the model "stages of change" (or transtheoretical model) (Prochaska and DiClemente, 1983); (2) the five A's using nicotine replacement therapy (NRT) (Fiore et al., 1996); and (3) the main principles of motivational interviewing techniques (Miller and Rollnick, 2002).

In brief, the model includes recording every patient's tobacco use history, followed by a brief interview of no more than five minutes. The main aim of these interviews is to help tobacco-using patients to move from pre-contemplation to contemplation, and further to preparation, action, and maintenance stages. The routine use of a tobacco use history form as well as a record sheet to monitor tobacco use intervention is suggested (Figure 3.9). Patients' tobacco use history may be recorded on this form regarding intensity, time since cessation, and duration of each period, and they may be asked about their readiness to quit. According to a number of authors, current and former tobacco users should be asked about (1) the type of tobacco used, (2) the intensity of use (quantity per day), (3) the duration of use (years), and (4) time since cessation (years) (Dietrich and Hoffmann, 2004; Ramseier, 2003).

The evidence-based perspective: Evidence from a systematic review (Carr and Ebbert, 2007) suggests that behavioral interventions for tobacco use conducted by oral health professionals incorporating an oral exam component in the dental office and community setting increase tobacco abstinence rates.

Behavioral Support

People who want to kick the smoking habit do not always take part in state-of-the-art nicotine withdrawal programs in linear fashion from start to finish. Nevertheless, simple instructions, such as those offered in the "Assist" and "Arrange" programs, can be a valuable tool for physicians supporting patients in their attempts to quit smoking.

Some smokers are so euphoric about stopping smoking that they tend to move from one step to the next in a premature, i.e., unprepared manner. Even if this approach works for some smokers, others require varying amounts of behavioral support. This behavioral support can be given in an individual manner by adopting the following steps:

1. Asking the patient to complete a tobacco use journal (Figure 3.10): Every smoker has his individual smoking habits. To pinpoint the behavioral changes required in the particular case, it is advisable to keep a tobacco use journal for several days.
2. Evaluate the tobacco use journal: Reading through the journal entries later, the patient will notice smoking patterns and assessments of which she was previously unaware. These can serve as the basis for deciding which habits she must change to give up smoking (ideally without withdrawal symptoms) and replace the old habit with new patterns of behavior. During the control period, it is advisable to reduce nicotine consumption only down to a level where the "sacrifice" is bearable.
3. Behavioral changes: The process of successfully replacing smoking habits with other activities can be difficult and time consuming. Each patient should name an action that is good for him. It might be wise to arrange additional consultations at this point so that enough time can be devoted to this important step.

Pharmacotherapy

Kotlyar and Hatsukami (2002) have reviewed the management of nicotine addiction (Kotlyar and Hatsukami, 2002). The use of nicotine replacement therapy in dental tobacco use cessation was recently reviewed by Ramseier (2003) and Christen et al. (2003) (Ramseier, 2003; Christen et al., 2003). On the quit date, the patients should be sent home from the dental practice as "former smokers." It may be worthwhile to give each individual patient a written recommendation con-

