PRIMARY PREVENTIVE DENTISTRY - 6th Ed. (2004)

Front Matter

TITLE PAGE

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PREFACE

This is the sixth edition of the text, *Primary Preventive Dentistry*. The successive editions since 1982 have provided an excellent example of the fact that the useful lifetime of much knowledge is finite. At the time of the first edition even such dental essentials as mechanical and chemical plaque control, access to dental care and dental insurance were only being slowly accepted. Now, a new wave of dental visionaries is coming on the world stage to speak with confidence about future vaccines, genetic engineering and therapeutic stem cells. These are exceedingly important basic science subjects to all health professions and are only now creeping into the general dental lexicon and application.

Like in past editions, the information in the text and supporting references has been

greatly upgraded, although every effort has been made to retain original citations from past landmark research. An increased emphasis has been given to school programs because of the increasing number of school based health clinics (SBHC) that are being developed to care for children. Risk assessment is highlighted in the text as a necessity for determining at the time of an initial/annual clinical examination whether a patient's treatment is to be preventive or restorative. Remineralization of incipient caries, an old idea, but a relatively new weapon in the dentists' arsenal, offers a new preventive strategy for those seeking to maintain intact teeth for a lifetime.

Throughout this approximate last quarter-century of metamorphosis, the format of the book has remained constant. It is written in a style that is user-friendly, whether the user is a dental or dental-hygienist student, a dental assistant, a private- or public-health practitioner, a health educator, or a school nurse. The book and suggested learning strategies have been successfully used for class-paced study; they have been used for remedial programs; and they have been used for remote self-paced learning as well as for scheduled continuing education courses.

Each chapter commences with a series of objectives—subject matter that the authors consider essential. Key words and concepts are *italicized* in each chapter to help focus on information deemed important. Throughout the text, there are embedded clusters of true-and-false questions, as well as answers and fill-in-the-blank questions at the end of the chapter. These are included for student self-evaluation.

Following the class presentation of the subject matter it is recommended that about an hour-or-so should be spent outside the classroom to review the chapter. As each question is encountered for which the answer is not completely understood, a check mark should be made before reading on. At the end of the chapter, the marked questions should be again reviewed and the answers learned at the 100% level—not just memorized.

Prentice Hall has, with this sixth edition, established a website for the book that permits a student to take a "mock examination" at the end of each chapter. A personal or institutional computer is a requisite for Prentice Hall to respond to new true-or-false, essay, and to fill-in-the-blank type of questions. The true-or-false questions will be computer marked and returned immediately to the students e-mail address. The essay and fill-in-the blank questions will *not* be marked because of the variety of possible correct answers submitted, but will be immediately returned to the student along with the "school answers" for comparison. This exchange between the student and the Prentice Hall website is strictly between two computers. *No student records will be kept at the website*. The goal of the program is to provide the learner with a means of self-evaluation of his/her level of attainment. Student participation in this voluntary, non-jeopardizing, website program can result in a huge step towards achieving long-term mastery learning. The questions in the question bank are also available to teachers who might desire to use them for their own purposes

Since curriculum time allocations vary from institution-to-institution, the chapters do not need to be scheduled in a given sequence, being free standing for the indexed subject matter. The 23 chapters include the theory and practice of preventive dentistry in private and public health environments. One chapter discusses plaque formation, while two chapters each emphasize the importance of caries and periodontal disease

and disease prevention. To aid in combating these two plaque diseases, there are chapters on dentifrices, toothbrushing and auxiliary tooth cleaning devices used in accomplishing mechanical and chemical plaque control. Sugars, diets, and human motivation are included to facilitate better counseling of patients. A chapter is devoted to the use of pit-and-fissure sealants. Chapters on public health point out the responsibilities of a public health dentist, as well as two chapters on the oral health advantages of fluoride—water fluoridation, and topical applications—both of which are prime preventive tools of a public health dentist as well as for the private practitioner. Different age and health status groups are also considered in separate chapters—pedodontic, geriodontic, handicapped, and hospitalized individuals. Finally, there is a chapter on how to use risk assessment to integrate prevention into the total treatment plan.

In summary, the authors have contributed the chapters of updated information, the editors have established the learning system, while Prentice Hall has provided a website for worldwide user self-evaluation.

ACKNOWLEDGMENTS

For a multiauthored and multi-edition book text, there is a need for a lot of credit to go around. Lest we forget, the authors of the first edition established the foundation, from which the several later editions in preventive dentistry have been upgraded. Approximately 60 authors and authoresses have contributed of their knowledge and time through their writings from the first to the present sixth edition. These authors and authoresses have come from research laboratories, state and national public health agencies and teaching institutions in the United States and overseas. Authors from Canada, Korea, England, South Africa, Switzerland and Sweden are represented in the latter group. A spin-off Spanish edition of the fifth edition of the text has been published reflecting this multinational approach to the book. Manufacturers and dental-supply houses have contributed photos and information on their products, while journal publishers have given permission for use of copyright material. Teachers using the book, and students learning from the book, have both made suggestions that have enhanced the value of the texts.

Very few texts would be published without the help of a publisher. For this publication by Prentice Hall, there is Melissa Kerian who kept us on schedule, Amy Peltier who has lent her computer expertise, and Mark Cohen, the book editor, who harmoniously kept everyone staying the course. To those many other known and unknown individuals who helped develop this edition of the primary dental prevention text, the editors desire to voice heartfelt appreciation. Of a more personal nature, both editors wish to thank their wives, Katherine Garcia-Godoy and Grace Harris for their continuing support and encouragement.

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Chapter 1. Introduction to Primary Preventive Dentistry - *Norman O. Harris*

Objectives

At the end of this chapter, it will be possible to

1. Define the following key terms: health, primary prevention, secondary prevention, and tertiary prevention. Also, provide one specific example of each.

2. Name three convenient categories that aid in classifying dental disease and in planning oral-disease prevention and treatment programs.

3. Name four strategies and two administrative means for reducing the prevalence of dental caries and/or periodontal disease.

4. Cite two early actions that are essential for arresting the progression of the plaque diseases once primary preventive measures have failed.

5. Explain why the planned application of preventive-dentistry concepts and practices, including use of sealants and fluoride therapy, when coupled with early detection and immediate treatment of the plaque diseases, can result in a zero or near-zero annual extraction rate.

Introduction

In the year 2000, in the Executive Summary of the Surgeon Generals Report[a] on the "Oral Health in America," some of the major challenges facing American dentistry were listed.[1,2] It is appropriate to abstract a number of these problem areas in order to better understand the role that prevention can play in their solution.

1. Tobacco: This is a major societal health problem with very strong relationships to dentistry. Smoking has a very devastating relationship to periodontal disease and oral and pharyngeal cancer, while the use of chewing tobacco is associated with oral cancer as well as root decay (see Chapter 23).

2. The statistics of dental need:

Children

a. Dental caries is the most common chronic childhood disease.

b. Over 50% of 5- to 9-year-olds have at least one cavity or filling; by age 17, the percentage has increased to 78%.

c. As a part of childhood, children have many injuries to the head, face, and neck.

d. Twenty-five % of the children have not seen a dentist before entering kindergarten.

e. More than 51 million school hours are lost each year to dental-related illness.

Adults

a. Most adults show signs of periodontal or gingival diseases. Severe periodontal disease [measured as 6 millimeters of periodontal attachment loss (pockets)] affects about 14% of adults aged 45 to 54.

b. Employed adults lose more than 164 million hours of work each year because of dental disease and dental visits.

c. A little less than two-thirds of adults report having visited a dentist in the past 12 months.

Older adults

a. Twenty-three % of 65- to 74-year-olds have severe periodontal disease (characterized by 6 millimeters or more of periodontal attachment loss). At all ages, men are more likely than women to have more severe disease.

b. About 30% of adults 65 years and older are edentulous, compared to 46% 20 years ago.

c. Oral and pharyngeal cancers are diagnosed in about 30,000 Americans annually. Nine thousand die from these diseases each year. Prognosis is poor.

d. At any given time, 5% of Americans aged 65 and older (currently some 1.65 million people) are living in long-term care facilities where dental care is problematic.

[a]United States Public Health Service.

Throughout the entire Surgeon General's report, there is major emphasis on the great disparity *between those who get dental care and those that do not* have access to a dental facility.[3,4] These are the people who are poor,[5,6] are mentally handicapped,[7] those that are disabled,[8] children,[9-12] the aged,[13] and those without dental insurance. There are others living in underserved geographical areas,[14] and still others who do

not have access to dental care because of disease,[15] culture, or race.[16] To address these problems a national program and guidelines of dental care is needed that will include these dentally neglected groups. The questions then become, "What kind of a national program should it be? Is it possible to take care of *so many* people with *so few* dental health professionals?"

It is the goal of the dental profession to help individuals achieve and maintain maximum oral health throughout their lives. Success in attaining this objective is highlighted by the decline of caries throughout the Western world,[17] and the dramatic reduction of tooth loss among adults in the United States. This progress has been mainly attributed to the use of *water fluoridation* and *fluoride-containing products*—toothpastes and mouthrinses—and the growing acceptance and practice of primary preventive care.[18] Yet, dental caries remains a major public-health problem.

Untold millions of research hours and money have been invested in reaching our present capability to control the ravages of the *plaque diseases*. Effective strategies that can markedly reduce the number of carious teeth and better control of periodontal disease are now available. *They only need to be used*.

All health professions emphasize that patients should seek entry into well-planned preventive programs. For dentistry, lack of prevention results in more restorations, periodontal treatment, extractions, and dentures. The changeover in priority from treatment to prevention will require active leadership and health promotion by the dental profession, consumer advocates, public health educators, and health-policy planners. Public-health delivery systems, such as the military, national and state public-health services, and industrial organizations that provide benefits to their personnel, have usually been in the forefront of such change because of the economic advantages accruing to the provider and health benefits to the recipients. For example, in 1989, a report by Malvitz and Broderick[19] recounted the results following the change of focus toward a maximum emphasis on prevention for dental services by the Indian Health Service in the Oklahoma City area. The total number of visits increased by 10%, yet the number of dental personnel remained constant. The percentage of *preventive services increased*, along with a *decrease* of restorative procedures.

Benefits of Primary Preventive Dentistry to the Patient

For the patient who thinks in terms of economic benefits and enjoyment of life, prevention pays. Many studies document the *prevalence* of dental disease, but behind these numbers there is little mention of the adverse affects on humans caused by dental neglect. One study points out that 51% of dentate patients have been affected in some way by their oral health, and in 8% of the cases, the impact was sufficient to have reduced their quality of life.[20] If preventive programs are started early by the patient (or, preferably, by the parents of young children) long-range freedom from the plaque diseases is possible—a sound cost-benefit investment. After all, the teeth are needed over a lifetime for eating. Speech is greatly improved by the presence of teeth. A pleasant smile enhances personality expression. Teeth also contribute to good nutrition for all ages. At rare times, teeth have even provided a means of self-defense. On the other hand, the *absence* of teeth or presence of broken-down teeth often results in a loss of self-esteem, minimizes employment possibilities and often curtails social interaction.

Benefits to the Dentist

Possibly the first benefit of preventive dentistry is the fulfillment of the moral commitment to the Hippocratic Oath that was taken by health professionals at graduation "to render help to those in need, and to do no harm." Through ethics and training, the dentist should derive a deep sense of satisfaction by helping people maintain their oral structures in a state of maximum function, comfort, and aesthetics. A well-balanced practice that actively seeks to prevent disease but is also able to care for those individuals where prevention has failed should prosper. Patients can be outstanding public relations advocates if they are convinced that their dentist and staff are truly interested in preventing disease.

If for no other reason, a dentist should consider prevention to avoid possible *legal problems*. A now strongly supported law for medicine, but to a lesser extent for dentistry, requires that prior to treatment, all options—preventive as well as treatment—should be explained to secure *informed patient consent*. This discussion should include a comparison of health benefits and hazards, as well as the economic and the oral-health benefits of prevention. Long-term patients, the lawyers and the court system are taking a more unsympathetic attitude toward practitioners who have permitted a disease to progress over many years without having taken some accepted primary preventive actions to have slowed, or halted its progress. Patients no longer tolerate supervised professional neglect.[21]

What is Primary Prevention?

When discussing primary prevention, we must first define a few key words. Health is what we want to preserve, and it is defined as *a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity*. For instance, some individuals may actually be in excellent health but believe, for some reason logical to them, that they have oral cancer. Such individuals do not have an optimum mental well-being and will continue to worry until they are somehow convinced otherwise that they are indeed healthy. Another person may be functionally healthy, although facially disfigured, and as such be socially shunned throughout life.[22] Thus, health can at times be what the patient thinks and not the actual condition of the body. Even the terminology "preventive dentistry" has different connotations to different people. As a result, preventive dentistry can be arbitrarily classified into three different levels.

1. *Primary* prevention employs strategies and agents to *forestall* the onset of disease, to *reverse* the progress of the disease, or to *arrest* the disease process before secondary preventive treatment becomes necessary.

2. *Secondary prevention* employs routine treatment methods to *terminate* a disease process and/or to *restore* tissues to as near normal as possible.

3. *Tertiary prevention* employs measures necessary to *replace* lost tissues and to *rehabilitate* patients to the point that physical capabilities and/or mental attitudes are as near normal as possible after the failure of secondary prevention (Figure 1-1).

Figure 1-1 From natural teeth to denture teeth in three not-so-easy stages. (*Source:* Dr. Norman O. Harris, University of Texas Dental School at San Antonio.)

Question 1

Which of the following statements, if any, are correct?

A. The absence of a disease or infirmity is a good sign of physical health but not necessarily of mental and social well-being.

B. A professional football player who looks well, has no physical infirmities, but continually worries about his $10 million contract, can be considered in excellent health.

C. An amalgam restoration that is placed in a carious occlusal pit of a molar is an excellent example of tertiary prevention.

D. The avoidance of an etiologic factor for a specific disease—sucrose for instance to reduce caries—is an example of primary prevention.

