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The Importance of Peer Review

R Wilder, RDH, BS, MS

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Keywords: peer-reviewed journal, scientific journal, peer review, research journal



Many times I am asked what it means to be a "peer-reviewed" journal. After all, many journals and magazines claim to be peer reviewed. What makes ours different?

Recently I joined an organization called the World Association of Dental Editors (WAME). I found out about WAME from a medical colleague of mine who has a great deal of editorial experience. I had never heard of it before so I quickly visited their Web site at www.wame.org. WAME is free and open to all editors of peer-reviewed medical journals. As of May 2006, they have more than 1409 members representing 890 journals from 90 countries. I completed the paperwork, sent it in and now JDH is part of a prestigious list of medical and a few dental journals. WAME is an organization that was established in 1995 to do several things. First, the organization seeks to facilitate worldwide cooperation and communication among editors of peer-reviewed medical journals. Another goal is to improve editorial standards, to promote "professionalism in medical editing through education, self criticism and self regulation."

Another reason why I am honored to be a part of this organization is because the only editors who can be members are ones who are in charge of determining the scientific content of a "peer-reviewed biomedical journal." WAME defines a peer-reviewed biomedical journal as "one that has submitted most of its published articles for review by experts who are not part of the editorial staff."

I must take this opportunity at the beginning of my tenure as Editor-in-Chief of our scientific peer-reviewed journal to publicly thank the members of the JDH Editorial Review Board who have given their time and expertise and who are extremely dedicated to keeping high standards in the peer review process. These individuals are busy dental hygienists just like you and me! Most of them hold academic appointments and have advanced degrees of a master's degree or a doctorate. Most are heavily involved in their own research agendas. They are writers and teachers, in private practice or

public health. Some are nurses, dentists, physicians, physical therapists and they know the science. They did not get there overnight. They did not graduate from dental hygiene school with expertise in writing or conducting research, or even with the understanding needed to thoroughly read a scientific research paper. They have worked hard over the years to gain the expertise, to seek out mentors, and to realize that one has to make oneself vulnerable in order to become better at this process of writing for peer-reviewed publications. This hard work has assisted them as they have published their own papers and also as they critique the hard work of their peers.

I am in the process of forming a new Editorial Review Board, which will consist of previous members of the board as well as new members. These individuals hold high ethical standards to ensure that scientific quality is upheld. They possess knowledge and expertise in a multitude of areas to ensure that the manuscripts accepted for publication in JDH are of high scientific quality and are valuable to our profession and the building of our unique knowledge base. Lastly, they spend many hours to help make the manuscripts accurate, readable, and relevant to all dental hygienists. The reviews are not based on opinion but rather on science. Each article is reviewed by three members who are "blinded" to the information about the authors or the authors' affiliation. What is the value of peer review? True peer review lends credibility and respect to our Journal. You can be assured that papers published in your Journal have been read and approved by an exhaustive process to bring you the best that science has to offer in dental hygiene.

Sincerely,

Rebecca Wilder, RDH, BS, MS

Editor-in-Chief, Journal of Dental Hygiene

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Upfront

Katie Barge

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

No Amount of Secondhand Smoke is Safe, Warns Surgeon General

No amount of secondhand smoke is safe, according to a new U.S. Surgeon General's report, *The Health Consequences of Involuntary Exposure to Tobacco Smoke*, issued Tuesday, June 27, 2006. The report concluded that the only way to protect nonsmokers is through 100% smoke-free environments. Separating smokers and nonsmokers within the same air space or relying on sophisticated ventilation systems just doesn't cut it.

"Science has proven that there is no risk-free level of exposure to secondhand smoke. Let me say that again: There is no safe level of exposure to secondhand smoke," said U.S. Surgeon General Dr. Richard H. Carmona in a prepared statement. "Only smoke-free environments effectively protect nonsmokers from secondhand smoke exposure in indoor spaces."

According to the report, nonsmokers who were exposed to secondhand smoke at work or at home had a 25% to 30% increased risk of heart disease and a 20% to 30% increased risk for lung cancer.

Peter G. Billings, the American Lung Association's vice president of national policy and advocacy responded to the report: "Essentially, the Surgeon General slammed the book on any scientific debate on secondhand smoke. The evidence is clear. Secondhand smoke is harmful and needs to be eliminated."

The sweeping report, which was based on the latest research on the topic, was the first comprehensive review of secondhand smoke by the U. S. Department of Health and Human Services since 1986. The 1986 report concluded that secondhand smoke causes lung cancer in nonsmokers.

According to the report, secondhand smoke, which contains more than 50 carcinogens and is a known human carcinogen, increases the risks of heart disease and lung cancer in nonsmoking adults as well as sudden infant death syndrome (SIDS), respiratory problems, ear infections, and asthma attacks in infants and children. While progress to control secondhand smoke has been made, some 126 million Americans are still exposed to it. Slightly more than 20% of children are exposed to secondhand smoke at home.

"Breathing secondhand smoke for even a short time can damage cells and set the cancer process in motion," Carmona said. "Brief exposure can have immediate harmful effects on blood and blood vessels, potentially increasing the risk of a heart attack. Secondhand smoke exposure can quickly irritate the lungs, or trigger an asthma attack. For some people, the rapid effects can be life-threatening. People who already have heart disease or respiratory conditions are at especially high risk."

Nearly half of all nonsmoking Americans are regularly exposed to secondhand smoke. In 2005, as a result of exposure to secondhand smoke, 3000 adult nonsmokers died from lung cancer, 46 000 adult nonsmokers died from coronary heart disease, and 430 newborns from SIDS

The report also found that living with a smoker increases a nonsmoker's risk of lung cancer and heart disease by up to 30%. At this point, evidence linking secondhand smoke and breast cancer is only suggestive.

"The good news is that, unlike some public health hazards, secondhand smoke exposure is preventable," said Carmona. "A proven method exists for protecting nonsmokers from the health risks associated with secondhand smoke exposure: Avoiding places where secondhand smoke is present."

The American Dental Hygienists' Association (ADHA) supports a tobacco-free environment in all public places. "My personal hope is that more dental hygienists can use that policy to push for more smokefree workplaces as other states and communities work toward going smokefree. Dental hygienists are perfect advocates to push for such laws since we have daily opportunities to promote smoking cessation as a means of health promotion and disease prevention," said Diann Bomkamp, RDH, BSDH, CDHC, vice president of ADHA, and a participant in Tobacco-Free Missouri, a group working to promote a smokefree environment in Missouri.

"Being a dental hygienist advocate has four benefits: promoting us as knowledgeable health professionals; being involved with coalition building with other likeminded groups; promoting better health policies; and getting our oral health messages to the public. Dental hygienists can do them all effectively!" said Bomkamp.

The Surgeon General recommends the following tips on protecting yourself, friends, and family from the effects of secondhand smoke:

- Make your home and car smoke-free.
- Ask people not to smoke around you or your children.
- Make sure that your children's daycare center or school is smoke-free
- Patronize restaurants and other businesses that are smoke-free.
- Teach children to stay away from secondhand smoke.
- Avoid secondhand smoke exposure especially if you or your children have respiratory conditions, if you have heart disease, or if you are pregnant.

U.S. Women are in the Dark about Lung Cancer

A new survey published by the U.S. National Lung Cancer Partnership (NLCP) revealed that American women are greatly uninformed about lung cancer and how it can affect them. The 2006 survey of more than 500 women discloses not only the statistical realities of lung cancer in this county but also the widespread lack of awareness by millions.

"This survey is a current snapshot of women's attitudes and beliefs about lung cancer, and it's frightening-especially considering the extensive media coverage on the topic after Peter Jennings' and Dana Reeve's deaths," said Regina Vidaver, executive director of the NLCP. "Women need to know the truth about lung cancer."

Lung cancer affects more than 80 000 American women annually, with over 70 000 cases resulting in death. Although breast cancer is often thought of as the leading cause of death among women, 30 000 more women die annually from lung cancer than from breast cancer. In fact, lung cancer claims the lives of more women than breast, uterine, and ovarian cancers combined. Vital findings from the survey include:

- Only 41% of women know that lung cancer is the leading cancer killer in the United States
- Only 8% of women understand that exposure to radon gas is the second leading cause of lung cancer. Instead, 60% of women share the mistaken belief that exposure to secondhand smoke is the number two cause (the number one cause being smoking).
- Only 36% of women are aware that lung cancer kills more women than breast cancer.
- Only 29% of women know that lung cancer kills more women than breast, ovarian, and uterine cancers combined.
- Only 41% of women know that one in every 17 women will develop a lung malignancy in her lifetime.

- Only 18% of women know that women make up the majority of young-under the age of 40-lung cancer patients.
- Only 4% of women know that women typically do better than men following lung cancer treatment.

Why are so many women dying from this disease? According to the NLCP, 25% of women "mistakenly believe there is a standard screening test to detect lung cancer in its early stages. Although such tests are in development, there is no clinically-approved screening test of this nation's top cancer killer."

"Lung cancer is often perceived as a man's disease, yet it affects tens of thousands of women, and we're very concerned that women seem to be in the dark when it comes to the facts about lung cancer and the significant impact lung cancer can have on their lives," said Dr. Joan Schiller, president of NLCP.

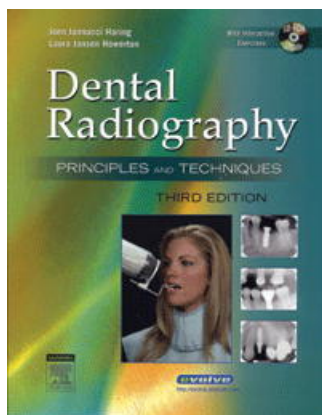
For more information, please visit <http://www.4woman.gov/faq/lung.htm>, the U.S. Department of Health and Human Services, Office on Women's Health Web site on women and lung cancer.

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Review of: Dental Radiography: Principles and Techniques

Jacqueline Brian, LDH, MEd

Reviewed by Jacqueline Brian, LDH, MEd, professor, Indiana University-Purdue University Fort Wayne, Fort Wayne, Indiana.



Dental Radiography: Principles and Techniques

Third Edition

Haring JJ and Howerton LJ

W. B. Saunders Company, 2006

St. Louis, Missouri

544 pages; illustrated; indexed; softcover

ISBN: 0-72161-575-9

\$59.95

The third edition of *Dental Radiology: Principles and Techniques* has quickly become the premier resource for comprehensive-yet clear and concise-fundamental concepts of dental radiology. The authors have wrapped these positive learning features into short chapters to facilitate student learning and ease in understanding.

The text is divided into 6 parts: Radiation; Equipment, Film and Processing; Dental Radiographer; Technique; Normal Anatomy and Film Mounting; and Radiographic Interpretation.

Radiology concepts are difficult to master, so the step-by-step procedures for proper techniques, which include rationales and charting notes, allow students to easily gain a solid understanding of these procedures. Other learning-friendly features are the quiz questions for each chapter and example boxes.

In addition, 750 strategically placed, detailed illustrations are combined with a clear writing style to explain concepts in a simple, student-friendly way. These tools are essential for the comprehension of critical material

The authors also recognized the need for students to play an active role in their own learning, and thus have expertly expanded beyond the basic teaching medium. They've included a companion, interactive CD-ROM, a new Web site, which includes a 110-question self-study exam, as well as a series of patient case studies that are in the same format as the National Board Dental Hygiene Exam. Animations also help the students visualize and learn key concepts and theories that are so difficult to comprehend.

All these learning tools are important to link the essential information to the application for development of self-directed student learning. In updating this new text, the authors have included the latest advances in radiography, including additional chapters on Descriptive Terminology and Interpretation of Restorations and Dental Materials. This additional material will better prepare students for future National Board Dental Hygiene Exams.

Extending beyond student needs, the authors have provided instructors with a new, online resource manual that will save class preparation time. This will be a valuable course management tool for instructors who may be unfamiliar with this subject material.

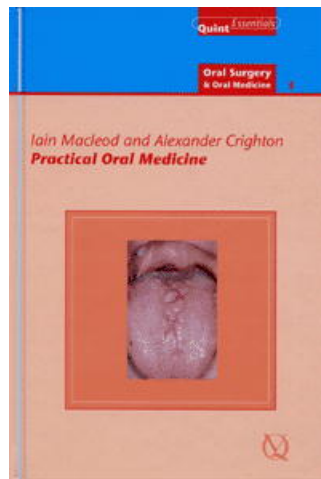
As leaders in their field, the authors have provided an impressive resource for dental hygiene and dental assisting students. Classroom instructors will want to include this text as required reading.

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Review of: Practical Oral Medicine

Ruth Fearing Tornwall, RDH, MS

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



Practical Oral Medicine

First Edition

Macleod I and Crighton A

Quintessence Publishing Co., Ltd., 2006

London, England

164 pages, color illustrations, indexed, hardcover

ISBN: 1850970653

\$54.00

Practical Oral Medicine is a new book in the Quintessentials for the General Dental Practice series from Quintessence. This book, like others in this series, is concise, easy-to-absorb, and up-to-date with color illustrations. The book is meant to be an easy to understand chairside reference text, providing oral medicine information and advice. The level of writing is appropriate for dental health professionals.

The book includes 11 chapters, with the first being an introductory chapter and the last covering therapies in oral medicine. Each chapter includes an aim, outcome, introduction to the area, the lesion or condition being discussed, diagnosis, management or treatment, a conclusion or summary, and references for further reading.

The introductory chapter describes in detail the procedure for an oral medicine consultation. The authors stress the fact that this meeting sets the tone for all remaining visits. The consultation includes the greeting, the purpose of the appointment, the information gathering format, a review and discussion of the key points of the examination, the conclusion of the findings, and the discussion of any future appointments, if necessary. At the end of this meeting, both the patient and the practitioner should have a clear understanding of their future care plan.

Chapter 2 reviews immunological problems, including the oral effects of allergy along with the oral mucosal effects of immunological reactions to the oral mucosa. Chapter 3 examines the various oral and perioral lumps and swellings, their diagnosis, and management. The authors conclude that these lesions are a common finding with most being benign. Clinically though, many of these lesions are similar and need to be differentiated histologically. The authors state there are some instances when a lesion would warrant further investigation.