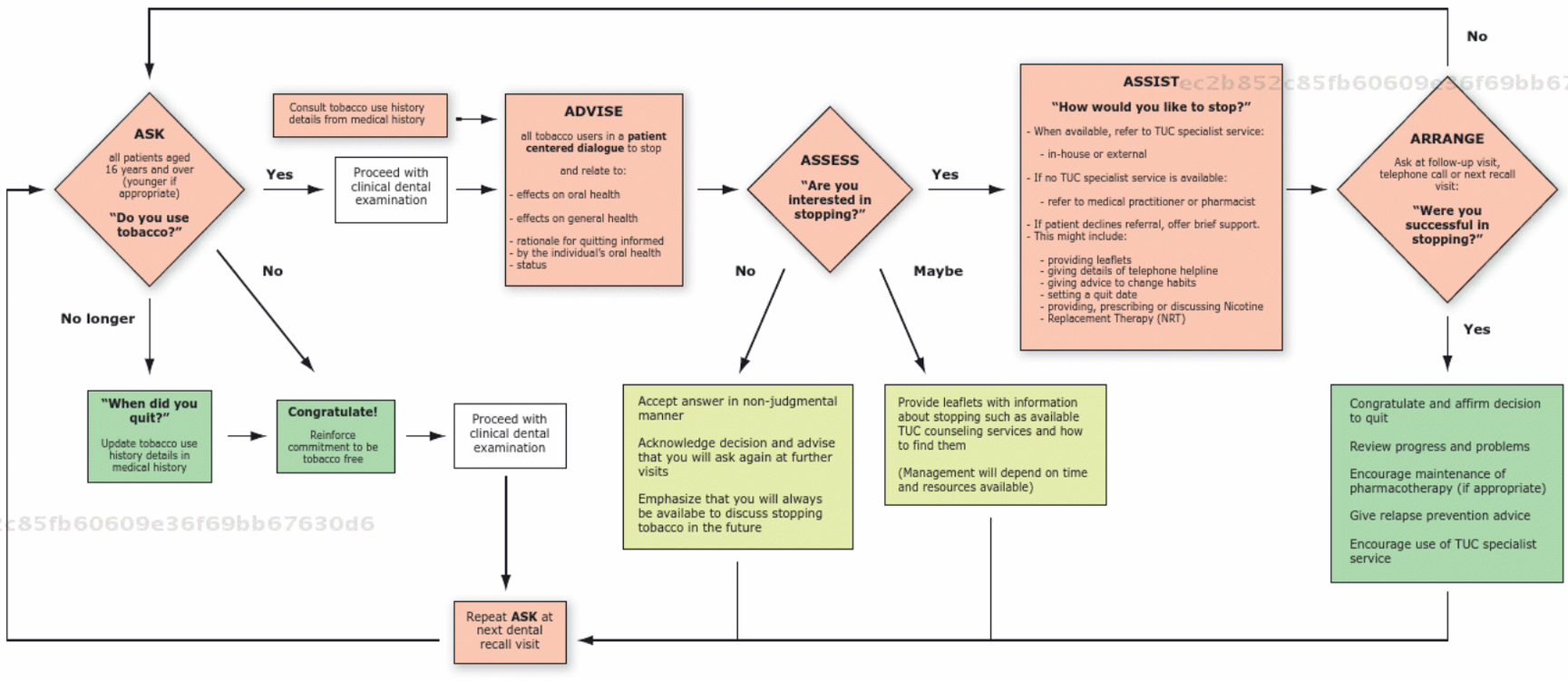

Figure 3.8. Tobacco use cessation care pathway for the dental practice.

Tobacco use history

Last / first name: _____ Date: _____

1. Have you ever smoked more than 200 cigarettes?	☐ yes ☐ no (go on with question 6)
2. At what age did you start to smoke regularly?	_____ years
3. Are you currently smoking cigarettes?	☐ yes (go on with question 5) ☐ no
4. In which year did you quit smoking?	_____
5. How many cigarettes do you smoke per day?	_____
6. Have you used other tobacco products regularly?	☐ no (go on with question 8) ☐ yes, the following: **Cigar** ☐ never ☐ in the past ☐ now **Pipe** ☐ never ☐ in the past ☐ now **Chewing tobacco** ☐ never ☐ in the past ☐ now **Other** ☐ never ☐ in the past ☐ now
7. How often have you already tried quitting tobacco use?	☐ never ☐ once ☐ 2 - 4 times ☐ more than _____ times
8. Are you currently thinking of quitting tobacco use?	☐ no ☐ yes, within the next _____ months
9. Personal information **a. Age** **b. Sex**	Date of birth: _____ ☐ female ☐ male

Figure 3.9. Tobacco use history form.

TOBACCO USE JOURNAL Date: _____

Cig.	Time	Place or activity	Accompanied by	Importance	Alternative
1					
2					
3					
3					
4					
5					
6					
7					
8					
9					
10					

Front

TOBACCO USE JOURNAL Date: _____

Cig.	Time	Place or activity	Accompanied by	Importance	Alternative
11					
12					
13					
13					
14					
15					
16					
17					
18					
19					
20					

Back

Figure 3.10. Tobacco use journal.

cerning the use of nicotine replacement products during the next three months (Figure 3.11).