E. Preventive dentistry, in its broadest sense, embodies primary, secondary, and tertiary prevention.

In going from primary to tertiary prevention, the cost of health care increases exponentially, and patient satisfaction decreases proportionately. An excellent example of the comparative cost of these two levels of care was the treatment of an individual with poliomyelitis. It has only been a few years ago that the cost of the polio vaccine was only a few dollars. The use of the polio vaccine to prevent the onset of the disease was *highly* effective. But, for someone not adequately immunized, the cost of treatment for poliomyelitis and subsequent rehabilitation approximated $50,000 or more for the first 7 weeks of hospitalization and outpatient care.[23] Yet, the individual receiving the $50,000 worth of tertiary preventive treatment and the attendant disability was certainly not as happy as the one who benefited from only a few dollars' worth of primary preventive care. The payoff of the worldwide drive to eliminate polio promises to have this disease follow smallpox into oblivion. Another appropriate example is the fluoridation of drinking water. This costs approximately $0.50 per year per individual, yet it reduces the incidence of dental caries in the community by 20 to 40%. If this primary-preventive measure is not available, the necessary restorative dentistry (secondary prevention) can cost approximately 100 times more, or about $50.00 per restoration.[18] Finally, if restorative dentistry fails, as it often does, prosthetic devices must be constructed at an even greater cost. This great disparity between the lower cost of prevention and the much higher cost of treatment must be seriously considered *if* the United States is to develop an affordable national health program in which dentistry is represented.

This text emphasizes primary prevention, and specifically focuses on primary prevention as it applies to the control of dental caries and periodontal disease. On the other hand, it must be recognized that primary prevention often fails for many reasons. When such failure occurs, *two actions* are essential to contain the damage:

(1) *early identification* of the disease (diagnosis) and (2) *immediate treatment* of the disease.

Categories of Oral Disease

For planning purposes, dental diseases and abnormalities can be conveniently grouped into three categories: (1) dental caries and periodontal disease, *both* of which are acquired conditions, (2) acquired oral conditions *other than* dental caries and periodontal disease (opportunistic infections, oral cancer, HIV/AIDS), and (3) craniofacial disorders which would include a wide variety of conditions ranging from heredity to accidents.[24,25] For instance, the ordinary seat belt and the air bags in a car exemplify how a simple preventive measure can greatly reduce the facial injuries of car accidents. Looming in the not-too-far distant future is the very real possibility that many acquired health problems will be corrected or ameliorated for total populations by use of vaccines, genetic engineering, or specifically targeted drugs ("magic bullets").

The treatment of caries and periodontal disease (and their sequelae) accounts for most of the estimated $60 billion U.S. dental bill for the year 2000.[26] *Both* caries and periodontal disease are caused by the presence of a pathogenic dental plaque on the surfaces of the teeth and hence are known as the *plaque diseases*. Any major reduction in the *incidence* of caries and periodontal disease will release resources for the investigation and treatment of conditions included in the acquired and craniofacial category.

The ideal, or *long-range* planning objectives for coping with both dental caries and periodontal disease should be the development of a preventive delivery system and methods to eventually attain a zero or near-zero *disease incidence* for the target population. However, a more realistic and feasible *shorter-term goal* is the attainment of a zero or near-zero rate of *tooth loss* from these diseases by integrated preventive *and* treatment procedures. Because of the varied etiology of the second and third categories, that is, other acquired conditions and craniofacial malformations and diseases, the planning for the control of each of these problem areas must be individually addressed and placed within the priorities of any overall health plan.

Question 2

Which of the following statements, if any, are correct?

A. A disfiguring facial deformity resulting from an automobile accident can be considered an acquired craniofacial problem.

B. The broad concept of preventive dentistry places major emphasis on primary preventive care but also considers the equal need for secondary and tertiary preventive care.

C. Because dental caries and periodontal disease are infectious diseases (true), they are acquired conditions.

D. The ideal or long-range objective for dentistry is an eventual zero annual extraction

rate; the more realistic, and much more encompassing *short-range objective* is to totally prevent the onset of any pathology requiring extraction.

E. Acquired conditions (*other than* caries or periodontal disease) and hereditary diseases account for the great proportion of income derived by the dental profession.

Strategies to Prevent the Plaque Diseases

Before providing an overview of methods used to implement primary prevention programs, it is important to point out that both dental caries and periodontal disease are *transmissible diseases*. If a child is considered at high risk for caries, one of the parents[27]—usually the mother—can usually be identified as high risk; if a child has periodontal problems, usually one of the parents is also afflicted. Any infectious (acquired) disease can only begin if the challenge organisms are in sufficient numbers to overwhelm the combined manmade and body defenses and repair capabilities. For this reason, all strategies to prevent, arrest, or reverse the ravages of the plaque diseases are based on (1) reducing the number of challenging oral pathogens, (2) building up the tooth resistance and maintaining a healthy gingiva, and (3) enhancing the repair processes.

In general, periodontal disease is a disease that involves the soft tissue and bone surrounding the affected teeth. Caries involves the demineralization and eventual *cavitation* of the enamel and often of the root surface. If the *incipient* lesions (earliest *visible* sign of disease) of caries and periodontal disease are recognized at the time of the initial/annual dental examination, they can often be reversed with primary preventive strategies. For caries, the *visible* incipient lesion is a *white spot*, which appears on the surface of the enamel as a result of subsurface acid-induced demineralization. For periodontal disease, the *visible* incipient lesion is *gingivitis*—an inflammation of the gingiva that is in contact with the bacterial plaque. Not all "white spots" go on to become caries, nor do all cases of gingivitis go on to become periodontal disease. In both cases, i.e., caries and periodontal disease, it should be noted *that if dental plaque did not exist, or if the adverse effects of its microbial inhabitants could be negated, the decrease in the incidence of the plaque diseases would be very dramatic. Based on these facts, it is understandable why plaque control is so important in any oral-health program*.

To control the *plaque diseases* with *available* methods and techniques, strong emphasis has been directed to four general strategies to reduce caries and two administrative requirements:

General Strategies

1. Mechanical (toothbrush, dental floss, irrigator, or rinse)

2. Chemical plaque control. Use of *fluorides* to inhibit *demineralization* and to enhance *remineralization;* use of antimicrobial agents to supress cariogenic bacteria.

3. Sugar discipline.

4. Use of pit and fissure sealants, when indicated, on posterior occlusal surfaces.

Administrative

5. Education and health promotion.

6. Establish access to dental facilities where diagnostic, restorative, and preventive services are rendered, and where planned recalls based on risk are routine.

A brief summary of each of these primary preventive procedures will serve as an introduction for the more detailed information presented in later chapters.

Plaque Control

Dental plaque is composed of *salivary proteins* that adhere to the teeth, plus *bacteria and end-products* of bacterial metabolism. Both cariogenic and periodontopathogens accumulate in the plaque located along the gingival margin, interproximally, and in the pits and fissures. Plaque collects more profusely in these specific areas because none of these locations is optimally exposed to the normal self-cleansing action of the saliva, the abrasive action of foods, nor the muscular action of the cheeks and tongue. Plaque decreases in thickness as the incisal or occlusal surface is approached. Little plaque is found on the occlusal surface except in the pits and fissures. As would be expected, plaque forms more profusely on malposed teeth or on teeth with orthodontic appliances, where access for cleaning is often difficult.

In the *gingival sulcus* between the gingiva and the tooth, little or no plaque normally accumulates *until* gingival inflammation begins, at which time the bacterial population increases in quantity and complexity. This is the beginning of *gingivitis* that, if continued, may eventually result in an *irreversible periodontitis*.

It is important to differentiate between the *supra*gingival and the *sub*gingival plaques. The *supra*gingival plaque can be seen above the gingival margin on all tooth surfaces; the *sub*gingival plaque is found in the sulcus and pocket below the gingival margin, where it is not visible. The supragingival plaque harbors specific bacteria that can cause supragingival (coronal) caries. The subgingival plaque microbiota is mainly responsible for periodontal problems. The bacterial populations of each of these plaques differ qualitatively and quantitatively in health and disease.[28] The pathogenicity of each of the plaques can vary independently of the other. For example, it is possible to have periodontal disease with or without caries, to have neither, or to have a shifting status of caries or periodontal disease, or both.

The pathogenicity of the subgingival plaque is becoming an increasing concern. Not only does it cause periodontal disease, which is a lifelong debilitating disease of the tooth supporting tissues, but it is now believed that there is a causal relationship between periodontitis[29] and such diverse conditions as, cardiovascular disease,[30] diabetis mellitus,[31] chronic respiratory disease,[32] and immune function.[33] There is also the possibility in some cases that this is a bi-directional association where the oral problem begins with a systemic condition, instead of vice versa.

In many cases, plaque is difficult for a patient to identify. This problem can be overcome, at least in the case of the supragingival plaque, by the use of *disclosing*

agents, which are harmless dyes such as the red-staining agent, FD&C Red. The dyes may be in solution and painted on the teeth with a cotton applicator, or they may be tablets which are chewed, swished around the mouth, and then expectorated. Once *disclosed*, most of the supragingival plaque and food debris can be easily removed by the daily use of a toothbrush, floss, and an *irrigator* (Figure 1-2). Plaque can also be removed at planned intervals by the dental hygienist or a dentist as part of an oral *prophylaxis*. This is a procedure that has as its objective the mechanical removal of all soft and hard deposits, followed by a polishing of the tooth surfaces. However, because daily removal of the plaque is more effective, it is *the individual*—not the hygienist or the dentist—who is vital for preserving lifelong intact teeth.

One site where neither the dentist nor an individual can successfully remove plaque is in the depth of pits and fissures of *occlusal* surfaces where the orifices are too small for the toothbrush bristle to penetrate (see Chapter 10). The flow of saliva or the muscular action of the cheeks and tongue also have little influence over the eventual development of caries in these areas. Not coincidentally, the occlusal surface is where the *greatest percentage* of caries lesions occur. *For this reason, it is recommended that all occlusal surfaces with deep convoluted fissures be sealed with a pit-and-fissure sealant.*

As soon as the plaque is removed from any surface of the tooth, it *immediately* begins to reform. This should not be unexpected, since by definition, dental plaque is composed of salivary residue, bacteria, and their end-products, all of which are always present in the mouth. Thus, a good plaque-control program must be continuous. It must be a daily commitment over a lifetime.

Figure 1-2 A. Flossing gets down under the gingiva and B.Flossing cleans the space between the teeth as well. (*Source:* Dr. Norman O. Harris, University of Texas Dental School at San Antonio.)

Question 3

Which of the following statements, if any, are correct?

A. Four general areas that form the basis for strategies for the primary prevention of dental diseases are (1) plaque control, (2) fluoride use, (3) sealants, and (4) restorations.

B. Plaque is found only on the smooth enamel surfaces of the tooth.

C. Plaque removal requires the use of instrumentation by a dentist or a dental hygienist.

D. Good flossing and toothbrushing techniques can completely remove the supragingival plaque from all five tooth surfaces.

E. The daily self-care removal of plaque by an individual is more productive than a semiannual removal by the dental hygienist.

Not only does the daily removal of dental plaque reduce the probability of dental caries; equally important, it also reduces the possibility of the onset of gingivitis. This

occurs when the metabolic end-products of the *periodontopatho-gens* that are contained in the plaque irritate the adjacent gingival tissues, producing an inflammation (i.e., gingivitis). If the inflammation continues, bleeding (hemorrhage) can be expected following even minimal pressure ("pink toothbrush"). This gingivitis can be arrested and reversed (cured) in the early stages by proper brushing, flossing, and irrigation, especially if accompanied with professional guidance.

Plaque concentrates mineralizing ions such as calcium, phosphate, magnesium, fluoride and carbonates from the saliva to provide the chemical environment for the precipitation and formation of *calculus*, a concretion that adheres firmly to the teeth. If the plaque is not removed by flossing and brushing before the calculus begins to form, the resultant mineralized mass provides a greater surface area for an even more damaging plaque accumulation. This *additional* mass of *periodontopathic* plaque covering the rough porous surface causes the stagnation of even more bacteria and is responsible for the damage to the periodontal tissues. Also, the hard, irregular calculus deposits pressing against the soft tissues serves to *exacerbate* the inflammation caused by the bacteria alone. The daily removal of plaque can successfully abort or markedly retard the build-up of calculus. Once the calculus forms, the brushing and flossing usually used for plaque control does *not* remove the deposits. At this time, the dental hygienist or dentist must intercede to remove the calculus by instrumentation.

To this point, only mechanical plaque control (i.e., use of a toothbrush, dental floss, and an irrigator) has been highlighted. Rapidly growing in importance as a supplement to mechanical plaque control (but not as a replacement), is *chemical plaque control*. This approach utilizes mouthrinses containing antimicrobial agents that effectively help control the plaque bacteria involved in causing *both* caries and gingivitis. For helping to control gingivitis, a popular and economical over-the-counter product is Listerine; the most effective *prescription* rinse is *chlorhexidine*. Many studies indicate that chlorhexidine is as effective in suppressing cariogenic organisms as it is effective in controlling gingivitis and periodontitis.[34,35]

Fluorides

The use of fluorides has provided exceptionally meaningful reductions in the incidence of dental caries. Because of water fluoridation, fluoride dentifrices, and mouthrinses, dental caries is declining throughout the industrialized world. Historically, the injection of fluoride into water supplies in the mid-20th century resulted in a decrement of approximately 60 to 70% in caries. Since that time, fluoride has been introduced into proprietary products such as dentifrices and mouthrinses. As a result, the caries decrement *directly attributable* to water fluoride over the past years has declined. Yet, the placement of fluoride into communal water supplies *still* results in an estimated 20 to 40% reduction in coronal caries, and a similar 20 to 40% decrease in root caries[36] (Figure 1-3).