Chapter 4 looks at common oral mucosal and facial infections. The authors break down the infections into categories of bacterial, viral, and fungal lesions. The main principle in treating these lesions is to identify the active organism and determine its sensitivity to antimicrobial therapy. Chapter 5 covers white patches, dividing them into 2 groups, those that can rub off and those that cannot. Chapter 6 describes the diagnosis and management of oral cancer and premalignant lesions; risk factors and possible risk factors are identified in the chapter. The authors suggest dental health professionals should be able to counsel patients about risk factors, as surviving oral cancer depends on its early detection.

Chapter 7 describes various disorders that can result in pigmentation of the oral mucosa. Chapter 8 examines various disorders that can affect the salivary glands and salivation. The chapter is divided into 3 categories: salivary flow disturbance, salivary gland infections, and salivary gland swellings. Most problems are a relatively common occurrence but all complaints warrant investigation. Chapter 9 covers facial pain. The chapter seeks to make the clinical pattern of different pain problems more familiar. The authors emphasize the importance of taking a careful history and highlight using quality of life as a measure of success. Chapter 10 reviews the common neurological problems that may occur in the head, neck, and mouth. Chapter 11 describes the range and value of the various complementary therapies that may be used in oral medicine. They conclude that the evidence base for many therapies is lacking and further research is needed in these areas.

The book contains an index as well as 2 appendices. The index includes most of the major headings and many terms used within the chapters. Appendix A reviews the features of 6 common oral medicine conditions and their protocols that should be used in conjunction with the general history and examination guidelines as suggested in Chapter 1. Each protocol concentrates on the more detailed history or examination that is needed when considering each diagnosis. These conditions include lichen planus, recurrent oral ulcerations, white patches, sore lips-cheilitis and angular stomatitis-oral dysaesthesia, and temporomandibular disorders. Appendix B lists topical and systemic steroid treatment protocols.

Overall the book succeeds in its goal to provide the reader with a succinct easy-to-use text on oral medicine. One area that could have been improved upon is the number of photos in the book to illustrate the lesion under discussion; but, the photos that are there are of high quality. This is a book that could easily fit into one's oral health library.

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Review of: Pocket Atlas of Oral Diseases

Margaret Six, RDH, MSDH

Reviewed by Margaret Six, RDH, MSDH, associate professor at West Liberty State College, West Liberty, West Virginia.



Pocket Atlas of Oral Diseases

2nd Pocket Edition

Laskaris G, MD, DDS, PhD

Thieme Medical Publishers, 2006

New York, New York

384 pages, softcover, indexed, illustrated

ISBN: 1588902498

\$39.95

Pocket Atlas of Oral Diseases is an excellent reference text for the dental professional. The second pocket edition, written by George Laskaris, MD, DDS, PhD, consists of 370 pages filled with 350 color photographs, with associated literature to describe conditions and diseases found in the oral cavity. Laskaris offers a comprehensive overview of local and systemic oral disease in a pocket-size book. The small size lends itself to easy storage in a lab coat pocket or the smallest space in a busy dental operator for quick review.

The contents of the text are well organized into 12 chapters. Three chapters are devoted to lesions categorized by the colors white, red, brown, and black, which assist the reader in quickly locating a photograph with associated facts particular to the lesion. Additional chapters include the categories of ulcerative, vesiculobullous, papillary, and lip lesions. The remaining chapters include soft tissue tumors and cysts, bone and neck swellings, and gingival enlargement.

Within each chapter, the definition, etiology, clinical features, laboratory test, differential diagnosis, and treatment guidelines for each oral disease or condition is described. These facts assist the reader in simple identification, yet provide adequate detail to assist the doctor in the diagnosis and treatment considerations specific to the lesion or condition.

In the educational setting, *Pocket Atlas of Oral Diseases* will provide the dental hygienist and/or dental hygiene student preparing for board examinations a reference text for the review of oral diseases and conditions. The color pictures are of excellent quality and particularly helpful.

Pocket Atlas of Oral Diseases is a complimentary text to 2 other major textbooks written by Laskaris. The third edition of *Color Atlas of Oral Diseases*, published in 2003, and the *Treatment of Oral Diseases*, recently available in 2005, provide additional clinical information for the dental professional seeking a more comprehensive text.

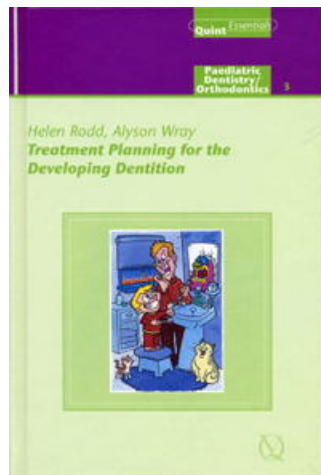
Any dental health professional will appreciate *Pocket Atlas of Oral Diseases*. The convenience of having a reference that contains conditions ranging from very common to the most obscure and rare provides a valuable resource for students, educators, and clinicians.

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Review of: Treatment Planning for the Developing Dentition

Lisa Shaw, RDH, MS

Reviewed by Lisa Shaw, RDH, MS, residential health care coordinator for the James M. Rozanski Dental Residency Program, St. Luke's Memorial Hospital, Utica, New York.



Treatment Planning for the Developing Dentition

Rodd H and Wray A

Quintessence Publishing, 2006

London, England

137 pages, illustrated, indexed, hardcover

ISBN: 1-85097-081-5

\$54.00

I liked this book from the beginning. Its diminutive size and multicolored, cartooned cover immediately drew me in. Once there, I was not disappointed. What followed was articulate text that didn't set out to "dictate rigid treatment" plans, but rather to generate an understanding of the "basic principles behind good decision-making." In her forward, Nancy Wilson, the editor-in-chief, reminds us that "good treatment planning for the developing dentition gives the child patient life-long benefits." Authors Rodd and Wray provide a framework for realizing these benefits that is "realistic, personalized, holistic, flexible, progressive and forward thinking," with goals that benefit not just the patient, but also the family and the practitioner.

Rodd and Wray divide the book into 6 sections that include the following

1. The First Visit And Information Gathering
2. Interceptive Orthodontic Treatment
3. Planning For Prevention
4. The Restorative Phase Of Treatment
5. Management Of The Dental Emergency
6. Recall Strategy

Each chapter begins with clearly stated aims and objectives and culminates with a list of recommended readings. The text is comprehensive and provides articulate dialogue about common questions and scenarios. Chapter 1, for example, asks the age old question about whether or not parents should accompany the child in the room for treatment. Rodd and Wray don't subscribe to any fixed dogma, but rather provide the reader with information regarding age-appropriate separation anxiety and about individual behavioral development that can effect the decision regarding parent accompaniment. In addition, they identify the parent's role in the process and provide a table that lists the pros and cons of parental accompaniment.

In another example, Chapter 3 is devoted to their belief that every child should have a "personalized preventive program that reflects their individual social, medical, and dental needs." The authors provide a table that outlines patient social, medical, and dental variables that may influence a preventive strategy. They also provide a dietary advice table as well as a list of dietary advice do's and don'ts.

Chapter text is expanded by the use of numerous illustrations, highlighted "tip" boxes, and tables. In addition to the aforementioned tables, the following tables, among others, provide valuable information in a way that can be easily accessed and recalled:

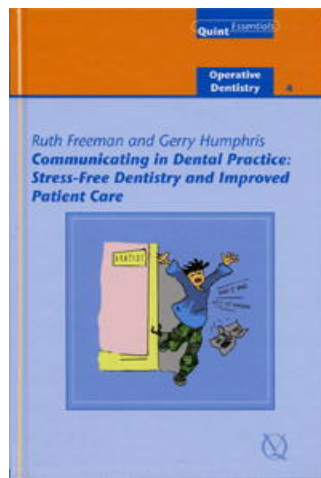
1. Implications Of Some Common Clinical Findings
2. A Guide To The Appropriate Use Of Radiographs In Children
3. Caries Risk Assessment Factors
4. Treatment Plans Based On Caries Risk
5. An Outline Of Possible Problems To Watch Out For In The Primary, Early And Late Mixed Dentition
6. Eruption Tables For Primary And Permanent Teeth
7. Guidelines On When To Balance And When To Compensate First Permanent
8. Molar Extractions In The Mixed Dentition
9. Management Of Gingival And Periodontal Conditions In Children
10. Recommended Fluoride Supplement Dosages
11. Dos And Don'ts Of Fluoride Supplement Prescription
12. Working Examples Of Individualized Preventive Programs
13. Tips For Giving A Successful Local Anesthetic To Children
14. Decision-making For Material Selection for Primary Tooth Restoration
15. Common Pulpal Therapies for Primary Molars
16. Bleaching Or Tooth-Whitening For Children
17. Special Restorative Challenges For Children
18. Common Oralfacial Infections In Children
19. Key Points For Dental Trauma Management

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Review of: Communicating in Dental Practice: Stress-Free Dentistry and Improved Patient Care

Mary Danusis Cooper, RDH, MSED

Reviewed by Mary Danusis Cooper, RDH, MSED, professor in the dental hygiene program at Indiana University-Purdue University, Fort Wayne, Indiana.



Communicating in Dental Practice: Stress-Free Dentistry and Improved Patient Care

Freeman Ruth and Humphries GM

Quintessence Publishing Co.

Chicago, Illinois, 2006

116 pages 24 illustrations, indexed, hardcover

ISBN: 1-85097-099-8

\$54.00

One of the major obstacles in dentistry is communicating effectively with patients. Many patients seen in the dental office are anxious; some can be difficult, while others may be dissatisfied. This book focuses on the many challenges faced when communicating with dental patients and how those challenges can be overcome. Principles and actions are introduced to enhance communication between the patient and staff member that will, in turn, help with difficulties encountered in the dental setting.

The text has 9 chapters written in an easy-to-read format. At the end of each chapter is a list of additional readings offered to the reader. Chapter 1, the introduction, addresses interaction problems that may be evident between staff and patients. These include difficult and demanding patients, the management of pain, dentally anxious patients, and how to encourage patients to adhere to oral health recommendations made by the dental staff.

Chapter 2 addresses basic communication skills. There are 6 key elements to communication. They include understanding nonverbal communication, listening, engaging others to talk, acknowledging other's feelings, asking questions and obtaining feedback, and giving feedback. Questions used to obtain more information about a patient's needs include open-ended and close-ended questions.

In chapter 3, advanced communication skills are noted. How does one gain consent or break bad news to a patient? How do staff members handle complaints? An interesting fact noted in the book is that 90% of litigation in the health service is attributed to poor communication skills. This proves how essential it is to practice good communication skills.

Chapter 4 focuses on communicating with certain groups of patients such as the older population, those with learning disabilities and mental illness, ethnic minorities, as well as the homeless.

Chapter 5 addresses ways to deal with the anxious and/or demanding patient. As stated in the text, "dental anxiety is the most important psychological factor dental staff will meet in practice." All dental staff members have dealt with the anxious patient and know the importance in working with this patient in order to make the appointment as pleasant as possible-both for the practitioner and patient. Techniques recommended to reduce anxiety include tell-show-do, hypnosis, biofeedback, and relaxation.

In chapter 6, "Understanding and Finding Solutions: The 'Difficult' and Dissatisfied Patient," the emphasis is placed on managing patients with difficulties that are not necessarily dental in origin. Recommendations are given on to how to manage these patients once the source of their difficulties is defined. Patients can easily displace other problems, such as a recent divorce, into the dental setting.

Chapter 7 deals with preventive health principles. Strategies are addressed for all areas of prevention-primary, secondary, and tertiary. In addition, several models that have influenced oral health education are presented.

Chapter 8 deals with integrating oral health education into the dental care of the patient. This area should be most comfortable to the dental staff since it encompasses oral hygiene care-an area essential for preventive results. In particular, the areas on fluoride, dietary and plaque control, erosion, advice for denture wearers, and oral cancer offer an excellent review for all staff members. Lastly, chapter 9 emphasizes the importance of improving patient care through excellent communication skills.

This text provides an excellent tool for every dental office. I have already recommended this book to several dentists. Upon an initial hire of an employee, I believe this book should be a mandatory read. As a dental hygiene educator, I feel this book should be implemented into the curriculum. All students, dentists, and dental personnel would benefit from the material presented in the text. Communication is essential in reducing stress and complaints from patients and, in turn, will improve the overall clinical outcomes.

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Oral Malodor: A Review of the Literature

PK Pratibha, MDS, KM Bhat, BSc, MDS and GS Bhat, MDS, MFGDF

Pratibha PK, MDS, associate professor, Department of Periodontics, Manipal College of Dental Sciences, Manipal Academy of Higher Education, Manipal, India; K Mahalinga Bhat, BSc, MDS, professor and department head, Department of Periodontics, Manipal College of Dental Sciences, Manipal Academy of Higher Education, Manipal, India; G S Bhat, MDS, MFGDF (UK) professor, Department of Periodontics, Manipal College of Dental Sciences, Manipal Academy of Higher Education, Manipal, India.

Oral malodor or halitosis is any unpleasant odor emerging from the mouth that is detected by others. Many patients experience extreme discomfort and embarrassment and therefore seek help for this problem.

Oral causes, such as poor oral hygiene, periodontal disease, tongue coating, food impaction, unclean dentures, faulty restorations, and dry mouth, are far more common than nonoral causes of malodor. Management may include simple measures such as scaling and root planing, instructions in oral hygiene, tongue cleaning, and mouth rinsing.

This paper reviews the current knowledge, etiology, diagnosis, and possible treatment strategies for oral malodor. Emphasis is placed on the recognition of the dental hygienist as a specialist in aspects of patient care and instruction, which relate to the prevention and control of oral malodor.

