Source: Journal of Dental Hygiene, Vol. 80, No. 2, April 2006

The Journal of Dental Hygiene: Tried and True

R Wilder, RDH, BS, MS

Rebecca S. Wilder is an associate professor at the University of North Carolina Chapel Hill School of Dentistry and Director of the Master of Science Degree Program in Dental Hygiene Education. She is the current editor-in-chief of the Journal of Dental Hygiene.

Keywords: Leadership change, Dental hygiene research journal, Peer reviewed research publication, Original research

I have been reading the *Journal of Dental Hygiene* for almost 30 years. The *Journal* was initiated in 1926 so that means I have been reading it for almost a third of its existence! When I started out as a dental hygiene student, the *Journal* was called *"Dental Hygiene."* Later on in my career, it was changed to reflect its true stature as **The** *Journal of Dental Hygiene*. As a dental hygiene student, I did not think too much about what it all meant. After all, I was unsure about research and certainly had no idea about what it meant to be a peer reviewed publication. As I went through my graduate program in dental hygiene education, I began to see the value in a peer reviewed journal. It became clearer to me the importance of having my work appear in a publication that had been reviewed, critiqued, applauded, and yes-criticized by my peers. When my graduate class completed our thesis work-all of us wanted to publish our work in the *Journal of Dental Hygiene*. After all-it was *our* Journal and we wanted to support it and contribute to the dental hygiene research base. Numerous publications later and several years older-I still feel that way. I am always proud to see my work appear in the *JDH*.

Why is this Journal important to our profession? Research molds a profession. It adds to the scientific body of knowledge through the process of scientific inquiry. It means that we have unique information worth reporting to over a 120 000 dental hygienists and millions of health care professionals throughout the world. Although we have several publications in our profession, this one is the most important in my opinion because it is what sets us apart and elevates dental hygiene and hopefully what will take us to the next level. It is my intent to continue to do the work of my predecessors and make this *Journal* a true contribution to members of ADHA and to all who have a vested interest in oral health. It is my personal goal to have every member of our profession read every issue of the *Journal of Dental Hygiene*.

I am so honored to be the new Editor-in-Chief of the *Journal of Dental Hygiene*. I am in awe of those who have been in this position before me and have worked diligently to make the *Journal* what it is today-a well respected, peer reviewed research publication that is read by thousands of health professionals and held in high esteem by academic institutions

throughout the world. I want to personally and publicly thank Mary Alice Gaston, RDH, MS, for the time and energy she put into making the *Journal* what it is today. She worked tirelessly to bring us a high quality scientific publication. I intend to follow in her footsteps and maintain the integrity of the *Journal*.

The *Journal* will continue to focus on publishing first rate original research publications of basic, applied, clinical, health services, and educational research. We will continue to accept high quality literature reviews and seek to publish systematic reviews of interest to our profession. We will also continue to publish book reviews-which have been very popular over the years. However, it is a "new day" for the *Journal of Dental Hygiene*. Changes will come about with the *Journal* in the months to follow. Look out for some exciting additions starting with the fall edition. While the *Journal* is *"Tried and True"* my desire is to see it expand and grow as our profession is growing and changing.

I appreciate all the well wishes I have received from dental hygienists from all over the world and want to express my hope that you will always feel free to email me or contact me with any comments or suggestions you might have for ***our*** *Journal*. I will strive to make it the most professional publication in our profession and one that you will be proud to read and share with your colleagues. Thank you for the warm welcome as your new Editor-in-Chief of the *Journal of Dental Hygiene*.

Sincerely

Rebecca Wilder, RDH, BS, MS

Editor-in-Chief, Journal of Dental Hygiene

Source: Journal of Dental Hygiene, Vol. 80, No. 2, April 2006

Upfront

Katie Barge

Katie S. Barge is staff editor of the Journal of Dental Hygiene and staff writer for Access

HPV Test Best for Detecting Cervical Cancer

A test to detect the human papillomavirus (HPV), the virus that causes cervical cancer, is more sensitive, more effective, and easier to conduct than the traditional Pap smear. This test should be adopted as the worldwide standard for detecting HPV, reported the authors of a study appearing in the April 3, 2006, issue of the *International Journal of Cancer*.

"We are reporting many studies here, which are being brought together," said study author Jack Cuzick, the John Snow professor of epidemiology at the Centre of Epidemiology, Mathematics and Statistics at Cancer Research UK, Queen Mary School of Medicine, in London. "Hopefully, seeing the overwhelming effect of all results together will change practice."

Cervical cancer causes 300 000 or more deaths worldwide each year, with the most deaths occurring in areas where there is currently no screening at all. In the United States alone, some 10 400 women will be diagnosed with cervical cancer this year, and 3700 will die from the disease.

According to Cuzick, international implementation of the HPV test would greatly reduce the number of deaths caused by cervical cancer. He estimated a reduction of 50% above and beyond that achieved by the conventional Pap test in the United Kingdom. Cuzick believes the impact in the developing world would be even greater. "If this test could be applied there as well, 80% to 90% of cancers and deaths might be prevented."

HPV is one of the most common sexually transmitted diseases. An estimated 20 million men and women in the United States are infected with at least one type of HPV. According to the National Institute of Allergy and Infectious Diseases, there are high-risk and low-risk types of HPV. High-risk HPV may cause abnormal Pap smear results, and could lead to cancers of the cervix, vulva, vagina, anus, or penis. Low-risk HPV may also cause abnormal Pap results or genital warts.

Currently, early detection is the only way to prevent cervical cancer. However, researchers are close to finalizing a vaccine against HPV. "We are learning the viral implications of female genital tract malignancies," said Dr. Jay Brooks, chairman of hematology/oncology at the Ochsner Clinic Foundation in Baton Rouge, La. "In the future, this will be the way that individuals will be screened for the risk of many genital malignancies."

At this time, the gold standard test for cervical cancer involves collecting cells from the cervix via a Pap smear, then examining the cells under a microscope for abnormalities. In the study, this technique, which is known has cytology, had a sensitivity rate of 53%; HPV testing had a sensitivity rate of 96%. HPV testing was less specific for women under the age of 35, which lead to an increased number of false-positive readings for this age group.

The authors of the study feel it is "very realistic" to adopt this test worldwide. "Cheap forms of HPV testing are well into development to deal with cost issues in the developing world," said Cuzick. "In the developing world, the main barrier is inertia and an unfounded belief in the accuracy of cytology, which hopefully this paper will put to bed."

For more information about cervical cancer, please visit the American Cancer Society's Website at http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_is_cervical_cancer_8.asp or the National Institute of Allergy and Infectious Diseases Web site at http://www.niaid.nih.gov/factsheets/stdhpv.htm.

An Active Teen May Be a Safer Teen

Parents who have been looking for an alternative way to keep their teenager out of trouble may be in luck. Researchers at the University of North Carolina at Chapel Hill (UNC) found that teens who take part in many different kinds of physical activity - particularly with their parents - are less likely to get involved in high-risk activities, including drinking, drugs, and violence than teens who spend a lot of time in front of the television.

"Adolescents who spend a lot of time watching TV or playing computer video games tend to be at higher risk for engaging in all of these risky behaviors," said study co-author Dr. Penny Gordon-Larsen, assistant professor of nutrition, a department housed jointly in UNC's schools of public health and medicine, and a fellow at the Carolina Population Center.

The study, which was published in the April issue of the journal Pediatrics, compared 7 distinct clusters of adolescents, defined according to the types of physical or sedentary activities they participated in on a day-to-day basis.

Examples of clusters include:

- Adolescents who frequently played sports with their parents, who also spent a lot of time playing sports overall;
- Skaters/gamers, who did a lot of skating, skateboarding, bicycling and playing video games;
- High TV/video viewers, who made their own decisions about TV viewing and did a lot of it;
- Teens who often use neighborhood recreation centers; and
- Adolescents who often participated in school activities, including sports, clubs and physical education.

The study also asked questions about self-esteem, finding that the teens who were less physically active tended to have lower self-esteem. The remaining clusters were groups of adolescents who often used community recreation centers, as well as the group who participated frequently in school activities. Both also tended to have high self-esteem, compared to adolescents who watched a lot of TV.

This study revealed that kids who focused on fitness and activity were less likely to take up drinking, illicit drug use, violent behavior, sex and delinquency. On the other hand, the researchers found that adolescents who spend a lot of time watching TV or playing computer video games tend to be at higher risk for engaging in risky, unsafe behavior.

"Anything we can do to get kids to be physically active will help them in terms of their physical health, but this research suggests that engaging in a variety of activities may also have social, emotional, and cognitive benefits, including reduced likelihood of engaging in risky behaviors such as drinking, drugs, violence, smoking, sex, and delinquency," Gordon-Larsen added.

The current study also asked questions about self-esteem, finding that the teens who were less physically active tended to have lower self-esteem. The remaining clusters were groups of adolescents who often used community recreation centers, as well as the group who participated frequently in school activities. Both of these groups tended to have high self-esteem in comparison to the adolescents who watched a lot of TV.

The study also found the skaters/gamers to be at a relatively low risk, which may seem surprising given the bad rap skateboarding generally receives because schools don't generally sponsor it, many public places ban it, and not a lot of adults participate in it. "But we found that adolescents who skateboard actually fared well in terms of self-esteem and were less likely to engage in risky behaviors compared to teens who watch a lot of TV," Gordon-Larsen said.

"I think that parents should find ways to participate in sports and physical activities with their children," Gordon-Larsen said. "So, instead of having family TV time, build in time that the family is together and active. It's also extremely important for communities and schools to provide safe and affordable recreation facilities and opportunities for physical activity."

Although activity patterns among teens have been studied in the past, this study focused on the many benefits of physical activity. "Our previous research revealed physical activity and sedentary behavior patterns that vary among teens, and these activity patterns go beyond highly active and not active," said first author Dr. Melissa C. Nelson, who received her doctoral degree from UNC and now is assistant professor of epidemiology and community health at the University of Minnesota.

Researchers are still trying to understand all of the benefits of being active, according to Nelson. "This research leads us to believe that those benefits extend well beyond physical fitness. It could be that active teens are being exposed to more opportunities for team-building, engaging in more social interactions with others, or seeing the benefits of hard work and practice."

"We also suspect that all teens might not benefit similarly from the same kind of activity - it's not a one-size-fits-all kind of thing. Helping to provide kids with the opportunity to get involved in any number of physical activities, instead of staying at home and watching TV, may provide a kind of resilience against engaging in these other risky behaviors."

Cocoa Intake Associated with Lower BP and Cardiovascular Mortality

Although it has often been said that the key to a woman's heart is chocolate, Dutch researchers recently found that chocolate may also be the key to a healthy heart.

Sipping a cup of hot chocolate, or eating a candy bar, has been associated with reduced blood pressure for older men in Holland and a reduced risk of cardiovascular and all-cause mortality, reported the researchers in the February 27, 2006, issue of *Archives of Internal Medicine*.

Cocoa, which is rich in flavanols, has been associated with heart health since the 1700s; however, scientific evidence is now available to back this claim. "To our knowledge, this is the first epidemiological study to report an inverse relationship of cocoa intake with blood pressure and cardiovascular and all-cause mortality," said Brian Buijsse, MSc, and colleagues at the National Institute for Public Health and the Environment, Bilthoven.

The research team examined links between cocoa and cardiovascular health in 470 men ranging from 65 to 84 years of age. The men had physical examinations and were interviewed about their diet at the start of the study in 1985, and then again in 1990 and 1995. The researchers concluded that over a 15-year period, men who ate cocoa regularly - including chocolate - had significantly lower blood pressure compared with those who did not consume cocoa on a regular basis.

The delectable treat might even help ward off death. Over the course of the study, 314 men died, with 152 of those deaths blamed on heart disease. The men who consumed the highest amount of cocoa were half as likely to die from cardiovascular disease as those who ate little or no cocoa. Furthermore, men who ate the most cocoa were less likely to die from any causes.

The Dutch team said that the decrease in cardiovascular deaths could not be attributed to lower blood pressure. The decrease in cardiovascular mortality could perhaps be related to the improvement in endothelial function by flavan-3-ols in cocoa. Cocoa products may also inhibit platelet function and low-density lipoprotein oxidation, which could also account for the decrease in cardiovascular-related deaths.

"Cocoa is the most concentrated source of bioflavonoid antioxidants readily available in our diets," said Dr. David L. Katz, an associate professor of public health, and director of the Prevention Research Center at Yale University School of Medicine. However, moderation is vital, warned Katz. "Cocoa comes in foods that tend to be energy-dense, and the harm of excess calories could readily offset the benefit of antioxidants."

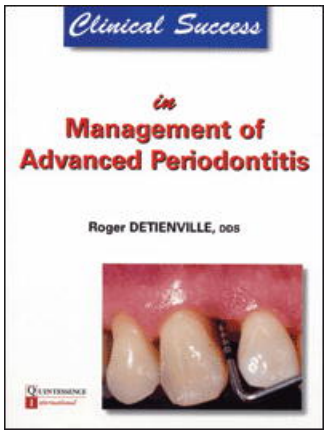
Katz also stressed that cocoa's heart-healthy benefits only come from bittersweet dark chocolate and in concentrated cocoa beverages, which contain an effective dose of antioxidants, along with magnesium, arginine, and fiber.

Source: Journal of Dental Hygiene, Vol. 80, No. 2, April 2006

Review of: Clinical Success in Management of Advanced Periodontitis

Lisa Shaw, RDH, MS

Reviewed by Lisa Shaw, RDH, MS, residential health care coordinator at St. Luke's Memorial Hospital Dental Service in Utica, New York.



Clinical Success in Management of Advanced Periodontitis

Detienville R

Quintessence International, 2005

Paris, France

120 pages, illustrated, indexed, soft cover

ISBN 2-9125-5041-6

$78.00

The treatment of periodontal disease is a major focus in the practice of dental hygiene. Approximately 80% of all American adults show evidence of some degree of periodontitis. Chronic periodontitis affects approximately 30% of the population; 10% of the population experience severe chronic periodontitis. Periodontal disease is responsible for 40% of all tooth extractions and is the major cause of tooth loss for individuals over 45 years of age. As such, it is imperative that dental hygiene students and practicing dental hygienists be aware of current classifications for periodontal disease, current evidenced-based treatment methodologies, individual risk factors for periodontal disease, and behavioral models that effect change.

Clinical Success in Management of Advanced Periodontitis by Roger Detienville, DDS, is an English translation of his 2002 French text. In it, he outlines the severity, prevalence, pathogenesis, diagnosis, infection control, and treatment strategies of periodontal disease. In his opening chapter, Detienville clearly lays out his intent: "Currently, there is a strong incentive toward the application of evidence-based solutions and techniques. However, statistical analysis is more difficult to carry out in the clinical setting than in the context of pure research. Clinical practice can therefore demonstrate its efficacy and ultimately point out elements of scientific truth through interpretation of scientific information." Detienville goes on to define "clinical proof" indicators that support the success or failure in the treatment of periodontal disease. Positive indicators include "durable elimination of clinical signs of inflammation and long-term maintenance of periodontal support structures...[as well as] attachment gain and reduced probing depths." Negative indicators include "continuous, progressive loss of periodontal support."

I chose to evaluate this text not on a chapter-by-chapter basis, but rather by how it succeeded or failed relative to the following criteria:

- Is the information technically and factually correct?
- Do references support the text?
- Does the book cover each topic adequately and clearly?
- Is the level of writing appropriate for dental hygienists?
- Does the book succeed in its goals?

Is the information technically and factually correct? Do references support the text?

Throughout the text Detienville makes statements that are not supported by citations. In particular, I found concern with his discussion regarding the prevalence of periodontal disease, infection control in periodontics, and in his discussion of aggressive periodontitis. Regarding prevalence, the chapter only makes reference to 4 studies but then generalizes the findings of these very different and geographically diverse studies to conclude that "8% to 15% are likely to develop an aggressive form of periodontal disease..." In addition, he states "low socioeconomic indices...seem to increase the risk for developing an aggressive form of periodontitis" but provides no citations or references to studies that had these findings.

Regarding infection control in periodontics, Detienville makes absolute statements about the survival rate of Actinobacillus actinomycetemcomitans, the use of local anesthesia only in the second phase of scaling and root planing, and the effects on biofilm to brief exposure to chlorhexidine, all without citation. In his discussion of aggressive periodontics he, again, makes absolute statements regarding etiology without citation and states "aggressive treatment [of aggressive periodontitis] under local anesthesia should be avoided because it may hinder cellular and tissue reorganization during tissue response and repair." I was unable to find support for this statement in the literature. A statement such as this should be supported with references.

Does The Book Cover Each Topic Adequately And Clearly?

Overall, I found this text to be confusing and poorly formatted. In his chapter on clinical signs and symptoms he states "a treatment is considered successful when it changes the intensity of all features of disease. Some signs are totally reversible, others are not," yet the very next line states " treatment is considered successful when all reversible signs of disease have disappeared." I would suppose that the intensity of an indicator could change without it having disappeared.