Approximately 126 million individuals in the United States consume fluoridated water through *communal water* supplies and another 9 million are drinking *naturally* fluoridated water. It is estimated that 65% of the U.S. population, therefore, is receiving fluoride through drinking water.[37] Many times during the past years, it has not been possible to fluoridate city water supplies because of political, technical, or

financial considerations. In such cases, it is still possible to receive the *systemic* benefits of fluoride by using *dietary supplements* in the form of fluoride tablets, drops, lozenges, and vitamin preparations. Some countries permit fluorides to be added to table salt.[38] Elsewhere, ongoing research studies are being conducted to determine the anticariogenic effect of fluoride when placed in milk,[39,40] and even sugar.[41]

It is also possible to apply fluoride directly *to the surface* of the teeth by use of cotton pledgets, and/or by use of fluoride-containing dentifrices, gels, varnishes or mouth rinses. Such applications to the surface of the teeth are referred to as *topical applications*. The extent of caries control achieved through topical applications is directly related to the number of times the fluoride is applied and the length of time the fluoride is maintained in contact with the teeth. Research data also indicate that it is better to apply *lower* concentrations of fluoride to the teeth *more often* than to apply higher concentrations at longer intervals.

Fluorides and chlorhexidine are the most effective agents used by the profession to combat the plaque diseases. The fluorides help prevent demineralization and enhance remineralization, while chlorhexidine severely suppresses the mutans streptococci that cause the demineralization. Chlorhexidine also helps suppress bacteria causing the inflammation of periodontal disease.

Figure 1-3 Water fluoridation reduces cavities in the population by 20 to 40%. (*Source:* Dr. Norman O. Harris, University of Texas Dental School at San Antonio.)

Question 4

Which of the following statements, if any, are correct?

A. Prophylaxes and chlorhexidine are effective in the partial control of *both* caries and gingivitis.

B. Even after calculus becomes attached to the tooth, it can still be removed by good home self-care plaque control programs.

C. The addition of fluoride to communal water supplies is now accompanied by a 20 to 40% decrease in caries incidence.

D. The topical application of higher concentrations of fluoride at longer time intervals is more effective than lower concentrations of fluoride at shorter intervals.

E. The topical application of fluoride can only be accomplished by a dentist or a dental hygienist.

Neither the action of topically applied nor of systemic (ingested) fluoride in preventing dental caries is completely understood. It is believed that fluoride has several key actions: (1) it may enter the dental plaque and affect the bacteria by depressing their production of acid and thus reduce the possibility of demineralization of the teeth; (2) it reacts with the mineral elements on the surface of the tooth to make the enamel less soluble to the acid end-products of bacterial metabolism; and (3) it

facilitates the remineralization (repair) of teeth that have been demineralized by acid end-products. The latter is probably the most important of these three effects.

The natural source of minerals such as calcium and phosphate, fluoride and others needed for this remineralization is the *saliva*.

Sugar and Diet

The development of dental caries depends on four interrelated factors: (1) diet, (2) inherent factors of host resistance, (3) the number of challenge bacteria located in the dental plaque, and (4) *time* (Figure 1-4). Without bacteria, no caries can develop. For the bacteria in the plaque to live, they must have the same amino acids, carbohydrates, fatty acids, vitamins, and minerals that are required for all living organisms. Because these nutrients are also required by the cells of the body, the food that is ingested by the host or that which later appears in the saliva in a metabolized form, provides adequate nutrients for bacterial survival and reproduction. With three *well-balanced* meals per day, however, the usual plaque bacteria probably would not release a sufficient quantity of metabolic acids to cause caries development (Figure 1-5A). But, as soon as *sugar and sugar products* are included in the diet of the host, bacterial acid production markedly increases in the plaque. This release of acid end-products is the major cause of the initiation and progression of caries.[42] Of even greater importance *than* the *total intake* of refined carbohydrates is the *frequency* of intake and the *consistency* of the sugar-containing foods.[43] The continuous snacking of refined carbohydrates that characterizes modern living results in the teeth being constantly exposed to bacterial acids (Figure 1-5B). For example, the prolonged adherence of sugar products to the teeth, such as that experienced after eating *taffies* and *hard candies*, results in prolonged production of the plaque acids that are in direct contact with the tooth surface. Thus, if caries incidence is to be reduced, all three factors—*total intake* of sugar, *consistency* of the cariogenic foods, and especially *frequency* of intake should be considered.

Possibly one of the most promising means of reducing caries incidence in the United States has been the wide-scale acceptance of sugar substitutes such as NutraSweet, Sweet'n Low, and Splenda. In the Nordic countries, there is considerable enthusiasm for use of xylitol—a sugar alcohol. Xylitol has been found to inhibit decay, reduce the amount of plaque and plaque acid, inhibit growth and metabolism of streptococci,[44] reduce decay in animal studies, and contribute to remineralization. It is considered noncariogenic and cariostatic.[45] All the Nordic dental associations recommend its use. Since the 1970s, one of the favorite ways to take advantage of xylitols unique anticaries property, has been to use it to sweeten chewing gum, a product that is a popular item among school children.[46]

Two other dental uses of xylitol chewing gum have come out in Scandinavia:

1. Chlorhexidine can dramatically suppress the number of mutans streptococcus in the saliva. However, after discontinuing use of the product, there is a rapid repopulation of the bacteria. This repopulation can be arrested or greatly slowed by the use of xylitol chewing gum.[47]

2. Previously it was mentioned that a child's flora often reflected that of the mother.

To help minimize this mother-child transmission of cariogenic bacteria, mothers have been urged to chew xylitol gum.[48]

This creditable background of xylitol has prompted Anasavice to ask, "Are chlorhexidine, fluoride, fluoride varnishes, and xylitol chewing gum under ustilized preventive therapies?"[49]

Pit and Fissure Sealants

Approximately 90% of all the carious lesions in the mouth occur on the occlusal surfaces of the posterior teeth.[50] These surfaces represent only 12% of the total number of tooth surfaces, so that occlusal surfaces with their deep pits and fissures are approximately *eight times as vulnerable* as all the other smooth surfaces. The availability of sealants offers an alternative to a restoration. With the use of sealants, a thin layer of a plastic, called Bis-GMA, is flowed into the deep occlusal pits and fissures of teeth not having open carious lesions. This action effectively isolates these areas from the oral environment (Figure 1-6). Since no cavity preparation is necessary, no pain or discomfort accompanies sealant placement. Following the placement of the sealant in the deep fissures, the newly created fossae can be effectively cleaned with a toothbrush.

As long as the sealants are retained, no bacteria or bacterial acids can affect the sealed areas. If they are not retained, no damage to the teeth results from a retreatment. The lost sealant can be easily replaced. One 10-year study demonstrated a 57% retention of the original sealants.[51] In another study, approximately 95% retention occurred over 2 years.[52] With these performances, the average life of the sealant approximates the 10 years projected for an amalgam.[53] It should be emphasized that sealant placement should be followed by a topical fluoride application to the teeth, because *fluorides are most effective in protecting the smooth surfaces and least effective on the occlusal surfaces, a situation that is the reverse of the results expected of the sealants*.

Figure 1-4 Caries is a *multifactorial* disease caused by bacteria, a supporting host diet of refined carbohydrates, decreased host resistance, and time for the cavity to develop. (*Source:* Dr. Norman O. Harris, University of Texas Dental School at San Antonio.)

Figure 1-5 A. This balanced meal does not provide the bacteria with enough energy to produce acids.

Figure 1-5 B. Snacks such as this expose teeth to bacterial acids.

Figure 1-6 Molar A. without and B. with a clear plastic sealant to protect the deep occlusal fissures. (*Source:* Dr. Norman O. Harris, University of Texas Dental School at San Antonio.)

Question 5

Which of the following statements, if any, are correct?

A. Using fluoride to remineralize incipient lesions, one can expect the reminerlized lesion to be more resistant to future demineralization than incipient lesions before

remineralization.

B. The calcium and phosphate that is lost from the tooth in demineralization can be replaced during remineralization.

C. The development of dental caries depends on four essential factors: (1) diet; (2) inherent factors of host resistance; (3) bacteria; and (4) time.

D. Refined carbohydrates alone provide sufficient nutrition for cariogenic bacteria.

E. Sealant longevity closely coincides with the longevity of amalgam restorations.

Public Dental Health Education

If the profession of dentistry can control caries effectively through plaque control, systemic (ingested) and topical (local application) use of fluorides, dietary control, and the use of plastic sealants, two important questions need to be asked.

1. Why do we not have a more effective dental caries-control program in the United States?

2. If daily toothbrushing, flossing of teeth, and irrigation removes plaque and food residue, why are these simple procedures not used effectively to control both caries and periodontal disease?

Probably the best answer to these questions is that people must first *know* what they need to do as well as *how* it is to be done. Unfortunately, the public has relatively little information about the tremendous potential of primary preventive dentistry for reducing their adverse exposures to the plaque diseases. Without this information, *it is difficult to convince people that they can greatly control their own dental destiny*. Many individuals think of dentistry as a treatment-oriented profession that specializes in periodontal treatment, restorations, endodontics, exodontics, and prosthetics. An expanded public education and promotion program is essential to ensure the success of any preventive dentistry program in which an individual or a community is asked to participate.

In dentistry, a one-on-one relationship between the patient and the health professional is still a basic approach to patient education and motivation. This approach makes the task impossible because there are 250 million people in the United States and only approximately 165,000 practicing dentists, plus 120,000 dental hygienists and 175,000 assistants.[54,55] The main thrust of public dental-health education and oral-health promotion is provided by the various dentrifice manufacturers advocating the daily toothbrushing routine and biannual visits to the dentist for a checkup. The effectiveness of this approach was underlined by the long-running advertisement for the first marketed, stannous fluoride containing, Crest toothpaste, "Look Mom, no cavities."

Knowing facts and applying the information are two separate processes. The application of knowledge by an individual requires a personal commitment; it is at this point of personal commitment that most primary preventive-dentistry programs

fail. *If* people embraced the daily use of mechanical and chemical plaque control regimens, the risk of caries and gingivitis would be minimized. *If* people would exercise reasonable sugar discipline the possibility of caries development would be further reduced. *If* individuals rejected the use of cigarettes as well as smokeless ("spit") tobacco, oral and pharyngeal cancer and periodontitis would be much less prevalent. Clearly, education, motivation, and behavior modification are a necessary part of enjoying good oral and general health.

A sound, well-planned program of dental-health education and promotion is lacking in the curriculum of the great majority of primary and secondary schools. Few people can discuss the advantages and disadvantages of water fluoridation and the topical application of fluorides. Few have any detailed information about the dental plaque and the disease-inducing potentialities of this bacterial film.[b] Few people know why sugar is cariogenic. Even fewer people know that gingivitis can be *cured*, but that if allowed to progress, there is the possibility of a life long future of periodontal disease treatment and maintenance. Finally, the public has not been adequately informed that the timely use of sealants and remineralization therapy provides a hope of possessing a full intact dentition for life. Even though the Internet has greatly expanded the delivery of health education, there is always the question of the quality of information (or misinformation) that is disseminated.[56]

Ideally, school-based and public-education programs should exist to help people to *help themselves* in applying primary preventive procedures. The same programs should also teach all individuals to *recognize the presence of oral disease*. With proper instruction that can be provided by *schoolteachers*. The general public can be taught to understand that they must assume major responsibility for their own oral health (see Chapter 19). Only the individual can seek immediate treatment when pain or disease occurs. Public dental-health education might benefit if there was a consumer organization such as an American Oral Health Association that could promote oral health education, much like the American Cancer Society and the American Heart Association.

[b]Biofilm = A collection (film) of living organisms attached to a solid base, such as algae to the bottom of a swimming pool, or dental plaque to a tooth. Both terms are used in the book, but dental plaque is preferred because of public familiarity and understanding of "dental plaque."

Access to Comprehensive Dental Care

This factor is probably the most important of all preventive options. Without the benefit of a routine periodic dental examination, it is difficult for individuals to realize that they are vulnerable to oral disease. The first indication of a dental problem is pain, which is the wrong starting point for prevention. An example of the benefits of combining prevention with the advantages of early identification, prevention and treatment is seen in the New Zealand school-dental-nurse program. In the New Zealand School Dental Service, a dental nurse visits every primary and secondary school in the country at approximately 6-month intervals. At that time, all children receive a dental examination. If necessary, the dental nurse applies fluoride varnish to achieve remineralization of incipient caries, removes visible calculus, or when indicated, refers the child to a dentist for more complex treatment requirements.[57]

As a result of this program, the average rate of extractions dropped from 19 per 100 students in 1960, to 2 per 1,000 in 1979. From 1973 until 1992, the average decayed, missing, or filled permanent teeth (DMFT) for 12- to 14-year-old children plummeted from 10.7 to 1.88 per child. Approximately 96% of all New Zealand schoolchildren are enrolled in this program. Unfortunately, relatively few comprehensive primary-preventive dentistry school programs are being conducted in the United States school systems. However, there are more than 1,400 School Based Dental Health Clinics (SBDHC) now in operation in the United States (see Chapter 19).

Prognostic and Diagnostic Tests

Several methods for preventing the onset or progress of caries and periodontal disease have been discussed. Because it is impossible to apply vigorously *all* the preventive procedures to *all* the people *all* the time, it would be desirable to have some tests to indicate the extent of caries and periodontal disease *risk* of an individual at any given time. This need is highlighted by the fact that an estimated *60%* of all carious lesions in schoolchildren *occur in 20%* of the stu-dents.[58] It would save much time to be able to identify this 20% group of high-risk students without having to examine an entire school population. Although no tests are 100% correlated with the extent of caries activity or periodontal disease, several test procedures are sufficiently well correlated with either condition to be of interest. To be successful, such screening tests should be simple to accomplish, valid, economical, require a minimum of equipment, be easy to evaluate, and be compatible with mass-handling techniques.

Laboratory methods exist for counting the number of bacteria in the saliva. If the caries-causing mutans streptococci or lactobacilli counts are high, the individual from whom the sample was derived can be presumed to have a higher risk for dental caries, whereas a low count permits the opposite assumption.[59] A second general method for estimating caries susceptibility is by use of a refined-carbohydrate dietary analysis to (1) evaluate the patient's overall diet with special attention to food preferences and amounts consumed and (2) to determine if the intake of refined carbohydrates is excessive in quantity or frequency (see Appendix 23-2). A well-balanced diet is assumed to *raise host resistance* to all disease processes, whereas a frequent and excessive intake of refined carbohydrates (i.e., sugar) has been associated with a high risk of caries development. The dietary analysis is very effective when used as a guide for patient education.