Keywords: Bad breath, malodor, halitosis, volatile sulfur compounds

Introduction

Oral malodor, also called halitosis or bad breath (feto ex ore) is a universally experienced condition affecting humankind since ancient times. Historic references include the Jewish Talmud, as well as Greek and Roman writings.¹ Islam stresses the importance of fresh breath as part of good oral hygiene.² Ladanum (mastic), a resin derived from the 'Pistacia lentiscus' tree used in the Mediterranean region for breath freshening for thousands of years, has been mentioned in the book of Genesis.³ Parsley, cloves, guava peels and egg shells have been considered as traditional remedies for bad breath in various countries across the world.³

The overall prevalence of oral malodor in the adult population is uncertain. According to Tonzetich and Ng,⁴ bad breath is a common condition found in approximately 50% of the adult population as a severe chronic problem.^{5,6} Most individuals experience personal discomfort and social embarrassment leading to emotional distress.⁷ The consequences of oral malodor may be more than social; it may signal the presence of disease.⁷

Review of the Literature

Etiology of Oral Malodor

Offensive odors emanating from the oral cavity have been attributed to a variety of etiologic factors, including local and systemic disorders. Oral malodor caused by normal physiologic processes and behaviors is usually transitory.⁸ Extrinsic causes include tobacco, alcohol, and certain foods, such as onions, garlic, and certain spices.^{9,10} Substances absorbed into the circulatory system may be released in pulmonary air or saliva as volatile odoriferous compounds.¹¹ These are best controlled by eliminating the intake of such offensive substances.¹⁰

Intrinsic causes¹² of bad breath are both oral (90%) and systemic in origin (10%).¹ Oral sites in which microbial accumulation and putrefaction occur are suspect.¹³ These may include the interdental and subgingival areas, faulty restorations, dentures, sites of food impaction, and abscesses.^{3,10,14} The coating on the dorso-posterior region of the tongue is also a primary cause for halitosis.^{15,16} The bacteria on the tongue were found to correlate strongly with malodor.⁶

Extraoral sources¹⁷ of odor may include sinusitis, mucous secretions, polyps, postnasal drip, tonsillitis, etc. An increased oral malodor in some women during ovulation, menstrual cycles, pregnancy, and menopause has been reported.¹⁸ Many non-oral diseases, including bronchial and lung infections, kidney failure, various carcinomas, trimethylaminuria, metabolic dysfunction, and biochemical disorders, can result in bad breath.¹⁴ Furthermore, there are people who do not have bad breath, but are convinced that they have oral malodor (halitophobia).¹⁹ However, all these diseases taken together affect a very small percentage of people.³

Certain chemical end products of bacterial putrefaction known as volatile sulfur compounds (VSCs) smell foul and have been determined to be responsible for the offensive odor.²⁰ Volatile sulfur compounds, such as hydrogen sulfide (H₂S), methyl mercaptan (CH₃SH), dimethyl sulfide ((CH₃)₂S), dimethyl disulfide, and sulfur dioxide (SO₂), make up more than 90% of the putrid odors from the oral cavity.²⁰ Methyl mercaptan has a lower threshold of objectionability and is more unpleasant than hydrogen sulphide. Concentrations greater than 0.5ng of methyl mercaptan and 1.5ng of H₂S per 10 ml of air sample have been found objectionable.²⁰ Nonsulfur containing compounds such as cadaverine, putrescine, indole, and skatole have also been implicated in oral malodor.²¹

Relationship of Periodontal Disease to Oral Malodor

The accumulation of plaque and debris and the stagnation of saliva occur most commonly in areas where tooth and tissue crevices lend themselves to stagnant micro-environments, like the posterior dorsum of the tongue, interdental spaces, and subgingival areas. Dental plaque progresses from aerobic, gram-positive colonization to one that is anaerobic, favoring gram-negative growth.²² As the bacterial plaque matures, the oxygen level drops to zero, favoring reduced conditions and the production of odoriferous volatiles.²³ Oxygen depletion is attributed to the bacteria that use oxygen to oxidize substrates (anerobes) from saliva and crevicular fluid.²³

Yaegaki and Sanada²⁴ found that bleeding on probing and periodontal pocket depths positively correlated with production of volatile sulfur compounds (VSCs). Deep periodontal pockets tend to harbor and promote the growth of VSC-producing gram-negative microorganisms like *T.denticola*, *P. gingivalis*, *T. forsythensis* and *Fusobacterium nucleatum*.²⁵ Bosy et al,⁶ have found oral hygiene levels and not periodontal pockets to be more indicative of oral malodor.

Studies have suggested that periodontitis increases the severity of oral malodor.^{15,25} The bleeding tendency of the periodontal tissues may provide essential substrates for odor production.²⁶ The inflamed periodontal tissues provide more methionine, which is converted into methyl mercaptan at a higher rate than in healthy gingival tissues.¹⁵ The increased gingival crevicular

fluid flow in periodontitis may be a continual source of methionine.¹⁵ Increased salivary putrefaction may occur due to a higher concentration of disintegrated epithelial cells.²⁶ Some studies suggest that the production of VSCs by these gram-negative microorganisms may contribute to the progression of periodontal disease via breakdown of the oral mucosa leading to bacterial invasion.²⁷

The average amount of tongue coating was also 6 times greater in individuals with periodontal disease.²⁴ This coating is comprised of epithelial cells, leukocytes, and microorganisms released from periodontal pockets.²⁸

Diagnosis of Oral Malodor

A number of methods have been used to detect the presence of oral malodor.³ Direct tests include sniffing of bad breath and determination of the odoriferous sulfur containing substances by halimetry or gas chromatography.²⁹ Indirect methods identify the odor producing microorganisms or assess their by products in vitro.²⁹ The primary reference standard for the detection of oral malodor is the human nose (organoleptic or hedonic assessment).³⁰ The organoleptic evaluation of oral malodor depends on the person who makes the evaluation and the technique used.

Whole mouth breath assessment (method of choice)²

The subject is instructed to breathe out through the mouth at a distance of approximately 10 cm from the nose of the judge, who is blinded.

Spoon test (assesses odor from the dorsum of the posterior tongue)³

A plastic spoon is used to scrape and scoop material from the back region of the tongue dorsum. The spoon odor is evaluated after five seconds at a distance of approximately 5 cm from the examiner's nose.

The dental floss odor test³ (determines the presence of interdental plaque odor)

Unwaxed floss is passed through interproximal contacts of the posterior teeth and the examiner assesses the odor by smelling the floss at a distance of approximately 3 cm.

The saliva odor test

The subject expectorates approximately 1 to 2 milliliters of saliva into a Petri dish. The dish is covered immediately, incubated at 37°C for five minutes and is then presented for odor evaluation at a distance of 4 cm from the examiner's nose.³¹

A scale commonly used in malodor research is the 0-5 intensity scale used by Rosenberg et al.³² In this organoleptic scale,³³ 0 indicates a concentration of odorant that is below a threshold, and 5 indicates concentrations that are extremely strong. (0-absence of odor, 1-questionable odor, 2-slight malodor, 3-moderate malodor, 4-strong malodor, 5-severe malodor.)

Objective instrumental analysis includes the use of gas chromatography to measure the presence of specific volatile sulfur compounds in expelled mouth air.²⁴ Gas chromatography²⁴ coupled with flame photometry detection is considered the gold standard for measuring oral malodor, because it is specific for volatile sulfur compounds, the main cause of oral malodor.^{15,34,35} The disadvantage, however, is the high cost, time, expertise required, and lack of portability.¹⁶ Odoriferous gases such as cadaverine,²¹ putrescine, and skatole can also be detected by gas or liquid chromatography.

Sulfide monitors^{2,36} analyze total sulfur content of the subject's mouth air using an electrochemical, voltametric sensor, which generates a signal when it is exposed to sulfide and mercaptan gases, measuring the concentration of H₂S gas in parts per billion. Advantages include its portability, low cost, rapid analysis time, and training required.²⁹ The main disadvantages are that it is incapable of distinguishing among individual sulfide compounds, and that measurements are not reliable in the presence of alcohol or essential oils.³⁷

The 'Electronic Nose' is a handheld device, developed to rapidly classify the chemicals in unidentified vapor.³⁸ It has the potential to be used as a diagnostic tool to detect odors.³⁸

Attempts have also been made to measure oral malodor using indirect methods, such as cultures of bacterial isolates, direct bacterial smears, and detection of periodontal pathogens using BANA hydrolysis.³⁹ BANA test is used chairside to determine the proteolytic activity of certain oral anaerobes that contribute to oral malodor.³⁹ Samples of plaque or tongue scrapings are incubated with N - benzoyl - DL - arginine - naphthylamide (BANA), which is a synthetic trypsin substrate.^{16,22} If the organisms have enzymes that degrade BANA, a colored compound is produced within roughly 5 minutes to 15 minutes that indicates a positive BANA test.²⁹ This test may be useful for patient education.^{16,39}

Indices were developed to compare the severity of oral malodor with the extent of coating on the tongue.^{8,5} The Winkel Tongue Coating Index (WTCI) divided the dorsal surface of the tongue into sextants, 3 in the posterior and 3 in the anterior part of the tongue. (Scoring criteria: 0-no coating, 1-presence of light coating, 2-presence of a distinct coating.) The resulting WTCI is obtained by adding all 6 scores.⁸ A greater thickness/extension of tongue coating was assumed to be associated with increased oral malodor. The Miyazaki Index was used to assess the efficacy of various treatment procedures in reducing oral malodor by reducing the coating on the tongue.⁵ A single score for the entire tongue, as well as score per area (anterior and posterior to the sulcus terminalis, each region further divided into left and right sides) is given. The scores ranged from 0 to 3. (0-no coating, 1- < 1/3, 2- < 2/3, and 3- > 2/3 of the surface coated)⁵

Treatment of Oral Malodor

Oral malodor is a multifactorial problem that requires a well-defined approach to diagnosis and treatment. Identification of the major and minor contributing factors and institution of appropriate measures is essential for successful treatment.²⁹ A thorough medical, dental, and halitosis history is necessary to determine whether the patient's complaint of bad breath is due to oral causes or not. If it is determined that the source of malodor is not in the oral cavity, the patient should be referred to a physician for further treatment.^{22,40}

The simplest way to distinguish oral from nonoral etiologies is to compare the smell coming from the patient's mouth with that exiting the nose.³ If the odor is primarily from the mouth, an oral origin may be inferred.³ The first step in treating oral malodor is to assess all oral diseases and conditions that may contribute to oral malodor, including large carious lesions.²⁹

For disease-free people, the aim of treatment is to reduce the overgrowth of microorganisms in the oral cavity, with concomitant reduction in the formation of volatile compounds.⁴⁰ This may be accomplished by mechanical or chemical methods.⁸ Mechanical reduction⁴¹ of microorganisms through improved oral hygiene procedures, both professional and personal, has been associated with reduced oral malodor.^{7,42,43} All patients should be instructed in proper toothbrushing, flossing, use of interdental aids, and tongue cleaning.

The dorso-posterior surface of the tongue has been identified as the principal location for the intra oral generation of volatile sulfur compounds (VSCs).²⁴ Fissures and crypts of the tongue harbor large amounts of *Porphyromonas gingivalis*, *P. intermedia*, spirochetes, etc.¹⁶ These surface irregularities protect the bacteria from the flushing action of saliva and possess low oxygen levels, which facilitate their growth.²⁶ This is an excellent putrefactive habitat for gram-negative anaerobes that metabolize proteins as an energy source.⁴⁴ The bacteria hydrolyze the proteins to amino acids containing sulfur functional groups, which are the precursors to volatile sulfur compounds.⁸

Brushing the tongue significantly reduced concentrations of VSCs, such as methyl mercaptan, and to a lesser extent, hydrogen sulfide.⁴⁵ Some papers suggest a significant reduction in bacterial load^{46,47} in contrast to others, which showed that bacterial load on the tongue was not influenced by intense periodontal therapy including tongue brushing.²⁶ The

beneficial effect of tongue cleaning on oral malodor is therefore primarily related to the removal of the substratum for bacteria, and not to the reduction of the bacterial load.⁴⁴ Taste sensation also improved by removing the thick layer of tongue coating.⁴⁴ Hence, cleaning the tongue is a very effective measure for improving physiologic halitosis.

Use of either a toothbrush or scraper for tongue cleaning is debatable. People, generally, accept a brush to clean the tongue because it does not require an additional tool.⁴⁸ The toothbrush bristles sweep between papillae and remove microorganisms reducing malodor effectively.⁴² Various scrapers were examined for reduction of VSCs following tongue cleaning.⁴⁹ All the scrapers were found to be less effective than tongue brushing.⁴⁹ However, plastic loop scrapers resulted in less gagging and more comfort.⁴⁴ Pedrazzi et al compared the tongue cleaning efficacy of a tongue scraper and soft bristle toothbrush. The tongue scraper showed a 75% reduction in VSCs while the toothbrush only achieved a 45% reduction in VSCs. Patients complained of nausea and tongue mucosal trauma with the toothbrush.⁷ In another study, hemoglobin was detected in saliva after 3 brush strokes among subjects who brushed the dorsal surface of their tongues with a regular toothbrush and 100g force.⁵⁰ The results indicated that use of a regular toothbrush could damage the dorsal tongue and cause microbleeding.⁵⁰ An infant toothbrush would thus be more appropriate.⁵¹

Tongue cleaning is best done before going to bed, because scraping early during the day may induce retching.¹⁰ It must be performed gently in order to remove the mucous and lightly bound debris without doing any damage to the tongue itself.¹⁰ Patients with psychological conditions may overzealously scrape or brush the tongue till bleeding starts.⁵¹ It is important to demonstrate to patients the position of the terminal sulcus of the tongue and the anatomical limits for cleaning.⁵¹ Patients should be instructed to brush from the terminal sulcus to the front of the tongue.⁵² Cleaning the tongue before brushing or asking the patient to momentarily stop breathing may prevent a gag reflex.⁵²

Treatment of periodontal disease⁵³ and improved oral hygiene measures⁴¹ can reduce malodor considerably. It is imperative to correct overhanging restorations and treat deep periodontal pockets for maintenance of periodontal health.³ Scaling and root planing procedures can be effective for patients with periodontitis.⁵³ Scaling and root planing of all pockets in combination with chlorhexidine irrigation resulted in dramatic improvement in the organoleptic malodor ratings.²⁶ This one-stage, full-mouth disinfection resulted in significant improvement when compared to a fractionated periodontal therapy.²⁶

Various chemical plaque control agents have been used as a supplement to combat oral malodor. Chlorhexidine digluconate^{54,55, 56} is useful in decreasing plaque and gingivitis and therefore has shown significant improvement in reducing oral malodor when compared to periodontal therapy alone. Reduced breath odor with significant reduction in microbial load could be observed only when mechanical therapy was combined with chlorhexidine or chlorhexidine-CPC mouthrinsing.⁴³ Mechanical debridement and improved plaque control alone resulted in minor reduction in anerobic species.⁴³ However, routine use of chlorhexidine is discouraged because of reported side effects.⁵⁷