In his photo presentation of an individual with chronic periodontitis having a positive culture for Fusobacterium nucleatum his treatment regimen does not include the Metronidazole regimen that he suggests, "effectively suppresses these bacterial species" in his discussion of biofilms and bacterial complexes. Further, in his initial discussion of the use of the Amoxicillin/Metronidazole regimens, he discusses 2 different protocols within the same paragraph and then discusses a third protocol in a conclusion statement.

In addition, while he references the new classification system for periodontal disease, throughout the text he continues to use old terminology (eg, table of microbiology of healthy and pathologic periodontal tissue includes refractory periodontits).

His section on infection control in periodontics focuses mainly on the use of scaling and root planing, surgical intervention, antibiotic therapy (Amoxicillin/Metronidazole), and chlorhexidine. His chapter on adjunctive therapy focuses mainly on guided tissue regeneration, bone grafting, and splinting. There is no discussion about the local delivery of antimicrobials, the use of other systemic antibiotics, or the use of other agents such as NSAIDS, as adjunctive therapies.

Is the level of writing appropriate for dental hygienists?

I believe that overall, this text lacks detail in many areas, in particular the discussion of the pathogenesis of periodontal disease. I am confident that today's dental hygiene student (and yesterday's) will want a more comprehensive discussion of host response than what is contained in this book. In addition, he dedicates only 2 paragraphs to the discussion of periodontitis and systemic disease, and notes only the association of bacterial load and bacteremias as etiologic risk factors.

His statement, "in terms of efficacy, there is no difference between manual instrumentation and ultrasound devices," deserved more discussion, as well. While he referenced Sherman et al (1990) in his statement, "best results are obtained when both methods are combined," I believe the student or practicing dental hygienist would be better served by a more comprehensive discussion that referenced current literature regarding this matter (eg, American Academy of Periodontology position paper on Sonic and Ultrasonic Scalers in Periodontics, 2000).

His discussion of high-risk individuals covers only 2 and a half pages and dedicates only 1 paragraph to diabetes and 2 sentences to stress. He offers very little in his discussion of periodontal maintenance and includes tooth polishing as a procedure in periodontal maintenance. His discussion on daily maintenance therapy is very simple, and suggests that patients acquire new daily oral hygiene habits (eg, brushing 3 times daily, the use of interdental brushes) but provides no detail or direction regarding patient education methodologies or behavior change models. Finally the glossary in this text is very inadequate and provides only 1 page with 17 entries.

Does the book succeed in its goals?

Overall, I believe the goal of any dental hygiene text can be two-fold:

1. Furthering an understanding of the biology and current treatment methodologies regarding periodontal disease.
2. Furthering knowledge of methodologies and strategies that will foster behavior change in patients.

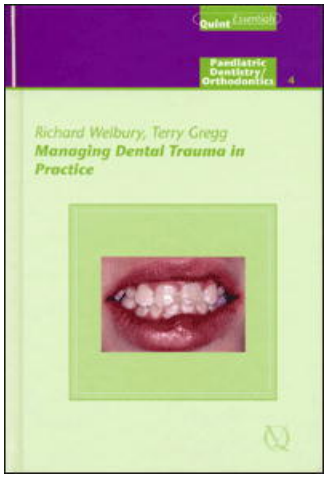
I don't believe that this text will benefit either the dental hygiene student or the practicing dental hygienist in accomplishing the aforementioned goals.

Source: Journal of Dental Hygiene, Vol. 80, No. 2, April 2006

Review of: Managing Dental Trauma in Practice 24th in the series Quintessentials of Dental Practice

Cindy Merback, RDH, MS

Reviewed by Cindy Merback, RDH, BS, clinical instructor.



Managing Dental Trauma in Practice, 24th in the series Quintessentials of Dental Practice

Welbury RR and Gregg TA

Quintessence, 2006

Britain

128 pages, illustrated, hardcover

ISBN: 1-85097-082-3

$54.00

This volume is the 24th in the Quintessentials for General Dental Practitioners Series and is dedicated to dental trauma across the life span, but most specifically targets the pediatric patient and as it states, the older patient. This well-organized book begins with a one-page table of contents describing the chapter contents and ends with an index as well as future editions in the series.

Managing Dental Trauma in Practice is published in London and references numerous British and Scandinavian professional journals and therefore its information is based on those populations, including sports and cultural activities of those regions. It does, however, make a general reference to global trauma seen in boys and girls. Dental trauma, including injuries to

the primary and permanent dentitions, is simply and clearly stated, with chapters often including before and after treatments. Each chapter is structured in an easy-to-read format, beginning with Aim, Objective, and Introduction to the topic, and ending with Prevention, Key Points, and Further Reading sections. Chapter topics include: History, Examination, Diagnosis, Treatment Planning, Primary and Permanent Dentition Injuries, Soft Tissue Injuries, Complicated Crown Fractures, Root Fractures, and Dento-alveolar Fractures.

The photographs complement the text and aid in the comprehension of the trauma and treatment prescribed. In addition, film representation is used to inform the reader about the extent and location of the traumatized area. The book expresses a clear defining line where treatment can be provided in the dental office or would require referral to a secondary treatment center. Trauma both intra- and extraorally is discussed and a special section is devoted to the identification of child abuse along with the role of the dental professional in child protection.

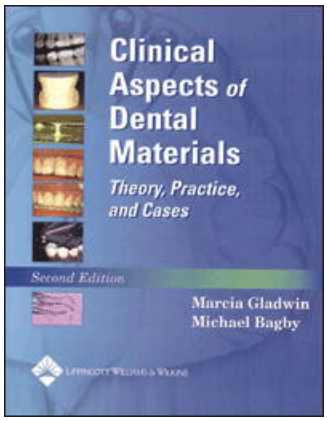
Managing Dental Trauma in Practice is an invaluable reference tool for all dental professionals as well an important read for parents and patients in the waiting room. There are multiple prevention strategies discussed that can serve to educate the staff as well as all patients. Sports trauma photographs, while graphic, are worthy of being shown to all parents who feel mouthguards are unnecessary. In addition, specific predispositions to trauma are mentioned, which can be used to alert patients to potential dental injury. Dental hygienists play a key role in educating patients about the prevention of dental trauma and this book will be an important asset in the teaching process.

Source: Journal of Dental Hygiene, Vol. 80, No. 2, April 2006

Review of: Clinical Aspects of Dental Materials: Theory, Practice, and Cases

Michele R Sweeney, RDH, MS

Reviewed by Michele R. Sweeney, RDH, MS, assistant professor of dental hygiene, West Liberty State College, West Liberty, West Virginia.



Clinical Aspects of Dental Materials: Theory, Practice, and Cases

Second Edition

Gladwin M and Bagby M

Lippincott, Williams, & Wilkins

Baltimore, Maryland

425 Pages indexed, soft cover

ISBN: 0-7817-4344-3

$49.00

The study of dental materials presents ever changing information due to constant improvements in techniques and material composition. Gladwin and Bagby's second edition of *Clinical Aspects of Dental Materials: Theory, Practice, and Cases* includes 5 new chapters, a new section of case studies, and edited former chapters to bring the dental hygiene profession up to date on the latest in dental material usage.

The text is divided into 3 sections followed by 2 appendices. The first 3 sections include 35 total chapters and 4 case studies. Part I has 22 chapters dealing with theoretical perspectives. Part II has 13 chapters covering laboratory and clinical

applications. Part III introduces 4 individual case studies examining aspects of dental materials through patient charting, photos, radiographs, and questions. Appendix 1 consists of answers and justifications to review questions found in each of the theoretical chapters found in Part I and the case studies in Part III. Appendix II consists of 35 skill evaluations that may be used in a laboratory/clinical setting.

Part I, with its 22 chapters, is written in an easy-to-follow outline form beginning with an introduction and concluding with a summary. Materials science and physical and mechanical properties are discussed in the first 2 chapters. Subsequent chapters include information on adhesives, direct polymeric restorative materials, amalgam, direct metallic restorative material, dental cements, impression materials, gypsums, removable and fixed prostheses, dental implants, specialty materials, polishing materials, tooth whitening, and oral appliances. In addition, chapters are included on clinical detection of restorative materials during scaling and polishing, infection control, disinfection of impression materials, dentures, and oral appliances, as well as general rules for handling dental materials. Each chapter includes behavioral objectives and a list of key words and phrases. Throughout the chapters the key words and phrases are highlighted in bold for the reader. Chapters conclude with a list of learning activities, review questions, and supplemental readings. Most supplemental readings are current (within the last 5 years). Some of the readings are reference materials (such as dictionaries) or respected texts that are somewhat older. Each of the 22 theoretical chapters covers the materials, their components, their history, and their correct usage. Photos, diagrams, illustrations, and charts further enhance comprehension of the subject matter. Black and white photos are of good to excellent quality. The chapters are written at an appropriate level for the dental hygiene student.

Part II follows with 13 chapters presenting laboratory and clinical applications of 13 different materials and procedures. This section is the cookbook of the text. Using the text as a "how to" guide can easily be accomplished through this lab and clinical application section. Each chapter is preceded by objectives and keywords and phrases. Throughout the chapters, those keywords and phrases are highlighted in bold. The chapters are written in an outlined format just as the theoretical chapters were organized Subjects include how to handle mixing liners, bases, cements, applying rubber dams, removing rubber dams, pit and fissure sealants, amalgam finishing, amalgam polishing, taking alginate impressions, fabricating study models, trimming study models, fabricating custom trays, elastomeric impressions, vital tooth whitening, debonding orthodontic resins, placing periodontal dressings, removing periodontal dressings, removing sutures, and temporary crowns. Photos and diagrams enhance the reader's comprehension of how to manipulate the dental materials. In each chapter there are handy boxed in areas titled "Tips for the Clinician," "Armamentarium," "Precautions," and "Summary." In the boxed section of "Tips for the Clinician," the authors review important hints that aid in properly handling the chapter's dental material. Armamentarium sections list the needed equipment and materials needed for each procedure. Precaution sections heed warnings about the vulnerabilities of materials. Summary sections review the manipulations and procedures discussed in the chapter. The chapters are easy to follow and give enough information to easily follow the steps for manipulating a material or completing a procedure. Laboratory and clinical application chapters conclude with supplemental readings that include many sources that are less than 5 years old.

Part III includes 4 patient case studies. The section is an excellent review for dental hygiene board case study questions. Each case asks a number of questions regarding restorations, appliances, or other elements dealing with dental materials. Questions are multiple choice and in a format similar to the dental hygiene board exam. Cases include a brief description of the patient, intraoral photos, charting, and/or study models. Each of the cases includes 5-7 questions. All answers and justifications for those answers can be found in Appendix 1 of the text.

Appendix 1 contains answers and justifications to the review questions at the end of the theoretical chapters in Part I. Appendix 1 also contains answers and justification to the case study questions in Part III. Questions in both of these parts are set up in dental hygiene board format. The questions in the chapters and the answers found in the Appendix will be a valuable study tool.

Appendix II will be a bonus for dental materials instructors. The Appendix contains 15 skill evaluations that can be implemented into a laboratory or clinical setting. Each page is perforated for ease of removal and use. Skills evaluated include mixing glass ionomer, mixing zinc-oxide eugenol, applying/removing rubber dams, pit and fissure sealants, amalgam finishing/polishing, alginate impressions, trimming/finishing study models, custom impression trays, elastomeric impressions, constructing bleaching trays, debonding orthodontic resins, placing/removing periodontal dressings, removing sutures, and constructing temporary crowns. The evaluations are set up as grids that include criteria, instructor, and student

evaluation. Each skill lists criteria needed for satisfactory manipulation of material or completion of procedure. The instructor and student may then choose to evaluate according to satisfactory or unsatisfactory performance, accordingly. Both lab and clinical competency levels can be identified on the form. Forms include areas for comments and instructor's signatures.

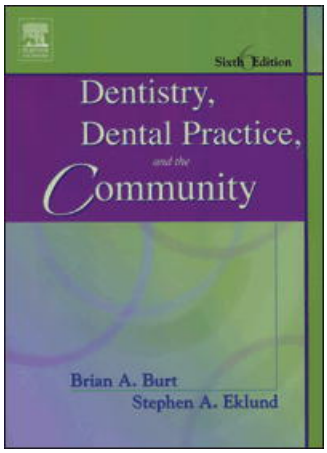
Clinical Aspects of Dental Materials: Theory, Practice, and Cases will be a valuable asset to both the dental hygiene student and faculty. Students will find the text easy to follow and comprehend. The educator will find the skills evaluations valuable for use in lab and clinical settings. The information provided by the review and case study questions will be of great help in preparing for the dental hygiene board exam. Gladwin and Bagby have presented this material in a way that can be easily digested for learning.

Source: Journal of Dental Hygiene, Vol. 80, No. 2, April 2006

Review of: Dentistry, Dental Practice, and the Community

Ruth Fearing Tornwall, RDH, MS

Reviewed by Ruth Fearing Tornwall, RDH, MS, Instructor IV at the Lamar Institute of Technology in Beaumont, Texas.



Dentistry, Dental Practice, and the Community

Sixth edition

Burt BA and Eklund SA

Elsevier Saunders, 2005

St Louis, Missouri

425 pages, illustrated, indexed, soft cover

ISBN: 0-7216-0515-X

$49.95

This is the sixth edition of *Dentistry, Dental Practice, and the Community*. The text provides the reader with a comprehensive overview of community oral health. Its purpose is to "present dentistry and dental practice against the backdrop of social events: economic, technological, and demographic trends, as well as the distribution of the oral diseases that dental professionals treat and prevent." The text is written for both dental hygienists and dentists. Like the previous edition, the authors' guiding principle is to lay out the facts on all matters discussed and interpret them as they see them. As a result, many changes have been made in the book to provide the reader with a comprehensive array of subject matter in dentistry. An exceptional feature of the book is its extensive references that provide the readers a chance to pursue further issues that interest them. The references become the basis for an interpretation of the more controversial issues.

The book has 30 chapters and is divided into 5 parts: Dentistry and the Community, Dental Practice, the Methods of Oral Epidemiology, the Distribution of Oral Diseases and Conditions, and Prevention of Oral Diseases in Public Health.

Part I includes 5 chapters and provides content about the dental professions and the public they serve while discussing such topics as ethics, the public-private partnership, public health practice, and health promotion. In Chapter 4, the authors do an excellent job of setting the stage for defining public health , the development of public health in the United States, dental public health, and differences between personal and community health care. Chapter 5, Oral Health Promotion, discusses oral health in the community and among individual patients. Furthermore, the chapter explains what is meant by health promotion and differentiates it from health education.

Part II includes 7 chapters and deals with the structure and financing of dental practices, types of personnel in the dental workforce, infection control and mercury safety, and a new chapter on access to dental care. The chapter on "Reading the Literature," is now attached to a new chapter on "Evidence-based Dentistry." Of interest in chapter 6, is the discussion of quality assurance, its evolution, the recent emphasis on increasing the quality of patient care, and cost control.

Part III includes 6 chapters on oral epidemiology, including information on research designs and survey methods for dental caries, periodontal diseases, dental fluorosis, other conditions and the various indexes used to measure oral disease. Chapter 13, Research Designs in Oral Epidemiology, provides a basic presentation of the essentials of valid research reports to prepare the reader to interpret epidemiological studies, particularly clinical trials, and of other studies involving human subjects.

Part IV, includes 5 chapters and presents content on the distribution of the oral disease in the population along with information on various risk factors. The first chapter in Part IV discusses loss of teeth, the end product of oral disease, the issues and trends in tooth loss, and the reasons why people lose teeth. Other chapters include content on dental caries, periodontal diseases, dental fluorosis, oral cancer, and other oral conditions. Each disease is extensively covered and provides a historical perspective as well as future trends in the area.

Part V of the book covers the prevention of oral diseases and conditions. It includes chapters on fluoride, fluoridation of drinking water, other uses of fluoride in caries prevention, fissure sealants, diet and plaque control, prevention of periodontal diseases, and restricting the use of tobacco. Each chapter comprehensively discusses the background and all the issues surrounding prevention of oral disease in public health.

Burt and Eklund have successfully fulfilled their purpose and their guiding principle in this new edition. This text is an important resource and has practical value for all dental hygienists involved in the dental field. The book is very readable and should be of interest to all dental professionals practicing in this complex environment we now live in.

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The Effect of Periodontal Therapy on TNF-α, IL-6 and Metabolic Control in Type 2 Diabetics

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Purpose.This pilot study investigated if scaling and root planing (S&RP) was an effective intervention in reducing levels of inflammatory markers TNF-α and IL-6 in a type 2 diabetic population

Methods. Twenty-five patients with type 2 diabetes, 18-64 years of age were enrolled having 4 or more sites with pocket depths ≥ 5mm and 2 or more sites with attachment loss ≥ 3mm. Participants received S&RP following collection of gingival crevicular fluid and serum which were analyzed for TNF-α and IL-6. After 3 months post-treatment levels were collected. Serum pre-and post-treatment levels were analyzed using a paired t test at a significance level of p≤ 0.05. Mean TNF-α was 1.7pg/ml at baseline and post-treatment was 4.0pg/ml. Mean IL-6 was 2.8pg/ml at baseline and post-treatment 6.0pg/ml.