The onset of *gingivitis* is much more visible than the early demineralization that occurs in caries. The sign of impending periodontal disease is an inflammation of the gingiva that can be localized at one site, or generalized around all the teeth. Red, bleeding, swollen, and a sore gingiva are readily apparent to dentist and patient alike.

Remineralization of Teeth

Both demineralization and remineralization occur *daily* following the cyclic ebb-and-flow of the caries process during and after eating meals and snacks. An eventual caries lesion develops *over a period of time* when the rate of acid-induced demineralization of teeth exceeds the capability of the saliva to remineralize the damaged enamel components. A negative mineral balance at the enamel-plaque

interface, if continually repeated, results in an incipient lesion that eventually can become an overt lesion. It often requires months, or even years, for the overt lesion to develop.[60,61] During this time, under proper conditions, remineralization can reverse the progress of the caries front, with the mineral components coming from the saliva. There is a physiological precedent for such a mineralization. Immediately after eruption of the teeth the outer layer of the enamel is not completely mineralized; the maturation (mineralization) of this outer layer requires approximately 1 year, during which time the tooth is continuously bathed in the saliva.

The point at which a developing caries lesion is no longer reversible is considered to be when *cavitation* occurs; clinical experience indicates that as long as the lesion is incipient (i.e., with *no* cavitation), remineralization is possible.[62] The need to exploit this possibility to the benefit of all patients was emphasized by Koulourides's statement many years ago that "there is a wide gap between current practices of many dental clinicians and the potential application of present scientific knowledge to arrest and reverse incipient carious lesions."[63]

The outstanding electron microscope research contributions of Silverstone several decades ago clearly demonstrated that demineralized tooth structure could be remineralized.[64] No longer was a simple interproximal x-ray radiolucency a signal to place an interproximal restoration. Several reports from Scandinavia now indicate that even when the caries front of an incipient lesion extends *past* the dentino-enamel junction, it can be remineralized. Foster (England) has recommended "… that operative intervention (be) considered for approximal lesions which extend *deeper* that 0.5 mm into the dentine, while *preventive treatment and re-assessment* may be considered for *shallower lesions*."[65] Missing at the present time is an accurate predictive test for caries that would permit the targeting of individuals who would be candidates for remineralization therapy (see Chapter 23).

The conditions for optimum remineralization are the same as for preventing the initiation of a lesion: (1) plaque control to reduce the number of cariogenic bacteria, (2) a strict self-imposed sugar discipline to minimize the number of acidogenic episodes, (3) the use of sealants to interdict bacterial entry into deep pits and fissures, and (4) the use of topical and/or systemic fluoride to inhibit demineralization and to potentiate the remineralization process. Thus, with the same primary preventive dentistry routines using fluoride, an individual can *simultaneously* protect the tooth into the future by prevention, as well as to compensate for limited past damage through reversal strategies.

Question 6

Which of the following statements, if any, are correct?

A. Sealants are most effective in preventing smooth-surface caries, whereas fluorides are most effective in preventing caries in the deep occlusal pits and fissures.

B. There are enough dentists and dental auxiliaries in the United States to provide approximately 1 hour per year of educational lectures to each of the 250 million citizens of the United States.

C. Caries activity indicators (tests) are indicative of a patient's vulnerability at the time of the test.

D. Plaque control, sugar restriction, and topical-fluoride therapy not only are effective in preventing demineralization, but they also can enhance remineralization.

E. The process of natural mineralization (maturation) of enamel during the first year after eruption is a precedent for man-initiated remineralization (repair) of incipient lesions.

Summary

Each year more than $60 billion is spent in the United States for dental care, mainly for the treatment of dental caries and periodontal disease or their sequelae. Yet, strategies now exist that with patient knowledge and cooperation, could greatly aid in preventing, arresting, or reversing the onset of caries or periodontal disease. The six general approaches to the control of both caries and periodontal disease involve (1) plaque control, (2) water fluoridation and use of fluoride products for self-care and for professionally initiated remineralization procedures, (3) placement, when indicated, of pit and fissure sealants, and (4) sugar discipline. Supporting these measures are (5) public and private enterprise financed media distributed programs extolling the benefits of oral health and proprietory products for family prevention; and (6) access to a dental facility where diagnosis, comprehensive preventive, restorative treatment, and planned recall and maintenance[c] programs are available. The zeal and thoroughness with which these preventive measures should be prescribed and used are indicated by the information obtained from the clinical and roentgenographic oral examination, dietary analysis, patient history, and laboratory tests.

If at the time of the clinical and roentgenographic examinations, emphasis was placed on searching out the incipient lesions ("white spots") and early periodontal disease (gingivitis), preventive strategies could be applied that would result in a reversal or control of either/or both of the plaque diseases. It is essential that both the profession and the public realize that biologic "repair" of incipient lesions, and "cure" of gingivitis is a preferred alternative to restorations or periodontal treatment.

Even if these primary preventive dentistry procedures fail, tooth loss can still be avoided. In practice, the early identification and expeditious treatment of caries and periodontal disease greatly minimizes the loss of teeth. When such routine diagnostic and treatment services are linked with a dynamic preventive-dentistry program that includes an annual dental examination and recall program based on risk assessment, tooth loss can realistically be expected to be reduced to zero or near-zero.

This introductory chapter has briefly pointed out some of the problems of dentistry and the means by which the dental profession can make primary preventive dentistry its hallmark. The remaining chapters provide the detailed background that can make this challenge become a reality.

[c]There is a trend to consolidate the two terms, "recall" and "maintenance", into the word, "recare".

Answers and Explanations

1. A, D, and E—correct.

B—incorrect. With salaries now escalating, maybe the poor fellow has something to worry about; the true answer is that continued worry is not healthy.

C—incorrect. An amalgam restoration is an excellent example of secondary prevention, not tertiary.

2. A, B, and C—correct.

D—incorrect. It is easier to reduce the extraction rate to zero or near-zero by the combined application of treatment and preventive procedures, than to reduce the incidence of disease by preventive procedures alone.

E—incorrect. The major income of a dentist is derived from treatment of the plaque diseases and their sequelae.

3. E—correct.

A—incorrect. Restorations are not a primary preventive-dentistry option; rather they are the mainstay of secondary prevention.

B—incorrect. Plaque is found in the pits and fissures of the occlusal surfaces.

C—incorrect. Plaque can be removed by use of toothbrush and floss; it is calculus removal that requires instrumentation.

D—incorrect. It is not possible to remove oral debris from deep pits and fissures.

4. A and C—correct.

B—incorrect. Once calculus has formed, professional intervention is required for its removal.

D—incorrect. It is vice versa—the more often that fluoride is applied topically (dentifrices), the more effective it is.

E—incorrect. Remember, brushing with a fluoride dentifrice constitutes a topical application.

5. A, B, C, and E—correct.

D—incorrect. Bacteria require carbohydrates, fats, proteins, minerals, and water to exist; they need the carbohydrates for their energy needs, which, in turn, results in their acid production and cariogenicity.

6. C, D, and E—correct.

A—incorrect. Just the opposite. The sealants are used to seal off the convoluted pits and fissures of the occlusal surfaces.

B—incorrect. The only means to promote preventive dentistry to a *total* population is by the use of the schools and the popular media.

Self-evaluation Questions

1. Health is defined as _____.

2. If primary prevention fails, the two sequential actions necessary to minimize progression of a disease process are _____ and _____.

3. For planning purposes, oral diseases and abnormalities can be grouped into three general categories: (1) _____, (2) _____, and (3) _____.

4. Five strategies used to attain primary prevention in caries control are: (1) _____ (2) _____, (3) _____, (4) _____, and (5) _____.

5. Of the six general methods for caries control, the two that are also valuable in periodontal disease control are (1) _____ and (2) _____.

6. Plaque control in a home environment requires essential items or devices: (1) _____ and (2) _____ and (3) an irrigator.

7. Caries development depends on four interrelated factors: (1) _____, (2) _____, (3) _____ and (4) time.

8. Fluoride is most effective in preventing caries on (smooth)(occlusal) surfaces of the teeth, whereas plastic sealants are most effective in preventing caries on (smooth)(occlusal) surfaces of the teeth.

9. "Biologic repair" of a tooth results from a positive mineral balance at the enamel surface; the process of replacing the ions lost in demineralization is known as _____.

10. Name three American sugar substitutes and one foreign anticariogenic sugar alcohol used for sweetening: _____, _____, _____ and _____.

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Chapter 2. The Development and Structure of Dental Plaque (A Bacterial Biofilm), Calculus, and Other Tooth-adherent Organic Materials - *Max A. Listgarten Jonathan Korostoff*

Objectives

At the end of this chapter it will be possible to

1. Differentiate between organic coatings of *endogenous* and *exogenous (acquired)* origin.

2. Explain why dental plaque is not unique among naturally occurring microbial layers.

3. Discuss some of the mechanisms proposed to explain *bacterial adhesion* to the *acquired pellicle.*

4. Distinguish between *primary* and *secondary* bacterial colonizers in dental plaque, and cite examples of each.

5. Identify the prime sites of calculus formation, explain how calculus forms, and detail the differences between *supragingival* and *subgingival* calculus.

6. Explain the basis for the involvement of the acquired pellicle, bacterial dental plaque, and dental calculus in caries and the inflammatory periodontal diseases.

Introduction

The dental profession has to deal with two of the most widespread of all human maladies, dental caries (tooth decay), and inflammatory diseases of the periodontium (i.e., the supporting tissues of the teeth), gingivitis, and periodontitis (Figure 2-1). These conditions are known to have a bacterial etiology. Unlike some other infectious diseases, these diseases are not caused by a single pathogenic microorganism. Dental caries and inflammatory periodontal diseases result from the accumulation of many different bacteria that form *dental plaque*,[1,2] a naturally acquired bacterial biofilm that develops on the teeth (Figure 2-2). Some bacterial species in dental plaque may be of greater relevance to caries and periodontal diseases than others. Dental plaque *cannot* be removed by rinsing alone but can be removed by mechanical debridement. The *proportions* of different bacteria in plaque from a healthy mouth are different from those in plaque associated with caries, and both are different from the dental plaque of

an individual with inflammatory periodontal disease.[3,4]

If the role of dental plaque in caries and inflammatory periodontal diseases is to be understood,[5,6] the logical place to start is by examining how dental plaque forms and, as will be discussed in later chapters, how changes in the proportions of different plaque bacteria lead to oral disease.

Figure 2-1 A 13-year-old female with dental caries on facial surface of the maxillary incisors and swollen, discolored gingival tissues around the mandibular incisors, characteristic of chronic gingivitis.

(Courtesy of Dr. WK Grigsby, University of Iowa College of Dentistry.)

Figure 2-2 The dental plaque on these teeth has been stained with a discoloring solution and rinsed. Note the presence of plaque interproximally and adjacent to the gingiva, but relatively absent closer to the incisal edge. (Courtesy of Dr. WK Grigsby, University of Iowa College of Dentistry.)

Dental Plaque as One of Many Microbial Biofilms

Most natural surfaces have their own coating of microorganisms or biofilm adapted to its individual habitat. The features of dental plaque formation are by no means unique and merely reflect a single instance of a widespread and ancient natural phenomenon. One of the first known examples of life are mineralized bacteria or algae[7,8] found on rocks from the Precambrian era. These are quite similar to dental calculus. The physicochemical and biochemical interactions that underlie bacterial adhesion elsewhere in nature are the same as those observed in plaque formation.[9-11] For example, all living cells, including bacterial cells, have a *net negative surface charge.* The cells can, therefore, be attracted to oppositely charged surfaces on such items as rocks in a stream, skin, or teeth. As with plaque bacteria, organisms in other environments can produce extracellular coatings or slime layers, or a variety of surface fibrils or appendages extending from their cell walls that mediate their attachment to the substrate.[9,12]

In response to environmental conditions and interactions with other members of the microbial community, biofilm bacteria behave differently from planktonic (liquid-phase) cells. This has significant clinical implications. Current research indicates that bacteria growing in biofilms are more resistant to the effects of host defense mechanisms and exogenous antimicrobial agents when compared to the same cells in a liquid suspension.[13,14,15] Thus, it becomes of paramount importance to mechanically disturb the biofilm when utilizing antimicrobial therapy.

Bacterial Colonization of the Mouth

Microorganisms initially colonize the mouth during birth, being naturally acquired from the *mother.* Thereafter, bacteria are acquired from the atmosphere, food, human contact, and even from animal contacts (e.g., pets). The bacteria subsequently colonize interfaces between saliva and both oral soft (e.g., gingiva, tongue, cheeks, and alimentary tract) and hard tissues (e.g., erupted teeth). Mucosal surfaces of the tongue and tonsils may serve as reservoirs for dental plaque-forming organisms, including those related to disease.[16]

With increasing age and improper toothbrushing, gingival recession may occur and result in the exposure of root cementum and dentin. These surfaces, like enamel, may become colonized by oral bacteria that can trigger dental caries.

Prior to eruption, the enamel is lined by remnants of the enamel-forming organ, namely the *reduced enamel epithelium and the basal lamina* that connects it to the enamel surface. The basal lamina is also continuous with organic material that fills the microscopic voids in the superficial enamel. This subsurface material appears as a fringe attached to the basal lamina and is composed of residual enamel matrix proteins (Figures 2-3, 2-4). It is referred to as a *subsurface pellicle.* Because it originates from local cells during tooth formation, it is considered to be of *endogenous* origin. When the tooth emerges into the oral cavity, the remnants of the reduced enamel epithelium are worn off or digested by salivary and bacterial enzymes. The underlying enamel becomes exposed to saliva and the oral microbiota. Salivary components become adsorbed to exposed enamel *within seconds,* forming a microscopic coating over the exposed tooth surface. This thin coating can subsequently become colonized by oral bacteria. Because this coating originates from salivary proteins rather than the dental organ, it is considered to be of *exogenous* origin. Thus, the tooth surface is almost always coated by a variety of structures that are either of endogenous origin (i.e., derived from cells of the dental organ) or of exogenous origin (i.e., acquired following eruption of the teeth into the oral cavity).[17]

Figure 2-3 This transmission electron micrograph demonstrates remnants of the subsurface pellicle (SSP) and the acquired pellicle (AP) between the enamel (ES) surface and the bacterial cells (B) of the dental plaque. (Courtesy of Dr. MA Listgarten, University of Pennsylvania School of Dental Medicine.)