The use of zinc rinses has also been recommended.⁵¹ VSCs are inactivated and converted into non malodorous compounds by formation of zinc salts.^{35,8,58,59} A zinc containing mouthwash reduced VSCs by 80% to 90% for 3 hours after rinsing.⁶⁰ Baking soda^{61,62} and/or zinc-containing toothpastes greatly reduced VSCs in mouth air.^{61,62} Chlorine dioxide mouthrinse eliminates odorogenic microorganisms.^{10,45,63,64} Commercially available mouthrinses contain sodium chlorite since chlorine dioxide readily loses its activity.⁵⁸ Other antiseptics, such as triclosan rinses,¹⁰ cetylpyridinium chloride,⁸ essential oils,³⁷ and hydrogen peroxide,⁶⁵ have also been tried. Hydrogen peroxide mouthwash was effective in reducing oral malodor, but its oxidative activity may be harmful to the oral soft tissues.⁵¹ Oral rinsing with 3% hydrogen peroxide produced impressive reductions in three breath sulfur gases, which persisted for 8 hours.⁴⁵ A combined zinc and triclosan rinse had a cumulative effect reducing malodor, which increased with duration of the product use.⁵⁸

Certain lozenges,⁶⁶ chewing gums and mints, toothpastes, and breath strips have been reported to reduce tongue dorsum malodor. Seventy percent of people who are concerned with halitosis use chewing gum in order to reduce their malodor.⁶⁷ Chewing gum increases salivation and thus oral cleanliness.^{1,10} Chewing gum containing sugar was shown to reduce VSCs in mouth air by altering the pH of the oral cavity.¹³ Mint did not change the concentration of methyl mercaptan, and sugarless chewing gum increased it slightly.⁵² Gum has only short-term effects, masking halitosis with its flavors.⁶⁸

The use of probiotics to suppress oral malodor is now being recognized. Probiotics, as defined by the Food and Agriculture Organization (FAO), are live microorganisms administered in adequate amounts that confer a beneficial health effect on the host.⁶⁹ Kazor et al compared the bacterial populations on the dorsal surface of the tongue in healthy subjects and people with halitosis.⁷⁰ *Streptococcus salivarius* was found to be the predominant species in healthy subjects, but was typically at low levels or absent in those subjects suffering from halitosis.⁷⁰ Hence, probiotic bacteria may have potential application as adjuncts for the prevention and treatment of halitosis.⁷¹

Other methods of managing malodor include chewing parsley, mint, cloves, or fennel seeds.^{1,10} Some herbs like alfalfa, cardamom, chamomile, myrrh, rosemary, and sage are also known to reduce halitosis.¹⁰

Since bad breath is worse when the mouth dries out, patients should be encouraged to drink ample amounts of water.³ Salivary stimulation or use of saliva substitutes, nasal mucous control methods, avoidance of odoriferous foods, and the medical management of systemic diseases have also been recommended as measures to control halitosis.¹⁰

Role of the Dental Hygienist

Oral hygiene care as provided by the dental hygienist becomes an integral part of the total care of the patient.⁷² The dental hygienist may be responsible for recording information for use in diagnosis and treatment planning and for comparison during continuing care evaluations. Early recognition of lesions may prevent the development of conditions favoring malodor. Specific clinical procedures, such as oral prophylaxis, scaling and root planing, correction of overhangs, and temporary restorations can aid in eliminating areas where food debris and plaque are retained, thereby preventing halitosis. In conjunction with these specific clinical procedures, the dental hygienist provides instructions and supervises the patient in assuming plaque control measures. They assist in motivating the patient to develop adequate habits for personal oral care. They may demonstrate the use of proper brushing technique, tongue cleaning, and method of flossing or even assist the patient in selecting a suitable toothbrush or interdental device. The dental hygienist has the best opportunity to communicate and educate the patient regarding avoidance of smoking, odoriferous foods, such as onions, garlic, cabbage, and radishes. Patients with halitophobia need counseling, which will help them achieve self confidence.

Patients debilitated from medical illnesses such as arthritis, dementia, stroke, or other neurologic disorders may not be able to brush and floss properly.⁷³ Regressive changes in the salivary glands affect the quantity and quality of saliva in the elderly.⁷⁴ Therefore, oral malodor is also of concern for the chronically ill, handicapped, or elderly patients.^{73,74} In these situations, dental hygiene techniques can be performed effectively at the bedside with manual instruments and powered toothbrushes. The vast array of chemical plaque control agents may also be used.

Conclusions

Oral malodor or feter ex ore is a foul or offensive odor emanating from the mouth and is a frequent cause for patients to seek treatment. Dental hygienists will likely encounter patients who approach them with this problem.

Intraoral and extraoral factors have been attributed to halitosis. In most cases bad breath originates from the oral cavity itself. Poor oral hygiene, periodontal pockets, faulty restorations, dry sockets, unclean dentures and abscesses are often overlooked as potential sources of volatile sulfur compounds (VSCs).^{3,10} VSCs result from proteolytic degradation by

anaerobic oral microorganisms found abundantly in periodontal pockets and on the surface of the tongue.^{22,29} A variety of methods like organoleptic assessment, gas chromatography, and sulfide monitoring have been used to assess oral malodor.¹⁹

To date, no specific treatment modality has shown consistent results in all cases because of the varied causes for oral malodor. Research is still underway to find a cure for this socially embarrassing problem. In most cases, good professional oral care combined with a daily regimen of oral hygiene, including interdental cleaning, tongue cleaning, and the optional use of a mouthrinse, can lead to improvement. Patient education about oral hygiene practices is crucial for treatment to be effective. Patient assistance and adequate oral health instructions by the dental hygienist can motivate patients to develop a greater interest in maintaining oral health. With increasing demand for dental care, and with continuing advances in dental education and research, there may be a greater potential for the dental hygienist to play a prominent role in the prevention and control of oral malodor.

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Notes

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Effects of Daily Oral Care with 0.12% Chlorhexidine Gluconate and a Standard Oral Care Protocol on the Development of Nosocomial Pneumonia in Intubated Patients: A Pilot Study

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Purpose. *The purpose of this pilot study was to determine if a difference existed between nosocomial pneumonia rates for intubated critical care unit (CCU) patients who received twice-daily oral hygiene care with 0.12% chlorhexidine gluconate and those who received the standard oral care.*

Methods. *Over seven months (February to August), CCU patients were identified through screening and informed consent procedures, and randomized into 1 of 2 groups. Over the 7 months, due to the critically ill nature of the patients, only 5 subjects were enrolled. While in the study, twice-daily oral hygiene care consisted of brushing the cheeks, teeth, and endotracheal tube with a suctioning toothbrush using an FDA-approved 0.12% chlorhexidine gluconate antimicrobial agent with the experimental group (2 intubated patients in the CCU). The control group (3 intubated patients in the CCU) received the standard oral care 6 times per day utilizing a soft foam swab and half strength hydrogen peroxide. All oral care was performed by the nursing staff. The number of persons developing nosocomial pneumonia was monitored until hospital discharge.*

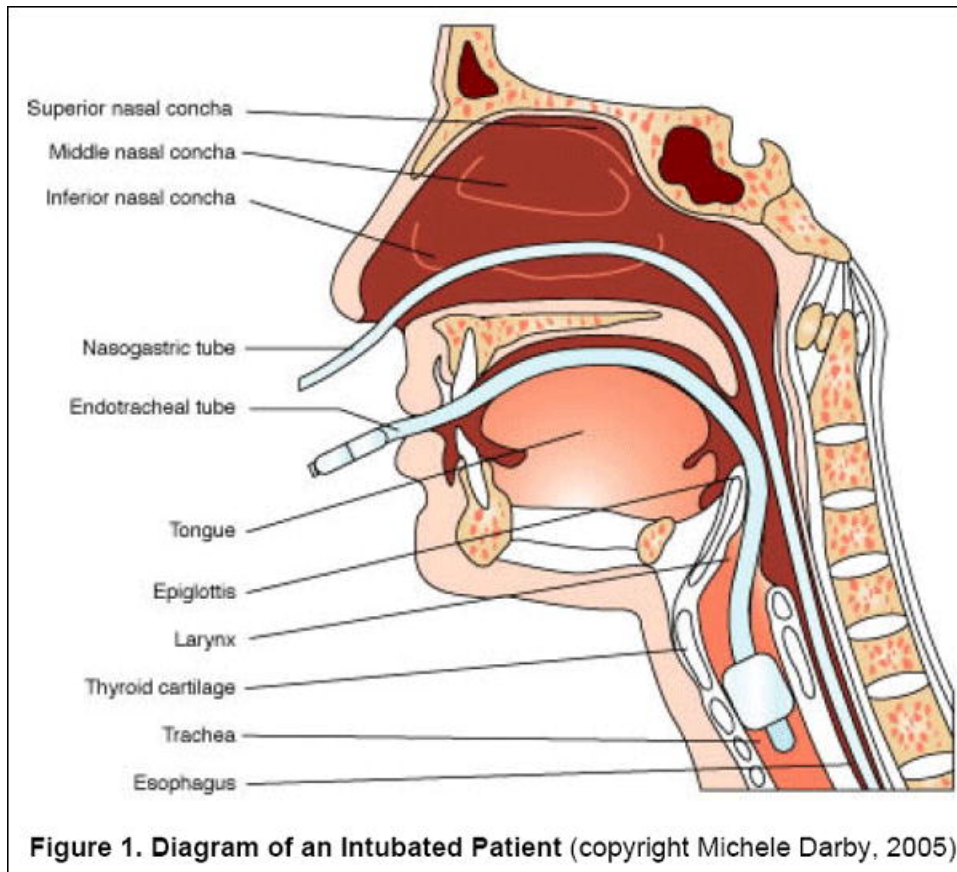
Results. *Results revealed that 1 person out of 3 in the control group was discharged from the hospital with a diagnosis of nosocomial (aspiration) pneumonia. Neither of the 2 subjects in the experimental group was diagnosed with nosocomial pneumonia. Preliminary findings suggest that twice-daily oral hygiene care with 0.12% chlorhexidine gluconate may reduce the risk of nosocomial pneumonia in intubated patients more than the 6-times daily standard oral care protocol. The standard oral care protocol does not include the use of an FDA-approved antimicrobial solution. However, the small size of the sample makes this finding inconclusive.*

Conclusion. *Twice-daily oral hygiene care with 0.12% chlorhexidine gluconate may hold promise as a nosocomial pneumonia reduction strategy within hospital CCUs; however, its application requires further testing.*

Keywords: nosocomial pneumonia, hospital-based dental hygiene, oral-systemic disease links, respiratory disease, oral disease

Introduction

A systematic review of articles on risk factors for nosocomial bacterial pneumonia suggests the oral cavity as a reservoir for nosocomial respiratory pathogens.^{1,2} The incidence of nosocomial pneumonia greatly increases morbidity and mortality and the length and expense of hospitalizations among critical care unit (CCU) patients. Currently, nosocomial pneumonia is the second most common nosocomial infection in the United States.³ At particular risk for hospital-acquired pneumonia are CCU patients undergoing intubation for airway management (See Figure 1). This disease occurs in 20% to 25% of patients treated with mechanical ventilation and is associated with a mortality rate of 50% to 80%.⁴



Current theories explaining this incidence center on sources of normal flora bacteria or nosocomial bacteria from the hospital environment that colonize the patient and are then aspirated into the lungs.^{5,6} Although theories about these mechanisms present strong cases for both nasopharyngeal and gastric colonization, this pilot study focused on the oral environment as the source of bacterial inoculation.

Patients in CCU settings are predisposed to develop colonies of more virulent pathogens than found in the normal oral environment of healthy people. Data supports an association between nosocomial pneumonia and poor oral health for persons with chronic obstructive pulmonary disease (COPD), congestive heart failure, diabetes mellitus, age greater than 70, mechanical ventilations, history of smoking, previous antibiotic treatment, immunosuppression, depressed consciousness, cross-infections, internal tube feeding, gastroesophageal reflux, a long preoperative stay, and/or prolonged surgical procedures.^{1,2,7,8} Predisposing conditions such as mucosal desiccation, xerostomia, reduced immunoglobulin, poor nutrition, severe stress, intubation mechanical injury from nasogastric and endotracheal tubes, and a compromised immune system allow respiratory bacteria to establish a population in the oral cavity. Rapid bacterial growth and mucosal adhesion occurs on pharyngeal mucosa.⁵ These bacteria are then aspirated leading to life-threatening respiratory infection. Various methods

of selective decontamination of the digestive tract using systemic and topical antibiotics have been studied with varying success;⁹ however, broad use of antibiotic therapy increases patient risk of developing resistant bacterial strains.⁹ Hospitals have implemented other strategies to reduce nosocomial pneumonia rates, such as meticulous handwashing by hospital staff, early extubation, frequent suctioning of patients, and semi-Fowler's positioning of patients. Since current research suggests that colonization of the oral cavity with respiratory pathogens precedes pulmonary colonization, the use of effective oral hygiene protocols and antimicrobial products might provide a noninvasive, cost-effective method to decrease the incidence of nosocomial pneumonia in the CCU environment.^{2,9}

The original purpose of this study was to collect preliminary data on a protocol for oral decontamination of intubated patients. The specific question addressed was: Does twice-daily oral hygiene care of intubated patients with 0.12% chlorhexidine gluconate reduce the nosocomial pneumonia rate within a hospital CCU to a greater degree than the standard oral care administered 6 times per day by the nursing staff? The exploratory hypothesis was:

Intubated patients who receive the twice-daily oral hygiene care with 0.12% chlorhexidine gluconate will experience a lower incidence of nosocomial pneumonia compared to those who received the standard oral hygiene care 6 times per day, as measured by overall nosocomial pneumonia rates. However, because of the small number of subjects enrolled, the hypothesis could not be tested and the results are reported as a pilot study. See Table I for terms associated with this topic.