Results. Both mean TNF-α and IL-6 were increased following S&RP ; however, the observed increases were not statistically significant. While participants improved on periodontal measures following therapy, systemic measures of inflammation (TNF-α and IL-6) did not show the hypothesized reductions.

Conclusion. Further studies are needed to determine effectiveness of S&RP on inflammatory mediators in a population with type 2 diabetes.

Keywords: type 2 diabetes, periodontitis, periodontal therapy, scaling and root planing, inflammatory mediators

Introduction

Diabetes is a metabolic disorder resulting in chronic hyperglycemia and hyperlipidemia that ultimately induces diverse multiple system pathologies, increasing the risk for atherosclerosis, coronary heart disease, stroke, myocardial infarction, renal disease and periodontitis.[1-5] Diabetes has become the fifth leading cause of death affecting approximately 17 million individuals or 6.2% of the population in the United States.[5,6] If left untreated or uncontrolled, diabetes will lead to heart disease, stroke, blindness, amputations, kidney failure, periodontal disease and death.[7]

Periodontitis is an infection caused by the gram-negative organisms in the plaque biofilm that affects 7% to 15% of the adult population.[8,9] An abnormal inflammatory response that is a hyper-inflammatory trait has been linked to diabetes where there is an increased susceptibility to infections such as periodontal disease.[10,11] The hyper-inflammatory trait is associated with an exaggerated secretion of inflammatory mediators (TNF-α and IL-6) and systemic markers of inflammation. It is suggested that this process mechanistically contributes to the pathology associated with this chronic disease process.[2-4,8,12,13]

Diabetes and chronic periodontitis are both common chronic diseases observed in a significant proportion of the adult US population.[3,4] It is well established that diabetes is a risk factor for poor periodontal health; however, recent studies have also suggested that periodontal disease adversely affects glycemic control in diabetics.[1-5,8,12] Diabetic complications have been attributed to the hyperglycemic state, which over time results in the irreversible covalent modification (glycosylation) of structural proteins and lipids that comprise the extracellular matrix and connective tissues, as well as the vascular tissues.[14-16] These structural changes result in impaired capillary function, poor blood perfusion of tissues and organs, and the release of reactive oxygen species (oxidative stress) triggering a systemic inflammatory process.[12,17] The activation of inflammation at a systemic level results in the chronic elevation of inflammatory mediators (IL-1, TNF-α, IL-6, and PGE_2) and acute phase reactants such as C-reactive protein, elevated fibrinogen, and lowered albumin, all hallmarks of the acute phase reaction (APR) observed in diabetes and periodontitis.[17-19] Thus, a hyper-inflammatory trait may predispose an individual to a more severe systemic disease that may occur as a result of over expression of inflammatory mediators and may ultimately lead to metabolic dysregulation.

The purpose of this study is to determine if periodontitis serves as a stimulus for a systemic-based inflammatory response that may represent a previously underestimated metabolic stressor, enhancing insulin resistance and impairing insulin secretion. Further, looking at the effect of non-surgical periodontal therapy (scaling and root planing (S&RP)) in patients with type 2 diabetes on the inflammatory mediators TNF-α and IL-6 and the relationship of these mediators to markers of insulin resistance may provide evidence of the importance to a successful periodontal treatment outcome on oral-as well as systemic-health.

Review of the Literature

Diabetes

Diabetes is a metabolic disease due to disturbances in insulin production resulting in abnormal fat, sugar, and protein metabolism producing a hyperglycemic state.[20] Insulin, a hormone produced by the pancreas, normally is released in small amounts on a constant basis. When a meal is consumed, insulin is then released in greater amounts. The body has the ability to remove the excess glucose and stores it in the liver and muscle or converts it to fat. , When needed, this stored glucose is released back into the blood stream, where insulin ushers the glucose back into the cells. In a patient with diabetes, this process changes; excess glucose builds up in the bloodstream because of insufficient insulin released by the pancreas or cells resistant to insulin.[7] Type 2 diabetes usually develops over time and typically involves reduced responsiveness of tissues to circulating insulin. Diabetes is often controlled by diet or hypoglycemic agents.[2-4,8,12] If not

controlled through blood sugar monitoring, healthy eating, and weight control, type 2 diabetes can contribute to an increased susceptibility to infection and inflammation such as that seen in periodontal disease.[2-4,8,12]

Prolonged exposure to hyperglycemia is the primary factor for the development of diabetic complications. The biochemical basis is Advanced Glycation Endproducts (AGEs). AGEs are chemically irreversible, glucose derived compounds that form slowly and continuously as a function of blood glucose concentration. AGEs accumulate in plasma and tissues of diabetic patients.[21,22] Macrophages have an affinity for AGE and usually help normal tissue turnover by binding to receptor advanced glycation endproducts (RAGE (a macrophage receptor)) activating the synthesis of TNF-α and IL-1. If the synthesis and secretion is increased, as in hyperglycemia, then connective tissue degradation occurs.[23]

The Centers for Disease Control (CDC) has stated that the number of Americans with diabetes is on the rise.[5] It is the fifth leading cause of death in America with approximately 800,000 new cases annually.[5] Global estimates by Zimmet and McCarty predict diagnosis of non-insulin dependent diabetes (NIDDM) at 216 million by 2010.[24] In 1986, Huse et al estimated the economic burden of NIDDM to be 19.8 billion dollars in the United States.[25] Other studies have estimated costs for all diabetics in the U.S. (NIDDM and IDDM) at 100 billion.[6,20,25-28] Overall, there are enormous costs related to the treatment and control of diabetes in this country.

It is estimated that there are 5.5 million type 2 diabetic cases that may remain undiagnosed until symptoms prevail.[28] Women, American Indians, Asians, Hispanics, and African Americans have shown an increased prevalence of type 2 diabetes.[7] Most cases of newly diagnosed diabetes may have had the disease for four to seven years, suggesting that undiagnosed diabetes may have adverse effects even though in a quiescent state.[29] There are risk factors for diabetes, including, gender, race, family history, and a sedentary lifestyle.

Type 2 diabetes was previously described as non-insulin dependent diabetes and was once considered a late-onset disease. Currently it is increasingly found in a much younger population.[5]

Biofilm

Plaque formation is the primary etiology for inflammation in periodontal disease. Plaque is comprised of several hundred bacterial species.[30] Dental plaque is a microbial biofilm that is formed by organisms tightly bound to one another and to the solid substratum by means of an exopolymer matrix into which they are embedded.[31] The bacterium in biofilm consists of gram-positive coccoid cells that divide and form microcolonies. Periodontal bacterial pathogens such as *B.forsythus*, *P.gingivalis*, *T. denticola*, *C.rectus*, *P.intermedia* cause the tissue to breakdown and hinder the healing response, thus increasing probing depths, bleeding, and bone loss.[31] After a few days of dental plaque growth, filamentous bacteria coaggregate to the initial colonizers and become embedded in a matrix composed of salivary components and high proportions of exopolysaccharides of bacterial origin.[32] To maintain the ecosystem, anaerobes anchor to each other by forming an aggregated bacterial mass.[33] Biofilms are complex and yield a challenge in understanding the many interactions between bacteria and substrate, and the bacterial components found in mature plaque.[34] Biofilms occurring in nature are firm clusters of bacteria adhering in layers to some kind of substrate.[35]

Bacteria are anchored to the tooth surface via a three-dimensional plaque matrix where more bacteria cluster and infiltrate, damaging tissue and destroying bone. The pellicle, a condensate of salivary proteins, forms first and then the bacteria adhere to that layer.[36] The bacteria then proliferate and communicate with each other.[37] Biofilm can form on restorations, implants, and hard and soft tissues soon after tooth debridement. Biofilm is difficult to remove with regular saliva flow, tongue movement, and antimicrobial agents.[35]

Supragingival plaque is distinct from subgingival plaque because it starts supragingivally and then progresses subgingivally.[34] Subgingival plaque repopulates rapidly and has been hypothesized to cause periodontal disease.[38] With the plaque growing

subgingivally and the bacteria disrupting the health of the tissues, deciding the course of effective treatment is of prime importance.

Understanding the makeup of the bacteria in plaque may provide a broader look at how periodontal disease can be stopped or controlled. There are at least 4 different approaches that can be taken: preventing biofilm formation, disrupting existing biofilms, preventing further biofilm growth, and killing specific organisms in the biofilm.[31] With this knowledge, planning a course of treatment for patients with type 2 diabetes may enhance their oral health and help to control the effect of diabetes on periodontal disease.

Periodontal Disease

Periodontal disease is a chronic inflammatory condition of gingival tissues causing destruction of periodontal tissues and loss of alveolar bone by *P. gingivalis*, and other anaerobic gram-negative pathogens.[10,19] These pathogens produce endotoxin lipopolysaccharide (LPS), a component of the outer membrane of bacteria. Periodontopathic organisms exhibit a number of virulence factors that enable them to evade neutrophil clearance and establish themselves as chronic subgingival inhabitants. Among these is LPS.[39] It is believed that when increased amounts of LPS are released it causes macrophages and fibroblasts to over-produce the inflammatory cytokines IL-6, IL-1, and TNF-α. This leads to the progression of periodontitis, which includes destruction of periodontal tissues, inflammation, and bone resorption, causing an immune response.[8,9,40] Once the bacteria has invaded the host clearance system, the host becomes exposed to an array of bacterial toxins. The interaction of the bacterial toxins with mononuclear phagocytic cells results in activation of an inflammatory cascade, with synthesis and secretion of TNF-α, IL-6, IL-1, and PGE_2.[41] Even though NIDDM and IDDM have different origins of disease (environmental versus genetic), chronic hyperglycemia in the presence of LPS is adequate for monocytic hypersecretion of cytokines and periodontal disease progression. The breakdown of connective tissue and alveolar bone in periodontal disease result mainly from an infection mediated pathway of cytokine upregulation.[42]

Hyperglycemia and hyperlipidemia have been pathologically implicated in complications of diabetes and periodontal disease. It has been shown that advanced glycation endproducts, which have formed as a result of hyperglycemia/hyperlipidemia, can alter the phenotype of cell types by receptor advanced glycation endproducts, a cell surface receptor. AGE then binds to RAGE and transforms macrophages into cells with a destructive phenotype producing inflammatory cytokines IL-1, IL-6, and TNF-α.[2-4, 8, 12] Periodontal infection-mediated cytokine synthesis and secretion may amplify the magnitude of the AGE-mediated cytokine response. As a result of the AGE/RAGE complex, there is a two-way relationship between diabetes mellitus and the infection caused by periodontal disease.[43]

Cytokines (IL-1, IL-6, and TNF-α) are soluble, biologically-active glycoproteins secreted by host immuno-inflammatory cells.[40] They have a role in the inflammatory process and are produced by lymphocytes, monocytes, macrophages, granulocytes, epithelial cells, endothelial cells, adipose tissue and fibroblasts.[44] Elevation of inflammatory mediators IL-6, IL-1, and TNF-α causes dysregulation of lipid metabolism and insulin resistance, thus breaking down gingival tissue, enhancing bone resorption by signaling osteoclasts and adding to long-term complications in the patient with diabetes.[8]

Acute infection results in the systemic challenge of pyrogenic cytokines, such as IL-1, TNF-α, and IL-6, which block lipoprotein lipase activity, resulting in decreased transportation of blood lipids from the circulating cells, eliciting hyperlipemia.[17,45,46] TNF-α promotes glycogenolysis and impairs glucose uptake by cells in the periphery, presumably by an effect on glucose transport receptor expression leading to hyperglycemia.[17,46] TNF-α and IL-6 target the hepatocyte to induce acute phase response (APR).[17,45,46] It is believed that these cytokines are detected in a periodontal lesion and can be heightened by the onset of diabetes.

Diabetes and Periodontal Disease

Historically, patients with diabetes have been shown to be at increased risk for infections. Increased periodontal risk is often related to the duration and adequacy of control of the diabetic state. It has been noted that individuals with type 2 diabetes have a three-fold increased risk of developing periodontal disease that can not otherwise be explained on the basis of age, sex, or oral hygiene.[47] Past and present studies have reported periodontal disease to be one of the most prevalent complications of diabetes.[2-4,48] The classic presentation of periodontal disease progression has been associated with accumulation of plaque and calculus on the tooth surfaces, and potent virulence factors produced by bacteria, causing destruction of periodontal tissues and resorption of alveolar bone.[10] Patients with diabetes have a compromised host response and ability to respond to bacterial infections, which in part, may increase their risk of periodontal disease. The reverse of this theory is that periodontal infections may exacerbate the diabetic condition.[42] Studies demonstrating the relationship between diabetes and the association of microbial organisms in prevalence and severity of periodontal disease show that the flora associated with diabetes does not appear to differ from patients without diabetes.[49] Patients who poorly control their diabetes and have periodontitis show an increase in progression of periodontitis. Patients who control their diabetes, receive timely care and control their blood sugar, are no more likely to develop severe periodontal disease than patients without diabetes.[2-4] The literature clearly supports that diabetes increases the risk for severe periodontitis and an increased incidence of periodontal disease progression by approximately 2 to 3 times than that observed in healthy patients.[2-4,12,50,51] Traditionally, these complications have been attributed to the hyperglycemic state, which over time, results in glycosylation of structural proteins and lipids that comprise the extracellular matrix and connective tissues, as well as the vascular tissues. These structural changes result in impaired capillary function and poor blood profusion, triggering a systemic inflammatory process. The activation of inflammation at a systemic level results in the chronic elevation of C-reactive protein (CRP), a hallmark of APR.[17,45,46] Studies of patients with diabetes typically demonstrate an elevation of APRs, which tend to correlate with the degree of glycemic control.[17,18,45,46] Thus, it has been generally hypothesized that elevated APR markers in type 2 diabetes are a direct consequence of diabetic metabolic dysregulation.[52] This suggests that periodontal disease may actually contribute to the development of metabolic imbalance, which may result in insulin resistance or impair insulin secretion and type 2 diabetes.[52] In a longitudinal study, participants with type 2 diabetes and severe periodontal disease at baseline demonstrated significantly worse glycemic control than participants with diabetes who have minimal periodontal destruction.[49]

Studies have shown that the Pima Indians, a population who have a high incidence of type 2 diabetes, rank as one of the highest populations in the world with this systemic disease.[53] They also have a high prevalence of periodontal disease.

Current Therapies for Treatment

Preventing dental plaque (biofilms), which holds the bacteria and causes destruction of the periodontal tissues, can be achieved by inhibiting the attachment of bacteria, altering the structure of deposit, or interfering with the pattern of plaque development.[54,55] Once the plaque deposits have formed, primary prevention could be accomplished through reducing existing plaque, preventing the formation of new plaque, selectively inhibiting particular bacteria associated with disease, or inhibiting the expression of virulence determinants.[56] Periodontal disease is a plaque-induced infection. Patients are generally not skilled at removing the plaque and are unable to remove subgingival deposits of plaque and calculus; thus, professional debridement and scaling is needed to maintain the health of the periodontium.[57] Not everyone responds to therapies or is able to maintain the health of the periodontium after S&RP. There are many reasons why traditional therapies do not control disease such as poor compliance with home care, patients not returning for regular maintenance visits, insufficient debridement by the clinician or reinfection by the bacteria, and above all, systemic diseases such as type 2 diabetes.[42,58-60] By maintaining regular visits for oral care and proper home care, the microbiota can be kept under control and damage to the periodontium will be decreased. Studies show that a reduction in inflammation after periodontal treatment will reduce the insulin a patient requires.[61] By including a therapeutic approach to reducing the bacteria the health of the diabetic patient may improve and reduce blood sugar levels.

Anti-infective therapy includes both mechanical and chemotherapeutics to eliminate or decrease biofilms. Mechanical therapy, supra- and subgingival scaling and root planing, consists of debridment of the roots by hand or power-driven scalers to remove plaque, endotoxin, calculus, and other plaque-retentive local factors.[62] Mechanical S&RP to remove plaque and calculus is essential to decreasing the inflammatory response. This therapy is increasingly important for patients with type 2 diabetes who have an increased susceptibility to inflammation.[38] Non-surgical periodontal therapies, such as S&RP and S&RP with antimicrobial therapies, have been shown to decrease periodontal disease progression.[42, 58] By using manual or sonic driven scalers in subgingival pockets there is a profound shift in the composition of microbial flora.[63-65] By effectively removing the endotoxins in the subgingival areas, healing of the tissues can occur and a reduction in probing and attachment levels can be attained.

