SMOKING AND THE PERIODONTAL PATIENT

ABSTRACT

Evidence from cross-sectional and case-control studies in various populations demonstrates that adult smokers are approximately three times as likely as non-smokers to have periodontitis. The association between smoking and attachment loss is even stronger when the definition of periodontitis is restricted to the most severely affected subjects. Smokers have a diminished response to periodontal therapy and show approximately half as much improvement in probing depths and clinical attachment levels following non-surgical and various surgical modalities of therapy. Implant failures in smokers are twice those of non-smokers, with a higher failure rate in the maxillary arch accounting for the majority of the difference. Tobacco-induced alterations in microbial and host factors contribute to these deleterious effects of smoking on the periodontium. In longitudinal studies, the rate of periodontal disease progression is increased in smokers, but decreases to that of a non-smoker following tobacco cessation. Likewise, recent non-smokers respond to periodontal therapy in a manner similar to patients who have never smoked. Data regarding the impact of smoking on periodontal status included in this review will be helpful to dental health professionals as they counsel their patients regarding tobacco use. The role of dental health professionals in tobacco cessation is discussed, including the use of the five A's: ask – identify tobacco users; advise – advise them to quit; assess – evaluate the patient's readiness to quit; assist – offer assistance in cessation; and arrange – follow up on the patient's cessation efforts. The addition of pharmacotherapy to behavioral therapy, including nicotine replacement therapy and bupropion, can increase cessation rates. The most popular form of nicotine replacement therapy is the patch, and its use has been shown to double cessation rates compared to behavioral therapy alone. Use of bupropion in combination with nicotine replacement therapy may be particularly helpful for heavy smokers or smokers who have experienced multiple failed attempts at cessation. The American Academy of Periodontology *Parameters of Care* include tobacco cessation as a part of periodontal therapy, and the *2000 Surgeon General's Report on Oral Health in America* encourages dental professionals to become more active in tobacco cessation counseling. Doing so will have far-reaching positive effects on our patients' oral and general health. *J Periodontol 2004;75:196-209*.

METHODS

To identify literature regarding the role of smoking as a risk factor for periodontitis, a MEDLINE literature search was conducted using the key words smoking, tobacco, and smoking cessation in combination with periodontitis or periodontal disease. MEDLINE searches were conducted to gather literature regarding the effects of smoking on various treatment outcomes using combinations of the key words smoking, tobacco, or smoking cessation with periodontal therapy, scaling/root planing, local delivery, metronidazole, amoxicillin, doxycycline, gingival grafts, gingival recession, guided tissue regeneration, bone grafts, dental implants, and sinus grafting. Prospective and retrospective studies that had at least five smokers among the patients treated were included, and studies in which the number of smokers was not enumerated were excluded. The MEDLINE literature search for cessation was conducted using the key words smoking, smoking cessation, and tobacco cessation. Searches were limited to the English language, and the primary focus was on current literature (1996 to 2002).

SMOKING AND PERIODONTITIS

Smoking and Periodontitis in Adults

As proposed by Gelskey,[7] smoking meets the majority of nine criteria for causation according to Hill[8] to varying degrees (Table 1). Abundant cross-sectional data support the relationship between smoking and periodontitis,[9-22] the strength of which varies, depending upon the criteria used to identify periodontitis and whether the effects of plaque and confounding variables are addressed. A meta-analysis of six studies concluded that smokers are almost three times as likely to have severe periodontitis compared to non-smokers.[23] In general, in studies where plaque accumulation was similar in smokers and non-smokers or was adjusted, current smokers had deeper probing depths,[9,20,22,24-26] greater attachment loss,[12,15,20,25,26] more bone loss,[13,27-29] and fewer teeth.[15,25] Smokers also exhibit more supragingival calculus deposits,[30] and the majority of these studies show a trend toward decreased clinical signs of inflammation.

Evidence for Smoking as an Etiologic Factor in Periodontitis

Criterion*	Evidence
Strength of association	Cross-sectional and case-control studies demonstrate a moderate to strong association between smoking and periodontitis.
Consistency	Multiple studies of various designs (cross-sectional, case-control, and longitudinal) and in various populations have demonstrated an association between smoking and periodontal attachment loss.
Specificity	Disease progression slows in patients who quit smoking as compared to those who continue to smoke.
Temporality	Longitudinal studies show that smokers do not respond as well to periodontal therapy as non-smokers.
Biologic gradient	There is a dose-response effect in that heavy smokers have increased disease severity compared to light smokers.
Biologic plausibility	The biologic plausibility of the explanation of the relationship between smoking and periodontitis is supported by tobacco's adverse impact on microbial and host response parameters.
Coherence	The effects of smoking on periodontitis are consistent with our knowledge of the natural history of periodontal disease.
Analogy	Periodontal effects of smoking are analogous to other adverse smoking-related general health effects.
Experiment	Evidence not currently available.