Table I
Terms used in this study

Intubation	The insertion of a tube via the oral or nasal cavity, into the larynx. The purpose of intubation varies with the location and type of tube inserted; generally the procedure is done to allow for drainage, to maintain an open airway, or for the administration of anesthetics or oxygen. For the purposes of this study, intubation was used to ensure a patent airway for the delivery of oxygen. Intubation was determined by the physical presence of an oral endotracheal tube in critical care patients for 48-hours or longer.
Nosocomial Pneumonia	An infection and inflammation of the lung with consolidation and exudation that is pertaining to or originating in a hospital. The development of nosocomial pneumonia was diagnosed by the attending physician and recorded in the patient's chart.
Nosocomial Pneumonia Morbidity	Assessing the condition of acquiring pneumonia within the hospital critical care setting. Nosocomial pneumonia morbidity was measured by the frequency at which intubated subjects developed pneumonia while in the study. This was the original outcome variable measure of the study.
Ventilator-Associated Pneumonia	The development of pneumonia due to or resulting from the presence of an endotracheal tube. The infection can be classified as primary, secondary, or aspiration pneumonia.
Mechanical Ventilation	The placement of an endotracheal tube, through the oropharynx, that is attached to a ventilator in order to assist a patient to breathe.
Oral Hygiene Protocol with 0.12% Chlorhexidine Gluconate	The twice-daily brushing of the oropharynx, including the cheeks, teeth, endotracheal tube with an FDA approved 0.12% chlorhexidine gluconate solution and a suctioning toothbrush. This was the protocol used with subjects in the experimental group.
Standard Oral Care	This is the oral care provided to critical care patients at the site hospital. It included the brushing of the mouth with a soft foam swab dipped in half strength hydrogen peroxide or Listerine every four hours. This was the protocol used with subjects in the control group.

Review of the Literature

Nosocomial pneumonia affects up to 40% of all critically ill or immunocompromised patients with fatality rates reported ranging from 13% to 55%.¹⁰ Patients receiving mechanically-assisted ventilation have higher mortality rates than do patients not receiving ventilation support;⁴ however, other factors such as the patient's underlying disease and organ failure are stronger predictors of death in patients who have pneumonia.¹¹ In addition, nosocomial pneumonia increases the time required for hospitalization by 5 days to 7 days, resulting in increased hospital charges of approximately \$1.3 billion per year.^{3,12} Nosocomial pneumonia is a major infection control issue because of its reported frequency, high fatality rate, and associated costs.⁷

The majority of nosocomial pneumonia cases are associated with extremes of age, underlying medical and respiratory conditions, compromised immune systems, and trauma.^{13,14} Intubation increases the risk of nosocomial infection because it interferes with the body's initial reflexes to dispel aspirated bacteria.¹⁵ Intubation interferes with the body's cough reflex and its mucociliary clearance; intubation also stimulates excess mucous secretions.^{3,13,15} Placement of an endotracheal tube impairs the gag and cough reflexes that normally act to prevent organisms from entering the lower respiratory tract.¹³ In addition, inspired air is no longer heated and humidified by the upper respiratory tract, but instead is artificially heated and humidified by the ventilator. As a result, mucociliary clearance is impeded.¹⁰ In hospital settings, ventilator-associated pneumonia usually occurs as a result of the colonization of microorganisms in the patient's oropharynx or gastrointestinal tract at the time of admission or within a short time of admission.⁴

Numerous factors contribute to an unhealthy oral environment for an intubated patient: the patient's inability to perform oral care; medications that cause xerostomia alter the body's host-response to infection or modify the normal bacterial flora; presence of nasogastric and endotracheal tubing; trauma from the insertion of the endotracheal tube; lack of time for oral care; and ineffective hand washing techniques by hospital staff.¹⁶⁻²⁰ Such opportunities for contamination by the oropharyngeal flora along with the microbial colonization of this compromised environment by more virulent pathogens increases the probability for aspiration and subsequent infection in the lower respiratory tract.

Current evidence-based measures to control nosocomial pneumonia include disinfection of the hospital environment, sterilization of critical care unit (CCU) equipment, pneumococcal vaccines, and education of all health care workers on handwashing to further prevent cross-contamination of patients.²¹ Attention has also focused on decreasing the intubation time, the continuous aspiration of subglottic secretions (CASS), and semi-Fowler's patient positioning. No clearly defined, constantly used, evidence-based protocol has been developed for oral decontamination of intubated patients.¹⁹⁻²⁰

Studies have looked at interventions to reduce the levels of oropharyngeal and gastrointestinal microorganisms, but most methods utilize topical and systemic antibiotics termed selective decontamination of the digestive tract (SDD).²² The goal of SDD is to reduce the number of microorganisms in the oropharynx and gastrointestinal tract; however, it may contribute to antibiotic-resistant microorganisms and superinfections.²³ One study could be found that tested the use of a topical antimicrobial, such as 0.12% chlorhexidine gluconate, for oral decontamination of intubated patients.²⁴ Twice-daily rinsing with 0.12% chlorhexidine has been used successfully for many years in healthy patients to control dental plaque and gingival inflammation.²⁵ Chlorhexidine gluconate mouthrinse has been shown to be beneficial in reducing oral infections and severe mucositis during cancer therapy,²⁶ and to control oral soft tissue inflammation in patients with AIDS.²⁷

Pneumonia may be caused by bacteria that are not normally residents of the oropharynx, but enter from the CCU environment.^{1,9,24,28} The colonization of these microorganisms first takes place in the oropharynx with subsequent aspiration into the lungs.^{1,12,21,24} If the oropharyngeal microorganisms are the primary contributors, then utilizing effective oral antimicrobial decontamination twice-daily may decrease the risk of nosocomial pneumonia and decrease pneumonia rates in CCU patients.²⁴ Development of an effective oral hygiene protocol for intubated patients without the use of SDD could feasibly provide a safe, efficient, and cost-effective way to diminish the morbidity, mortality, and expense of

ventilator-associated pneumonia and nosocomial pneumonia in intensive care unit (ICU) patients.^{19,20,29} The oral care protocol may also have implications for reducing respiratory infections in the elderly and in nursing home residents.^{13,14,30,31}

Methods and Materials

Each orally and nasally intubated patient who entered the critical care unit (CCU) during the 7-month study had an opportunity to participate, pending informed consent from the patient or legally authorized representative/ medical decision maker. Many patients admitted to the CCU were unable to provide informed consent to participate. Upon admission, potential subjects were screened to determine if it was possible for the subject to make the decision to participate, or if a legally authorized representative or health care decision maker would make the decision. To minimize risks, approximately 20 potential subjects with the following characteristics were excluded from enrolling: currently taking metronidazole; a history of allergy to chlorhexidine gluconate; sensitivity to alcohol; moderate or high risk for infective endocarditis; congenital heart disease; a history of rheumatic fever or previous endocarditis; a surgically constructed pulmonary shunt; hypertrophic cardiomyopathy; history of joint replacement within the past 2 years; history of previous joint infection; a prosthetic heart valve; mitral valve prolapse; a joint replacement and immunosuppressed by medications taken for rheumatoid arthritis; systemic lupus erythematosus; hemophilia; insulin dependent diabetes; uncontrolled diabetes; sickle cell anemia; a ventriculoatrial shunt; and /or were admitted to the hospital with pneumonia and were subsequently intubated.

The original desired minimum sample size was 30 to 60, but this number depended on the number of intubated patients who entered the CCU, met the inclusion criteria, and agreed to participate during the study period. At the completion of the study in August 2002, only 5 patients had completed the study (N=5), although approximately 10 other patients met enrollment criteria but declined to participate.

Subjects ranged in age from 28 years of age to 81 years of age. Four of 5 subjects were 50 years of age or older. One hundred percent of the subjects were Caucasian. Two of the subjects were male and 3 of the subjects were female. These 5 patients were randomly assigned to either the control or experimental treatment by using the flip of a coin. A coin flipped that landed on "heads" indicated that the patient be placed in the experimental group; "tails" indicated that the patients be placed in the control group. Patients in the experimental group (n=2) received the twice-daily oral hygiene care with 0.12% chlorhexidine gluconate during their intubation period. The patients in the control group (n=3) received the standard oral care (Table II).

Table II
Characteristics of Intubated Subjects in the Study (N=5)

	EXPERIMENTAL GROUP (n=2)	CONTROL GROUP (n=3)
GENDER	FEMALE FEMALE	FEMALE MALE MALE
MEAN AGE	40 YEARS	73.7 YEARS
AGE RANGE	28 TO 52	62 TO 81
RACE	CAUCASIAN	CAUCASIAN
MEAN LENGTH OF TIME INTUBATED	5.5 DAYS	5 DAYS
RANGE	2 TO 9	4 TO 7
MEAN LENGTH OF TIME IN CRITICAL CARE UNIT	18 DAYS	10.3 DAYS
RANGE	3 TO 33	7 TO 17

The original plan was to utilize a randomized, 2 groups, post-test only experimental design. The 2 independent variables included the twice-daily oral hygiene care with 0.12% chlorhexidine gluconate that the experimental group would receive and the standard oral care that the control group would receive 6 times daily from the critical care nursing staff during their entire intubation time. Twice daily administration of the experimental protocol was used because of the substantivity of chlorhexidine, because dental patients are likely to clean their mouths morning and night, and to accommodate nursing staff efficiencies. The outcome variable was the nosocomial pneumonia rate as determined by the attending physician and recorded in patients' charts.

Although the structure of the study design remained intact, the investigation was modified to a case study due to the final small sample size. Upon discharge, the nursing staff and principal investigator completed a demographic data sheet for each of the 5 subjects enrolled in the study. This information was compiled to analyze the characteristics of the participants in the sample descriptively.

The CCU nursing staff attended an educational session conducted by the 2 dental hygiene researchers on the twice-daily oral hygiene protocol, recruited case study participants at the time of admittance to the critical care unit, provided the twice-daily oral hygiene care protocol with chlorhexidine gluconate to the experimental group or the standard oral care protocol that the hospital already followed to the control group, and kept a record of the oral hygiene administration and adverse effects to the subjects.

The principal investigator visited the CCU every 3 days to 4 days to monitor record keeping and to note adverse effects. No adverse effects were noted. The record of oral hygiene administration was kept with each patient's hospital chart. The equipment and materials utilized for the twice-daily oral hygiene protocol in this study included the Plak-Vac™ Oral Evacuator Brush distributed by Trademark Corporation (Figures 2 & 3) and 0.12% chlorhexidine gluconate distributed by the Discus Dental Company. The materials utilized for the standard oral care protocol included a suctioning foam swab, hydrogen peroxide, and oral lubricant (Figure 4).

Figure 2
Plak-Vac™ Oral Evacuator Brush

Matter and plaque are loosened by the unique Star-shaped filament

Matter loosened by soft bristles is removed along with fluids through suction head.

Matter and fluids evacuate through the hollow handle and pass through the vacuum tube to be collected at wall or portable suction.

Finger control suction port located on back easily regulates suction with your finger tip.

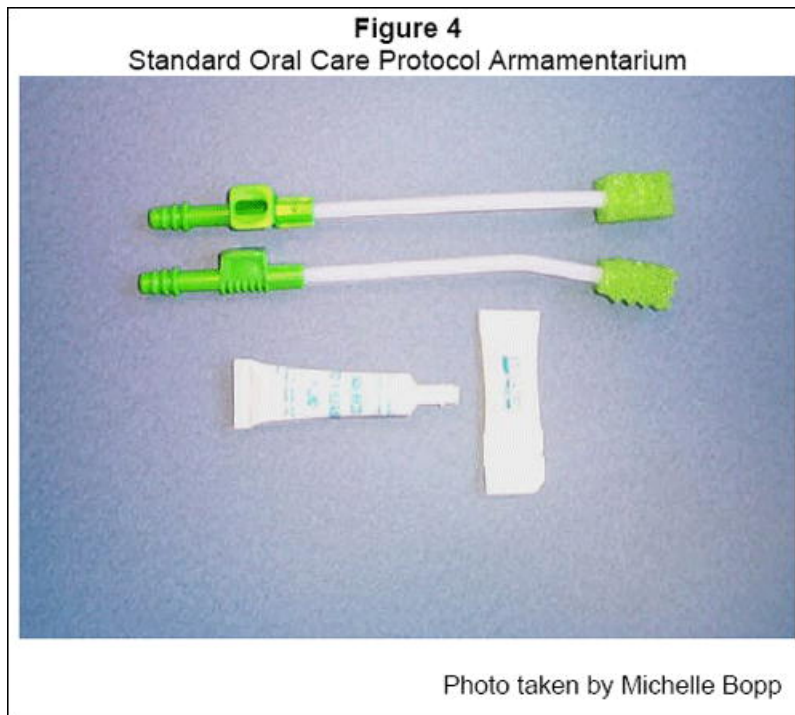
A ten minute demonstration video tape is available FREE from Trademark Medical with the purchase of 12 Plak-Vac! Plus \$5.00 S&H.

Courtesy of Trademark Medical ®

Figure 3
Participant Receiving the Experimental Treatment, The Twice-Daily Oral Hygiene Care with Plak-Vac™ and 0.12% Chlorhexidine Gluconate, From A CCU Nurse.