Figure 2-4 Junction of reduced enamel epithelium and enamel. The reduced ameloblasts (RA) are attached to the enamel by hemidesmosomes (HD) and a basal lamina (BL). EM, enamel matrix remnants form a subsurface pellicle; ES, enamel space. ×45,000. (Courtesy of Dr. MA Listgarten, University of Pennsylvania School of Dental Medicine.)

The Acquired Pellicle

The coating of salivary origin that forms on exposed tooth surfaces is called the *acquired pellicle.*[18,19] It is acellular and consists primarily of glycoproteins[a] derived from saliva (Figure 2-3). The pellicle also occupies the millions of microscopic voids in the erupted tooth caused by chemical and mechanical interactions of the tooth surface with the oral environment. Collectively these organic fringe-like projections form a *subsurface pellicle,* which is of *exogenous* or acquired origin. Oral fluids and small molecules can slowly diffuse through the acquired pellicle into the superficial enamel. If the pellicle is displaced, for example by a prophylaxis, it begins to reform immediately.[20,21] It takes about a week for the pellicle to develop its condensed, mature structure which may also incorporate bacterial products.[22-24]

An acquired pellicle also forms on artificial surfaces, including dental restorations and dentures. These organic coatings are similar to the pellicles on natural teeth and may be colonized by bacteria.[25-27] Colonization of the acquired pellicle can be beneficial for the bacteria because the pellicle components can serve as nutrients.[28] For example, proline-rich salivary proteins may be degraded by bacterial collagenases,[29] releasing

peptides, free amino acids, and salivary mucins that may enhance the growth of dental plaque organisms, such as actinomycetes and spirochetes.[30]

The carbohydrate components of certain pellicle glycoproteins may serve as receptors for bacterial-binding proteins such as *adhesins*, thereby contributing to bacterial adhesion to the tooth.[31-33] There is competition for the binding sites on the pellicle, not only by receptors on bacteria, but also from *host proteins*, including immunoglobulins (i.e., antibodies), proteins of the complement system, and the enzyme *lysozyme*. These host proteins originate from saliva and gingival sulcus fluid.[34,35] Once a pellicle site is occupied by one of the competing entities, occupancy by another is inhibited.[36] Not only does competition arise for occupancy of binding sites, but an antagonistic relationship often exists between different types of bacteria competing for the binding sites. For example, it has been shown that some streptococci synthesize and release *bacteriocins*, which can inhibit some strains of *Actinomyces*[37] and *Actinobacillus* species.[38]

[a]A glycoprotein is a protein molecule that includes an attached carbohydrate component.

Dental Plaque Formation

All bacteria that initiate plaque formation come in contact with the organically coated tooth surface fortuitously. Forces exist that tend to allow bacteria to accumulate on teeth or to remove them. Shifts in these forces determine whether more or less plaque accumulates at a given site on a tooth. Many factors influence the build-up of plaque,[39] ranging from simple factors, such as mechanical displacement, stagnation (i.e., colonization in a sheltered or undisturbed environment), and availability of nutrients, to complex factors, including interactions between the microbes and the host's inflammatory-immune systems. Bacteria tend to be removed from the teeth during mastication of foods, by the tongue, toothbrushing, and other oral-hygiene activities. For this reason, bacteria tend to accumulate on teeth in sheltered, undisturbed environments (sites at risk), such as the occlusal fissures, the surfaces apical to the contact between adjacent teeth, and in the gingival sulcus.

Question 1

Which of the following statements, if any, are true?

A. The acquired pellicle is a layer of cells on the *external surface* of the clinical crown of the tooth.

B. Salivary glycoproteins are a major source of organic materials *in* the acquired pellicle.

C. Bacteria produce enzymes that may degrade some of the acquired pellicle components such as proteins.

D. It usually takes *several days* before the acquired pellicle is reformed after a prophylaxis.

E. The presence of immunoglobulins in the acquired pellicle guarantees that the acquired pellicle will remain free from bacterial colonization.

Therefore, it is no coincidence that the major plaque-based diseases, caries and inflammatory periodontal diseases, arise at these sites where plaque is most abundant and stagnant. Initial plaque formation may take as long as 2 hours.[40] Binding sites and individual strain affinity for a given surface vary considerably.[41,42] Colonization begins as a series of isolated colonies, often confined to microscopic tooth surface irregularities.[23] With the aid of nutrients from saliva and host food, the colonizing bacteria begin to multiply. About 2 days are then required for the plaque to double in mass, during which time, the bacterial colonies have been coalescing.[43] The most dramatic change in bacterial numbers occurs during the first 4 or 5 days of plaque formation.[44,45] After approximately 21 days, bacterial replication slows so that plaque accumulation becomes relatively stable.[46] The increasing thickness of the plaque limits the diffusion of oxygen to the entrapped original, oxygen-tolerant populations. As a result, the organisms that survive in the deeper aspects of the developing plaque are either *facultative* or *obligate anaerobes.*[b]

The forming bacterial colonies are rapidly covered by saliva.[47] When seen with the scanning electron microscope, growing colonies protrude from the surface of the plaque as *domes,* giving the appearance of a cluster of igloos beneath newly fallen snow (Figure 2-5). In individuals with poor oral hygiene, superficial dental plaque may incorporate food debris and mammalian cells such as desquamated epithelial cells and leukocytes. This debris is called *materia alba* (literally, "white matter"). Unlike plaque, it is usually removed easily by rinsing with water.[18] At times, the plaque demonstrates staining, with the discoloration being caused by sources including tea, heavy metal salts, drugs, and chromogenic bacteria.

[b] Facultative anaerobes can exist in an environment with or without oxygen; obligate anaerobes cannot exist in an environment with oxygen.

Figure 2-5 Scanning electronmicrograph of dome formation in the plaque. (From Brady, J. M. *J Periodontol*, 1973, 44:416-428.)

Molecular Mechanisms of Bacterial Adhesion

The initial bacterial attachment to the acquired pellicle (Figure 2-6 A) is thought to involve physicochemical interactions (e.g., electrostatic forces and hydrophobic bonding)[48-51] between molecules or portions of molecules, such as the side chains of the amino acids phenylalanine and leucine. It has been suggested that the hydrophobicity of some streptococci, a major plaque group, is caused by cell wall-associated molecules including *glucosyltransferase,* an enzyme that converts the glucose portion of the sugar, sucrose, into extracellular polysaccharide. Some glucosyltransferases have been designated as *hydrophobins.*[52]

Another molecular mechanism of bacterial adhesion is *calcium bridging*[53-55] which links *negatively* charged bacterial cell surfaces to the *negatively* charged acquired pellicle (Figure 2-6 B) via interposed positively charged, divalent calcium ions from the saliva. Calcium bridging may only be important in early plaque formation, because recently formed plaque is readily disrupted by exposure to a calcium-complexing (*chelating*) agent, such as ethylenediaminetetraacetic acid (EDTA).[56]

Some of the streptococci in plaque use the enzyme glucosyltransferase to synthesize extracellular polysaccharides (ECP). Among these are "sticky" *glucans* that, through hydrogen bonding, are thought to contribute to the mediation of bacterial adhesion (Figure 2-6 C).[57] Once the bacteria adhere, they are often "entombed" as additional glucan is produced.[58]

Bacteria also exhibit external cell surface proteins termed *adhesins*,[33,59] that have lectin-like[c] activity as they can bind to carbohydrate components of glycoproteins.[32,60] These molecules, which some researchers have suggested may be located on bacterial surface appendages, such as fimbriae[61] (Figur e 2-6 D), are believed to facilitate colonization of the acquired pellicle.[62] Fimbria-associated adhesins probably mediate bacterial adhesion via ionic or hydrogen-bonding interactions. Adhesins and fimbriae may function together to promote bacterial attachment to pellicle-coated surfaces.[63] For example, *pilin*, a structural protein that constitutes the bulk of some fimbriae, is hydrophobic because of its amino-acid content.[64] These fibrillar surface appendages extend from the bacterial surface and *may reduce* or *mask* the repelling effect of the net negative surface charges. Carbohydrate-binding adhesins have been shown to link actinomycetes to streptococci in early dental-plaque formation.[65,66]

While some or all of the above-described mechanisms may play a role in the attachment of bacteria to one another and to the tooth surface, the nature of the actual linking molecules in plaque, or between plaque and tooth surface coatings is not known.

[c]Lectins are plant proteins with receptor sites that bind specific sugars.

Figure 2-6 This diagram illustrates some of the possible molecular mechanisms mediating bacterial attachment to teeth during dental plaque formation. A. A side chain of a phenylalanine component of a bacterial protein interacts via hydrophobic bonding with a side chain of a leucine component of a salivary glycoprotein in the acquired pellicle. B. The negatively charged carboxyl group of a bacterial protein is attracted to a positively charged calcium ion (i.e., electrostatic attraction), which in turn is attracted to a negatively charged phosphate group of a salivary phosphoprotein in the acquired pellicle. C. The host's dietary sucrose is converted by the bacterial enzyme, glucosyltransferase, to the extracellular polysaccharide, glucan, which has many hydrophobic groups and can interact with amino acid side-chain groups, such as serine, tyrosine, and threonine. D. The fimbrial surface appendage extends from the bacterial cell to permit the terminal adhesin portion to bind to a sugar component of a salivary glycoprotein in the acquired pellicle.

Bacteria in the Dental Plaque

Plaque bacteria vary in number and proportions from time to time and from site to site within the mouth of any one individual. The diversity is even greater between individuals,[67] between races,[68] and between supra- and subgingival plaques.[69] The only abundant bacteria found almost universally in the mouths of humans and animals are streptococci and actinomycetes.

The bacteria colonize the teeth in a reasonably *predictable* sequence. The first to

adhere are *primary colonizers*, sometimes referred to as pioneer species. These are *microorganisms* that are able to stick directly to the acquired pellicle. Those that arrive later are *secondary colonizers.* They may be able to colonize an existing bacterial layer, but they are unable to act as primary colonizers. Generally speaking, the primary colonizers are not pathogenic. If the plaque is allowed to remain undisturbed, it eventually becomes populated with secondary colonizers that are the likely etiologic agents of caries, gingivitis, and periodontitis, the destructive form of inflammatory periodontal disease.

The earliest colonizers are overwhelmingly *cocci* (i.e., spherical cells),[1,69,70] especially streptococci, which constitute 47 to 85% of the cultivable cells found during the first 4 hours after professional tooth cleaning.[71] These tend to be followed by short rods and filamentous bacteria. Because of stagnation, the most abundant colonization is on the proximal surfaces, in the fissures of teeth, and in the gingival sulcus region.[72]

Cocci are probably the first to adhere because they are small and round and, therefore, have a smaller energy barrier to overcome than other bacterial forms.[73] The first or primary colonizers tend to be *aerobic* (i.e., oxygen-tolerant) bacteria including *Neisseria and Rothia.* The streptococci, the Gram-positive facultative rods, and the actinomycetes are the main organisms in both early fissure and approximal plaque.[73-75] As plaque oxygen levels fall, the proportions of Gram-negative rods, for example fusobacteria, and Gram-negative cocci such as *Veillonella* tend to increase.

Of the early colonizers, *Streptococcus sanguis* often appears first,[76] followed by *S. mutans.* Both depend on a sheltered environment for growth and the presence of extracellular carbohydrate (e.g., sucrose). Sucrose is used to synthesize *intracellular* polysaccharides that serve as an internal source of energy, as well as external polysaccharide coats.[77,78] The polysaccharide coating helps protect the cell from the *osmotic* effects of sucrose. In addition, it reduces the inhibitory effect of toxic metabolic *end products,* such as lactic acid, on bacterial survival.

Whereas nonmotile cells, including streptococci and actinomycetes, come into contact with the tooth randomly, motile cells such as the spirochetes are likely to be attracted by *chemotactic* factors (e.g., nutrients). Surface receptors probably provide a means of attachment for secondary colonizers *onto the initial bacterial layer.*[79] Bacteria that cannot adhere easily to the tooth initially via organic coatings can probably attach by strong lectin-like, cell-to-cell interactions with *similar* or *dissimilar bacteria* that are already attached (i.e., the primary colonizers).[33,80,81]

Gram-negative, *anaerobic* (e.g., oxygen-intolerant) species predominate in the *subgingival* plaque during the later phases of plaque development,[82] but they may also be present in early plaque, for example, *Treponema, Porphyromonas, Prevotella,* and *Fusobacterium* species. There is evidence that oxygen does not penetrate more than 0.1 mm into the dental plaque,[83,84] a fact that may explain the presence of anaerobic bacteria in early plaque.

Question 2

Which of the following statements, *if any,* are correct?

A. An important criterion for successful bacterial colonization of teeth is the availability of an unoccupied binding site.

B. Sites on teeth at risk for dental plaque formation include the occlusal fissures, approximal surfaces *apical* to the contact point between adjacent teeth, and the gingival sulcus region.

C. An operational definition for materia alba is "the adherent material on tooth surfaces that can be removed by *rinsing*."

D. During initial formation of dental plaque, negative charges on bacterial cells are attracted to the negative charges of the acquired pellicle.