To date, no one treatment has been truly successful. In a study by Christgau et al, HbA1c levels were measured prior to and after S&RP. The treatment group received S&RP and gingival curettage while the control group did not receive any treatment. In the treatment group the levels of HbA1c were measured pre- and post- treatment and then again at 9 months; HbA1c levels improved by 6.7% in the control group and 17.1% in the treatment group. This study reported that mechanical therapy had no effect overall on levels of HbA1c.[66] A study by Westfelt et al looked at the maintenance of patients with diabetes 5 years after S&RP and found no alterations in HbA1c levels.[67] Rodrigues et al found that S&RP and S&RP in combination with amoxicillin/clavulanic acid reduced HbA1c levels, especially in patients who had an increased severity of diabetes and periodontal disease. The HbA1c levels were taken at baseline and after 3 months following therapy. Both groups improved but the group with antimicrobial therapy had a greater reduction in HbA1c values. Thus, the use of antimicrobial therapy along with S&RP was found to improve the levels of glycemic control in patients with type 2 diabetes.[44] Stewart et al did a retrospective study and found that there was an improvement in glycemic control in participants with type 2 diabetes mellitus following S&RP.[68] Antimicrobial therapy, minocycline gel, when introduced along with S&RP, decreased TNF-α, subgingival bacteria, and HbA1c levels; probing and attachment levels were also reduced.[69] There are no studies where the levels of TNF-α and IL-6 have been assessed following S&RP. Most studies that investigated S&RP and its effect on patients with type 2 diabetes' oral health did show that S&RP alone improved probing and attachment levels but they did not assess inflammatory mediator levels.[43,66,67] Many of the studies that investigated TNF-α and IL-6 levels studied adjunctive therapies with antimicrobials.[44,66,70] Thus, there is a need to look at the effect S&RP has on decreasing the levels of inflammatory mediators.

Investigators are also taking a closer look at the role of glycemic control. While it is well established that S&RP can influence levels of HbA1c, what is not clear is how S&RP influences levels of inflammatory mediators. For example, TNF-α has been identified as a strong antagonist to the cell surface insulin substrate.[71] This activity, by TNF-α blocking of the insulin receptors, can contribute to the level of insulin resistance by inhibiting glucose transport and insulin action.[19] Recent evidence has suggested that chronic infection via periodontitis can influence insulin resistance.[50,72] Therefore, it is hypothesized that toxins from subgingival bacteria can produce chronic increases in inflammatory mediators such as TNF-α, which have been implicated in patients with type 2 diabetes and inflammation. This increase in inflammatory mediators is believed to increase the severity of diabetes and negatively influence diabetic control.

The overall objective of this pilot study was to improve the understanding of the mechanisms by which infection contributes to the metabolic dysregulation associated with the diabetic state and to provide primary prevention strategies. The magnitude for additional research initiatives and clinical interventions for diabetes is very apparent with expenditures climbing higher and higher and no cure available. Years of research has produced evidence about the increased frequency and severity of periodontal disease in patients with type 2 diabetes.[2-4,8,12,48] Recent studies have reported that a controlled periodontal condition may positively influence a patient with diabetes' glucose level.[2-4,8,12] If this is the case, it is of great interest to explore the inflammatory response in the patient with diabetes, mechanisms to control it, and the possibility that reduction of periodontal disease through mechanical therapy might lead to a better control of blood glucose. This research will contribute to the body of knowledge within the national dental hygiene research agenda for promoting health in a population with type 2 diabetes. It will also enhance the knowledge of how inflammation affects diabetes, specifically insulin resistance

and inflammatory mediators TNF-α and IL-6, and the long term effects that this plays on total body health. In obtaining a greater understanding of diabetes and periodontal disease, dentists and hygienists can better inform patients with type 2 diabetes about nonsurgical periodontal therapies.

Methods and Materials

Twenty-five participants with type 2 diabetes were recruited through advertisements and e-mail messages. All participants had to have type II-IV periodontal disease as defined by the American Academy of Periodontology (AAP).[73] Participants could not have had periodontal therapy (scaling and root planing or periodontal surgery) within 6 months prior to enrollment in the study.

Participants were first appointed for a screening visit to determine eligibility in the study. If eligible, a full-mouth series of x-rays (FMX) were taken to determine bone loss. Participants were then appointed for three additional visits. Inclusion and exclusion criteria were as follows.

Inclusion Criteria:

1. 4 or more sites with probing pocket depths ≥ 5mm.
2. 2 or more sites with attachment loss ≥ 3mm.
3. 18-64 years of age.

Exclusion Criteria:

1. < 20 teeth.
2. Systemic disease (systemic lupus erythematosis, HIV, AIDS).
3. Immunosuppressive Therapy (Cortisone, steroids, cancer chemotherapy).
4. Recent periodontal surgery or scaling and root planing (S&RP) within the past 6 months.
5. Chronic liver disease including Hepatitis.
6. BMI ≥ 40.
7. Pregnant.
8. Current abuse of alcohol or drugs.

Procedures

Medical and dental information was collected at the screening visit and updated at each subsequent appointment. At visit 1 and visit 2, participants fasted for a minimum of 6 hours. Blood was collected for serum to analyze TNF-α and IL-6, HbA1c, fasting insulin, and glucose. Gingival crevicular fluid (GCF) was collected to assess TNF-a and IL-6. The periodontal evaluation consisted of plaque index (PI),74 gingival index (GI),74 probing depths (PD), bleeding on probing (BOP), and clinical attachment level (CAL) measured from cementoenamel junction (CEJ) to gingival margin (Figure 1). After visit 1, participants were reappointed for the treatment visit (S&RP) in which anesthetic was administered by the dentist, if the patient requested it. After treatment was completed, participants were reappointed for visit 2; this occurred 3 months after treatment (S&RP) was completed. Visit 2 consisted of the same treatment as in visit 1. Oral hygiene instructions were not provided and compliance with home care was not followed. The following is a list of all the procedures that were conducted during each appointment (Figure 2).

Figure 1.

Clinical Indices Performed on each Participant

1. Plaque Index (PI): supragingival plaque recorded on an ordinal scale of 0-3[74]
2. Gingival Index (GI): the degree of buccal inflammation recorded on an ordinal scale 0-1[74]
3. Probing Depth (PD): the distance from the free gingival margin to the base of the pocket measured in millimeters
4. Bleeding on Probing (BOP): the presence or absence of bleeding recorded as 0 (no bleeding) or 1 (bleeding present)
5. Clinical Attachment Loss (CAL): measurement from the cementoenamel junction to the gingival margin
6. Gingival Crevicular Fluid (GCF): a fluid that is found in small amounts in the gingival crevice.

Figure 2

Screening

1. Determined if patient was eligible for the study through probing depths and CAL
2. Informed consent, HIPPA
3. FMX

Visit 1*

1. Fasting blood draw for serum TNF-a and IL-6, HbA1c, fasting insulin and glucose
2. Probing Depths (PD)
3. Clinical Attachment Loss (CAL)
4. Gingival Index (GI)74
5. Bleeding Index
6. Plaque Index74
7. Oral Examination (caries, oral lesions, missing teeth)

*Collected by examiners

Treatment

1. 1. Treatment (S&RP) with or without anesthetic

* Registered dental hygienist completed S&RP

Visit 2*

1. Same as visit 1.

*Collected by examiners

This study utilized three examiners who were all calibrated to the gold standard and to each other as set forth by University of North Carolina-Chapel Hill School of Dentistry and based on the kappa statistic.[75] Two examiners were research dentists (examiner 1 and examiner 2). A dental hygienist with 7 years of clinical experience performed all scaling and root planing procedures. The dental hygienist (clinician) and examiner 2 performed all treatment (S&RP). The examiners were calibrated to themselves (intra-rater) as well as to a gold standard and to each other (inter-rater), and percent agreement and kappa scores were determined.

Gingival Crevicular Fluid

GCF was taken at visit 1 and visit 2 in 4 quadrants starting with the distal of the second premolar, then the mesial and distal of the first molar and then the mesial of the second molar. If these teeth were unavailable, the teeth in succession with each other were utilized (the next more mesial tooth in proximity). Each site was assayed for TNF-α and IL-6. Each site was sampled using sterile cotton forceps and a strip of PerioPaper (Ora Flow, Inc. Plainview, New York). The PerioPaper was grasped by the orange strip and inserted into the gingival sulcus until a resistance was felt. Care was taken to ensure that contamination by food or blood was limited. The PerioPaper remained in the sulcus for approximately 10 seconds or until the paper was beginning to look saturated. The PerioPaper was removed from the sulcus and inserted between the calibrated Periotron (Ora Flow, Inc., Plainview, New York) sensors. The sensors were closed and a reading was obtained after a 16-second measuring cycle. The reading obtained was between 30 and 180. Any readings that were below 30 or over 180 were thrown out and a new reading was obtained. After the proper reading was obtained, the PerioPaper was wrapped in aluminum foil to prevent evaporation. The wrapped aluminum foil was placed in a cryovial, which had been labeled for each patient enrolled in the study and then immediately placed in a canister containing liquid nitrogen. The canister of liquid nitrogen was used to transport the GCF samples to the Center for Oral and Systemic Disease, where the samples were stored at -80°C until they were analyzed.

Results

Patient Population

Upon approval by the University of North Carolina School of Dentistry Institutional Review Board, 28 participants were enrolled in the study. However, 2 participants did not return for post-evaluation, one subject did not complete the initial clinical evaluation and collection (visit 2), and one patient did not fast for visit 2; thus, complete data was collected on 23 participants. Nevertheless, because some data was collected on 25 participants, some variables will have an N =25. See Table 1.

Table I. Demographics of Participants

		n	Percent	Mean/SD
Gender	Male	12	48%	
	Female	13	52%	
Race	African Am.	14	60.87%	
	White	9	39.13%	
BMI		24		33.05±7.03
Age (years)		25		49.92±8.41

Clinical Measurements

A paired *t* test was used to determine significance in the mean change between the baseline and post-treatment values for all clinical parameters. Significant improvements were found in all clinical parameters. Mean probing depths at baseline were 3.1 and post-treatment was 2.4 (p=0.02). Mean attachment loss at baseline was 3.0 and post-treatment 2.4 (p=0.05). Mean plaque index was 0.77 at baseline and 0.44 at post-treatment (p=0.01). Mean gingival index was 1.1 at baseline and 0.85 at post-treatment (p=0.04). Mean bleeding on probing was 0.57 at baseline and 0.42 at post-treatment (p=0.0005). (See Table II and Figure 3.)

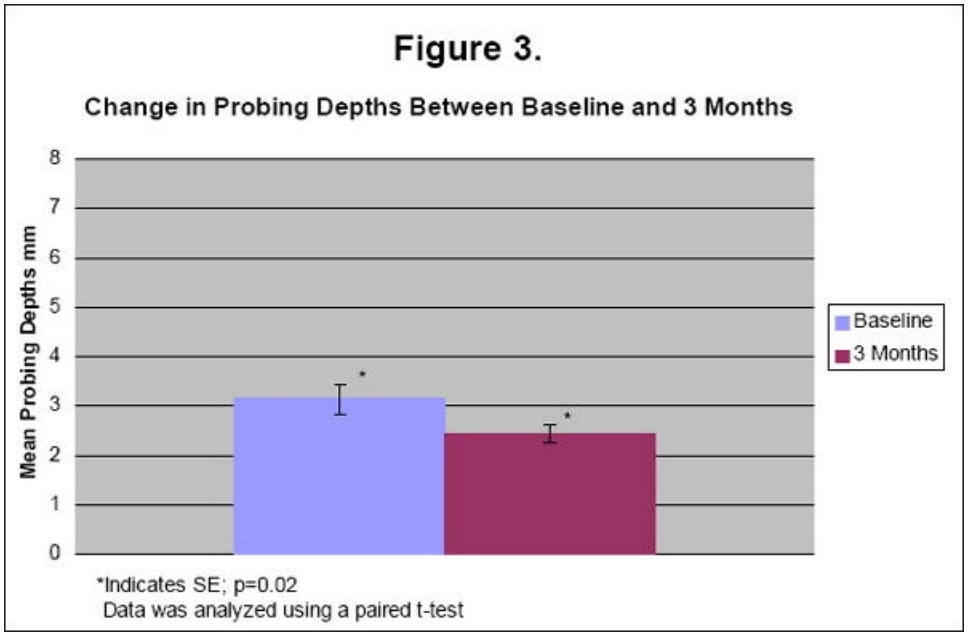


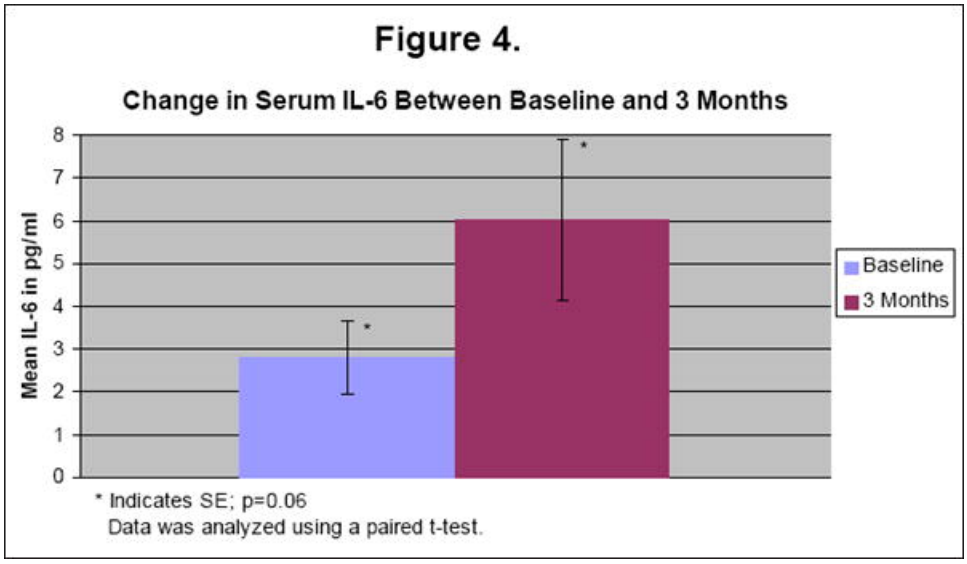
Table II. Baseline and Post Treatment Values For Outcome Variables

Baseline and Post-Treatment Values For Outcome Variables				
	n	Baseline Mean/SD	3 Months Mean/SD	P value
TNF-α	21	1.7±4.34	4.0±13.6	p=0.32
IL-6	23	2.7±3.9	6.0±9.0	p=0.06
HbA1c	25	8.2±1.8	8.3±1.9	p=0.66
Fasting Insulin	25	18.5±18.6	21.2±16.1	p=0.33
Fasting Glucose	25	177±66.9	193±70.0	p=0.24
Homa	25	134±126	161±128	p=0.34
Attachment Loss	24	3.0±1.6	2.4±1.1	p=0.05*
Plaque Index	24	0.77±0.55	0.44±0.35	p=0.01*
Gingival Index	24	1.10±0.58	0.85±0.63	p=0.04*
Bleeding on Probing	24	0.57±0.30	0.42±0.25	p=0.0005*
Probing Depth	24	3.1±1.5	2.40±0.90	p=0.02*

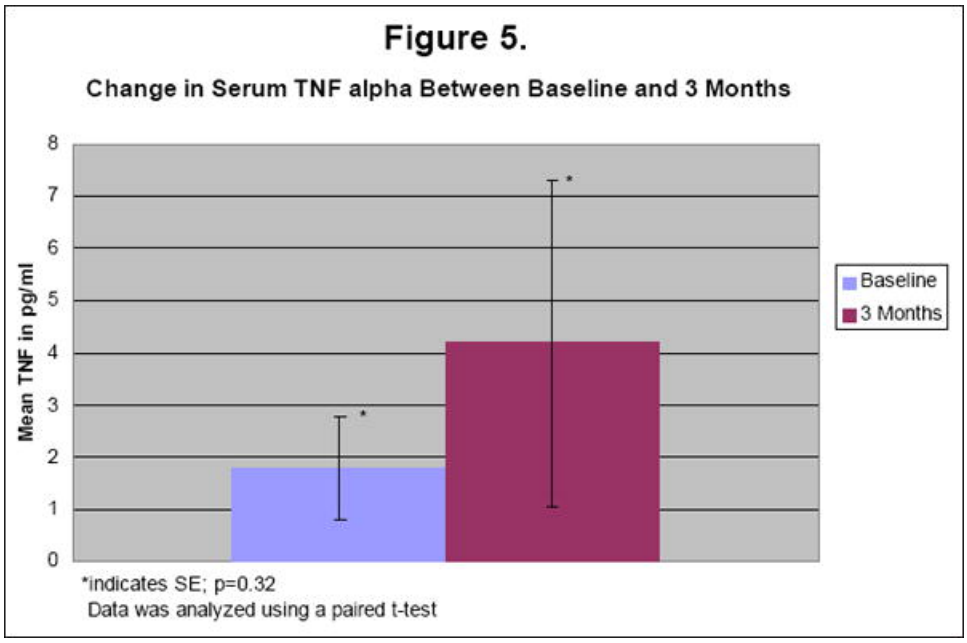
*alpha=0.05; Data was analyzed using a paired t-test

Serum TNF-α and IL-6

Mean IL-6 levels at baseline were 2.8 pg/ml and post-treatment levels were 6.0 pg/ml. Statistical analysis using a paired t test was used to look at the change in serum IL-6 levels from baseline to post-treatment. The observed increase in IL-6 was not significant (p=0.06). (See Figure 4.)