Photo taken by Michelle Bopp

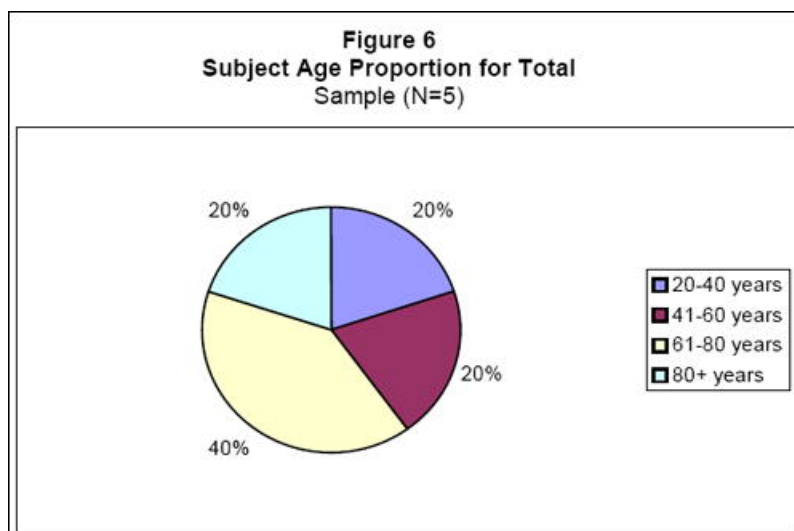
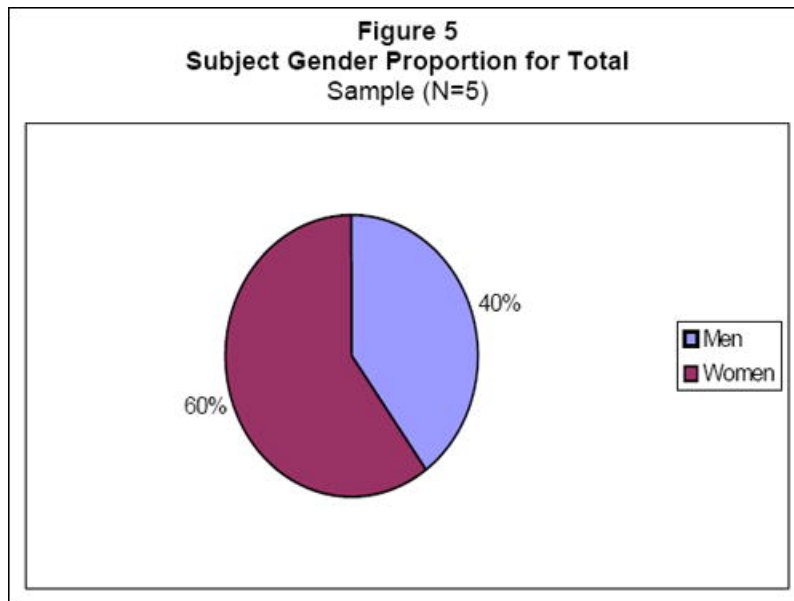


Nothing was requested of the eligible subjects except for their informed consent to receive a twice-daily oral hygiene care regimen with chlorhexidine gluconate or the standard oral care protocol. No modifications were made in the nursing care routine, other than the twice-daily oral hygiene care provided to the 2 intubated patients in the experimental group. Both oral hygiene protocols were conducted for as long as the 5 intubated patients remained in the CCU. These intubated subjects were a transient population to the CCU; none remained for the entire 7-month study period. The CCU nursing staff monitored each subject for nosocomial pneumonia with a diagnosis made by a physician.

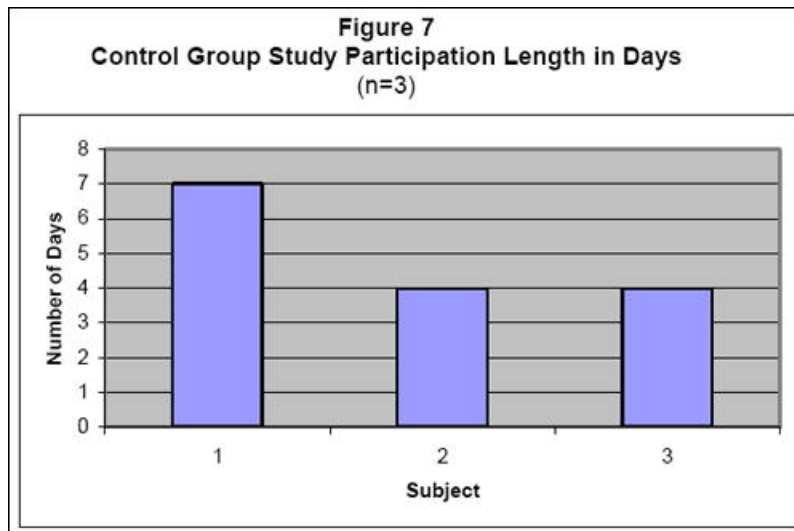
The small sample size prohibited the use of parametric statistical analysis and hypothesis testing; therefore, descriptive statistics, in the form of frequencies, percentages, and measures of central tendency were used. Demographic data were reported to thoroughly describe the patients in the study, to verify group equivalency, and to identify possible external factors that might have influenced the development of nosocomial pneumonia.

Results

Upon discharge from the critical care unit (CCU), the nursing staff and principal investigator completed a demographic data sheet for each of the 5 subjects enrolled in the study. Interestingly, one control group subject was diagnosed with aspiration pneumonia. Males comprised 40% (n=2) of the sample, while women represented 60% (n=3) of the overall sample population (Figure 5). Males comprised 67% (n=2) of the control group, while females comprised 33% (n=1) of the control group and 100% (n=2) of the experimental group. Subjects ranged in age from 28 years of age to 81 year of age (Figure 6). Four of the 5 subjects were 50+ years of age or older. The overall mean age of the subjects was 60.2 years. The mean age of the experimental group was 40 years and the mean age of the control group was 73.7 years. The ethnic/racial background of the subjects was 100 % Caucasian (N=5).



The sizes of the endotracheal tubes for all subjects ranged from 7mm to 8 mm. One hundred percent (N=5) of the subjects were orally intubated; 80% (n=4) had nasogastric tubes. The number of days spent at the hospital ranged from 9 days to 99 days, with a mean of 39.2 days from admission to discharge. The number of days spent in the CCU ranged from 3 days to 33 days, with a mean of 13.4 days. The length of intubation time for the subject who developed nosocomial pneumonia was 7 days. The number of days the control group (n=3) received the standard oral care ranged from 4 days to 7 days, with a mean of 5 days (Figure 7). The number of days the experimental group (n=2) received the oral hygiene protocol with 0.12% chlorhexidine gluconate ranged from 2 days to 9 days, with a mean of 5.5 days (Figure 8).



Discussion

The exploratory hypothesis that intubated patients who receive the twice-daily oral hygiene care with 0.12% chlorhexidine gluconate will experience less nosocomial pneumonia compared to those who received the standard oral care protocol, as measured by overall nosocomial pneumonia rates could neither be rejected nor retained because no inferential statistical analysis could be performed on data from only 5 subjects. However, the fact that one member of the control group developed pneumonia deserves some explanation. Perhaps a contributing factor is that the subject was taking steroids, which can mask the signs of infection, decrease the body's resistance to infection, and undermine the host-defense mechanism.³² The subject was also taking an antibiotic, which can change the normal flora leading to a superinfection.³ Length of intubation (7 days) may have played a role in the subject acquiring nosocomial pneumonia. According to Hixson et al³³ the risk of nosocomial pneumonia increased from 6.5% in those ventilated 10 days, to 28% in those ventilated 30 days. The longer a patient is intubated, the greater the risk of a nosocomial pneumonia infection. The subject's diagnosis upon admission to the hospital was respiratory failure, which is also a risk factor for the development of nosocomial pneumonia.²⁹ The patient was in the control group, which did not receive the 0.12% chlorhexidine gluconate with the Plak-Vac™ Oral Evacuation Brush. The control group received oral hygiene care with a foam swab and hydrogen peroxide. However, a toothbrush is a superior dental aid to a foam swab.^{34,35}

The use of a 0.12% chlorhexidine gluconate mouthwash has been shown to greatly reduce the bacterial load in dental plaque.^{24,25} DeRiso et al²² found that chlorhexidine gluconate reduced nosocomial pneumonia infection rates by 69%. Overall, respiratory infection incidence of gram-negative bacteria was reduced by 67%.²² Perhaps not having the chlorhexidine protocol further contributed to an already high-risk situation for the subject who developed nosocomial pneumonia.

In hindsight, the stringent exclusionary criteria kept too many patients from qualifying for the study, impeding the achievement of a large sample size. As reported by the critical care nursing staff, the patients admitted to the hospital CCU have multiple and complex medical conditions. The primary exclusionary criteria that kept critical care unit patients from qualifying for the study was a diagnosis of uncontrolled diabetes, closely followed by admission with pneumonia and subsequent intubation. Also, given this high degree of medically complicated patients, family members and medical decision-makers often were reluctant to consent to participation in the study. Many felt that their loved ones had been through enough and viewed the study as an unnecessary intrusion.

Demographically, the study sample comprised only Caucasian subjects. This lack of diversity does not favor the generalization of the results to diverse cultural backgrounds. The sample was comprised of a larger proportion of females (n=3). The majority of the subjects were over the age of 50 (n=4). Craven and Steger³⁶ reported that host factors, such as advanced age and underlying diseases, significantly increase the risk of pneumonia and colonization of the upper respiratory tract, but are often not effective targets for prevention. Subjects in the control group were far older than the subjects in the experimental group, which could also explain why one person in the control group developed nosocomial pneumonia.

Craven and Steger³⁶ also suggest the placement of oral gastric tubes in place of nasogastric tubes to decrease the incidence of nosocomial pneumonia. The subject in the control group who was discharged with a diagnosis of aspiration pneumonia did have a nasogastric tube in place. This apparatus may have increased the subject's risk of acquiring the infection. The serendipitous inclusion of the use of continuous suction endotracheal tubes for continuous aspiration of subglottic secretions (CASS) in the CCU may have confounded the study and reduced nosocomial pneumonia rates. CASS has been shown through previous research to be an effective component in the fight against nosocomial pneumonia.^{37,38} Fortunately, this new procedure was used in all the subjects in the case study and therefore its effects were balanced across both experimental and control groups.

Serendipitously, the nurses provided some feedback on their views of the experimental protocol. They noted that the gingival tissues of the experimental group subjects appeared healthier, with less redness and reduced mouth debris. They also felt the Plak-Vac™ Oral Evacuator Brush and chlorhexidine gluconate were easier and less time consuming to use than the standard oral care protocol they currently utilize.

The nursing staff also expressed that the complex medical conditions of the patient population at the hospital CCU were not conducive to subject enrollment. The exclusionary criterion, diabetes, should not have been used because most critically ill patients experience changes in their blood glucose levels temporarily while in the hospital. A reported nursing staff shortage interfered with having a clinical nurse specialist on site who could focus on subject screening, informed consent procedures, and oral hygiene administration. These limitations may also have negatively affected the sample size.

Conclusions and Recommendations

The most important finding of the case study was that no subjects receiving the experimental treatment were diagnosed with nosocomial pneumonia; however, one subject receiving the standard oral care did have an affirmative diagnosis of nosocomial pneumonia upon discharge from the hospital. Results, although inconclusive because of the small sample size and case study format, suggest that the twice-daily oral hygiene care of intubated patients with 0.12% chlorhexidine gluconate may hold promise as a nosocomial pneumonia reduction strategy within a hospital critical care unit (CCU); however, its application requires further testing.

Recommendations for future studies include the use of 0.12% chlorhexidine gluconate at multiple hospital-based sites so that the sample size and diversity can be increased and the findings can be generalized. Furthermore, instead of recruiting patients from the CCU, patients could be recruited in the step down unit or a department where patients have less complex medical conditions. Utilizing a patient population with less complex medical conditions would increase the number of eligible subjects and hence, the number of subjects enrolled, making the study more valid and reliable. Also, a hospital-based person specifically paid to recruit patients on a daily basis is needed for future research in this area. The utilization of a pre-procedural rinse with 0.12% chlorhexidine gluconate before endotracheal tube placement to reduce nosocomial pneumonia risk needs further study. Future research would also need to address the question of whether the reduction in nosocomial pneumonia rates came primarily from the 0.12% chlorhexidine gluconate mouthrinse or from the suctioning of oral secretions and mouth debris by the Plak-Vac™ Oral Evacuator Brush. Preliminary data warrant further investigation. Given the nursing shortage, if mouthcare by nurses can be reduced from 6 times per day to twice daily, then the hospital is likely to accrue savings in personnel time and mouth care supplies.

Acknowledgements

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Notes

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Bisphosphonate-Associated Osteonecrosis of the Jaw: A Literature Review and Clinical Practice Guidelines

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Background. Osteonecrosis of the jaw has recently been reported as a possible adverse drug effect from bisphosphonate therapy. Reports are coming from all over the world. Novartis, a pharmaceutical manufacturer of two implicated drug products, has notified dentists in the United States and made recommendations for dental management of cases.

Mechanism of Action. The exact mechanism of bisphosphonate effects leading to osteonecrosis of the jaw is unknown. The condition can affect both the maxilla and the mandible. Most cases developed following oral infection or dental treatment.

Prevention and Management. Clinical guidelines for prevention and management have recently been published. Dental hygienists have a major role in patient education related to awareness of the potential drug effect and to preventive oral health education.

Keywords: Bisphosphonate, osteonecrosis of the jaw, pamidronate disodium, zoledronic acid

Abbreviations:

ADR - adverse drug reaction, also called adverse drug effect

ONJ - osteonecrosis of the jaw, also called avascular bone necrosis

BIS - bisphosphonate

BON - bisphosphonate-associated osteonecrosis of jaw

IV - intravenous route of administration

FDA - Food and Drug Administration

Introduction

In May 2005 the drug company Novartis Pharmaceuticals Corporation sent a letter to dentists across the United States to warn them of reports of an adverse drug reaction (ADR), osteonecrosis of the jaw (ONJ), observed in cancer patients receiving treatment with intravenous (IV) bisphosphonates (BIS), Aredia (pamidronate disodium) and Zometa (zoledronic

acid). Both drugs are manufactured by Novartis. The warning letter recommended that in dental patients who were being treated with these drugs "invasive dental procedures should be avoided if possible."¹ An additional recommendation was that cancer patients receive a dental examination prior to initiating therapy with IV administered bisphosphonates (Aredia, Zometa).

The letter cautioned that dental surgery may exacerbate alveolar destruction in patients who developed ONJ while being treated with the drugs. When ONJ was observed in patients using BIS, healthcare professionals were asked to submit a report of the serious ADR to the drug company via telephone (800-882-6577) or to the Food and Drug Administration's (FDA) MedWatch Adverse Drug Event Reporting program via telephone (800-FDA-1088) or online at www.fda.gov/MedWatch/report.htm. The FDA form to report an ADR can be downloaded from the FDA Web site, <http://www.fda.gov/MedWatch/getforms.htm>.