E. The observation that calcium-complexing agents release recently formed dental plaque from the teeth supports the argument for *calcium bridging.*

Dental-Plaque Matrix

A great variety of factors affect the colonization of the teeth by bacteria. Dental plaque consists of different species of bacteria that are not uniformly distributed, since different species colonize the tooth surface at different times and under different circumstances. The newly formed supragingival biofilm frequently exhibits "palisades" (i.e., columnar microcolonies of cells) of firmly attached cocci, rods, or filaments. The organisms are positioned perpendicular to the tooth surface,[1,69,85] the result of competitive colonization. The bacterial cells in the biofilm are surrounded by an *intercellular plaque matrix* (Figure 2-7).[56] The matrix is composed of both organic and inorganic components that *originate primarily from the bacteria.* Polysaccharides derived from bacterial metabolism of carbohydrates are a major constituent of the matrix while salivary and serum proteins/glycoproteins represent minor components. The bacteria in the subgingival biofilm consist of several motile species that do not form distinctive microcolonies. They tend to be located on the surface of the adherent bacterial layer and are separated by an abundant intercellular matrix. Some bacteria on the surface of the biofilm aggregate into distinctive structures that include arrangements of cocci ("corn-cob" configurations) and rods ("test-tube brush" configurations)[1,2,69,86] radially arranged around a central filament (Figure 2-8).

Figure 2-7 An electron micrograph showing palisades (P) of bacteria perpendicular to the enamel surface (ES), bacterial cells that are probably secondary colonizers (SC), the intercellular plaque matrix (IPM), and the acquired pellicle (AP). (Courtesy of Dr. MA Listgarten, University of Pennsylvania School of Dental Medicine.)

Figure 2-8 A. Cross section of "corn cob" from 2-month-old plaque. A coarse fibrillar material attaches the cocci (C) to the central filament (CF). Original magnification × 22,500. (From Listqarten, M. A., Mayo, H. E., Tremblay, R. *J Periodontol,* 1975; 46:10-26.) B. Coarse "test-tube brush" formations consisting of central filament (CF) surrounded by large, peritrichously flagellated filamentous bacteria (LF). Background consists of a spirochete-rich microbiota (S). Original magnification × 4,300. (From Listgarten M. A. *J Periodontol.* 1976; 47, 1-18.)

Dental-Plaque Metabolism

For metabolism to occur, a source of energy is required. For the caries-related *S. mutans* and many other acid-forming organisms, this energy source can be *sucrose.*[87] Almost immediately following exposure of these microorganisms to sucrose, they produce (1) acid, (2) intracellular polysaccharides (ICP), that provide a reserve source of energy for each bacterium, much like glycogen does for human cells,[88] and (3) extracellular polysaccharides including glucans (dextran)[89] and fructans (levan).[90] Glucans can be viscid substances that help anchor the bacteria to the pellicle, as well as stabilize the plaque mass. Fructans can act as an energy source for any bacteria having the enzyme levanase.[91,92] Quantitatively, the glucans constitute up to approximately 20% of plaque dry weight, levans about 10%, and bacteria the remaining 70 to 80%. As mentioned previously, the glucans and fructans are major contributors to the *intercellular plaque matrix.*[92]

Plaque organisms grow under adverse environmental conditions. These include pH, temperature, ionic strength, oxygen tension, nutrient levels, and antagonistic elements, such as competing organisms and the host inflammatory-immune response. To cope with this hostile environment, the plaque organisms must find a safe haven in relation to their neighbors and the oral environment. Such a favorable location is termed an *ecologic niche.*[5] Normally, once the niches are established, the bacteria of the resident microbiota coexist with the host and the surrounding microcosm. This symbiosis results in a resistance to colonization by subsequent *nonindigenous* organisms. In this manner, the resident microbiota can protect the host against infection by major primary pathogens, e.g., *Corynebacterium diphtheria* and *Streptococcus pyogenes.*

With dietary sugars entering the plaque, *anaerobic glycolysis* results in acid production (*acidogenesis*) and accumulation of acid in the plaque.[5] If no acid-consuming organisms (e.g., *Veillonella)* are available to utilize the acids, the plaque pH drops rapidly from 7.0 to below 4.5. This drop is important because *enamel begins to demineralize between pH 5.0 and 5.5.* One possible outcome of the drop in pH may be the dissolution of the mineralized tooth surface adjacent to the plaque, resulting in carious *cavitation* of the tooth.[77] This process provides the bacteria access to the inorganic elements (e.g., calcium and phosphate) needed for their nutritional requirements. By adhering to the tooth surface via an organic layer of salivary origin, dental plaque bacteria can gain access to a supply of organic nutrients, a widespread phenomenon.[47] The same search for nutrients may explain the extension of bacteria from the supragingival plaque into the gingival sulcus.[93,94] To prevent or reduce subgingival colonization, the host tissues defend against the bacterial challenge with antibacterial strategies, such as the passage of antibodies and the emigration of polymorphonuclear neutrophils from the adjacent connective tissue into the gingival sulcus. The continued metabolic activity of plaque in the subgingival environment initiates the inflammatory response of the gingival tissues (gingivitis)[95] and also may eventually lead to progressive destruction of the periodontium (periodontitis)[96].

Until supragingival plaque mineralizes as dental calculus, it can be removed by toothbrushing and flossing.[97] As the plaque matures, it becomes more resistant to removal with a toothbrush. In one study, at 24, 48, and 72 hours after formation, 5.5, 7.8, and 14.0 g/cm^2 of pressure, respectively, were required to dislodge the plaque—almost three times as much pressure to remove it on the third day as on the

first.[98] Once dental calculus is formed, professional instrumentation is *necessary* for its removal.

Question 3

Which of the following statements, if any, are correct?

A. Dental plaques typically exhibit uniform structures, composition, and properties.

B. The intercellular dental plaque matrix is probably formed by a combination of host *materials*, such as salivary proteins, *and* bacterial metabolites.

C. The term "corn-cob" configuration, describes one of several possible aggregates between different kinds of bacterial cells in the dental plaque matrix.

D. The acid dissolution of tooth mineral supplies calcium for both bacterial nutrition and for calcium binding.

E. Gingival inflammation is generally caused by bacteria that reside in dental plaque adjacent to the tooth.

Dental Calculus

A last stage in the maturation of some dental plaques is characterized by the appearance of mineralization in the deeper portions of the plaque to form dental calculus[99]. The term *calculus* is derived from the Latin word meaning pebble or stone. The lay term, *tartar,* refers to an accumulated sediment or crust on the sides of a wine cask. Some people do not form calculus, others form only moderate amounts, and still others form heavy amounts.

Calculus itself is not harmful. However, a layer of unmineralized, viable, metabolically active bacteria that are closely associated with the external calculus surface is potentially pathogenic. Calculus *cannot* be removed by brushing or flossing. It is often difficult to remove all the calculus, even professionally, without damaging the tooth, especially the softer root cementum. However, calculus needs to be removed because its presence makes routine oral hygiene more difficult or even impossible by forming calculus *spurs* (Figure 2-9). These structures may contribute to plaque accumulation and stagnation. Calculus removal is also a prerequisite to regenerate lost or damaged periodontal tissues following treatment.

In addition to local factors, behavioral and systemic conditions may affect calculus formation. For example, smoking causes an accelerated formation of calculus.[100] Children afflicted with asthma or cystic fibrosis form calculus at approximately twice the rate of other children.[101] Similarly, non-ambulatory, mentally handicapped individuals, tube-fed over long periods, may develop heavy calculus within 30 days, despite the fact that no food passes through the mouth.[102] Conversely, medications such as beta-blockers, diuretics, and anticholinergics can result in significantly reduced levels of calculus. The authors of the latter study concluded that either the medications were excreted directly into the saliva, affecting the rate of crystallization, or altered the composition of the saliva and thus indirectly affected calculus

formation.[103]

Calculus formation is related to the fact that saliva is saturated with respect to calcium and phosphate ions.[104] Precipitation of these elements leads to mineralization of dental plaque giving rise to calculus. The crystals in calculus include hydroxyapatite, brushite, and whitlockite, all of which have different proportions of calcium and phosphate in combination with other ions, such as magnesium, zinc, fluoride, and carbonate. *Supragingival calculus* forms on the tooth *coronal* to the gingival margin, and frequently develops opposite the duct orifices of the major salivary glands. It is often found where saliva pools on the lingual surfaces of the mandibular incisors (Figure 2-10), and can form in the fissures of teeth. Subgingival calculus forms from calcium phosphate and organic materials derived from serum, which contribute to mineralization of subgingival plaque.

One of the means by which formation and growth of calculus may be studied is by ligating thin plastic strips around the teeth and then removing the strips at various intervals.[105] Within 12 hours after placement, x-ray diffraction studies demonstrate mineral elements in the forming plaque. By 3 to 4 days, the concentration of calcium and phosphate is significantly higher in the plaque of those with heavy calculus formation than in the plaque of those with no calculus formation.

Subgingival calculus is about 60% mineralized, whereas supragingival calculus is only about 30% mineralized.[106] Because it is harder, thinner, and more closely adapted to tooth surface imperfections, subgingival calculus can be more difficult to remove than supragingival calculus. The two types of calculus may differ in color. Supragingival calculus, which derives its mineral content from saliva, usually appears as a yellow to white mass with a chalky consistency. Subgingival calculus, which derives its mineral from the inflammatory exudate in the sulcus and periodontal pocket, appears gray to black in color and has a flint-like consistency. The dark coloration may be caused by bacterial degradation of components of the hemorrhagic exudate that accompanies gingival inflammation.

Alkaline conditions in dental plaque may be an important predisposing factor for calculus formation.[107] Calculus formation is not restricted to one bacterial species, or even to those growing at neutral or slightly acidic pHs. This is evidenced by the fact that caries-related streptococci may mineralize.[108] Not all plaques mineralize, but a plaque that is destined to mineralize begins to do so within a few days of its initial formation, even though this early change is not detectable at a clinical level. Mineralization usually begins in the *intercellular plaque matrix* but eventually occurs *within* the bacterial cells (Figure 2-11). Bacterial phospholipids and other cell-wall constituents may act as initiators of mineralization,[109] in which case mineralization may begin *in the cell wall* and subsequently extend to the rest of the cell and into the surrounding matrix (Figure 2-12). Calculus may also form on the tooth surfaces of germfree animals.[110] This type of calculus consists of an organic matrix of nonmicrobial origin which becomes mineralized.

Attachment of Calculus to the Teeth

At the tooth interface with calculus, the enamel or root cementum are never perfectly smooth and invariably contain a variety of surface imperfections. These normal

irregularities such as the *perikymata*[d] and the point of origin of *Sharpey's fibers*[e] on the cementum appear to aid calculus attachment. Other defects in the enamel and cementum, including areas of demineralization and cemental tears,[111] may also contribute to a stronger calculus attachment to the tooth. Electron micrographs indicate a very close relationship between the matrix of the tooth surface and the matrix of calculus; the crystalline structures of both are also very similar.[112]

[d]Perikymata are the numerous, small, transverse ridges on the exposed surface of the enamel of the permanent teeth.

[e]The tooth is anchored by connective tissue fibers that extend between the cementum and the bone; the ends embedded in the cementum and bone are known as Sharpey's fibers.

Inhibiting Calculus Formation

Several agents are currently available to reduce calculus formation, including dentifrices that contain pyrophosphate, or metal ions such as zinc.[113,114] One dentifrice contains two soluble phosphates, *tetrasodium pyrophosphate* and *disodium dihydrogen pyrophosphate*, in addition to fluoride.[114,115] The pyrophosphate ion not only serves as a structural analog of the orthophosphate ion, disrupting the formation of calcium phosphate crystals, but also inhibits some bacterial growth at concentrations significantly lower than the levels found in dentifrices.

Figure 2-9 Radiograph demonstrating a "spur"- shaped deposit of calculus (C) on the distal side of the maxillary left first molar apical to the overhanging metallic restoration (R). The arrow (G) marks the coronal level of the gingival tissues indicating that this is a subgingival deposit of calculus. (Courtesy of Dr. WK Grigsby University of Iowa College of Dentistry.)

Figure 2-10 Deposits of supragingival calculus on the lingual surface of incisors and canines that could not be removed by brushing. (Courtesy of Dr. WK Grigsby, University of Iowa College of Dentistry.)

Figure 2-11 Typical pattern of dental plaque mineralization in which the initial mineralization occurs in the interbacterial plaque matrix (M), with bacterial cells (B) becoming mineralized secondarily, × 40,000. (Courtesy of Dr. MA Listgarten, University of Pennsylvania School of Dental Medicine.)

Figure 2-12 Atypical pattern of dental plaque mineralization in which bacterial cells (B) act as foci of initial mineralization, with the matrix (NM), becoming mineralized secondarily, ×25,000. (Courtesy of Dr. MA Listgarten, University of Pennsylvania School of Dental Medicine.)

Question 4

Which of the following statements, if any, are correct?

A. Intracellular polysaccharides are a source of energy available to bacteria, but levans are available only to the synthesizing bacteria.

B. An operational definition of calculus might be that it is "a mineralized dental plaque that cannot be removed from the tooth by brushing or flossing."

C. The flow of saliva over the tooth surfaces near the major salivary gland ducts keeps those teeth free of calculus deposits.