* Based on Hill's criteria for causation[8] as reviewed by Gelskey.[7]

The most recent and largest epidemiological study on smoking and periodontal disease[16] is based on data from the National Health and Nutrition Examination Survey (NHANES) III, which included a nationally representative sample of 12,329 U.S. adults, 18 years of age or older. Periodontitis was defined as the presence of one or more sites

with attachment loss and probing depth ≥4 mm. Among this sample, approximately one-half of periodontitis cases were attributable to either current (41.9%) or former (10.9%) smoking. The investigators also estimated that among smokers, approximately 75% of periodontitis cases were due to smoking. After adjusting for age, race or ethnicity, income and educational level, current smokers were four times as likely to have periodontitis as compared to non-smokers.[16] Additional analyses, based on the same data set and a more restrictive definition of periodontitis, found an even higher adjusted odds ratio and attributable fraction of attachment loss associated with current smoking. Among 20 to 49 year olds, the adjusted odds ratio for a mean attachment loss of 1 to 1.99 mm among current smokers was 2.29, whereas the odds ratio for attachment loss ≥3 mm was over 18.[21] This suggests that smoking is particularly important in the etiology of severe periodontal attachment loss. This is substantiated by results from a Swedish database that examined the impact of smoking on periodontal disease as a function of definition and the prevalence of probing depths ≥5 mm.[22] Using a broad definition of disease, such as 1% of probing depths 5 mm or greater, the smoking-associated odds ratio was 3. However, a more restrictive definition of 15% of probing depths 5 mm or deeper resulted in a smoking-associated odds ratio of 12.1.[22] These studies and others[12,13,20,31] have shown a strong dose-response relationship between the amount smoked and the severity of periodontal destruction, which further supports the role of smoking as a risk factor for periodontitis.

The most marked difference between smokers and non-smokers in probing depths or attachment loss occurs in the maxillary lingual area[25,32,33] and mandibular anterior teeth,[25] suggesting a local effect of smoking. Further evidence of the role of smoking in periodontal destruction is provided by retrospective and prospective studies which show that the rate of periodontal disease progression[34-38] and tooth loss[39-44] is greater in current smokers compared to non-smokers.

Smoking and Periodontitis in Young Adults

Smoking is more prevalent among young patients (≤35 years) with the generalized form of aggressive periodontitis compared to those with the localized form or with healthy periodontal tissues.[45,46] Several studies have shown that compared to non-smokers, young adult smokers aged 19 to 30 years have a higher prevalence and severity of periodontitis, despite similar or lower plaque levels.[19,26,47] Haber et al.[19] reported that the prevalence of periodontitis, defined as having a site with attachment loss ≥2 mm and probing depths ≥4 mm, was three to four times higher in young smokers 19 to 30 years of age compared to non-smokers. The high “periodontal cost” of smoking has been calculated as 27 years of disease progression; in other words, a 32-year-old smoker has similar periodontal attachment loss as a 59-year-old non-smoker.[25] Recent statistics indicate that more than 23% of high school students are current smokers,[48] which does not bode well for the future health of this generation.

Interaction Between Smoking and Systemic Health Status

The combination of smoking with other systemic factors further enhances the risk of periodontal destruction. In the Erie County study, diabetics were approximately twice as likely to exhibit periodontal attachment loss compared to non-diabetics,[12] and the combination of diabetes and heavy smoking in an individual over the age of 45 years who harbored *Porphyromonas gingivalis* or *Tannerella forsythensis* (formerly *Bacteroides forsythus)* resulted in an odds ratio of attachment loss that was 30 times that of a person lacking these risk factors.[12] Smoking also increases the risk of attachment and/or bone loss in postmenopausal women[49,50] and AIDS and HIV-seropositive patients.[51] In a retrospective private practice study, heavy smoking and

interleukin (IL)-1 genotype individually increased the risk of tooth loss over a 14-year period by 2.9 and 2.7, respectively, but the combination of these two factors increased the risk of tooth loss by 7.7.[52] Collectively, these studies suggest that smoking interacts with various systemic conditions; the end result is not purely additive, but can be synergistic, resulting in greater disease severity than either factor alone.

FACTORS CONTRIBUTING TO THE NEGATIVE IMPACT OF SMOKING ON THE PERIODONTIUM

Various factors contribute to the deleterious periodontal effects of smoking, including alterations in both microbial and host response factors (Table 2). Systemic innate and immune responses are impacted by smoking, and tobacco components have toxic effects for local cell populations, and impact local host responses.