Aredia is used to treat conditions such as hypercalcemia of malignancy, Paget's disease, osteolytic bone metastases of breast cancer, and osteolytic lesions of multiple myeloma. Zometa is indicated for hypercalcemia of malignancy, bony destruction from multiple myeloma, and bone metastases from solid tumors. Patients treated for malignancy can have accelerated reduction in bone density leaving them at an increased risk for fractures, especially of the hip and vertebrae. The use of a BIS is considered the standard of care for treatment of moderate to severe hypercalcemia associated with malignancy and for metastatic osteolytic lesions associated with breast cancer and multiple myeloma. The company has added the following statement to the US package insert for both drugs, in the *Precautions* section:

"Osteonecrosis of the jaw has been reported in patients with cancer receiving treatment regimens including bisphosphonates. Many of these patients were also receiving chemotherapy and corticosteroids. The majority of reported cases have been associated with dental procedures such as tooth extraction. Many had signs of local infection including osteomyelitis. A dental examination with appropriate preventive dentistry should be considered prior to treatment with bisphosphonates in patients with concomitant risk factors (eg, cancer chemotherapy, corticosteroids, poor oral hygiene). While on treatment, these patients should avoid invasive dental procedures, if possible. For patients who develop osteonecrosis of the jaw while on bisphosphonate therapy, dental surgery may exacerbate the condition. For patients requiring dental procedures, there are no data available to suggest whether discontinuation of bisphosphonate treatment reduces the risk of osteonecrosis. Clinical judgment of the treating physician should guide the management plan of each patient based on individual risk assessment."²

Novartis published a patient brochure entitled "Taking Care of Yourself While Living With Cancer" in April 2005, which includes information on dental health during cancer treatment and ONJ as a potential ADR. A Dental Consultation Form is included in the brochure to assist the client in coordinating medical and dental care. This brochure is free from the company and can be obtained by calling 800-521-9445 (publication #ONC-8155(03)). As the discussions of the association between BIS drug therapy and ONJ have emerged, another anachronism has been proposed, namely bisphosphonate-associated osteonecrosis of the jaw (BON). The overall prevalence of BON is unknown. The prevalence of BON in patients treated for cancer and who received IV BIS therapy at Sloan-Kettering Cancer Center was reported by the Dental Service of Memorial Sloan-Kettering Cancer Center to be 10.5%.³ Currently, BON can only be considered to be *associated* with BIS, as causality has not been determined.

ONJ in patients who have taken a BIS closely resembles the occupational disorder formerly referred to as "fossy jaw," which occurred in workers in match factories using white phosphorus in manufacturing. In the process of making matches, the worker would often put the match in the mouth to shape the end, thereby introducing phosphorus into the system. The condition was distressingly painful, refractory to treatment, and disfiguring to the extent that some sufferers committed suicide. One author has noted similarities between osteonecrotic lesions in phosphorus-related cases with lesions of BON and has coined the term "bis-phossy jaw" to describe the recent reports.⁴ Both the mandible and the maxilla are affected, with most cases found in the mandible, especially lingual to the prominent mylohyoid ridge. The signs and symptoms that can occur before the appearance of clinically evident ONJ include changes in the health of periodontal tissues, nonhealing mucosal ulcers, loose teeth, and unexplained soft tissue infection.⁵

Other drugs in the BIS class are taken by mouth (tablets) and include alendronate (Fosamax), risedronate (Actonel), etidronate (Didronel), tiludronate (Skelid) and a new BIS approved for once per month dosing, ibandronate (Boniva).

Fosamax, Actonel, and Boniva are approved to treat or prevent osteoporosis and are widely prescribed. In January 2006, the FDA approved an IV dose form of Boniva for the client who cannot swallow or sit upright for 30 minutes. In the early published reports, it was thought the ADR only occurred in the IV dose form, but as more reports emerged, it was discovered that BON developed in some patients taking the oral dose form of the medication. Product labeling for oral dose forms was recently revised to include the possibility of BON. The oral dose forms are manufactured by a variety of pharmaceutical companies. So far, only the Novartis group has issued a warning letter to dentists. It is important for the oral health professional to be aware of this potential ADR in a client reporting a history of taking a BIS because most of the affected patients had jaw disease that was not detected by their physicians or oncologists. Diagnosis was made after the oral problems developed (pain, loose teeth, loss of mucosa, and/or exposed bone) and the patient sought out dental consultation or was referred for dental evaluation by the medical practitioner.

This article will give the timeline of the reports of BON (also referred to by some authors as avascular necrosis of the jaw) and the proposed mechanisms of action leading to ONJ. Prevention and treatment guidelines recently published by the American Academy of Oral Medicine will be provided.

History of ONJ Associated with Bisphosphonates

The first report of dentally-related problems in a patient with osteoporosis being treated with alendronate (Fosamax) was published in 1995. In this case report, 5 fully integrated dental implants failed 6 months after initiation of BIS therapy.⁶ The authors attributed the failure to the bisphosphonates (BIS) and suggested that prolonged use of the medication may represent a contraindication to implant placement.



The first published reports of osteonecrosis of the jaw (ONJ) secondary to BIS drug therapy appeared in September 2003. One report was contained in a lengthy letter by an oral surgeon associated with a medical college in Florida.⁷ The other was a paper written by a group of oral surgeons from the University of California-San Francisco.⁸ Robert Marx, DDS, Division of Oral & Maxillofacial Surgery at the University of Miami School of Medicine, called the condition *avascular necrosis of the jaw* and described it as "a growing epidemic" associated with the intravenous route of administration (IV) of BIS. In his report of 36 cases, 24 had received pamidronate, 6 were treated with zoledronate, and 6 received both drugs.⁷ Although all of the patients were taking other medications, some of which could reduce wound healing, the one single drug class taken by *all* patients was a BIS. The report described painful exposure of bone in the mandible (80.5%), maxilla (14%), or both locations (5.5%), resulting in dental abscess, "toothaches," denture sore spots, osteomyelitis, and/or exposed bone. The typical presentation upon referral was an extraction site that failed to heal and exposed alveolar bone that progressed to formation of sequestrum, with associated localized swelling and purulent exudate. The author explained that

all cases were unresponsive to surgical or medical treatments (antibiotics). The dental procedure that most commonly seemed to exacerbate the appearance of the above signs was tooth extraction; however, 8 (22.3%) patients had a spontaneous manifestation of bone exposure unassociated with prior dental disease. Twenty-two (61%) patients were taking a systemic corticosteroid and 24 (67%) patients were on maintenance chemotherapy for cancer. Only 4 patients had a history of radiation therapy.

A report by Wang et al described 3 patients who were receiving cancer chemotherapy and BIS therapy and who subsequently developed ONJ in the maxillofacial region of the jaw.⁸ In their discussion, they dismissed the relationship of ONJ to BIS therapy due to a lack of reports of the adverse drug reaction and the metabolic action of BIS to reduce bone loss. They attributed the ONJ event as being secondary to taxoid classification chemotherapy drugs (Taxol, Taxotere) used in cancer treatment. Later, however, in a letter to the editor the following year they attributed their cases to BIS therapy.⁹

Later that year (November 2003) Migliorati reported on 5 patients who developed intraoral bone necrosis while taking pamidronate (Aredia) or zoledronic acid (Zometa).¹⁰ Three of these patients experienced spontaneous necrosis of the mylohyoid bony plate in the mandible and 2 patients developed ONJ following extraction of molars (arch unidentified).

A large case series was published in 2004 when Ruggiero reported that over a 3-year-period (February 2001 to November 2003) his clinic had treated 63 cases of bisphosphonate-associated osteonecrosis of jaw (BON).¹¹ Both males and females were affected. The age range was from 43 years to 89 years. Most patients had a history of various malignancies and received IV administered BIS, but 7 cases had no history of malignancy and were being treated with an oral dose form for osteoporosis. This was the first report of the oral dose form leading to BON. The small number of cases in the report may indicate that the risk of the complication is very low with the oral dose form, or it may represent underreporting of BON in patients taking oral dose forms. The disease was described as resembling osteoradionecrosis seen following radiation to the head and neck. The most common presenting symptoms were either a nonhealing extraction socket or an alveolar bone exposure. Oroantral fistula formation secondary to necrosis in the posterior maxillary area occurred in several patients. Radiographs revealed a mottled radiographic appearance with evidence of sequestrum. Six patients had abnormal bone in pre-extraction radiographs, suggesting alveolar changes prior to tooth extraction. Since some patients had received BIS as part of cancer chemotherapy, it was thought that the necrosis may represent metastatic cancer. However, a biopsy of lesions showed no evidence of malignancy. Most patients presented with pain and exposed bone following previous tooth extraction, but 9 patients (14%) had no history of recent dental surgery and had spontaneous breakdown and exposure of necrotic alveolar bone with no known cause. Conservative debridement of the bone and administration of antibiotics were not effective in resolving the disease and most patients required surgery to remove affected bone. Five patients had persistent necrosis of alveolar bone or new regions of bone necrosis, although they were no longer taking BIS, so discontinuing the drug didn't seem to help resolve the condition. All patients on oral BIS were managed with sequestrectomy (removal of exfoliated bone); none had to have resection of the affected area. This infers, perhaps, less serious damage to alveolar bone with the oral dose form. The authors concluded that dentists should be aware of this formerly unrecognized potential complication of BIS therapy and should monitor patients for untreated dental disease as a strategy to prevent the future need for tooth extraction or osseous surgery. A follow-up of Ruggiero's cases includes information that he now has 130 cases, with 16 cases of ONJ in patients taking oral dose forms of BIS (S. Ruggiero, DDS, email communication, September 25, 2005). This is still a very small group since over 21 million prescriptions for alendronate were prescribed in the United States in 2004 (<http://www.pharmacytimes.com/article.cfm?ID=2534>) and over 6 million prescriptions of risedronate. Risk factors leading to development of BON are unclear at this time and the appearance of the ADR developing spontaneously in clients with no dental disease is troubling.



In February 2005, Bagan et al published a report of 10 cases of BON that developed following cancer chemotherapy.¹² All 10 cases had mandibular involvement and 5 had maxillary involvement as well, with an average of 2 painful areas of exposed bone. In 7 patients, tooth extraction preceded the onset of ONJ. Two patients developed oroantral communications and another developed a cutaneous fistula to the neck with suppuration. Histopathological diagnosis was chronic osteomyelitis with no evidence of metastatic disease in the jaws. All patients had received a BIS as part of the cancer chemotherapy and a BIS was the only drug used by all patients. They concluded that there appears to be a relationship with the use of BIS and subsequent development of ONJ.

Another case report in February 2005 from Canada described the development of ONJ following tooth extraction in a client with a history of BIS therapy.¹³ They recommended that medical professionals should request that a dental examination be completed before prescribing BIS therapy.

In April 2005, a report from Australia, Adverse Drug Reactions Advisory Committee, Department of Health and Ageing, identified 13 cases of BON reported to the Agency.¹⁴ Twelve patients received IV administered BIS therapy as part of cancer chemotherapy and one took oral alendronate (Fosamax) for osteoporosis. Time from administration of drug therapy to onset of ONJ ranged from one month to more than 4 years. Four cases involved the mandible, 2 in the maxilla, and the others did not specify the location, other than the "jaw." Four reports documented that dental extraction occurred in the months before the onset of BON. One patient had teeth extracted because they became loose during BIS therapy. A further report to the Agency stated that onset of ONJ occurred, in some cases, before dental extraction. They reported that the condition caused chronic pain, dysfunction and disfigurement, and that no treatment has been consistently effective. Withdrawing the BIS did not seem to hasten recovery. Six cases had not recovered following dental management, one case was slowly improving, and 6 case reports did not comment on the outcome of therapy. The medical officer in charge of the governmental committee recommended that necessary dental treatment be completed before BIS therapy begins, as a measure to prevent BON.



Images courtesy of Sal Rugiero, DMD, MD

In the same issue of the medical journal, a group of oral surgeons reported on 5 ONJ cases from 2003 who were treated in the Oral and Maxillofacial Surgery Unit at Royal Adelaide Hospital, South Australia, with painful exposed bone in the maxilla or both the maxilla and mandible.¹⁵ Bisphosphonates involved included IV pamidronate (Aredia) or an oral daily dose of 40 mg alendronate (Fosamax), which is the recommended dose for Paget's disease. Duration of therapy was 6 months to 6 years. Histology revealed no evidence of metastatic malignancy or Paget's disease from the affected sites in the jaws. Only 2 of the 5 cases resolved with treatment and those were treated with local debridement and removal of sequestrum. Of the cases that did not resolve with treatment, 2 received hyperbaric oxygen treatment. Hyperbaric oxygen therapy has been reported to be helpful in treating radiation-induced osteonecrosis of the jaw. The outcome following hyperbaric oxygen therapy was not identified in the report.

A recent report involving periodontally involved clients describes BON in 9 clients with a history of various malignancies who took IV administered BIS treatment.⁵ The mandible was affected in all cases and 2 cases had maxillary involvement, as well. All clients developed ONJ following tooth extraction of hopeless periodontally-involved teeth. In 2 clients, the lesions appeared spontaneously in edentulous areas. The duration of BIS therapy ranged from 10 months to 70 months. The time of extraction to diagnosis of BON ranged from 3 months to 12 months. All cases, but one (who died due to progression of metastatic bone disease), were treated with debridement and cyclic oral antibiotics. The majority of patients experienced regression of pain and local infection, although despite treatment, areas of exposed bone persisted in all cases.

The most recent case report by Marx and associates includes 119 cases.¹⁶ The vast majority of patients had received IV administered BIS as part of cancer chemotherapy and 3 cases took alendronate (Fosamax) for osteoporosis. The mean time from initial BIS administration to symptomatic bone exposure ranged from 9.4 months (IV dose forms) to 3 years (oral dose form). The posterior molar area of the mandible was involved most often, although some patients had BON in both arches or exclusively in the maxilla. Most patients presented for dental examination due to pain, although BON was discovered during routine oral examination or through self-examination in about 30% of patients. Precipitating events leading to development of BON, from those most common to least common, included tooth extraction, periodontitis, periodontal surgery, dental implants, and endodontic surgery. Spontaneous bone exposure with no recent history of dental treatment occurred in about 25% of cases. Pain was controlled using a regimen of antibiotics and chlorhexidine mouthrinse, although exposed bone remained in all cases.



Reports of BON are being published in a variety of countries outside the United States There are reports in Australia (www.mja.com.au/public/issues/182_08_180405/pur10144_fm.html), (www.mja.com.au/public/issues/182_08_180405/car10429_fm.html); Canada (www.cda-adc.ca/jcda/vol-71/issue-2/111.pdf); Singapore (www.annals.edu.sg/pdf200409/V33N4p48S.pdf); Europe (<http://annonc.oxfordjournals.org/cgi/content/extract/16/7/1207>); Israel¹⁷, Germany,¹⁸ and Italy.⁵

Most likely, an accurate report of the worldwide prevalence of BON is unknown since only 1% to 10% of ADRs are reported to the FDA and other drug safety reporting agencies (FDA Medwatch, personal e-mail communication, November 7, 2005).