TNF-α mean levels at baseline were 1.8 pg/ml and post-treatment 4.2 pg/ml. Statistical analysis was done using a paired t test to look at the change in serum TNF-α levels from baseline to post-treatment. No significance was shown p=0.32. (See Figure 5.)



Discussion

This study attempted to assess the effect non-surgical periodontal therapy (scaling and root planing (S&RP)) would have on inflammatory mediators TNF-α and IL-6 and the relationship of these mediators to markers of insulin resistance. The results of this pilot study showed non-significant increases in TNF-α or IL-6 pre- and post-treatment. Statistical significance was shown in reduction of pocket depth, gingival index, attachment loss, plaque index, and bleeding on probing.

Patients with uncontrolled type 2 diabetes with periodontal disease are systemically stressed by the bacteria in plaque, the increase in circulating inflammatory mediators, and an increase in insulin resistance. In this study the clinical periodontal parameters improved, while the patients did not show improvement in measures of systemic inflammation (IL-6 and TNF-α). One plausible explanation for this apparent contradiction is that S&RP produced systemic stress because bacteria were released into the blood stream. As a result, the patient's systemic inflammatory system may have tried to combat the inflammatory process. The alternative explanation for the improvement in periodontal clinical parameters while the systemic mediators appeared to be increasing is that the observed results occurred purely by chance.

In this study the mean Body Mass Index (BMI) for participants was 33. Because TNF-α and IL-6 are produced in the adipose tissue, the higher BMI may have increased the participants metabolic stress, which could have caused the elevation in TNF-α and IL-6. Visceral obesity is quite common in patients with type 2 diabetes and could be a contributing factor to why there was not a reduction in these mediators nor HbA1c levels. Nishimura et al studied levels of TNF-α in correlation to obesity levels of participants and found that there was a correlation between weight and mediator levels.[50]

The addition of an adjunctive antimicrobial agent to the S&RP regimen may have produced a different effect on the bacteremia theory. Previous studies of patients with type 2 diabetes have shown positive results with the addition of local and systemic drugs which decreased the serum levels of TNF-α and IL-6. Iwamoto's study reported a decrease in serum levels of TNF-α, circulating insulin concentrations and HbA1c levels in a diabetic population by placing 10 mg of local minocycline in every pocket and mechanical debridement of plaque once a week for a month.[69] As Grossi and Genco reported in a review of the literature, S&RP alone showed improvement in periodontal status but when systemic antibiotics plus S&RP were incorporated there was an improvement in blood glucose levels, a decrease in gram-negative bacterial levels and a reduction in inflammatory mediators.[42]

In future studies, having a larger patient sample size to assess serum levels of IL-6 and TNF-α could possibly increase the significance of serum TNF-α and IL-6. In this study, serum was assessed prior to scaling and root planing and then again 3 months after scaling and root planing. By having a shorter sample time period between treatment and re-sampling, more information could be provided on TNF-α and IL-6 as it relates to patients with type 2 diabetes.

In addition to having a shorter interval between S&RP and post-treatment, oral hygiene instructions may have enhanced or provided more significant results. Most patients have poor compliance or lack the motivation needed to succeed with oral hygiene aids.[58] By providing reinforcement of oral hygiene aids prior to, during, and post- S&RP, the patient may begin to develop the understanding of how important regular home care is and its role in decreasing the progression of periodontal disease thus controlling the diabetic state.

A control group was not utilized in this pilot study. Control groups decrease error and reduce bias. A control group is an asset to any study because it provides data on a healthy population that can be compared with a population with disease. The present study sought to collect pilot data on the effects of S&RP on inflammatory mediators TNF-α and IL-6 in a population with type 2 diabetes. A future study will incorporate an experimental and control group to assess the differences.

This study has shown that further investigations are needed to look at what role TNF-α and IL-6 have on the markers of insulin resistance in the population with type 2 diabetes. In future studies, increasing the sample size, adding a control group, having a run-in period where patients would serve as their own control to show stability of the measures during the run-in period, and the addition of additional therapies would be an enhancement to the study design.

Conclusion

It has been recognized that scaling and root planning (S&RP) is an effective therapy in reducing pocket depths and decreasing bleeding on probing, thus restoring periodontal health to the patient and then in turn decreasing insulin resistance.

This pilot study investigated the effect of S&RP in patients with type 2 diabetes on inflammatory mediators TNF-α and IL-6 and the relationship of these mediators to markers of insulin resistance. A second objective was to determine if periodontitis serves as a stimulus for systemic-based inflammatory response that may represent a previously underestimated metabolic stressor, enhancing insulin resistance and impairing insulin secretion. At visit 1 and visit 2, clinical assessments along with gingival crevicular fluid, and serum samples were taken to examine TNF-α and IL-6 levels.

The results of this study show that S&RP is a beneficial treatment in decreasing the clinical periodontal parameters; but, in this population, systemic reduction of TNF-α and IL-6 was not significant. These results could have been affected by an already systemically stressed population of participants. Performing S&RP could have increased the participants' systemic stress load through the release of bacteria into their bloodstream.

Further studies are needed to assess the effect of non-surgical periodontal therapy to inflammatory mediators TNF-α and IL-6 and how they affect the markers of insulin resistance in patients with type 2 diabetes.

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Notes

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Characteristics of Adolescent Smoking in High School Students in California

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Purpose.This pilot study assessed smoking-related behaviors, experiences, and beliefs among a sample of continuation high school students in California to inform dental hygienists about adolescent tobacco use and to assist with the development of effective tobacco cessation intervention strategies.

Methods. After gaining informed consent, we conducted a self-administered questionnaire among 117 adolescent volunteers in rural northern California. The questionnaire assessed demographic variables, lifetime tobacco use, current alcohol use and tobacco use status, early smoking experiences and sensations, factors that might motivate a quit attempt, depression index scores, and other psychosocial variables associated with adolescent smoking. Means and frequencies were generated to evaluate characteristics of tobacco use in this sample of adolescents. Variables of interest were stratified by regular and social smokers. Univariate association of smoking with alcohol use is described by an odds ratio with a 95% confidence interval. Wilcoxon rank sum tests were used to evaluate gender differences among mean depression index scores.

Results. Fifty percent of subjects were current smokers, 21% had tried smoking, 5% were former smokers, and 24% had never smoked. Current smokers were 8 times more likely to drink alcohol compared to nonsmokers (OR = 8.0; 95% CI 3.1 - 21.2). Among current smokers, 32% were classified as regular smokers and 18% as social smokers. Sixty-three percent of regular smokers and 42% of social smokers reported smoking within 30 minutes of waking, an indicator of nicotine dependence. Patterns of smoking were variable in rate and frequency ranging from 1 to 30 cigarettes per day. Current female smokers scored higher on the modified Beck Depression Inventory (0-3) than current male smokers (p < 0.001). Oral health issues related to tobacco use, such as gum disease and tooth staining, were identified as factors that might motivate a quit attempt. Frequently reported reasons for use (eg, tension and craving) and reasons for relapse (eg, desire remained high, withdrawal symptoms) were related to nicotine dependence. Fifty-three percent of all smokers had tried to stop smoking but were unsuccessful. Intrapersonal characteristics such as risk-taking, rebellion, and impulsive spending appeared to be related to smoking.

Conclusion. In this sample of adolescents, smoking patterns were variable and many adolescents experienced symptoms of nicotine dependence. The dental hygiene care appointment provides a unique opportunity to discuss oral health effects of smoking, relate oral changes to smoking, and to deliver a brief smoking cessation intervention.

Keywords: Adolescence, smoking, smokeless tobacco, spit tobacco

Introduction

Tobacco use increases risk of lung disease, cancer, and heart disease.[1] In addition, smoking is highly associated with oral cancer,[1] periodontal disease,[2] failure of periodontal therapy,[3,4] failure of osseointegration of dental implants,[5] dental caries,[6] oral pain,[7] and decreased oral wound healing.[8] The dental hygiene care appointment provides dental hygienists with a "teachable moment" to discuss oral health effects of tobacco, relate oral changes to tobacco use, and to deliver a brief tobacco cessation intervention. For example, the oral cancer screening and the periodontal assessment provide a unique opportunity to ask all patients whether they use tobacco, advise users to quit, assess readiness to quit, assist with the quitting process, and arrange follow-up contact to check on the client's progress with stopping tobacco use.[9] Dental hygienists are well-suited to provide effective tobacco cessation services since they are educated in oral health promotion and disease prevention, including behavioral motivation and health education.[10, 11] In addition, because they come in contact with clients over an extended period of time, dental hygienists have the opportunity to provide repeated reinforcement, which is essential for tobacco users who often experience cyclic periods of abstinence followed by relapse.[9,11]

Review of the Literature

Although the prevalence of smoking among adults has declined from 40.4% in 1965 to 22.5% in 2004,[12] an estimated 3000 children and adolescents become regular smokers every day.[13] National epidemiological data on adolescent tobacco use indicates that 27% of 12th graders, 18% of 10th graders, and 11% of 8th graders report smoking in the past month.[14] Overall, nationally, 22% of high school students currently smoke cigarettes and 11% of high school adolescent males use oral snuff or chewing tobacco, also known as spit (smokeless) tobacco (ST).[15] Moreover, prevalence of smoking is higher among students who attend continuation high schools (vocational, technical, or alternative schools) compared with same-age students enrolled in academic high schools.[16]

Smoking among adolescents is a significant issue due to the fact that the initiation of smoking at a young age is correlated strongly to an increased risk of being a smoking-addicted adult.[17] Not only are those who begin to smoke before the age of 18 more likely to smoke longer and more frequently than those who start smoking after the age of 18, but they also have been identified as a group that has an increased difficulty in smoking cessation.[18] Since the negative health effects of smoking accrue the longer that one smokes, stopping smoking early in life is one of the best ways to reduce health risks.[18] As oral health care professionals, dental hygienists often see teenagers, a client population that is less likely to see other health professionals on a regular basis.[19] This contact provides an opportunity to provide a brief tobacco cessation intervention. The adolescent smoking cessation treatment field, however, is in its infancy and the literature addressing adolescent smoking cessation is limited.[20] Dental hygienists may need to address adolescents as a unique population since adolescence is a stage of life with numerous psychological, social, and physical changes and experimentation.[20] In addition, unlike adult smoking, there are more environmental constraints on youth smoking such as fines, school suspension, family consequences, and limited access.[20]

The purpose of this pilot study was to learn more about tobacco use among adolescents. In a sample of adolescents, we specifically assessed their patterns of tobacco use, reasons for use, symptoms of nicotine addiction related to first use and current use of tobacco, factors that would motivate quit attempts and reasons for relapse, and other psychosocial variables related to tobacco use. To inform dental hygienists about adolescent tobacco use and to assist with the development of effective tobacco cessation intervention strategies for adolescents, this paper reports the results of that pilot study.

Methods and Materials

This cross-sectional descriptive study was approved by the Institutional Review Board (IRB) at the Committee on Human Research at the University of California, San Francisco. The population for this study consisted of a convenience sample of male and female continuation high school students in Lake County, a rural area of California. The high school principal was contacted to explain the study and to gain permission to recruit students at the school to participate in a 40 minute to 60 minute, self-administered questionnaire. After agreeing to allow the school to participate in the study, the principal mailed consent forms to all parents of students with a cover letter explaining the purpose, benefits, risks, and methods of the study and provided a toll-free number for parents to call to have their questions answered by a study investigator. Parents who did not want their child to participate in the study were instructed to sign the refusal statement on the consent form and return it to the principal by a specific date. No parents refused consent for study participation. At least 2 weeks after the parental consent deadline, teachers at the high school made announcements in their classes and study investigators posted signs at the school indicating dates and times when study-related meetings would be held outside of class time. At the meetings, study investigators explained the study, answered questions, obtained student informed consent, and provided pizza and soft drinks. All eligible students (ie, those with no parental refusal forms) who signed and returned a consent form were enrolled in the study and completed the questionnaire under the supervision of a study investigator. Number 2 pencils were passed out to students for use in completing the questionnaire and a standardized paragraph of instructions on how to complete the questionnaire was read to the assembled students prior to their completion of the questionnaire. Only one experienced investigator administered the questionnaire. Thus, no study investigator calibration in administering the measurement instrument was conducted. At the end of the assessment session, all questionnaires were reviewed by the study investigator to ensure completeness.

Attached to the questionnaire was a face page where the name of each study participant was collected. After completing this face page, students were instructed to separate it from the questionnaire and return it to the investigator prior to completing the questionnaire. The face page and the questionnaire were linked by coded identification numbers to ensure confidentiality of questionnaire responses. Matching names on face pages to consent form signatures ensured that individuals participating in the study had provided informed consent to participate in the study.

Questionnaire Measures

The questionnaire items included several self-report measures developed by our group and used over numerous studies, as well as, several self-report measures used by other investigators in other studies. Specifically, questionnaire items assessed demographic factors (ie, age, gender); lifetime tobacco use (ie, cigarette, cigar, chewing tobacco, dip/snuff with 4 response options ranging from "never" to "100+"); and current alcohol and tobacco use (defined as use within the last 30 days). Students also were asked about the age they first began using tobacco regularly and their frequency of use per day. In addition, the questionnaire assessed early and current smoking experiences and sensations, level of nicotine dependence, sensations experienced when unable to smoke, factors that might motivate a quit attempt, history of quit attempts and reasons for relapse, reasons for smoking, and other psychosocial factors associated with adolescent smoking. The following more specifically explains these latter categories and variables assessed on the questionnaire.

Early tobacco use experiences and sensations

Early tobacco use experiences were assessed by asking students to "Think back to the time when you first began experimenting with tobacco, and answer the following questions as best you can remember." The list of questions asked about age first tried; where first tried (response options were "Home," "School," "On the way to school," "Don't remember," and "Other"); and with whom first tried (response options were "Friends/peers," "Family," "Alone," "Don't remember," and "Other").

In addition, students were asked about sensations experienced when tobacco was first tried. A list of the following potential sensations were presented: "Pleasant sensations," "Unpleasant sensations," "How much nausea you experienced," "How much dizziness you experienced," "How much of a rush or buzz you experienced," "How much coughing you experienced," and "How much difficulty inhaling you experienced." Response options for each item were: "None," "Slight," "Moderate," "Intense," and "Don't remember."

Current tobacco use experiences and sensations

To assess current tobacco use experiences and sensations and to compare them with those reported for early tobacco use, we asked similar questions as those asked for early use (described above).

Level of nicotine dependence

Level of nicotine dependence was assessed by asking students to estimate how many minutes after they wake up in the morning do they usually have their first cigarette, dip, or chew. Use of tobacco within 30 minutes of waking[21,22] has been reported to be a measure of nicotine dependence in tobacco users. In addition, students were asked to estimate how many minutes they could go without using tobacco without encountering a problem.

Sensations experienced when unable to smoke

To assess sensations experienced when unable to smoke, a list of the following potential sensations were presented: "Depressed mood," "Trouble falling asleep," "Irritability/frustration/anger," "Anxiety," "Difficulty concentrating," "Restlessness," "Increased appetite/weight gain," "Loneliness," "Headaches," and "A racing heart". Students also were asked to rate the extent to which they experienced each of the sensations when unable to use tobacco due to either restriction on using tobacco or because they were trying to quit. Response options were "Not at all," "Mild," "Moderate," or "Severe."

Factors that might motivate a quit attempt

Motivational factors that might influence the student to stop using tobacco in the next 3 weeks were assessed by presenting a list of possible situations and asking students to check "Yes" or "No" if the situation might motivate them to try to stop their tobacco use.

History of quit attempts and reasons for relapse

To assess personal quitting history, the students were asked the following questions. Have you ever tried to quit before ("Yes" or "No")? If so, how many times? Have you ever tried to quit for more than one month ("Yes" or "No")? If so, how many times. Have you ever tried to quit for more than a week, but less than one month ("Yes" or "No")? If so, how many times? Reasons for relapse were assessed by presenting a list of statements and asking students to indicate "Yes" or "No" if each statement applied to their starting to use tobacco after having quit for a period of time.

Reasons for smoking

To assess reasons for smoking, we used a modified version Horn's Smoker's Self-Test using the Tomkins model.[23] The questionnaire presented a list of reasons for smoking and asked students to indicate how much each statement applied to their smoking (four possible responses: "Not at all," "A little," "Quite a bit," and "Very much so"). Each statement listed was measured on a scale of 0 to 3, with a score of "0" indicating "Not at all," and a score of "3" indicating "Very much so."

Psychosocial factors associated with adolescent tobacco use

In addition, as described below, students were asked about personal opinions and feelings about themselves. To assess depression, the students were asked questions based on the modified Beck Depression Inventory.

Personal opinions and feelings

A list of statements reflecting students' personal opinions and feelings about themselves was presented and students were asked to indicate "Yes" or "No" if each statement applied to their perception of themselves.