TABLE 2

Proposed Mechanisms for the Negative Periodontal Effects of Smoking

- Vascular alterations
- Altered neutrophil function
- Decreased IgG production
- Decreased lymphocyte proliferation
- Increased prevalence of periopathogens
- Altered fibroblast attachment and function
- Difficulty in eliminating pathogens by mechanical therapy
- Negative local effects on cytokine and growth factor production

Smoking and Microflora

There are conflicting reports on the effects of smoking on the microflora which, in part, is explained by differences in methodology and statistical expression of the data. Some studies report no difference in the prevalence of subgingival bacteria associated with periodontitis.[53-55] However, data from the large Erie County study showed that the proportions of subjects positive for *Actinobacillus actinomycetemcomitans, P. gingivalis*, and *T. forsythensis* were higher among smokers,[56] and there are other reports of a higher prevalence of certain organisms in smokers.[57-59] Furthermore, increased counts of exogenous flora (*Escherichia coli* and *Candida albicans*) have been reported in smokers.[60]

Smoking and the Host Response

It is widely accepted that smoking impairs various aspects of the innate and immune host responses.[61,62] Numerous functions of oral or peripheral neutrophil are negatively affected by smoking or nicotine exposure, including phagocytosis,[63] superoxide and hydrogen peroxide generation,[64,65] integrin expression,[66] and protease inhibitor production.[67] The immune response is also impaired by smoking. Alterations in gingival crevicular fluid[68-70] and peripheral blood mononuclear cell[71] levels of various cytokines in smokers, tipping the balance in favor of tissue breakdown, have been noted. Smoking decreases salivary IgA[72] and serum IgG,[73] and specifically reduces IgG2 to *A. actinomycetemcomitans*.[74] The ability of tobacco products to decrease the proliferative capacity of T and B lymphocytes might contribute to this diminished production of protective antibodies.[61]

Local Effects of Nicotine

The oral tissues of smokers are exposed to high nicotine concentrations that negatively affect local cell populations. Gingival crevicular fluid nicotine concentrations can be up to nearly 300 times[66] that of nicotine plasma concentrations in smokers (20 ng/ml).[75] The vasoconstrictive properties of nicotine are hypothesized to impair gingival blood flow; however, studies that have examined the effects of smoking on gingival blood flow in humans have shown either no change or increased flow as measured by laser Doppler flowmetry.[76-78] This may be due to smoking-induced elevation in blood pressure, which overcomes any vasoconstrictive effects of smoking.[78] Smoking has been shown to impair revascularization during soft[79] and hard tissue wound healing,[80] which is critical for periodontal plastic, regenerative, and implant procedures.

Nicotine binds to root surface in smokers,[81] and in vitro studies show it can alter fibroblast attachment[82,83] and integrin expression,[84] and decrease collagen production while increasing collagenase production.[85] Root surfaces of teeth extracted from smokers show reduced periodontal ligament (PDL) fibroblast attachment as compared to those from non-smokers.[86] Cultured gingival keratinocytes[87] and fibroblasts[88] exposed to nicotine produce higher amounts of the proinflammatory cytokines IL-1 and IL-6, respectively. Furthermore, there is evidence of a synergistic effect on inflammatory mediator production when bacterial lipopolysaccharide is combined with nicotine.[88,89] Taken together, these factors could contribute to the increased tissue destruction observed in smokers. Furthermore, animal studies have shown that local nicotine delivery negatively impacts bone healing,[90] which may be related to inhibited expression of various growth factors[91] and delayed revascularization.[80] These findings might help explain the diminished treatment response to surgical periodontal procedures, especially those involving tissue regeneration.

EFFECTS OF SMOKING ON PERIODONTAL THERAPY

Non-Surgical and Surgical Therapy

Numerous studies have shown that smoking compromises probing depth and/or attachment gain outcomes following non-surgical or surgical therapy.[92-102] Collectively, these studies show that probing depth reduction and clinical attachment level improvements in smokers are 50% to 75% those of non-smokers following non-surgical and surgical periodontal therapy. The numerical differences between smokers and non-smokers become more pronounced in probing depths ≥5 mm, where smokers demonstrated 0.4 mm[94] to 0.6 mm[97] less improvement in clinical attachment levels following scaling and root planing. Following flap debridement surgery, smokers experienced up to 1 mm less improvement in clinical attachment levels in probing

depths initially ≥7 mm.[101] In terms of dose response, a trend, albeit not significant at most time points, has been noted for heavy smokers (≥20 cigarettes per day) to respond less favorably than light smokers (<20 cigarettes per day).[100] Additional studies are needed to further clarify the effects of smoking dose on treatment outcomes.