Mechanism of Action of Bisphosphonates and ONJ Complication

Bisphosphonates (BIS) are prescribed for prevention of bone resorption in metastatic malignant disease and other osteolytic diseases, and to increase bone density and reduce fractures in osteoporosis. The precise mechanism attributed to inhibition of bone resorption of BIS is unclear. BIS bind to bone and are very slowly metabolized. They are internalized by osteoclasts in the process of bone resorption and the life span of these cells and their activity are diminished. Additionally, osteoclastic breakdown of necrotic bone is inhibited. These combined effects reduce bone resorption which is the reason for Food and Drug Administration (FDA) approval of BIS to treat osteoporosis and other disease conditions where osteolysis occurs. BIS are reported to be helpful in preventing loss of bone density and fractures in the hip, spine, and other skeletal regions, but in the jaw, where osteoclastic remodeling of bone is natural in the dynamic cycle of alveolar bone maintenance, this depression of osteoclastic activity may impair the structure of jaw bone in a manner as yet unidentified. As diseased bone is not removed (by depression of osteoclasts) and new bone is formed (by unaffected osteoblasts), the result is that diseased bone is "walled off" and healthy bone is laid over diseased bone.⁴ It was believed up until recently that oral BIS protected long bones and spinal bone and that only the jaw was affected adversely by BIS drugs. However, a group from the Center for Mineral Metabolism and Clinical Research recently reported 9 cases of spontaneous nonspinal fractures in patients after 1 year to 8 years of alendronate (Fosamax) treatment.¹⁹ Six of these cases displayed either delayed or absent fracture healing for 3 months to 2 years during therapy. Histological examination revealed diminished matrix synthesis, suppressed bone formation, with absent or reduced osteoblastic surface in most patients. They concluded there is a possibility of severe suppression of bone turnover during long-term alendronate therapy, resulting in increased susceptibility to and delayed healing of nonspinal fractures.

Physiology of Alveolar Bone Turnover.

It is well known that remodeling of bone in the jaws via osteoclastic and osteoblastic activity is a normal physiologic process. The osteoblast secretes both the collagen and mineral, which form the mineralized bone. When osteoblasts are

entrapped within the developing bone and become surrounded by this mineralized bone, they are termed osteocytes. The cellular change results in the osteoclast surrounded by this mineralized bone within cavities called lacunae. The life span of the osteoclast is about 150 days. The osteoclast resorbs the nonvital bone and releases growth factors, which induce differentiation of osteoblasts capable of new bone formation. The interruption of this cycle by the bisphosphonates' (BIS) action, which inhibits bone resorption, may result in an accumulation of nonvital osteoclasts and microfractures of old mineral matrix.⁸

The mechanism involved in bisphosphonate-associated osteonecrosis of jaw (BON) is unknown. The leading theory to explain the mechanism suggests that it is caused by cessation of bone remodeling and bone turnover by the inhibition of osteoclasts by BIS.¹⁶ One postulation is that it may be due to an antiangiogenic effect leading to inhibition of blood vessel formation within bone.²⁰ During the process where a BIS reduces the vascular supply to bone (by inhibiting the formation of blood vessels, endothelial cells, fibroblast growth factor, and endothelial growth factor), this may promote an avascular bone necrosis effect. Because daily bone remodeling related to the periodontal ligament results in a greater blood supply and a faster bone turnover rate in the jaw than in other bones in the body, BIS are highly concentrated in the jaws. Thus, exposed necrotic bone in the jaws may represent a direct result of BIS action on the daily remodeling and replenishment of bone.¹⁶ The rich blood supply in the oral cavity generally promotes rapid healing of oral wounds. Reduction of this blood supply, leading to delayed wound healing, may be a major factor in BON. Observations of "bone that does not bleed normally" have been reported.¹¹

Prevention and Management

The American Academy of Oral Medicine has proposed clinical guidelines for prevention and management of bisphosphonate-associated osteonecrosis of jaw (BON).²¹ The recommendations are based on expert opinion as there are no available randomized controlled trials to support prevention and management strategies. Prevention of BON is not possible at this time, but the incidence possibly can be reduced by treating all dental disease prior to initiating bisphosphonate (BIS) therapy. It has been noted that the most common clinical history associated with this process is absent or delayed hard- and soft-tissue healing after dental extractions.^{7,11,22} In patients who develop BON spontaneously, the most common initial complaint is the sudden presence of intraoral discomfort and the presence of roughness irritating the tongue in the area of necrotic bone. Nonsurgical dental procedures should be used to manage osteonecrosis of the jaw in patients reporting a history of BIS therapy.¹⁶ The majority of patients (90.1%) gained a pain-free state (although exposed bone remained) using a regimen of antibiotics (when secondary infection developed) and a daily 0.12% chlorhexidine mouthrinse. The recommendations of the AAOM include implementation of measures to *prevent* the condition in patients at risk for developing BON with the dentist and physician working collaboratively.²¹ When the collaborative relationship is established, a preventive/management protocol should include:

Prevention:

- A dental examination should be recommended by the MD/oncologist before intravenous route of administration (IV) BIS therapy is initiated.
- " Patients who have been given oral forms of a BIS within the last 3 months should have a complete dental evaluation.
- Dental therapy to remove disease or inflammation should be provided before the risk of developing BON increases.
- Dentists should follow existing guidelines for a dental consultation for the prevention of oral complications of cancer therapy (chemotherapy, radiation therapy, pre-hematopoietic stem cell transplantation). Elimination of all potential sites of infection must be the primary objective of the consultation. The goal of therapy should be to attain oral and dental health so that during the active phase of BIS therapy, only 3 to 6 months of maintenance hygiene appointments will be necessary.

- A comprehensive extraoral and intraoral examination should be performed by a dental professional, including a full radiographic series and panoramic radiograph, to assist in the diagnosis of dental disease, the evaluation of third molars, and the identification of metastatic cancer and other bone pathology.
- The periodontal health status should be determined and appropriate therapy provided. Pocket elimination is of importance to reduce plaque accumulation, minimize chronic periodontal inflammation, and minimize acute periodontal infections.
- Extraction of teeth should be completed as soon as possible.
- Restorative treatment to eliminate caries and defective restorations should be completed. Crowns and extensive fixed prosthodontic appliances may not be appropriate for some patients. Prosthodontic appliances should be evaluated for fit, stability, and occlusion, and adjustments made when necessary.
- Prophylaxis should be performed and oral health instructions given, including the possible, but uncommon adverse effect of BON in patients who have taken BIS medications. Instructions should include early signs of BON so the patient may be able to recognize the condition should it develop. This is an essential component of the regimen and one in which the dental hygienist can play a major role.
- " Periodic maintenance visits should be included in the treatment plan to reinforce the importance of oral hygiene maintenance and to conduct a new oral examination. It has been estimated that BIS can remain within the bone for at least seven, possibly more, years.

Management:

When lesions suggestive of BON are reported the following is recommended:

- Routine restorative care may be provided and local anesthetics used as necessary.
- Scaling and prophylaxis should be done as atraumatically as possible, with gentle soft-tissue management.
- Avoid dental extractions if possible unless the teeth have a mobility score of 3 or greater. Extractions should be performed as atraumatically as possible. Patients should be followed up weekly for the first 4 weeks afterward, then monthly until the sockets are completely closed and healed. If there is an indication for antibiotic use (due to secondary infection), amoxicillin (alone or in combination with clindamycin) may help to reduce the incidence of local infection.
- Teeth with extensive decay should be considered for endodontic therapy. They should be prepared as overdenture abutments. The crown should be excised at the gingival margin. This is especially important in patients with a history of previous extractions that resulted in BON. In these patients, extraction should be avoided.
- The area of BON should be treated only with the objective of eliminating sharp edges of bone that may traumatize soft tissues, especially when the lingual aspect of the posterior mandibular arch is involved. Superficial debridement may be performed, as needed, to eliminate areas that may further traumatize adjacent tissues.
- Clinicians should follow up with the patient every 2 weeks to 3 weeks to reevaluate the areas and to ensure that suppuration has not developed.
- If the area around the exposed bone exhibits tender erythema and suppuration and/or sinus tracts, the patient should be treated with antibiotics until the areas resolve. Microbiologic culture and sensitivity tests may be helpful; however, the clinician must realize that culture results do not always guarantee microbiological etiology since host oral flora can colonize the necrotic bone surface.
- The use of a chlorhexidine mouthrinse 3 or 4 times a day is recommended to reduce bacterial load and colonization. Irrigation of areas of BON with chlorhexidine may be performed in the evaluation visit.
- A surgical approach with the goal of removing necrotic bone and closing the site with a healthy mucosa may be considered for patients with multiple myeloma who require hematopoietic stem cell transplantation. In a patient with exposed necrotic bone, the risk of undergoing high-dose conditioning chemotherapy in preparation for transplantation is unclear. The necrotic area may act as a portal of entry for bacteria; it may traumatize the adjacent soft tissues and cause ulceration, forming another portal for bacterial contamination. Furthermore, surgical manipulation may not

lead to the closure of the necrotic site but to further increase of the osseous breakdown and dehiscence. If a surgical procedure is needed, patients should be informed of the potential risks and benefits.

- The role of hyperbaric oxygen therapy in BON is unknown at this time.
- Soft vinyl appliances or obturators may help cover exposed necrotic bone to prevent further trauma to soft tissues. These appliances should not rest on the necrotic bone. The interior portion of the flanges must be relieved so as not to deliver pressure to the diseased tissues, but rather to serve as a barrier to protect them. Appliances should not be designed for use during mastication.
- Existing prosthetic appliances should be reevaluated to ensure they are properly fitted. Relining a denture with a soft liner to promote a better fit and to minimize soft-tissue trauma and pressure points is recommended.
- Odontogenic infections should be treated aggressively with systemic antibiotics. When possible, identification of the responsible microorganisms and respective antibiogram (chart produced by laboratories, which documents the percentage of microbes that are sensitive to particular antibiotics) is indicated. If empiric therapy is to be used, although penicillin is the first-choice antibiotic in dentistry, amoxicillin and/or clindamycin provide better bone penetration and a wider spectrum of coverage.

At this time, management of BON is empirical and based on individual experience. As new information becomes available and the results of well-designed clinical trials are known, better guidelines and management protocols based on results of scientific evidence will be developed. At the present time, dentists, physicians, and oncologists should establish communication to ensure patients are referred for oral examination prior to initiation of BIS therapy. The dental hygienist should be involved in the consultations to facilitate the communication of appropriate information in the oral health education care plan. Evidence indicates that the risk of BON may persist for years after discontinuation of the drug, so long-term follow-up is needed. The dental hygienist must be able to recognize signs of BON and assist the dentist in the maintenance and periodic evaluations of patients at risk for the condition. Patients who are receiving BIS drug therapy should be informed of the possibility of developing BON following routine dental treatment. A consensus must be reached among the patient, the dentist, and the physician before dental therapy is initiated.

Future Research

Research should be initiated to determine if there are radiographic features to identify the client at risk for BON. Since not all patients develop BON, determination of cofactors that increase the risk for development of the condition need to be made. Histologic studies to determine the extent of damage to alveolar bone should be completed in order to answer the following questions:

- Is alveolar bone adversely affected in all people who have taken a bisphosphonate (BIS)?
- Is healthy bone ever available in the jaw when BIS drugs have been received?
- How long after taking a BIS is bone likely to be affected?
- What factors place a person at risk for developing BON?

Reports continue to be published on bisphosphonate-associated ONJ.^{23,24,25,26} The International Myeloma Foundation conducted a Web-based survey in July 2004 to determine prevalence of ONJ with IV administered BIS (http://www.myeloma.org/main.jsp?type=article&tab_id=1&menu_id=0&id=1259). They report a 10% incidence among patients receiving zoledronic acid and a 4% incidence among patients receiving pamidronate.²⁷ Dental authors are informing the medical community.²² Dentists are being cautioned regarding the need for accurate pulpal and periradicular testing procedures to establish a clear diagnosis before proceeding with endodontic treatment when clinical signs of ONJ are present in patients with a history of taking BIS drug therapy.²⁸ In this report of 2 cases referred for endodontic therapy, medical evaluation determined that both clients with periapical disease who developed ONJ had received BIS therapy. Endodontic therapy and long-term antibiotic therapy did not resolve BON in these patients.²⁸

Conclusion

Many questions remain concerning the degree of risk for future manifestation of bisphosphonate-associated osteonecrosis of jaw among the huge population who are taking bisphosphonate (BIS) drugs or who have done so in the past. The association between osteonecrosis of the jaw and BIS is not considered by some authors to be causal at this point. Further research needs to be completed to determine causality. Fortunately, there are other non-bisphosphonate drugs for prevention and treatment of osteoporosis that have not yet been reported to cause ONJ. These agents include raloxifene (Evista), calcitonin (Micalcin Nasal spray) and teriparatide (Forteo). Both Evista and Miacalcin affect the osteoclast to reduce bone resorption, but since there are no reports of ONJ with either drug, hopefully the action of these drugs is insufficient to cause the effect.

Note: Since this article was accepted for publication, a systematic review on BON has been published (Woo et al. *Ann Intern Med.* 2006;144:753-761), which represents a position paper of the American Academy of Oral and Maxillofacial Pathology. Recommendations for dental management concur with AAOM guidelines discussed in this article.

Other drugs in the bisphosphonate class are taken by mouth and include alendronate (Fosamax), risedronate (Actonel)... and a newly approved bisphosphonate for once/month dosing, ibandronate (Boniva). In January 2006 an IV administered dose form of Boniva was approved for patient who cannot swallow or sit upright for 30 minutes.

It is important for the oral health professional to be aware of this potential ADR in a client taking bisphosphonates because most of the affected patients had bone disease that was not detected by their physicians nor their oncologists.

Instructions should include early signs of BON so the patient may be able to recognize the condition should it develop. This is an essential component of the regimen and one in which the dental hygienist can play a major role.

Web Resources

Maxillofacial Center for Diagnostics & Research, Morgantown WVA [<http://maxillofacialcenter.com/NICOhistory.html>] Accessed 10/2005 International Myeloma Foundation (<http://www.myeloma.org>) Food and Drug Administration www.fda.gov/medwatch/safety/2005/zometa_deardentite_5-5-05.pdf Accessed 10/2005

Acknowledgements

Notes

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