Depression

The questionnaire, using the modified Beck Depression Inventory,[24] presented a series of 20 situations and asked about the frequency the students may have experienced each. The purpose was to assess whether a student might be suffering from depressive symptoms. Responses to items presented were: often (score = 3), sometimes (score = 2), rarely (score =

1), or never (score = 0). Response scores associated with each item were combined into a mean score used to classify adolescents as having or not having depressive symptoms.

Overall, the questionnaire consisted of 37 items. However, many of the items had subsections, so that in total, 176 responses were required. For example, item 3 assessing lifetime use of tobacco products had 4 subsections; item 4 on current use of tobacco products had 5 subsections; item 15 on early experiences and sensations with cigarettes had 8 subsections; item 23 on experiences and sensations with cigarettes in a typical day during the past year had 8 subsections; item 24 on factors that might motivate adolescents to stop smoking had 15 subsections; item 25 on reasons for smoking had 41 subsections; item 31 on sensations experienced when unable to smoke had 10 subsections; item 35 on reasons for relapse had 13 subsections; item 36 on personal opinions and feelings about themselves had 52 subsections; and item 3, the modified Beck Depression Inventory, had 20 subsections.

Data Analyses

Current smokers were categorized into regular smokers (ie, self-reported smoking on at least 22 days of the previous 30 days) or social smokers (ie, self-reported smoking on at least one, but no more than 21 days of the previous 30 days). Regular and social smokers were compared by early smoking experiences and sensations, level of nicotine dependence, sensations experienced when unable to use tobacco, factors that might motivate a quit attempt, personal quitting history, reasons for relapse, reasons for smoking, and other psychosocial factors (ie, personal opinions and feelings and depression). We computed means and frequencies and generated descriptive tables. Since this is a pilot study, we did not screen all variables for statistical association or employ statistical modeling. However, as a measure of the association between smoking and the likelihood of alcohol use, we calculated an odds ratio (OR) and a 95% confidence interval (CI). An odds ratio is a measure of the strength of a hypothesized association. Odds ratios that are significantly different than one (that is, the lower and upper bounds of the 95% CI exclude 1.0) provide evidence of a statistically significant association. To assess for a significant gender difference in mean depression index scores, we employed the Wilcoxon rank sum nonparametirc test. Data processing was accomplished utilizing the software package Epi Info, version 6.04b.[25]

Results

A total of 117 high school adolescents (51 males, 66 females) with a mean age of 16 years participated in the study. Table I shows lifetime history of cigarette, cigar, and spit (smokeless) tobacco (ST) (ie, oral snuff or chewing) use for adolescents in the study. Twenty-five percent reported never smoking a cigarette in their lifetimes with no gender differences (male = 24%; female = 24%). In contrast, more females than males reported never using ST (male = 55%; female = 92%). The mean age of initiation of smoking cigarettes was 12.1 years (n=83; SD=2.4) and of using ST was 13.7 years (n=26; SD=2.2). Overall, 61% (71) reported use of alcohol in the last 30 days.

Table I. Lifetime use of various forms of tobacco and mean age started.

	None		1-19		20-99		100+		Age Started (Yrs)
Type	%	(n)	%	(n)	%	(n)	%	(n)	Mean
Cigarette	25	(29)	21	(25)	9	(11)	44	(52)	12.1
Cigar	44	(52)	33	(39)	16	(19)	6	(7)	13.5
ST*									
female	92	(61)	8	(5)	0	(0)	0	(0)	14.4
male	55	(28)	31	(16)	10	(5)	4	(2)	13.6

*oral snuff & chewing tobacco also known as spit (smokeless) tobacco (ST)
row percentages may not add to 100 due to rounding

The distribution of participants by self-reported smoking status is shown in Table II. Fifty percent (59) reported smoking in the last 30 days and were classified as either regular or social smokers based on number of days they had smoked in the last 30 days. In our entire sample of continuation high school students, 32% (37) were classified as regular smokers and 18% (22) were classified as social smokers.

Table II. Self-reported smoking status

Tobacco Use Status*	**Male** (N=51) %	**Female** (N=66) %	**Combined** (N=117) %
Regular Smoker[+]	37	27	32
Social Smoker[+]	24	15	18
Trier	16	24	21
Former	0	9	5
Never Smoker	24	24	24

[+]Smoked within the past 30 days
*Four subjects that reported current smokeless tobacco use also currently smoked and are included in the regular or occasional smoker groups.
column percentages may not add to 100 due to rounding

Patterns of smoking were variable in rate (number of days they smoked in the past month) and frequency (the number of cigarettes smoked per day). For example, among the 3 students who reported they smoked 30 cigarettes per day (the highest number reported), 2 reported having smoked on all of the last 30 days and one reported smoking on only one day of the last 30 days. Overall, among regular smokers, the mean number of cigarettes smoked per day was 13.5 and duration of use was 4.6 years. Among social smokers, the mean number of cigarettes smoked per day was 11.5 and duration of use was 3.4 years. Sixty-three percent of regular smokers and 42% of social smokers reported smoking within 30 minutes of waking, an indicator of heavy nicotine dependence. (Data not shown in a table.)

In addition, 83% of current smokers also drank alcohol in the past 30 days, while only 38% of nonsmokers drank alcohol. Current smokers were 8 times more likely to drink alcohol compared to nonsmokers (OR=8.0; 95% CI 3.1-21.2). Among current smokers, 4 (6%) also reported current ST use. The mean age of smoking experimentation (ie, first began experimenting with cigarettes) for current smokers was 10.3 years. (Data not shown in a table.)

Table III presents mean depression index scores separately for current smokers, former smokers, triers, and never smokers as measured by the modified Beck Inventory of Depression.[24] This Inventory uses a scale of 0 to 3, with a score of 3 indicating the most depressive symptomatology. In Table III, mean depression scores, of the entire study sample overall and stratified by gender, are presented under the category entitled "All Students." Overall, within our sample of adolescents, there is a statistically significant gender difference in depression scores. Males had an overall mean depression index score of 0.78 which was significantly lower than the overall female depression score of 1.19. Upon further examination, however, when data were stratified by smoking status, the main gender difference was found to occur only among smokers, regardless of number of days smoked in the last 30 days. In contrast, there is no difference in mean depression scores between males and females who never smoked. These findings suggest that females may be more likely to smoke to cope with depression compared to males.

Table III. Mean Beck Depression Index (BDI) scores by smoking status

	Male	Female	All Students
	Mean Score (0-3) (n)	Mean Score (0-3) (n)	Mean Score (0-3) (n)
• **Current Regular Smoker**** (smoked on > 21 of last 30 days)	**0.75** (19)	**1.39** (18)	**1.07** (37)
• **Current Social Smoker**** (smoked on <22 of last 30 days)	**0.67** (12)	**1.20** (10)	**0.91** (22)
• **Former Smoker** (lifetime > 100 times)	**NA** (0)	**1.65** (6)	**1.65** (6)
• **Triers** (lifetime < 100 times)	**0.68** (8)	**0.95** (15)	**0.85** (23)
• **Never Smoked Tobacco**	**1.01** (12)	**1.01** (16)	**1.01** (28)
All Students**	**0.78** (51)	**1.19** (65)	**1.01** (116)

** Wilcoxon rank sum test for gender difference in BDI for all students: $p < 0.01$

Although we do not have a large enough sample for gender comparisons among former smokers, the 6 female former smokers scored the highest mean depression index (1.65) of any of the groups analyzed. Finally, a tendency toward a gender difference also is observed among adolescents who reported that they had tried smoking in the past, but were not ever regular or social smokers. Males who had only tried smoking tended to have depression scores similar to male current regular and social smokers. In contrast, within the female group who had only tried smoking, depression scores were similar to those females who had never smoked. Thus, in general, only female current smokers and former smokers scored high on the index for depression.

Table IV compares early and current smoking experiences among the 59 current smokers in the study sample. Most reported that during early smoking experiences, they smoked with peers outside the home such as at a friend's house (19%) or at some place other than home (20%). Only 29% reported smoking at home. In addition, only 2% reported smoking on the way to school and nobody reported early smoking experiences at school.

Table IV. Comparison of early and current smoking use experiences (N=59)

Where	Early	Current
	%	%
Home	29	56
Friend's house	19	0
Way to school	2	20
School	0	5
Other	20	5
Don't remember	19	2
Missing	12	12
Whom		
Friends/Peers	61	22
Family	19	14
Alone	10	49
Other	0	0
Don't remember	0	3
Missing	10	12

column percentages may not add to 100 due to rounding

During current smoking experiences, however, 56% reported that they smoked at home and 49% reported they smoked alone. No one reported that they smoked at a friend's house, but 20% said they smoked on the way to school, and 5% smoked at school. These findings suggest that over time, smoking becomes less social and increasingly more associated with smoking alone, in the morning (eg, on the way to school), and smoking in places where it is forbidden (eg, at school), suggesting a transition to nicotine dependence.

Moreover, as shown in Table V, both regular and social smokers reported experiencing negative sensations when they first tried smoking, however, compared to social smokers, a higher percentage of regular smokers recalled pleasant and relaxing sensations (25% vs. 43% and 37% versus 54%, respectively). Report of current sensations experienced revealed a decrease in negative sensations experienced (ie, symptoms of nicotine toxicity, such as, nausea and dizziness) for both groups, suggesting the development of adaptation to smoke irritation and physiological tolerance to nicotine. In addition, from the time smoking was first tried to the time of current use, pleasant and relaxation sensation increased in both groups, suggesting reinforcement for continued use.

Table V. Percentage of regular smokers (RS) and social smokers (SS) reporting moderate/intense sensations experienced when first tried and current use of cigarettes

Sensations	When First Tried		Currently	
	RS	SS	RS	SS
	%	%	%	%
Pleasant	43	25	46	50
Relaxation	54	37	81	47
Buzz	62	63	24	6
Unpleasant	30	25	19	0
Coughing	42	50	24	6
Nausea	38	44	14	0
Dizziness	65	75	11	6
Difficulty Inhaling	30	31	8	13

Table VI shows that over one-half of regular smokers experienced symptoms of nicotine withdrawal (eg, irritability, frustration, anger, and anxiety) when unable to smoke compared to about a one-third of social smokers. In general, regular smokers were about twice as likely as occasional smokers to experience withdrawal symptoms when unable to smoke.

Table VII ranks the categories of reasons for smoking from highest to lowest mean scores and stratifies by regular and social smokers. Findings indicate that tension reduction (mean = 2.15) followed by craving (mean = 1.46) and pleasure (mean = 1.15) are the main reasons reported for smoking.

Table VII. Reasons for current smoking* (N=53)

	Regular Smoker Mean score (N=37)	**Social Smoker** Mean score (N=16)		**All Current Smokers** Mean score (N=53)
Reduce Tension Four items	2.46	1.43		2.15
Crave Smoking Three items	1.72	0.82		1.46
Pleasure Seven items	1.21	1.00		1.15
Handling Four items	0.94	0.55		0.82
Habit Four items	0.87	0.62		0.79
Stimulation Seven items	0.92	0.41		0.77
Social Six items	0.74	0.59		0.69
Weight Control Three items	0.71	0.53		0.67

*Modified Horn Scale (0 = 'Not at all' to 3 = 'Very much so')

Factors that might motivate quit attempts among male and female smokers are shown in Table VIII. Overall, cosmetic and health factors were themes most often cited. Social factors (eg, friend preferred) were less influential. For example, over one-half of respondents indicated that perceived smoking-related facial disfigurement (89%), gum disease (87%), bad breath (55%), and stained teeth (53%) might motivate a quit attempt.

Table VIII. Factors related to tobacco use that might motivate a quit attempt among all current smokers (N=53)

Factor	%	n
Face seriously disfigured	89	47
Gum disease	87	46
Cancer	81	42
Sexual impotence	79	42
Stomach ulcer	74	39
Face wrinkles	73	38
Harm to others	72	38
Infertility	66	35
Change in voice	65	34
Free quit program	59	31
Girl/Boy friend asked	58	30
Bad breath	55	29
Stained teeth	53	28
Cough/Shortness of breath	46	24
Friend preferred	17	9

Fifty-three percent of all smokers (58% of regular smokers and 40% of social smokers) reported they had tried to stop smoking for more than a week, but less than a month. The mean number of times respondents tried to do so was 5.2. In addition, 49% (41% of regular smokers and 69% of social smokers) had tried to stop smoking for more than one month, but less than a year. The mean number of times respondents had tried to do so was 2.9. Finally, 32% of all smokers (32% of regular smokers and 31% of social smokers) had tried to stop smoking for more than one year. The mean number of times reported they had tried to do so was 1.5.

Reasons for relapse are reported in Table IX. The most frequently mentioned reasons were "continued desire for tobacco use" and "withdrawal symptoms" among regular smokers, and "boredom" and "personal problems" among social smokers.

Table IX. Percentage of regular smokers (RS) and social smokers (SS) reporting reasons for relapse

Reasons	**Regular Smoker** (N=34) % Yes	**Social Smoker** (N=16) % Yes
Desire Remained High	77	44
Withdrawal Symptoms	74	38
Personal Problems	71	63
Boredom	69	73
Enjoyment/No Good Substitute	68	31
Pressure From Friends	59	25
Job Pressure	41	31
Forgot Quit Resolution	41	31
Quitting Disrupted Life	32	19
No Health Problems	27	6
Concern About Weight Gain	21	27
Actual Weight Gain	21	25

Personal opinions and feelings about oneself as reported by regular, social, and never smokers are shown in Table X. Overall, more smokers than never smokers described themselves as thrill-seeking, rebellious, risk-taking, unmotivated,

lacking confidence, impulsive, and tense. There was a dose-response relationship related to rebellion and the experience of tension. Compared to regular smokers, however, social smokers described themselves more frequently as fun-seeking.

Table X. Percentage of regular (RS), social (SS) and never smokers (NS) who agree with statements of personal opinions and feelings about themselves

Opinion-related Personal Characteristic	Item	% Reporting True		
		RS (N=37)	SS (N=22)	NS (N=28)
Thrill Seeking	I often try new things just for fun or thrills even if most people think it is a waste of time.	68	82	46
Rebellious	I often break rules and regulations when I think I can get away with it.	62	48	29
Risk Taking	I usually stay calm and secure in situations that most people would find physically dangerous.	57	55	36
Lack of Self Confidence	I often avoid meeting strangers because I lack confidence with people I do not know.	38	36	61
Complacency	I am satisfied with my accomplishments and have little desire to do better.	42	23	21
Impulsive Spending	Because I so often spend too much money on impulse, it is hard for me to save money.	65	68	39
Tense, tired, worried	It is extremely difficult for me to adjust to changes in my usual way of doing things because I get so tense, tired, or worried.	49	36	22

Discussion

The purpose of this pilot study was to learn more about smoking among adolescents in order to inform dental hygienists about tobacco cessation intervention and treatment in this population. We found that smoking patterns in our sample of adolescents were variable in both rate and frequency. Sixty-three percent of current smokers in our sample were regular smokers and 37% were social smokers. Moreover, some regular smokers reported smoking only 1 or 2 cigarettes a day, and some social smokers reported smoking relatively large quantities per day but on only a few days. Our findings are consistent with those of others who report that compared with adult smokers, adolescent smokers are more likely to be sporadic or nondaily smokers, and to have more variable smoking patterns on days they do smoke.[20]

In general, in our study, regular smokers were twice as likely as social smokers to experience withdrawal symptoms when unable to smoke, and 63% of them reported smoking within 30 minutes of waking, and indicator of heavy nicotine dependence.[21,22] Nevertheless, over one-third of social smokers also reported experiencing nicotine withdrawal symptoms such as irritability, frustration, anger, and anxiety when they were unable to smoke, and 42% reported smoking within 30 minutes of waking. These findings support those reported by others that many adolescent smokers begin to experience symptoms of nicotine addiction early in their smoking careers even when they are smoking only sporadically or

occasionally.[26,27,28,29] Baker and colleagues[30] suggest that adolescence may be a time when the individual has greater vulnerability to nicotine dependence. This suggestion is supported by animal studies reporting that adolescent rats acquire nicotine self-administration behaviors much more readily than adult rats.[31,32] These findings suggest that processes involved in central nervous system maturation may play a critical role in the development of nicotine dependence.[30]

Moreover, in our study sample, tension reduction was the primary reason for smoking, and symptoms of nicotine addiction, was the main reason cited for relapse when trying to quit. Fifty-three percent of all smokers in our study sample had tried to stop smoking for more than a week, but less than a month. These data are consistent with those reported by others[33,34,35] suggesting that adolescents' interest in quitting is relatively high, but they may not be successful at quitting on their own. The evidence for nicotine dependence among smokers in our study sample suggests the need for nicotine replacement therapy to help adolescent smokers stop smoking. On the other hand, however, Hurt et al[36] reported very low quit rates for adolescents trying to quit using the nicotine patch. Further research is needed to clarify the effect of various types and dosages of nicotine replacement therapy on adolescent smoking cessation.