Antimicrobial Therapy in Smokers

Because of the diminished treatment response in smokers, clinicians may recommend adjunctive antimicrobial therapy for smokers. This practice may be justified by evidence that suggests subgingival pathogens are more difficult to eliminate in smokers following scaling and root planing.[94,95,103] To date, only a few studies have specifically addressed antimicrobial therapeutic outcomes in smokers, and the majority of these studies show that the clinical response in smokers is less favorable regardless of adjunctive systemic or local antimicrobial therapy.[104-108] Soder et al.[106] concluded that there was little adjunctive effect of systemic metronidazole on non-surgical therapy in smokers. On the other hand, in studies where adjunctive systemic amoxicillin and metronidazole[109] or locally delivered minocycline microspheres[108] enhanced the results of mechanical therapy, there was a greater difference between the control and experimental treatments within smokers as compared to within non-smokers. These enhanced results might be due to antimicrobial actions, and in the case of tetracycline derivatives, anticollagenase activity. The fact that gingival fibroblasts show increased collagenase activity when exposed to nicotine in vitro[85] suggests this is an area to be investigated.

A recent study reported a positive response to sub-antimicrobial doxycycline (anticollagenase) therapy in combination with scaling and root planing in a group of severe periodontitis patients that included smokers;[110] however, the comparative effectiveness of this host-modulatory therapy in smokers versus non-smokers has not been reported. Unique regimens that sequence systemic antimicrobial therapy or combine local antimicrobial delivery with host-modulatory therapy[110] might offer clinicians and patients options that address microbial and host response alterations in smokers.

Soft and Hard Tissue Grafting

There are relatively few studies on soft tissue grafting that have been designed to specifically address the impact of smoking on root coverage procedures, or that have included at least five smokers. In Harris' study of 100 consecutively treated recession sites using a connective tissue with partial-thickness pedicle graft, there was no difference in the percentage of root coverage achieved between light smokers (97%), heavy smokers (99%), or non-smokers (98%).[111] Likewise, Amarante et al.[112] found no difference in root coverage between smokers and non-smokers when recession defects were treated with a coronally repositioned flap alone or with a bioabsorbable membrane. On the other hand, when expanded polytetrafluoroethylene membranes were utilized in guided tissue regeneration procedures at recession sites, smokers had significantly less root coverage (57%) compared to non-smokers (78%).[113] The superior blood supply afforded by the subepithelial connective tissue graft might be more resistant to the effects of smoking as compared to the non-resorbable barrier membrane. Additional controlled studies with adequate numbers of subjects are needed to further investigate treatment outcomes for root coverage procedures in smokers.

Smoking is detrimental to regenerative therapy in interproximal and furcation defects, whether treatment includes osseous grafts alone,[114] membranes alone,[115-119] or membranes in combination with osseous grafts.[120] In these studies, the results have shown less than 50% as much improvement in clinical attachment levels in smokers

compared to non-smokers, which amounted to differences ranging from 0.35 mm[120] to 2.9 mm.[117] In studies that evaluated osseous changes by sound probing or reentry, vertical bone gain in smokers ranged from 0.1 to 0.5 mm, whereas non-smokers demonstrated 0.9 to 3.7 mm improvement.[115,118] In terms of stability of treatment results, Cortellini et al.[116] found that stability was related to patient factors; patients who smoked, were non-compliant with recall, and had deteriorating oral hygiene lost attachment (2.2 to 2.4 mm) following both guided tissue regeneration and scaling and root planing treatment modalities.

Implant Therapy

Based on a multivariate statistical model adjusted for age, gender, and jaw position, smoking is significantly associated with implant failure.[121] In the studies reviewed, 0% to 17% of implants placed in smokers were reported as failures as compared to 2% to 7% in non-smokers, with the majority of studies showing at least twice as many failed implants in smokers.[122-128]

The largest data set on the influence of smoking on implant success comes from the Dental Implant Clinical Research Group (DICRG) of the Department of Veterans Affairs (DVA);[126] this is an 8-year, randomized, prospective clinical study that includes more than 2,900 implants in more than 800 patients at 32 clinical centers. The 3-year data demonstrated that 8.9% of implants placed in smokers failed as compared to 6% in individuals who had never smoked or had quit smoking. The majority of implant failures in smokers occurred prior to prosthesis delivery;[125,126] thereafter, the differences between smokers and non-smokers tended to disappear.

Several investigators have confirmed that smoking more negatively impacts implants placed in the maxillary arch than in the mandible.[122,126,127] The DICRG reported that the percentage of maxillary implant failures among smokers (10.9%) was almost twice that reported for non-smokers or past smokers (6.4%). A number of studies show little difference in implant loss between smokers and non-smokers in the mandible.[126,127,129,130] It is important to note that even with an increased number of implant failures in smokers, the percentage of successful implants in most studies still ranged from the upper 80s to low 90s. However, considering the greater “investments” on the part of the patient and doctor, decisions regarding the treatment selection should be carefully made.