D. Subgingival calculus is usually more densely mineralized than supragingival calculus.

E. Calculus formation usually begins in the bacterial cell wall and extends to the intercellular matrix.

Summary

Bacteria in dental plaque are the direct cause of the most widespread of all human diseases—dental caries and inflammatory periodontal diseases. These diseases, however, are not classical infections. They arise because of complex changes in plaque ecology and are affected by many factors in the host's protective responses. To understand the role of dental plaque in disease and how to prevent or control the plaque-associated diseases, it is essential to understand the nature of dental plaque. Plaque forms initially on the organic layer coating the erupted tooth. This organic layer originates from salivary products that are deposited on the teeth, forming an acquired pellicle to which bacteria adhere. Adhesion is mediated by a variety of bonding mechanisms, including physicochemical and electrostatic interactions, and stereo-chemical interactions between bacterial adhesins and receptors in the acquired pellicle and bacterial surfaces. The earliest of the primary bacterial colonizers are mainly Gram-positive facultative cocci. They are followed by a variety of Gram-positive and Gram-negative species—the secondary colonizers. Caries-related bacterial species have a greater ability than others to adapt to excess sugars and their metabolites. Supragingival plaque is associated with caries and gingivitis, whereas subgingival plaque is associated with gingivitis and periodontitis. With higher pH (i.e., less acidity), some plaques mineralize to form supra- and subgingival dental calculus. In calculus formation, mineralization of dental plaque generally begins in the extracellular matrix and eventually spreads to include the bacteria. Rarely, mineralization may begin within the walls of bacterial cells and spread to the extracellular matrix. Calculus is generally covered by actively metabolizing bacteria, which can cause caries, gingivitis, and periodontitis. Regular toothbrushing and flossing can remove dental plaque and control its formation. Once dental plaque mineralizes to form calculus, professional instrumentation is necessary for its removal. Notwithstanding the contribution of calculus to inflammatory periodontal diseases, it is stagnation of pathogenic bacteria at critical sites that leads to both dental caries and periodontal diseases. Later chapters deal with the wide range of methods, mechanical and chemical, increasingly used to control plaque and calculus formation. All of these methods have the aim of preventing, arresting, or reversing the progression of dental caries and periodontal tissue inflammation.

Answers and Explanations

1. B and C—correct.

A—Incorrect. The acquired pellicle is "acellular," i.e., cell-free.

D—Incorrect. The acquired pellicle begins to reform immediately and is reestablished within several hours.

E—Incorrect. Even though some binding sites are occupied by immunoglobulins, many more are occupied by bacteria.

2. A, B, D, and E—correct.

C—Incorrect. Like charges (i.e., negative to negative or positive to positive) repel; unlike charges attract.

3. B, C, D, and E—correct.

A—Incorrect. So many factors affect plaque formation that composition, structure, and properties are greatly varied.

4. B and D—correct.

A—Incorrect. It should be the reverse, with intracellular polysaccharides available to the synthesizing bacteria, and levans to the surrounding bacteria with the enzyme levanase.

C—Incorrect. The presence of high concentrations of calcium and phosphate ions at the duct openings results in more, not less, calculus formation.

E—Incorrect. Calculus *usually* begins in the intercellular matrix, and spreads to engulf the cells.

Self-evaluation Questions

1. The presence of a preponderance of cocci is a sign of (early)(late) plaque formation.

2. Following prophylaxis, it takes about _____ (hours)(days) for the acquired pellicle to completely reform.

3. Two of the host's *defensive proteins that* compete with bacteria for receptor sites on the acquired pellicle are _____ and _____.

4. It takes approximately _____ (hours) (days) for the initial plaque to form and about _____ days to double in mass. Once formed, the growth is rapid for about _____ days and finally stabilizes in mass around the _____ day.

5. Given the choice of (water)(a toothbrush) or a (prophylaxis); which one is required to remove each of the following: (1) materia alba, (2) plaque, or (3) calculus?

6. Bacteria can attach to the acquired pellicle via _____ bonding, by calcium _____, via attachment to the sticky _____, and by surface proteins called _____.

7. The three places on the teeth where bacterial colonization is most abundant are _____, _____, and _____.

8. "Corn-cob" configurations are caused by (cocci) (rods) radially attached to a central rod, whereas the "_____" configuration is caused by rods radially attached to a central rod.

9. Between the cells of the plaque is the (extracellular polysaccharide)(intracellular polysaccharide) containing _____ and levans that serve as energy sources for the bacteria.

10. The "safe haven" where a bacterial colony can exist in the plaque environment is known as a(n) _____.

11. Calculus is mainly made up of calcified _____.

12. One condition causing accelerated calculus formation is _____. Reduced formation is seen after use of _____ (drugs).

13. *Supragingival* calculus derives its minerals from the _____; whereas *subgingival* calculus derives them from the _____.

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Chapter 3. The Developing Carious Lesion - *Norman O. Harris Adriana Segura*

Objectives

At the end of this chapter, it will be possible to

1. Name the four general types of carious lesions that are found on the different surfaces of the teeth.

2. Describe the histologic characteristics of enamel and dentin that facilitate fluid flow throughout a tooth.

3. Describe the four zones of an incipient caries lesion.

4. Describe the conduits (pores) that directly conduct acid from the bacterial plaque to the body of the lesion.

5. Name the two bacteria most often implicated in the caries process, and indicate

when each is present in the greatest numbers during the caries process.

6. Describe the series of events in a cariogenic plaque and subsurface lesion from the time of bacterial exposure to sugar until the pH returns to a resting state.

7. Discuss the characteristics of root caries and explain the differences and similarities to coronal caries.

8. List measures to prevent and to remineralize root and coronal caries.

9. Explain why so much time is taken by the profession in treating secondary caries.

10. Explain the relationship between pH and calcium and phosphorus saturation in caries development.

11. Discuss the protective relationship of calcium fluoride to hydroxyapatite and fluorhydroxyapatite during an acidogenic attack.

Introduction

Understanding Caries: Concepts

Every day there is a *normal, but minute, demineralization* of the hard tooth structures caused by bacterial acid production, as well as consuming acid foods such as fruit juices, vinegar, and soft drinks—even from the abrasion of toothbrushing.[1,2] So long as the demineralization is limited, the body's *remineralization* capabilities can replace the lost minerals from elements such as calcium, phosphate, fluoride and other elements that are found in the saliva. The *physiologic demineralization* does not become pathologic until the demineralization outstrips the remineralization over an indefinite period of time that leads to the onset of *cavitation*. A favorable balance between de- and remineralization is necessary to maintain the homeostasis needed for a lifetime of intact tooth retention.

When a cavity occurs, it can be defined as a localized, post-eruptive pathological process involving bacterial acid demineralization of hard tooth tissue, which if continued without a compensatory remineralization, results in the formation of a cavity.

The history of dental caries is as long as history itself. Probably one of the oldest and most whimsical theories of caries and toothache was that of the *tooth worm* which allegedly lived in the center of the tooth.[3] Many early barber-surgeons reported sighting the "worm," but none seemed to be able to capture the creature, nor could explain how it got into the tooth in the first place. In the late 1700s the worm theory was largely replaced by the *vital theory*, a theory that postulated that inflammation arising from *within* a defective tooth eventually caused a surface lesion. Robertson in 1835-England, and probably one of the first preventive-oriented dentists believed and published, that food impaction and fermentation might be the cause.[4] By the end of the 19th century, others in Europe began to indict bacteria as the culprit.

In 1890 W. D. Miller, an American dentist teaching in Germany, published his

chemicoparasitic theory of caries which (with many modifications) is still accepted in concept today.[5] As a result of his experimentation, Miller believed that the extraction of the "lime salts" from the teeth was a result of bacterial acidogenesis and was the first step in dental decay. Miller's work however, failed to identify dental plaque as the source of the bacteria and the bacterial acids. The chemicoparasitic theory became more cogent when taken in conjunction with the finding of other contemporary dental researchers, including G. V. Black (the "Grand Old Man of Dentistry") who described the "gelatinous microbic plaque" as the source of the acids.[6]

Caries lesions occur in four general areas of the tooth: (1) *pit and fissure* caries, which are found mainly on the occlusal surfaces of posterior teeth as well as in lingual pits of the maxillary incisors and buccal surfaces of lower molars; (2) *smooth-surface* caries, that arise on intact smooth enamel surfaces other than at the location of the pits and fissures; (3) *root-surface* caries, which might involve any surface of the root; and (4) *secondary* or *recurrent* caries that occur on the tooth surface adjacent to an existing restoration. Smooth-surface caries can be further divided into caries affecting the *buccal* and *lingual* tooth surfaces, and *approximal* caries, affecting the contact area of adjoining tooth surfaces (i.e., mesial or distal surfaces).

Dental caries is a *multifactorial* disease process, often represented by the three interlocking circles and an arrow depicting the passage of *time* (Chapter 1, Figure 1-4). For caries to develop, three conditions must occur simultaneously: (1) there must be a susceptible tooth and host; (2) cariogenic microorganisms must be present in quantity; and (3) there must be excessive consumption of refined carbohydrates. When exposed to a suitable substrate (usually *sugar or sugar-laden snacks or desserts)*, cariogenic bacteria present in the plaque produce acid. If this occurs over a sufficiently long period of *time*, a caries lesion develops. Each of these main factors includes a number of secondary factors and can be introduced to either protect or further damage the tooth. For example, fluoride incorporated into dental enamel increases tooth resistance (see Chapter 8 and 9). Conversely, a reduction in the saliva flow (*xerostomia*) greatly increases the caries risk.

Figure 1-4 Caries is a *multifactorial* disease caused by bacteria, a supporting host diet of refined carbohydrates, decreased host resistance, and time for the cavity to develop. (*Source:* Dr. Norman O. Harris, University of Texas Dental School at San Antonio.)

Embryology and Histology of Enamel

Before discussing the carious process further, it is *necessary* to briefly review the embryology and histology of enamel. Without this review it is very difficult to understand how de- and remineralization can occur in such a highly mineralized tissue as enamel.

The enamel is made up of billions of crystals that in turn make up millions of individual rods. The enamel rods, when viewed in cross section with an electron microscope, appear *not* as rods, but as *keyhole-shaped structures*, approximately 6 to 8 microns in diameter, with the enlarged portion of the keyhole called the *head* and the narrow portion the *tail*. With this configuration, each head fits between two tails. The tail is *always* positioned toward the *apex*. In the head of the rod the long axes of the *crystals*, called the *C axis*, are parallel to the enamel rod. However, as the

periphery of the rod is approached, the crystals assume an angle to the more central crystals; in fact, in the tail this angle may be around 30°. (Figure 3-1).

Each rod that extends from the dentoenamel junction (DEJ) to the tooth surface is completed start-to-finish by *one* ameloblast. The final enamel is approximately 95% inorganic, and 5% organic material and water. This 5% porosity forms a network of channels for fluid diffusion of ions and small molecules that are dispersed throughout the entire *enamel cap*.[a] The space available for this diffusion is found between the rods and even between the crystals. To further extend this intra enamel network throughout the enamel, there are morphologic structures in the enamel with a high protein content, such as the striae of Retzius, enamel lamellae, enamel tufts, pores, and enamel spindles. These several diffusion channels probably serve two very important purposes in preserving the teeth: (1) their teleological purpose was possibly to *permit physiological remineralization* throughout life, and (2) the voids and protein content in the enamel probably *cushion intense biting pressures* to help prevent fractures. Unfortunately, these same channels of diffusion also serve another purpose, viz., the conducting of plaque acids into the enamel interior to *cause demineralization*.

This brief summary also points out the *exquisite genetic control* exercised over the rapidly changing and complex tissue building that marks the development of enamel. The following review begins at the time when odontoblasts and ameloblasts are lined up opposite each other along the *future* dentino-enamel junction.

The initial event of the *secretory* stage occurs with an odontoblastic deposition of the first few microns of *predentin*. This is followed by the initiation of the secretory phase of the ameloblast. The first secreted enamel proteins do *not* accumulate as a layer, but instead, penetrates *into* the developing predentin and subjacent odontoblasts.[7] The microenvironment of the ameloblast at this time, is mainly one of proteins and water.[8] As the ameloblast retreats towards the future surface of the tooth, it uses these proteins to form an acellular and avascular matrix template upon which the future hydroxyapatite crystals are to be positioned.[9,10] This requires a very rigid *genetic control* over the sequence of events that will extend through matrix formation, crystal nucleation, and crystal growth; as well as rod formation (elongation, widening, and maturation).

The matrix is highly heterogenous because of the involvement of protein contributions from many different genes—amelogens, enamelin, ameloblastin, tuftelin, and various enzymes.[b] Possible functions of these proteins are nucleation (tuftelin), mineral ion binding, (amelogenin, enamelin), and crystal growth (amelogenin, enameling, ameloblastin).[11] If there is a failure of initiation or integration of action of any of these proteins, a dysplastic tissue can result, for example, amelogenesis imperfecta that is caused by a defect in the amelogen gene.[12] It should be emphasized that the ameloblast does *not* complete the matrix template from the dentino-enamel junction to the exterior of the tooth before enamel formation begins. Instead, while the ameloblast is *matrix-building* on the lateral sides, at the same time the Tomes process at the *basal end* of the cell is modulating the enamel-building from the time of initial secretion to the pre-eruption maturation stages.[13] This is a continuous process as the ameloblast moves outward. In an early *supersaturated* environment of high calcium and phosphorus initiated by the ameloblast, *octacalcium*

phosphate is laid down as a *precursor* to the *hydroxyapatite* crystal.[14] The early hydroxyapatite crystals are small and of poor crystalinity. As the ameloblast moves outward, the rod increases in length and thickness. Towards the end of the secretory stage, the matrix is *almost* completely *degraded*. Accompanying this event, there is a massive crystal growth.[15] The maturing enamel growth is now approaching the pre-eruptive state. The hydroxyapatite crystals are unusually large, uniform in size, and regularity positioned.[11] The enamel that was originally a soft product is now the hardest and most durable produced in the human body.

There are still a few more points about the life span of the amazing ameloblast. As the tooth approaches eruption, the columnar configuration of the ameloblasts flattens to form the *reduced enamel epithelium* that covers (and protects) the yet immature enamel. After eruption, the reduced enamel epithelium disappears and is succeeded by the *acquired (also called salivary) pellicle* that, in turn, is covered by the *dental plaque* (Chapter 2). Even at this time, the crystals of the rod are not yet fully mineralized. For the first year after eruption into the mouth, the rods undergo a post-eruptive maturation, with the additional tooth minerals being derived from the saliva. This temporary hypomineralization of the enamel with its greater porosity, in part explains why newly erupted teeth are more susceptible to caries than teeth that have been present in the mouth for some time.

[a]If an intact tooth was stripped of its pulp chamber, dentin and cementum, the only remaining structure is the enamel cap.

[b]At this point in time, it is not necessary to memorize the names and functions of genes. Just remember that the tooth morphology depends on genetic guidance.