Adult tobacco cessation treatments usually include adults who have regular, daily patterns of smoking. Our findings support the notion, however, that adolescents who smoke less than daily may be in need of smoking cessation treatment. In our sample, even social smokers reported symptoms of nicotine dependence, and as a result, may be at risk of becoming an adult smoker. Others have reported that even adolescents who smoke at relatively low levels (eg, only a few cigarettes a month) have a high probability of becoming regular adult smokers. In a large sample of adolescents, Chassin et al[37] found that the probability of adult smoking varied by smoking level in adolescence. Adolescents were assessed when they were approximately 15 years of age and assessed again about 7 years later when they were young adults (mean age of 22 years). Findings indicated that adolescents who had more experience with smoking were more likely to be adult smokers; yet, 25% of adolescents who had only smoked 1 or 2 cigarettes also became adult smokers (defined as smoking in the last week).

Given that the adolescent population is so vulnerable to long term tobacco use, access to them becomes important for early intervention to prevent initiation of smoking and to promote smoking cessation. Even though as an oral health care professional, dental hygienists are more likely to see teenagers than other health professionals on a regular basis,19 adolescents are a dental population that often falls through the cracks. To gain increased access to this group, dental hygienists, either individually or through their ADHA local components, could target local health fairs and/or vocational high schools to provide tobacco prevention and cessation community service programs for this population.

In our sample, the mean number of cigarettes smoked per day was somewhat similar among both regular and social smokers, (13.5 and 11.5, respectively) translating into about a half a pack per day. These findings are different than those of others[38] who reported that frequent adolescent smokers were 16.9 times more likely to report smoking at least half a pack of cigarettes on the days they smoked compared with nonfrequent adolescent smokers. In their study, frequent smokers were defined as those who smoked cigarettes on 20 or more days in the 30 days preceding the survey. In our study, however, regular smokers were defined as those who smoked on at least 22 days of the last 30 days. This difference in classification of regular/frequent smokers may explain the discrepancy between studies.

There are few studies on gender differences in tobacco cessation among adolescents. In a study of 1430 adolescent smokers (50% male, 50% female) from randomly selected classrooms from 24 California and Illinois high schools, male and female tobacco users did not differ in reasons for quitting, quit stage, or perceived likelihood of ever quitting smoking. However, a greater percentage of females than males reported that situations related to negative affect and nicotine withdrawal would tempt them to smoke.[39] In our study, adolescent female smokers scored higher on the modified Beck Depression Inventory[24] than adolescent male smokers. This finding suggests that depression in female smokers may need to be managed in a tobacco cessation program targeting adolescents.

Situations that might motivate quit attempts mentioned by a majority of smokers in our study sample included tobacco-related adverse effects on physical attractiveness, sexual potency, and oral health and hygiene factors, such as gum disease, stain, and bad breath. These findings are consistent with studies reporting that adolescents consistently rank physical attractiveness, dental concerns, and oral health as being of great importance.[40,41,42] Such findings are highly relevant to dental hygiene

practice. They suggest that when advising a tobacco-using adolescent to stop smoking, relating smoking to adverse effects associated with attractiveness and oral health may be more relevant and meaningful to an adolescent smoker than relating smoking to long-term health effects such as cardiovascular or lung diseases. Dental hygienists are well positioned to point out tobacco-related oral health and hygiene problems in the mouths of adolescents who smoke or use other forms of tobacco. In addition, when assessing the head and neck region, the dental hygienist could address tobacco-related facial wrinkling and aesthetic concerns as key areas of education. Incorporating this feedback as part of a brief tobacco cessation intervention in the dental hygiene care setting may encourage adolescents to try to stop smoking. Several studies have demonstrated that dental professionals are effective in helping their patients stop using tobacco when they point out spit tobacco-related lesions in the client's own mouth and provide brief tobacco cessation counseling.[43,44,45,46,47]

Consistent with previous research,[48] there was a strong association between smoking cigarettes and alcohol use in our sample of adolescents. This finding is of concern because the strongest data associating smoking with oral cancer were obtained in alcohol users.[49,50,51] Smokers who combine heavy smoking with heavy drinking of alcohol have been reported to have almost a 50 times greater risk of getting oral cancer than smokers who do not use alcohol.[50] Educating adolescents about the increased risk of oral cancer and related facial disfigurement among smokers who also use alcohol is a key patient education point to be addressed during the oral cancer screening and is consistent with the important role of the dental hygienist in oral cancer detection.

Compared to never smokers, smokers in our study tended to describe themselves as more thrill-seeking, rebellious, risk-taking, unmotivated, lacking confidence, impulsive, and tense. These findings are consistent with those of Gilpin and colleagues[52] who reported that rebelliousness was a characteristic of high school students that significantly predicted initiation of smoking among baseline never smokers.

Several limitations of the present study, however, must be considered when interpreting the present data. First, this study involved a small convenience sample of adolescents rather than a large random sample. Thus, one cannot generalize the results to all adolescents in rural areas of California or elsewhere. The outcomes reported, however, inform about patterns and correlates of smoking in a sample of high school students and suggest some important methodological issues to consider in smoking treatment outcome studies. In addition, the small sample size dictated limited data analysis with little power for detecting significant differences.

Moreover, while cross-sectional surveys are useful for tracking trends in smoking behavior, they cannot characterize completely which adolescents will transition from never smoking to smoking and from adolescent smokers to adult smokers. Longitudinal studies are needed to explore various predictors of adolescent smoking. Recent statistical advances in modeling[53] have been used in a small number of longitudinal studies to identify multiple age-related trajectories of smoking behavior among adolescents.[54,55,56] These trajectories have included an early tobacco-onset group (onset at ages 12 to 13) that shows a steep rise to heavy smoking; a late-onset group (onset after age 15) that smokes at more moderate levels; an experimenter group that tries smoking in adolescence but does not proceed to daily smoking and is developmentally limited to adolescence; and a group that quits smoking. Chasin and colleagues[37] point out that distinguishing smoking trajectories is an important methodological advance because it has the potential to illuminate diverse etiological pathways underlying different trajectories of tobacco use.

Another study limitation is that the self-reported smoking status in this study was not verified by biochemical assay and may be subject to under- or over-reporting. As anti-smoking norms become more pervasive in society, especially in California given its comprehensive tobacco control program, respondents may have been reluctant to admit to regular tobacco use. Nevertheless, although it is ideal to validate self-reported smoking status, biochemical validation is not feasible in adolescents who smoke infrequently.[57]

Finally, our findings are limited by the fact that our study was conducted among vocational technical students who are at higher risk for high smoking intensity compared with same-age students enrolled in academic programs.[16] Thus, our results may not apply to students in mainstream high schools. We chose to work with continuation high school students due to

the high prevalence of smoking reported in this population[16] to maximize exposure of our questionnaire to as many tobacco users as possible given limited time and resources.

The Public Health Service Guidelines for brief clinical interventions[9] recommend that all individuals seeking oral health care be asked if they use tobacco. Tobacco users should be advised to quit, assessed for willingness to quit, assisted appropriately based on willingness to quit, and follow-up arranged. Although the critical first step is to "ask" all clients their tobacco use status, a study of adolescents by Steinberg and colleagues[38] found that only 40.2% and 19.1% of adolescents studied reported being asked whether they smoked by their physician and dentist, respectively. Health care providers' advice to stop tobacco use has been shown to be an important motivator for patients to quit smoking.[9] Dental hygienists could not only deter experimental smoking among adolescents by discussing the dangers of addiction, but also could provide referral for treatment for highly dependent smokers.

Conclusion

Our pilot data suggests the following. (1) A large number of adolescents in rural continuation high schools in California smoke because of nicotine addiction and experience nicotine withdrawal symptoms when they are unable to smoke. As a result, use of pharmacological aids as a component of effective cessation treatment of adolescents needs further study. (2) There may be a need to define smokers in adolescent populations more broadly than "smoking in the last week" because many adolescent smokers appear to be sporadic or non-daily smokers with variable smoking patterns on days they do smoke. (3) There may be a need to intervene with social smokers, as well as regular smokers, among adolescents because they are at risk of becoming nicotine dependent and thus also are likely to become adult smokers; (4) Tobacco cessation treatment and measurement of cessation for adolescent smokers may need to be tailored specifically for adolescents because patterns of smoking among adolescents appear to differ from those of adults. (5) Lastly, dental hygienists can play an important role in preventing adverse health effects by promoting cessation of tobacco use among adolescents to whom they provide care. Pointing out oral health and hygiene problems caused by tobacco use in adolescent smokers' own mouths may motivate a quit attempt and prevent them from becoming addicted adult smokers, which puts them at high risk for the associated adverse health effects.

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Notes

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Validity of Oral Health Screening in Field Conditions: Pilot Study

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Purpose. This small pilot study examined the validity of visual dental hygiene screenings (VDHS) in conditions found in local communities.

Methods. A sample of 126 children in kindergarten through second grade was screened by 2 dental hygienists and inspected by a dentist. None of the assessors had more than minimal experience and training in epidemiological methodologies. Two denal hygienists noted teeth as decayed or not decayed using only a tongue blade and a goose-neck lamp (VDHS). The dentist noted decay by tooth and surface using a mirror, explorer, portable dental chair, as well as a goose-neck lamp; this examination is referred to as a mirror, tactile dental inspection (MTDI). The dentist's assessment (MTDI) was the "gold standard." Data were analyzed using frequency distributions, sensitivity, specificity, and kappa coefficient statistics, as well as other statistics to test the significance of differences and to investigate explanations for discrepancies between the VDHS and MTDI.

Results. Sensitivity and specificity for the VDHS for all teeth were 61% and 96%, with a kappa coefficient of 0.6. Analysis of the discrepancies between the VDHS and the MTDI suggest that, for primary teeth, the sensitivity of the VDHS is greater when: (1) lesions are large (i.e. multi surface) and (2) single surface lesions are located anteriorly. No statistically significant explanations were found for differences in permanent teeth.

Conclusion. In this study, VDHS demonstrated high specificity and moderate sensitivity for caries identification.

Keywords: dental caries, dentists, dental hygienists, oral health, mass screening, sensitivity and specificity

Introduction

Dental hygienists have a long history of providing oral health screenings in community settings, particularly in schools. However, the accuracy of these screenings has not been investigated relative to the standards of treatment by dentists in local communities. The intent of this small study was to investigate the validity of oral health screenings by dental hygienists in field conditions. Specifically, the study examined how well local hygienists could accurately classify school children as either having or not having decay, with reference to standards of treatment by dentists in the local community. This information would enable one to determine how well dental hygienists evaluate children who need dental care without over-referring.

Review of the Literature

Dental hygienists have been providing screenings for over 75 years. A 1927 book on school health elucidates this responsibility: "She [dental hygienist] makes thorough and detailed mouth examinations and records the needs of each individual."[1] In 1949, Williams and Abernathy stated that dentistry created the profession of dental hygiene to address the dental health aspect of school health after it became apparent that the prevalence and incidence of caries made it impractical to insist that only dentists conduct dental examinations in schools.[2]

Oral health screenings in schools are still needed. According to national data, low-income school-aged children have 1.2 to 2.2 times more decayed teeth than do their more affluent peers and while low-income children receive some dental treatment, it is insufficient to meet their needs.[3] In Iowa, as of 1999, 17% to 23% of low-income children aged 7-9 had untreated decay, as did 9% to 13% of non low-income children.[4] Furthermore, among those aged 6-11, 72% of low-income children had a dental visit in the past year compared with 92% of non low-income children. This 20% difference probably underestimates the number of low income children who had routine "check-up" visits to identify early carious lesions and prevent painful problems.

The American Dental Association identified four types of dental examinations:[5]

Type 1 Complete examination, using mouth mirror and explorer, adequate illumination, thorough roentgenographic survey.

Type 2 Limited examination, using mouth mirror and explorer, adequate illumination, posterior bitewing roentgenograms.

Type 3 Inspection, using mouth mirror and explorer, adequate illumination, and

Type 4 Screening, using tongue depressor, available illumination.

In public health settings, dental hygienists often use Type 4, a visual dental hygiene screening (VDHS); however,, dentists typically use Type 3, a mirror, tactile dental inspection (MTDI).

While studies comparing local dental Hygienists' screenings with local dentists' inspections were not found, 2 studies in the United States using epidemiological study methods compared a VDHS with a MTDI. In 1990, Mauriello et al tested the validity of a VDHS conducted by 4 hygienists on 5253 children in grades 1 and 5.[6] The dental hygienists had at least 5 years of private practice experience, extensive study-specific training over several days, and their inter-examiner screening skills calibrated on the first day of the screenings. The dental hygienists used a dental light and tongue blades. The MTDI was conducted by four dentists, each of whom had substantial experience in large-scale epidemiological studies. The dentists also had extensive study-specific training over several days and were calibrated for inter-examiner reliability on the first day of data collection. The dentists used a dental light, mirror, explorer, and compressed air. The dentists followed Radike's criteria and inspected by surface; the dental hygienists used criteria developed for the study and screened by tooth.[7] Using the kappa coefficient to compare the VDHS against the MTDI for all children in the study (n=5233), the kappa for presence of decay was 0.4 in permanent and 0.6 in primary teeth. This sample had a mean and standard deviation of 0.4 and 1.0 decayed teeth, respectively, for the permanent dentition, and a mean and standard deviation of 1.1 and 2.2 decayed teeth, respectively, for the primary dentition.

The other study was quite different in that the study-specific training of the dental hygienist only involved reading written materials and the screening protocol required assessment by quadrant until caries were found or not found. Beltran et al conducted this study in 1997, which compared the screening by one dental hygienist who had previous public health experience, to one dentist with previous experience and calibration with National Institute of Dental Research (NIDR) diagnostic criteria for decay.[8-10] The dental hygienist used a portable chair, flashlight, and tongue blade; the dentist used a portable chair, light, mirror, and explorer. They examined 309 children in kindergarten through fifth grade at a school where 67% of the children were eligible for free or reduced-price lunch.[11] Over one-third of the children had untreated decay. The sensitivity and specificity values of the VDHS were 95% and 94%, respectively, for presence of decay.

These studies involved several days of training for the dentists as did the dental hygienists in the Mauriello et al study.[6,8] It would be expensive and time consuming for local dentists and dental hygienists to undergo such training for local

assessment efforts. Further, the criteria of measurement of these 2 studies were based on the National Institutes of Health epidemiological protocols, as were their standard of validity. In contrast, this study used the level of dental care in the local community and did not provide study-specific training.

Data on the validity of the VDHS using local community dental hygienists without extensive training as "front line" individuals to develop and implement screening programs (12) would be useful in assisting communities accomplish the Healthy People 2010 objectives of: (a) reducing the proportion of children, adolescents, and adults with untreated decay; (b) increasing the proportion of children and adults who use the oral health care system each year; (c) increasing the proportion of low-income children and adolescents who received any preventive dental service during the past year; and (d) increasing the proportion of school-based health centers with an oral health component.[12,13] Weintraub also advocates that dental hygienists who can work without direct supervision in public health settings be utilized to conduct screenings for young children as a means of controlling early childhood caries.[14]

The unique goal of this study was to examine the validity of VDHS in typical field conditions to ascertain if children identified with decay had true treatment needs and those children identified as caries-free did not need treatment for caries consistent with the standards of treatment by a dentist in the local community. Specific aims were to: 1) determine the validity of oral health screenings (VDHS) in a community setting using local oral health care providers without training in public health and research data collection perspectives and methods and 2) explore explanations of possible discrepancies between the VDHS and MTDI based on size, surface, and location of decay.

Methods and Materials

The study population was comprised of 250 children in kindergarten through second grade (K-2) at one inner-city school in a predominantly white Iowa city. This school was identified as having a high risk population since more than half (68%) of the study population was eligible for free or reduced-price school lunch. Consent and cooperation were obtained from the school principal and district school nurse. Informational letters and parental consent forms were mailed to parents and non-respondents were contacted by teachers during parent-teacher conferences. This study was approved by the University of Iowa College of Dentistry's Institutional Review Board for Human Subjects Research.

Dental hygienists employed in the community were asked by the local public health dental hygienist to participate in the study. Interested dental hygienists attended a one-hour presentation to prepare for data collection. Four dental hygienists were available on the study day. Two dental hygienists (A and B) screened and 2 recorded concurrently. Dental hygienist A graduated in 1983 from a two-year dental hygiene program and hygienist B graduated in 1984 from a four-year program. The screenings (VDHS) were conducted one morning in the school art room where the children came one classroom at a time. Each child stood in front of the seated dental hygienist who used a tongue blade and goose-neck lamp to view each child's mouth. Dental hygienist A assessed 63 children, while dental hygienist B assessed the other 63 children. The hygienists were told to consider a tooth sound if in doubt. The dental hygienists recorded the total number of decayed teeth for each dentition. The screenings were completed in less than 2 hours.