The majority of the existing reports deal with machined titanium surfaces, and early evidence suggests that the currently, more popular roughened surfaces can partially compensate for the negative healing response in smokers.[131-133] A meta-analysis[132] reported that light smoking (average of 12 cigarettes per day) did not affect the success rate of either machined or dual acid-etched surface implants. Additional studies using rough-surfaced implants are needed to delineate interactions among smoking dose, loading protocols, and variables that have been reported to have an impact on implant outcomes in smokers, such as periodontal disease susceptibility and interleukin-1 genotype.[134,135]

Implant Placement in Grafted Sites

Emerging data indicate that the impact of smoking on implant therapy is more dramatic in grafted maxillary sinuses compared to non-grafted sites. Existing data are based on retrospective studies that have limitations including the use of multiple operators, various types of recordkeeping, and a variety of implant systems, as well as diverse grafting materials and techniques. Collectively, the percentage of implant failures in

grafted sinuses in smokers is 1.4 to 3.9 times greater than that of non-smokers, with the majority of studies showing at least 2.5 times the number of failed implants in smokers.[136-139] The 1996 report of the Academy of Osseointegration Sinus Graft Consensus Conference stated that the percentage of implant failures in grafted sinuses in smokers was 12.7% compared to 4.8% in the non-smoker group.[136] In a study that included complex grafting procedures with antral or nasal implant placement in severely resorbed maxillae, the failure rate was as high as 22% in smokers compared to 13% in non-smokers.

Limited data are available on the effect of smoking on ridge augmentation procedures. Jones and Triplett[140] reported that four of five smoking patients undergoing simultaneous onlay grafting and implant placement had impaired healing, as defined by loss of bone and/or implants. Another study reported that defect reduction in guided bone regeneration procedures around 36 implants placed in smokers was not significantly affected by smoking. In contrast to most other reports, smoking did not impact treatment success in these patients.[141] Additional studies, which include data regarding smoking dose and complications, are needed to expand these findings regarding smoking's impact on ridge augmentation procedures.

Impact of Smoking Cessation on Periodontal Status and Treatment Outcomes

While smoking cessation does not reverse the past effects of smoking, there is abundant evidence that the rate of bone and attachment loss slows after patients quit smoking, and that their disease severity is intermediate to that of current and non-smokers.[10,16,25,29,35,37,142] It is encouraging to note that former smokers respond to non-surgical and surgical therapy in a manner similar to never smokers.[94,100] In fact, among patients who had quit smoking 1 year or more prior to scaling and root planing, there was no relationship between the number of years since cessation and changes in probing depth or clinical attachment levels.[94]

Similarly, implant success rates for past smokers are similar to those for never smokers.[126] The majority of implant failures occur prior to prosthesis delivery; therefore, smoking cessation during the healing phase should be beneficial. According to Bain,[143] if patients quit smoking 1 week before and 8 weeks after implant placement, early implant failures were similar to non-smokers. Due to the highly addictive nature of nicotine, most patients will not be able to comply with a "cold-turkey" approach. Therefore, clinical studies should examine implant success rates in patients employing other smoking cessation strategies that include behavioral management and pharmacotherapy.

ROLE OF DENTAL HEALTH PROFESSIONALS IN TOBACCO CESSATION

Dentistry has a strong history of commitment to preventive education as a routine part of patient treatment. Dentists and dental hygienists have training in patient education that can be applied easily to tobacco use intervention methodologies, and dental professionals understand the nature of behavioral changes as gradual and requiring constant reminders. The practice of periodontics offers multiple opportunities for interaction with patients: during active treatment and especially in the ongoing long-term maintenance phase of care. Because of the negative impact of tobacco use on periodontal treatment, an additional motivation for cessation can be demonstrated over time and used effectively to help patients ultimately achieve a tobacco-free life.

Due primarily to increased public awareness of the negative effects of smoking, the United States has witnessed a decline in the percentage of adult smokers from 42% in the 1960s to approximately 23.3% in 2000.[144] The Healthy People 2010 objective is to cut this figure in half through numerous mechanisms, including increased use of tobacco cessation counseling in the dental office.[145] Of the 48 million adult smokers in this country, more than 70% express a desire to quit.[144] In a survey of general dentists, 65% claim to advise most or all of their patients who smoke to quit, but few provide cessation counseling.[146] There are many possible approaches to tobacco use intervention in the dental office, ranging from brief interventions to comprehensive cessation programs involving the entire office staff. These services can include determining patient tobacco use status; supporting abstinence; advising users to stop; and preparing users to stop and to remain tobacco free, in addition to offering cessation treatment.[147]

Nicotine dependence is classified as a chemical addiction by the American Psychiatric Association in the *Diagnostic and Statistical Manual of Mental Disorders* 1994 (DSM-IV).[148] It is a combination of physiological and psychological factors that must be addressed to help patients conquer the use of tobacco despite the extreme difficulty of the withdrawal process. Although tobacco use is a learned behavior with social implications and has characteristics of a habit, the main motivation behind continued use is relief of withdrawal symptoms. The symptoms can include irritability, anxiety, decreased heart rate, increased appetite, food cravings, restlessness, and difficulty concentrating.[149] A systematic approach that combines behavioral counseling with pharmacotherapy has been shown to achieve the highest rates of cessation, although each is also effective alone. These types of approaches used together address both the nicotine withdrawal symptoms and the psychological factors that must be faced to achieve abstinence.