There are various nicotine replacement products on the market such as gum, patches, sublingual tablets, inhalators, and nasal sprays. For the use of each product available, the reader is referred to the manufacturers' instructions.

The evidence-based perspective: There is strong evidence from a systematic Cochrane review that different commercially available forms of NRT can help people to quit smoking (Stead et al., 2008). NRTs increase the rate of quitting by 50% to 70%. The effectiveness of NRT seems to be largely independent of the intensity of additional support provided to the individual.

Other Risk Factor Modifications

Metabolic Control

The effects of metabolic control of diabetes mellitus on the periodontal status were evaluated exclusively on the basis of cross-sectional studies and a few prospective cohort studies. To date, the results seem to be conflicting and therefore no definite conclusion can be drawn (Bridges et al., 1996; Sastrowijoto et al., 1990; Taylor et al., 1998). From a clinical perspective, it is important to note that prevalence and severity of periodontal disease vary greatly within the diabetes mellitus population, just as it does in the non-diabetic population. Some diabetics may suffer from periodontitis because of inadequate oral hygiene and tobacco use rather than their diabetic condition (Haber et al., 1993).

Recommendation for use of Nicotine Replacement Therapy

Last name: _____ First name: _____

Level of nicotine dependency:

- ☐ very high
- ☐ high
- ☐ moderate
- ☐ low

Smoking behavior:

- ☐ smokes regularly through the day: recommendations: use of patch
- ☐ smokes only at specific times: recommendations: use of gum

From Day 1 of quitting:

	Patch (mg per day)	**Gum** (number per day)	**others** (number per day)
1st month			
2nd month			
3rd month			
After month 4			

Place, Date: _____ Signature: _____

Nicotine replacement	Low nicotine dependency	Moderate nicotine dependency	High nicotine dependency	Very high nicotine dependency
Patch		■	■ in combination with another nicotine preparation	■ in combination with another nicotine preparation
Gum	■ 2 mg	■ 2 mg	■ 4 mg	■ 4 mg
Sublingual tablets	■	■	■ in combination with patch	■ in combination with patch

Figure 3.11. Recommendations for use of nicotine replacement therapy.

To appoint the risk factor diabetes mellitus and the metabolic control of blood sugar levels in a periodontitis patient seems to be important for the understanding of the pathogenesis or the outcome of periodontal treatment. Once a patient is identified to be diabetic by a medical history a close collaboration with the diabetologist is advisable (Thorstensson et al., 1996).

Stress Reduction Therapy

Recently, an interesting approach on depression-related enhanced susceptibility of periodontitis was introduced (Breivik et al., 2006). According to this approach, treatment with an anti-depressant drug inhibited periodontal bone loss in an animal model of depression. Additionally, the individual coping behavior with stress in humans is shown to interfere with periodontal conditions (Genco et al., 1999; Wimmer et al., 2005). Coping behavioral training is likely to have a positive impact on disease severity or periodontal treatment outcomes. However, meaningful longitudinal clinical studies assessing the influence of psychological stress-related therapy on the outcome of periodontal treatment are currently not available.

Both risk factors for stress and poor coping behavior must be considered to achieve positive treatment outcomes in periodontitis patients. Once a patient is identified with stress or poor coping behavior, a close collaboration with a psychiatrist or psychologist may be advisable.

SUMMARY

Clinical research data indicate that risk factors associated with periodontitis can be identified. While certain non-modifiable factors are found, modifiable factors when amended may improve both periodontal conditions and the outcome of treatment. According to recent data, it appears reasonable to suggest that second to the removal of the bacterial biofilm, smoking cessation is the most important measure in periodontitis management. Consequently, periodontal health is to be supported by appropriate behaviors such as regular self-performed supragingival plaque control, avoidance of tobacco, and consumption of a healthy diet. The dental community involved with oral health care should gain an understanding of the health effects from inappropriate behavior to successfully target prevention and disease control. As a consequence, services for primary and secondary prevention on an individual level oriented toward the change of inappropriate behavior become a professional responsibility for all oral health care providers.

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