The MTDI was conducted by one local dentist. The same public health dental hygienist constructed a short list of dentists who worked in the city and whom she thought might participate. The first dentist contacted by the investigator volunteered. Originally, the protocol had been written to have the children examined in the dentist's office with radiographic data, Type 1 dental examination, just as a new patient would be examined.[5] However, it was not acceptable to the school administrator to transport the children from the school. Since it was not possible to safely accommodate radiographic exposure within the school, a Type 3 dental inspection was conducted in the same room at the school in 3 half-days (9:15 AM-12:30 PM) and completed within one month of the screenings.[5] In preparation, the dentist reviewed Radike's criteria for the diagnosis of dental caries and discussed the format with the investigator who instructed the dentist to examine the subject just as he would in his own practice, except without radiographs.[7] The dentist used a portable dental chair, a goose-neck lamp, a dental-operator chair, mouth mirrors, explorers, his own dental assistant seated in a four-handed-delivery mode, and slightly modified oral examination forms from his office. Students came to the art room in a manner that allowed for continuous

examining by the dentist and observation by subsequent children. In the MTDI assessment, the dentist noted the location of decay by tooth and surface.

Note that while decay status was determined by both the VDHS and the MTDI, they were assessed and recorded in different formats. With the VDHS, the total number of decayed teeth was recorded for each dentition for each subject; whereas, with the MTDI, the location of decay was recorded by tooth and surface for each subject.

All study findings were coded and entered into a computer and verified for accuracy. Initially, the data were analyzed by frequency distributions and cross-tabulations. To analyze the validity of the VDHS, the VDHS and MTDI were compared for presence of decay. Data regarding the number of decayed teeth were converted to dichotomous variables. The congruence of the VDHS and MTDI for these decisions was compared by sensitivity rate, specificity rate, and the kappa coefficient, using findings from the MTDI as the standard. According to Gordis, sensitivity is the ability of a test (VDHS) to identify correctly those who have the disease, while specificity is the ability of the test (VDHS) to correctly identify the non-diseased people as non-diseased.[15] The kappa coefficient is the extent to which the observed agreement (between the VDHS and the MTDI) exceeds that which would be expected by chance alone (numerator) relative to the most that the observers could hope to improve their agreement (denominator). Analysis for the permanent teeth was made only for the children who had permanent teeth (n=113). The chi-square statistic was used to test for significance of differences between the two hygienists.

Data were also analyzed using the Fisher exact test, Cochran *Q* test, and the Mantel-Haenszel test to explore explanations for the discrepancies between the VDHS and MTDI.[16] Three explanations were considered. 1) Lesion size was investigated to to determine if multiple-surface lesions (large lesions) were more accurately detected by the VDHS than single-surface lesions (small lesions). Lesion size was investigated separately for the primary and permanent dentitions. 2) Controlling for lesion size, the location of the lesion was analyzed to determine if lesions of approximately the same size were more accurately detected in locations most visible to the unaided eye (ie, the anterior vs. posterior teeth and mandibular vs. maxillary teeth for both the primary and permanent dentitions). Canines were categorized as posterior teeth because primary canines (there were no permanent canines in this K-2 sample) are normally located within the curvature of the arch and therefore are less visible to observation by the unaided eye. 3) The type of tooth surface, smooth vs non-smooth, was tested by single-surface lesion and by any type of lesion to determine if decay was detected more accurately on smooth surfaces than on non-smooth surfaces. For primary teeth, the analyses compared occlusal surfaces (non-smooth) with all other surfaces (smooth). For permanent teeth, developmentally pitted and fissured surfaces (the lingual surface of the right and left maxillary first molars and the buccal surface of the right and left mandibular first molars) were included with the occlusal surfaces as non-smooth surfaces. The data for the two hygienists were combined for this analysis.

Results

Of the 250 children in kindergarten through second grade, data were collected from 126 (50%) for whom parental consent was obtained. The study participants were not significantly different from the total population on any of the demographic variables available from school records. (Table I.) The largest percentage of students (35.7%) were in the second grade. Sixty-six percent of study participants were eligible for free lunch and 52% lived in a home without a father present.

Table I- Student characteristics of total population and study participants

	Total population N=250		Study participants n=126		
Variable	Number	Percent	Number	Percent	p value*
Economic status					
Self pay	78	31.2	30	23.8	.3
Reduced-price lunch	19	07.6	13	10.3	.6
Free lunch	152	60.8	83	65.9	.5
Not available	1	00.4	xxxxx	xxxxx	xxxxx
Family structure					
Single mom	119	47.6	66	52.4	.6
Single dad	20	08.0	11	08.7	.8
Mom and dad	111	44.4	49	38.9	.5
Gender					
Male	143	57.2	72	57.1	1.0
Female	107	42.8	54	42.9	1.0
Grade					
Kindergarten	91	36.4	41	32.5	.7
First	83	33.2	40	31.8	.9
Second	76	30.4	45	35.7	.4
Birth Year (age)					
1990 (5-6)	59	23.6	24	19.0	.4
1989 (6-7)	89	35.6	44	34.9	.9
1988 (7-8)	77	30.8	42	33.3	.8
1987 (8-9)	24	09.6	15	11.9	.6
1986 (9-10)	01	00.4	01	00.8	.3

* Percents rounded to nearest whole number for these calculations

The distribution of decayed teeth among the 126 children is presented in Table II. Of this K-2 population, 67.6% of the teeth were primary teeth. According to the MTDI, 67 children (53%) did not have any decay; while according to the VDHS, 87 children (69%) did not have any decay. Both approaches found similar numbers of children with 1, 2, and 3 decayed teeth; however, the MTDI found many more children with 4 to 8 decayed teeth.

Table II- Frequency of decayed teeth in children by VDHS and MTDI

Number of decayed teeth	Number of children as defined by VDHS	% children as defined by VDHS	Number of children as defined by MTDI	% children as defined by MTDI
0	87	69.0	67	53.2
1	18	14.3	16	12.7
2	10	07.9	9	07.1
3	6	04.8	8	06.4
4	3	02.4	10	07.9
5	1	00.8	7	05.6
6	1	00.8	2	01.6
7	0	00.0	6	04.8
8	0	00.0	1	00.8
TOTAL	126	100.0	126	100.1

The dental hygienists (VDHS) identified 79 decayed teeth in 39 children; the dentist (MTDI) identified 195 decayed teeth in 59 children. Among the 126 children, the mean number of decayed teeth was 1.6 according to the MTDI and 0.6 according to the VDHS. As expected, given the age of the children, there was more decay in primary teeth than in permanent teeth. While decay was found on all surfaces, the occlusal surface was most frequently decayed in the primary dentition and the buccal surface, followed closely by the occlusal surface in the permanent dentition. (Table III.)

Table III- Frequency, percent, mean and standard deviation of decayed teeth and surfaces among children from VDHS and MTDI

	All teeth (n=126)			Primary teeth (n=126)			Permanent teeth (n=113)		
VDHS	n	mean	sd	n	mean	sd	n	mean	sd
Decayed teeth	79	.6	1.2	70	.6	1.1	9	.1	.4
MTDI									
Decayed teeth	195	1.6	2.2	158	1.2	1.9	37	0.3	0.8
Decayed surfaces	343	2.7	4.1	289	2.3	3.4	54	0.5	1.4
occlusal	143	1.1	1.8	124	1.0	1.6	19	0.2	0.6
mesial	63	0.5	1.0	61	0.5	1.0	2	0.0	0.2
distal	58	0.5	0.8	53	0.4	0.8	5	0.0	0.3
buccal	44	0.4	0.8	22	0.2	0.5	22	0.2	0.6
lingual	35	0.3	0.7	29	0.2	0.6	6	0.1	0.3

The sensitivity and specificity for the VDHS in detecting decay was 61% and 96% for all teeth, 64% and 100% for primary teeth, and 15% and 97% for permanent teeth, respectively. Corresponding kappa coefficients were 0.6, 0.7, and 0.2, respectively. Dental hygienists A and B detected decay comparably except for sensitivity for permanent teeth, for which dental hygienist A was significantly (p .000) less able to accurately detect decay than dental hygienist B. (Table IV.)

Table IV. Sensitivity, specificity and Kappa of the VDHS regarding presence of decay compared with MTDI presence of decay

	Dental hygienist A			Dental hygienist B			Both dental hygienists		
	Sen*	Spec +	Kap-pa	Sen*	Spec+	Kap-pa	Sen*	Spec +	Kappa
All teeth	62	96	.6	60	95	.6	61	96	.6
Primary teeth	66	100	.6	62	100	.7	64	100	.7
Permanent teeth	0	98	.0	30	96	.3	15	97	.2

n=126 children, except for permanent teeth which were present in only 113 children, with hygienist A and B seeing 54 and 59 children respectively
Sen* Sensitivity
Spec+ Specificity

As determined by the MTDI, Table V summarizes carious lesions by primary and permanent dentition, maxillary and mandibular arch, anterior and posterior location, and surface involved. In addition, the table lists the number of subjects by each location of decay as determined by the MTDI. Of the 36 locations where decay was detected by the MTDI, the VDHS identified subjects as having decay in 32 of these locations. The four locations which were most problematic (0% of subjects detected) were the distal, buccal, and mesial single-surface locations of posterior primary teeth and one multiple-surface location in the primary posterior primary teeth.

Table V.- Frequency of decay by dentition, arch, location in the mouth and surface type as determined by MTDI and the ability of the VDHS to correctly identify cases with each type of lesion as having decay

LOCATION OF DECAY BY MTDI					NUMBER OF SUBJECTS		PERCENT CORRECT
Dentition	Arch	Position	Surface	Fre-quency	MTDI # children	VDHS # children	% correct by VDHS*
Primary	maxillary	anterior	mesial (M)	9	5	4	80
(Teeth #4-7)			distal (D)	1	1	1	100
			buccal (B)	3	2	2	100
			All	3	2	1	50
Primary	maxillary	posterior	lingual	6	4	3	75
(Teeth #1,2,3,8,9,10)			occlusal	15	12	6	50
			mesial	2	2	1	50
			distal	3	3	0	0
			buccal	1	1	0	0
			LO	6	5	5	100
			DO	11	9	7	78
			MO	14	10	6	60
			DL	1	1	1	100
			MLO	3	3	3	100
			MOD	1	1	1	100
			All	3	3	2	67
Primary	mandibular	anterior	All	1	1	1	100
(Teeth #14-17)							
Primary	mandibular	posterior	mesial	1	1	0	0
(Teeth #11,12,13,18,19,20)			occlusal	24	19	12	63
			distal	2	2	1	50
			buccal	1	1	1	100
			DO	19	14	12	86
			MO	16	12	8	67
			BO	3	3	3	100
			LM	1	1	0	0
			MOD	1	1	1	100
			BMO	1	1	1	100
			All	6	4	4	100
Permanent	maxillary	posterior	occlusal	4	3	2	67
(Teeth #3,14)			LO	1	1	1	100
			LD	3	2	1	50
			All	1	1	1	100
Permanent	mandibular	posterior	buccal	14	10	7	70
(Teeth #19,30)			occlusal	8	6	2	33
			BO	5	4	3	75
			All	1	1	1	100

* These statistics are for the combined data of hygienists A and B.

Results of testing for the 3 explanations for possible discrepancies between the VDHS and the MTDI are as follows. With regard to size, the ability of the VDHS to identify single-surface decay was significantly less than the ability to identify multiple-surface decay for primary teeth but not for permanent teeth. The odds of the VDHS detecting multiple-surface decay in primary teeth were 3.1 of those for detecting single-surface decay (OR=3.1; p=.0022; confidence interval (CI)= 1.5-6.9). The corresponding p value for the permanent teeth was 0.3887.

For the position of decay (anterior versus posterior) for the permanent teeth, tests could not be conducted due to lack of anterior decay in this dentition for this sample. For single-surface decay in primary teeth, the ability of the VDHS to identify posterior decay was significantly less than the ability to identify anterior decay. The odds of the VDHS detecting single-surface, anterior decay was 10 times that of detecting single-surface, posterior decay (OR=10.; p=.0117; CI=1.2-82.3). However, multiple-surface decay in primary teeth was detected equally well in either the anterior or posterior location (p=0.6473).

With regard to the position of decay in the maxillary versus mandibular arch, the VDHS was not significantly different from the MTDI in identifying either: 1) single-surface maxillary decay in the primary (0.7204) or permanent (0.4462) dentition, or 2) multiple-surface maxillary decay in the primary (0.5708) or permanent (0.3869) dentition.

Tests to compare smooth versus non-smooth surfaces could not be conducted for the permanent teeth due to lack of smooth surface decay in this young sample. For the primary teeth, the ability of the VDHS to identify pitted surfaces (including the occlusal surfaces) was not significant for either single-surface instances of pitted decay (p=0.2920) or instances in which a non-smooth surface was involved in any carious lesion (p=0.3715).

Discussion

As Beltran et al discussed at length, the level of congruence necessary to state that the VDHS is valid has not been widely discussed.[8] Landis established that kappa values <0.39 are "low," 0.4 to 0.6 are "moderate," and >0.61 are "substantial."[17] Stamm states that a test should have a sensitivity level of 0.75 or higher and a specificity level of 0.85 or higher.[18] These guidelines suggest that, as implemented in local community field assessment conditions, the VDHS had moderate to substantial kappa values in primary teeth, excellent specificity in both primary and permanent teeth, poor kappas in permanent teeth, and poor sensitivity in both primary and permanent teeth. Since the carious lesions most often missed by the VDHS were single-surface lesions in general and particularly in the posterior of the mouth, the use of the mirror in MTDI may account for much of the differences in findings.

The congruence level for detecting decay achieved in this study was remarkably similar to those reported by Mauriello et al.[6] This is a somewhat surprising finding given the extensive training for the screenings by both dental hygienists and dentists in the Mauriello study.[6] However, Mauriello's analysis was more precise than this study. Although the level of decay was similar in the 2 studies, the subjects in this study were younger and this may explain the lower kappa coefficient for permanent teeth (0.2 versus 0.4). In contrast, this study's sensitivity levels were considerably lower (0.6 versus 1.0) than those reported by Beltran et al.[8] The 2 studies had similar specificity levels (1.0 versus 0.9). The dental hygienist in Beltran et al had more study-specific training than the 2 dental hygienists in this study, and perhaps Beltran's sample had more extensive decay as slightly more than one-third of the Beltran et al subjects needed urgent dental care while only 15% of these subjects did.[8,9]

The primary limitations of this small-scale pilot study were sample size and the limited number of dental hygienists and dentists. Further, it is possible that the dentist may have over-identified decay knowing that the assessment was part of a research project and having been able to acquire data from use of the mirror and explorer. Also, directing the dental hygienists to consider questionable areas sound may have decreased their sensitivity rates. However, it must be kept in mind that this was a young sample with few erupted permanent teeth.

Without a doubt, the dental Hygienists' ability to detect caries would have been enhanced with a mirror and explorer. Dental hygienists and dentists detect caries at a comparable level when they utilize the same equipment and have the same study-specific training.[19,20] But, the purpose of this study was to investigate the VDHS in typical, local community situations. Analyses of discrepancies between the VDHS and MTDI in this study suggest that if dental hygienists give extra attention to identifying single-surface lesions and single- surface lesions in posterior teeth, the sensitivity of the VDHS would probably increase. If this more careful look is successful, it would be more cost-effective than adding mirrors which cost more than tongue blades and need to be sterilized. The primary advantage of the VDHS is low cost in terms of equipment, preparation, clean-up, and manpower. This study ascertained that child contact time for one dentist to perform the MTDI was more than twice that for one dental hygienist to perform the VDHS.

Some have advocated that dental hygienists who participate in screening programs, such as the Early Periodic Screening, Diagnosis and Treatment (EPSDT) program, need annual continuing education and clinical licensing tests in screening.[21] The present study and Beltran's study indicate that with short study-specific preparation, dental hygienists, as educated under current US accreditation standards, can achieve moderate to high levels of sensitivity and specificity with VDHS.[8] This suggests that that yearly clinical licensing tests and yearly continuing education are not necessary for dental hygienists to participate in oral health screenings.

These findings do suggest directions for further research on the VDHS. Larger sample sizes and a wider age group of children are needed to test the effects of location and size of decay on ability to detect decay with the VDHS. To increase the generalizability of study results, the study needs to be replicated in a variety of types of communities, and the number of local community dental hygienists performing the VDHS and the number of local community dentists performing the MTDI need to be increased. Investigators should consider comparing a visual dental hygiene screening to a dental hygiene screening that includes the use of mirrors. In addition to investigating the validity of the VDHS, cost-benefit analyses should be done. Until these studies are done, this study's findings and Beltran et al's indicate that the VDHS is a low-cost,

simple screening technique with reasonably good validity, and that dental hygienists with general private practice experience can adequately perform VDHS with minimal additional instruction.[8]

Conclusion

In this young sample (kindergarten through second grade) with mostly primary teeth, these dental hygienists using VDHS were able to identify children without carious lesions (specificity=96%). They were less successful in identifying children with caries (sensitivity=61%). For the dental hygienists in this study, it was particularly difficult to identify single-surface lesions and single-surface lesions in posterior teeth. Overall, the high specificity indicates that visual screenings by these 2 community, private-practice dental hygienists would not refer children for dental care who do not need care.

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Notes

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