TOBACCO INTERVENTION–MODELS FOR THE DENTAL PRACTICE

Brief Intervention Program

There are several barriers that have been identified which interfere with delivery of tobacco use intervention. A lack of information about treatment options, time constraints, lack of compensation, and unrealistic expectations are common reasons that prevent practitioners from offering these services.[150] In offices where time is an issue or where practitioners lack confidence in pursuing more comprehensive programs, a useful model for brief intervention that uses a five-step approach is recommended by the Agency for Health Care Research and Quality. The program is known as the five A's for smoking cessation. It includes: ask – systematically identifying the tobacco use status of all patients; advise – strongly advising all who use tobacco products to stop; assess – evaluating the patient's willingness to quit; assist – offering assistance in quitting; and arrange – following up on the patient's cessation efforts, especially early in the process.[151] The emphasis in this brief intervention is to offer information, encouragement, and support to patients, and to provide information about resources that may help the patient become tobacco free. All smokers benefit from the advice of a trusted health professional; in up to 10% of cases, the simple statement of encouragement to stop smoking will cause the patient to give up smoking.[152] Use of this model does not preclude developing a more comprehensive program as the office staff gains experience and knowledge in tobacco intervention. Professional continuing education is widely available and can range from extensive programs that give certification in nicotine dependence counseling to short courses that are overviews of the subject.

Comprehensive Intervention Program

A model for a comprehensive program in the dental office includes using the five A's and expanding the scope of intervention. Identification of an office coordinator for tobacco cessation activities is the first step in involving the entire office staff. The most ideal person to implement this program in the dental office is a dental hygienist. To create an environment of positive examples and to enhance the credibility of the message that patients are receiving, establishing a smoke-free office is also important. Implementing office systems to systematically identify tobacco users and to update tobacco use information regularly is a critical component of a comprehensive program in the dental office. It provides an opportunity to give regular reinforcement of the specific harmful effects of tobacco use. A cessation program tailored to the patient's needs should be offered, one that ideally combines counseling, pharmacological therapy using both nicotine replacement and other medications, and supportive follow-up.[153]

Expanding the Five A's for Comprehensive Intervention

Ask. Identification of the patient's tobacco use status (current, former, or never) is the first step in all interventions. The addition of tobacco use status to the traditional vital signs has been suggested as a way to initially assess and update this information,[154] and a question regarding tobacco use should be a part of the health questionnaire used in the dental office. Once a patient is identified as a tobacco user, additional information on the patient's level of addiction is useful. The Fagerström test is easily administered and is commonly used to assess nicotine dependence levels. The Fagerström test is scored based on answers to questions about timing of the first cigarette smoked in the day, difficulty in not smoking in forbidden areas, most important cigarette during the day, number of cigarettes smoked per day, timing of most intense smoking, and smoking when ill. Higher scores indicate more addicted smokers.[155]

Advise. All oral health professionals should advise patients who smoke of the associations between their oral disease and smoking, and advise them that smoking cessation would be beneficial. A good time to discuss this is after the periodontal examination has been completed and during a review of the etiologic factors involved in periodontal diseases. Facts regarding the strength of smoking as a risk factor for disease, its impact on treatment, and the positive impact of cessation are statements that can be included in a manner that is informative and not judgmental. The patient's responses during this discussion will provide insight into their interest in cessation and level of readiness for cessation. Educational resources for patients are available from several organizations, including the American Academy of Periodontology, American Dental Association, American Cancer Society, National Cancer Institute, American Lung Association, American Heart Association, and others. There are also numerous sites available on the Internet for information and support for smokers who are trying to quit.

Assess. The next step is to identify the patient's interest and readiness to attempt tobacco cessation. A transtheoretical model for readiness to change is useful for evaluation of addictive behaviors, and is used frequently for tobacco cessation counseling. It is a five-stage model that identifies behavior change as a process involving movement through these categories. This model is described as a spiral, and includes pre-contemplation, contemplation, preparation, action, and maintenance.[156] The most effective interventions can occur when the patient is in the preparation or action stage, but all patients can benefit from appropriate counseling based on their current stage of change. An intervention should be considered successful if some movement is made in the stages of change model, even if it does not lead immediately to cessation. Patients in the precontemplative or contemplative stages are currently

unwilling to stop using tobacco, and any intervention should focus on education, reassurance, and motivation to consider cessation. Patients in the preparation stage are willing to attempt cessation and are ready for behavioral intervention and pharmacological therapy. When patients are in the action or maintenance stages, relapse prevention is critical to continued abstinence.[157] Patients may cycle through these stages multiple times before they achieve success in becoming tobacco free.[158]

Assist. For any type of counseling that is performed with a nicotine-dependent patient, the most desirable techniques are those that are brief and non-judgmental. A style of behavior change counseling that has grown in popularity is motivational interviewing, a method that helps patients explore and resolve ambivalence about changing behaviors.[159] It is well suited for tobacco counseling, both for brief interventions and for more intensive counseling. The principles and techniques are particularly useful for health care practitioners who are not formally trained in counseling.[160] Motivational interviewing is described as the process of resolving ambivalence, using the patient's own reasons for concern and arguments for change. It involves creating a collaborative effort with the patient to overcome addictive behaviors.[161]

There are many other alternatives in behavioral therapy that have been studied and used successfully in tobacco use counseling. Some behavioral therapy attempts to break the association between smoking and pleasant events.[162] Stimulus control helps the patient develop behaviors to identify cues that trigger smoking, and then to create strategies to cope with these cues. Another popular technique is hypnosis, which uses suggestion, focused attention, and the therapeutic relationship to attempt to alter the patient's behavior. Meta-analysis of hypnosis in smoking cessation shows a treatment effect but no superiority over other behavioral techniques. Overall, the use of behavioral techniques has been shown to have a 2% to 14% effectiveness in cessation, defined as tobacco free for at least 6 months.[163] The best use of behavioral intervention might be in combination with pharmacological treatment.

Arrange. If the patient made a commitment to smoking cessation, follow-up from the office is critical. Methods of maintaining contact with the patient can range from appointments for office visits for monitoring and continued counseling, to letters or telephone calls confirming quit dates and encouraging follow-through with cessation. The most difficult time for patients is usually during the first week of cessation. Research shows that cessation rates are positively influenced by follow-up contact.[147]

Pharmacotherapy

The use of pharmacotherapy in tobacco cessation began in the 1980s, when nicotine replacement therapies were introduced. The U.S. Food and Drug Administration (FDA) currently approves nicotine chewing gum, nicotine lozenges, nicotine patches, nicotine nasal sprays, and nicotine inhalers for use in patients who are attempting cessation. The patches, chewing gum, and lozenges are available as over-the-counter products; the inhaler and nasal spray require prescriptions. Nicotine replacement products act as nicotine delivery systems in lieu of tobacco and can decrease withdrawal symptoms.[164-166] One non-nicotine medication, sustained-release bupropion, is also approved for tobacco cessation pharmacotherapy. In larger doses, bupropion is used as an antidepressant. These medications have been proven safe and effective, and have been extensively studied alone, in combination, and as an adjunct to behavioral therapy.[165-169] Barring complications, all patients attempting cessation should be treated with at least one form of pharmacotherapy. In general, the addition of medication to behavioral therapy doubles cessation rates.[170] Nicotine replacement products and

sustained release bupropion are considered first-line therapies (Table 3). Clonidine and nortriptyline are second-line pharmacotherapies that have been studied for cessation therapy, but have more side effects and are not approved at this time by the FDA for use in tobacco cessation.

TABLE 3

First-Line Pharmacotherapies for Smoking Cessation (adapted from Fiore et al.[151])

Pharmacotherapy	Precautions/ Contra-indications	Side Effects	Dosage	Duration
Bupropion sustained release	History of seizure History of eating disorder	Insomnia, dry mouth	150 mg every morning for 3 days, then 150 mg twice daily (begin treatment 1 to 2 weeks precessation)	7 to 12 weeks maintenance up to 6 months
Nicotine gum	Temporo-mandibular disorders exacerbated by chewing gum	Mouth soreness, dyspepsia	1 to 24 cigs/day-2 mg gum (up to 24 pieces/day) 25+ cigs/day-4 mg gum (up to 24 pieces/day)	Up to 12 weeks
Nicotine inhaler		Local irritation of mouth and throat	6 to 16 cartridges/day	Up to 6 months
Nicotine nasal spray		Nasal irritation	8 to 40 doses/day	3 to 6 months
Nicotine patch		Local skin reaction, insomnia	22 mg/24 hours 14 mg/24 hours 7 mg/24 hours 15 mg/16 hours	4 weeks then 2 weeks then 2 weeks 8 weeks
Nicotine lozenge		Heartburn, mouth soreness	2 mg lozenge (if first cigarette is 30 min. or more after awaking) 4 mg lozenge (if first cigarette is 30 min. or less after awaking) No more than 20 lozenges per day	Weeks 1 to 6: 1 lozenge every 1 to 2 hours Weeks 7 to 9: 1 lozenge every 2 to 4 hours Weeks 10 to 12: 1 lozenge every 4 to 8 hours

Indications for Use of Pharmacotherapy

In creating a personalized treatment plan for tobacco cessation, the patient's health history is important. The presence of other health problems may influence the approach that is used, and consultation and cooperative treatment in conjunction with the patient's physician are always appropriate. The use of nicotine replacement products should be related to the patient's current nicotine exposure, and to past experiences with cessation. Estimation of the daily amount of nicotine obtained from cigarettes can be calculated with accurate information about the daily number of cigarettes smoked. There are 3 mg of nicotine available in a single cigarette. On average, a smoker gets approximately 1 mg of nicotine from a cigarette leisurely smoked over about a 5-minute period. However, smokers who smoke more rapidly and inhale deeply can get up to 3 mg of nicotine from a cigarette.171 The ability to subconsciously titrate nicotine dosages is one of the reasons that patients who claim they have decreased the number of cigarettes smoked per day are still able to maintain the same blood levels of nicotine with fewer cigarettes, and successfully prevent withdrawal symptoms.

Nicotine Replacement Therapy

There are a variety of nicotine replacement products available, including gum, lozenge, patch, nasal spray, and inhaler. The selection of the type of nicotine replacement should be individualized based on the patient's smoking habits and preferences. For patients who smoke a pack (20 cigarettes) or less a day, the patch is the most popular form of replacement. Nicotine patches provide a steady delivery of nicotine throughout the time that the patch is in contact with the skin. Some patients may experience sleep disturbances if they wear the patch at night, but those effects often disappear over time.

Removing the patch at night is always an option. Nicotine patches come in several different dosages ranging from 7 to 22 mg. After the patient has been smoke free for at least 4 weeks or longer, tapering to lesser-strength patches at 2-week intervals has been recommended.[151]

Nicotine Replacement Combinations

Patients who smoke more than 20 cigarettes per day or who have had unsuccessful cessation attempts might benefit from a combination of nicotine replacement products to increase the blood levels of nicotine. Several combinations have been studied, but the usual recommendation is using the patch for constant nicotine blood levels, and adding one of the other products (gum, nasal spray, lozenge, or inhaler) as an addition for acute needs.[172] While the patient is using nicotine replacement products, they should not use any other forms of tobacco. If the patient has not stopped using tobacco products while using nicotine replacement, the therapy should be stopped, and a new treatment plan formulated.

Bupropion

Use of sustained-release bupropion has been shown to assist in cessation attempts, both alone[173] and in addition to nicotine replacement products.[174] The mechanism of action is unknown, but may be related to enhancement of dopamine and norepinephrine levels. Side effects include a lowering of the seizure threshold, so patients with known seizure disorders are not candidates for use. History of eating disorders, or concomitant use of wellbutrin or monoamine oxidase inhibitors are also contraindications to use of sustained-release bupropion. Although it is considered a first-line pharmacotherapy for smoking cessation and can be used alone, it is effective when used in combination with nicotine replacement products, and may be particularly helpful with heavy smokers or smokers who have experienced multiple failed cessation attempts. Bupropion use should be initiated at least 2 weeks prior to the patient's cessation date and subsequent initiation of nicotine replacement therapy, such as the patch. Therapy should start with 150 mg once per day for 3 days, then twice per day with at least 8 hours between doses.[173] The length of treatment can range from 7 to 12 weeks, but bupropion can safely be used for up to 6 months for maintenance therapy.[151]

Relapse Prevention

Since nicotine dependence is chronic, the tendency for relapse is high. Instead of seeing relapse as failure, it can be viewed by the practitioner as an indication that alternate treatment approaches might be indicated, just as the treatment of particularly challenging periodontal conditions requires a treatment plan unique to the patient's circumstances.[151] Smokers often experience several attempts at cessation before long-term abstinence is achieved and are more likely to have success when they have help with quitting.[175] All former tobacco users in the practice should be regularly encouraged to remain abstinent. For patients who have recently quit, discussions should include the benefits of cessation, the successes they have had, and problems they have encountered. Scheduling follow-up visits, sending notes, and making telephone calls of support are all examples of activities that can help patients remain abstinent.[151]

The opportunity for dentists and dental hygienists to become more active in evaluation of tobacco use by patients and more aggressive in offering counseling and cessation services can positively impact both the oral and general health of dental patients, and is strongly encouraged by the 2000 Report on Oral Health by the Surgeon General,[176] the recent clinical practice guidelines from the Public Health Service,[151] and the American Academy of Periodontology's Parameters of Care.[177] Of all the preventive services traditionally offered within a dental practice, those related to tobacco cessation are by far the most beneficial to the patient relative to general health and quality of life.

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