Figure 3-1 Enamel structure. A. The orientation of the enamel rods from the dentoenamel junction to the tooth surface. B. An arcade of rods seen at the section indicated by the line in A. C. The keyhole morphology of the rods. Shading differences represent different orientations of the crystals. (Courtesy of MWJ Dodds, University of Texas Dental School, San Antonio.)

Physical and Microscopic Features of Incipient Caries

The development of a carious lesion occurs in three distinct stages. The earliest stage is the *incipient* lesion, which is accompanied by histologic changes of the enamel; the second stage includes the progress of the demineralization front toward the dentino-enamel junction and/or into the dentin; while the final phase of caries development is the development of the *overt*, or *frank*, lesion, which is characterized by actual *cavitation*. If the time between the onset of the incipient lesion in one or more teeth, and the development of cavitation is rapid and extensive, the condition is referred to as *rampant* dental caries. Usually rampant caries occurs following either the excessive and frequent intake of sucrose, or the presence of a severe *xerostomia* (i.e., dry mouth) or both. From a preventive dentistry standpoint, the early identification of the incipient lesion is extremely important, because it is during this stage that the carious process *can be arrested or reversed*. The overt lesion can *only* be treated by operative intervention.

Clinically, it is often difficult to recognize and diagnose the early lesion, and for this reason it is important to be familiar with its features from etiologic and histologic

standpoints.[17] The incipient lesion is macroscopically evidenced on the tooth surface by the appearance of an area of *opacity*—the *white spot* lesion. At this earliest clinically visible stage, the subsurface demineralization at the microscopic level is well established with a number of recognizable zones. Probably a most important fact is that the surface of the enamel appears relatively intact (although the electron microscope shows a surface that is more porous than sound enamel). On the buccal and lingual surface of a tooth, the white spot may be localized, or it can extend along the entire gingival area of the tooth, or multiple teeth where food tends to lodge. *Interproximally*, the incipient lesion is usually first detected on a *bite wing x-ray*. It usually starts as a small lucency immediately *gingival* to the *contact point* and then gradually expands to a small kidney shape, with the indentation of the kidney contour directed coronally.[18] In fissure caries, the initial lesion comparable to the "white spot," usually occurs *bilaterally* on the two surfaces *at the orifice* of the fissure and eventually coalesces at the base[19] (Figure 3-2). Occasionally lesion formation begins along the wall of the fissure or at the base, either unilaterally or bilaterally.[20]

During the early stages the incipient lesion is not a surface lesion in which loss of outer enamel can be detected. Instead, the mature surface layer of 10 to 100 microns remains intact. If an explorer is used, the surface enamel feels hard and provides no indication of demineralization. However, microscopic pores extend through the mature surface layer to the point where subsurface demineralization occurs; the main body of the lesion is located and enlarges from this point.

The incipient lesion has been extensively studied and best described by Silverstone.[18] Many of the observations of the incipient lesion have been based on the use of a polarizing microscope, which permits precise measurements of the amount of space—called *pore space*—that exists in normal enamel and to a greater extent in enamel defects. Thus as demineralization progresses, *more* pore space occurs; conversely, as remineralization occurs, *less* pore space is present.

In the incipient lesion as described by Silverstone, four zones are *usually* present. Starting *from the tooth surface*, the four zones are the (1) *surface* zone, (2) *body of the lesion*, (3) *dark zone*, and (4) the *translucent* zone. (Figure 3-3)

Pore Spaces of the Different Zones

The translucent zone, the *deepest* zone is seen in approximately 50% of the carious lesions examined.[18] In this zone, which is the *advancing front* of the lesion, slight demineralization occurs, with a 1% pore space, compared with 0.1% for intact enamel. In contrast, the dark zone occurs in approximately 95% of carious lesions and has a pore volume of 2 to 4%. When teeth showing no dark zone are placed in a remineralizing solution, the dark zone becomes visible in its expected position between the translucent zone and the body of the lesion.[21] On the basis of this phenomenon, it is suggested that *this dark zone is the site where remineralization can occur* and that a wider dark zone indicates a greater amount, or a longer period, of remineralization.

Peripheral to the dark zone lies the main body of the lesion. In this zone, pore volume ranges from approximately 5% on the fringes of the lesion to about 25% in the center.[18] Despite this considerable amount of demineralization, the remaining crystals

still maintain their basic orientation on the protein matrix. Finally, the surface zone has a near-normal pore space of approximately 1%. It is the surface zone and the dark zone that are the remineralization zones of the incipient lesion.

Direct Connection of the Bacterial Plaque to the Body of the Lesion

Demineralization of the surface enamel produces a ragged profile when seen with the electron microscope (Figure 3-4). Small pores, or microchannels, have been observed by electron microscopy in the *surface zone* of incipient lesions. The initial attack may be on the rod ends, between the rods, or both.[22] There is a *widening* of the areas between adjacent rods.[23] When conditions are optimum, this ragged interface between surface and subsurface can be remineralized (repaired), either by the body defenses (calcium and phosphate and other ions from the saliva), or by man-made strategies (fluoride therapy and sugar discipline).

Figure 3-4 is an outstanding electron micrograph to aid in visually understanding caries initiation and progression beyond the details provided by Silverstone. For orientation, in the upper-left corner of the illustration, there is the bacterial plaque (B); immediately below is the salivary pellicle (SP), followed by the enamel (EN). The lighter area labeled CM leads *directly* from the bacterial plaque to the area that is, or will be, the expanding body of the lesion. In turn, the body of the lesion opens into many interrod spaces that continue uninterrupted to the dentino-enamel junction (DEJ). It is along these inter-rod spaces that the bacterial plaque fluids diffuse (Figures 3-5 A and B).

En route to the DEJ, the stria of Retzius allows lateral acid access out of the inter-rod space into the center of the intact or damaged rods and crystals. Once at the DEJ, any fluid flow whether causing de- or remineralization, can trichotomize[c] either along the hypomineralized DEJ, or into the dentinal tubules to the pulp chamber (Figure 3-6). The speed of progression of the caries front depends on such factors as ion concentration, pH, saliva flow, and buffering actions—all of which are continually changing. In summarizing, there is a trail of interconnecting channels for diffusion of fluids transiting from the bacterial plaque to the pulp chamber. Any chemical changes in the *plaque* can be soon reflected *throughout* the enamel and dentin as part of the incipient lesion.

These ultrastructural enamel defects—the pores—allow the exit of plaque acids direct to the subsurface region. The initial acid attack preferentially dissolves the magnesium and carbonate ions and is later followed by a removal of the less soluble calcium, phosphate, and other ions that are part of the crystal.

Eventually the undermined surface zone collapses. Concurrent with this change, the more soluble proteins are lost from the subsurface matrix. Once cavitation occurs, the zones of the incipient lesion become less clearly defined because of mineral loss and the presence of bacteria, bacterial end products, plaque, and residual substrate, which may support further lesion development. The lesion is *no longer* an incipient lesion; it is now an *overt caries lesion* requiring operative intervention.

[c]trichotomize = go in one of three directions—along the DEJ in either direction, or into the dentinal tubules.

Figure 3-2 Incipient caries in an occlusal fissure. The bilaterality of the lesion is evident in the microradiograph. (Courtesy of JS Wefel, University of Iowa College of Dentistry.)

Figure 3-3 A and B. The bilaterality of caries development. Note coalescence of two lateral carious areas at base of fissure. (From Konig, K.G. Dental morphology in relation to caries resistance with special reference to fissures as susceptible areas. *J Dent Res.* 1963. 42:461-476.)

Figure 3-4 From a mesial brown spot on a lower second molar. An 0.8 um defect filled with organic material (CM) extends from the plague (upper left, B), through the enamel (EN) as the surface zone, and continues into the larger area of the demineralized subsurface body of the lesion. On either side of the body of the lesion are areas with disoriented crystals that constitute the (demineralizing) translucent zones. (From Frank RM, Brendel A. Ultrastructure of the approximal dental plaque and the underlying normal and carious enamel. ArchOral Biol. 1996:11:909. Permission granted by Pergaron Press, Ltd., Oxford, England.) Teaching comment: This electron micrograph is very important to understanding the route of ions from the plaque of the tooth to the interior and vice versa, in de-and remineralization, respectively of an incipient lesion.

Figure 3-5 A. Electron micrograph of rod cut perpendicular to long axis, showing head (H) and tail (T) relationship. B. Electron micrograph of parallel to long axis showing two rods (R) and interrod area (I). Original magnification ×5000. (From Meckel AH, Griebstein WJ, Neal RJ. Structure of mature human dental enamel as observed by electron microscopy. *Arch Oral Biol.* 1965; 10:775-783.)

Figure 3-6 Diagram of a trichotomized lesion, due to diffusion of acids in both directions under the enamel, and directly into the body of the lesion in the dentin. T = translucent zone, B = body of the lesion, R = reactionary dentin, P = pulp. (Silverstone LM, Hicks MJ. The structure and ultrastructure of the carious lesion in human dentin. *Gerodontics*, 1985, 1:185-93.)

Question 1

Which of the following statements, if any, are correct?

A. All the following structures are involved in the passage of fluids in the enamel: interrod space, intercrystalline matrix, pores, and striae of Retzius.

B. The head of the enamel rod is always oriented toward the incisal or occlusal surfaces in both the maxillary and mandibular teeth.

C. A rampant caries attack implies a previous incipient lesion for each overt lesion that develops.

D. The incipient lesion usually starts incisal to the contact point in interproximal caries and at the base of the fissure in occlusal caries.

E. The dark and the translucent zones are the centers of remineralization when "biologic repair" of the tooth is occurring.

Know your Enemy, the Cariogenic Bacteria

Following Miller's works in the 1890s, it was not until 1954 that fundamental *experimental evidence proved* that bacteria were the agents of acid production. Orland and colleagues[24] demonstrated that *gnotobiotic*[d] rats did not develop caries when fed a cariogenic diet; they did develop caries when acidogenic bacteria, plus a *cariogenic diet were introduced* into the previous germ-free environment. The *transmissible* nature of caries in animals was later demonstrated by the experiments of Keyes[25] who showed that previously gnotobiotic, caries-inactive hamsters developed caries after contact with caries-active animals.

[d]Gnotobiotic = germ-free environments

Mutans Streptococci and Caries

For caries to develop, acidogenic (acid-producing) bacteria *must* be present, and a means *must* exist to prevent the acid from being washed away from the point where caries is to develop. Dental plaque fulfills both of these functions. It helps protect the bacterial colonies in a cocoon of (a gel-like) *glucan* from being flushed, neutralized, or effected by antimicrobials in the saliva or introduced by humans.

Of the 300-or-more species of microorganisms inhabiting the plaque, the great majority are not directly involved in the caries process. Two bacterial genera are of special interest in cariogenesis: (1) the *mutans streptococci*[e] and (2) the *lactobacillti*.[26,27] The mutans streptococci (MS) are a group of bacterial species previously considered to be serotypes of the single species, *Streptococcus mutans*.[26] These bacteria are characterized by their ability to produce *extracellular glucans* from sucrose and by their acid production in animal and human studies. *Streptococcus mutans* received its name in 1924 when J. K. Clarke in England isolated organisms from human carious lesions. He noted that they were more oval than round and assumed them to be a mutant form of a streptococcus.[28]

Mutans streptococci are now considered to be the major pathogenic bacterial species involved in the caries process. Innumerable surveys have indicated an association between the number of *S. mutans* and dental caries.[29-31] The counts have been repeated worldwide for over more than five decades for all ages—in the United States,[32] Sweden,[33] Latvia,[34] Finland,[35] and China[36]—with high MS bacterial counts being overwhelmingly correlated to the number of teeth with caries or restorations.

Mutans streptococci are usually found in relatively large numbers in the plaque occurring immediately over developing smooth-surface lesions. In one longitudinal study, specific sites were periodically sampled for the presence of MS, and the teeth later examined for caries. Teeth destined to become carious exhibited a significant increase in the proportions of MS from 6 to 24 months *before* the eventual diagnosis of caries.[37] Similarly, dental plaques isolated from sites overlying white spot lesions were characterized by a significantly higher proportion of MS than plaques sampled from sound enamel sites.[38] Increased numbers of MS in the saliva also parallel the development of the smooth-surface lesion. In another study, MS counts from the saliva of 200 children indicated that 93% with detectable caries were positive for MS, whereas uninfected children were almost always caries-free.[39]

Certain physiological characteristics of the MS favor their reputation as a prime agent in caries. These traits include the ability to *adhere* to tooth surfaces, production of abundant insoluble *extracellular* polysaccharides (glucan) from sucrose, *rapid* production of lactic acid from a number of sugar substrates, acid tolerance, and the production of *intracellular* polysaccharide (energy) stores. These features help the MS survive in an unfriendly environment due to periods of very low availability of substrate (i.e., between meals and snacks). As a general rule, the cariogenic bacteria metabolize sugars to produce the energy required for their growth and reproduction. The by-products of this metabolism are acids, which are released into the plaque fluid. The damage caused by MS is mainly caused by *lactic acid*, although other acids, such as butyric and propionic, are present within the plaque.[40]

[e]Originally, it was believed that Streptococci mutans was the only species of streptococci that caused caries; however, when it was found that other streptococci were also involved, they were all grouped under the umbrella designation of mutans streptococci. In the older references, the original terminology will be maintained.

Lactobacilli and Caries

Lactobacilli (LB) are cariogenic, acidogenic, and aciduric. Indeed, from the early 1920s until the 1950s, LB was considered *the* essential bacteria causing caries. It was not until 1954 when the gnotobiotic[f] studies of Orland demonstrated that if rodents living in a germ-free environment were infected with a lactic acid-producing *enterococci* (but *no* LB), they still developed caries.[24] This was the first time that it was *known* that LB were not requisite for caries development. Often, the number of lactobacilli isolated from either saliva or plaque was too low in number to be considered capable of producing the range of pH values required for caries initiation.[41] However, once a caries lesion develops, the stability of the immediate plaque population changes rapidly. The low pH environment of LB often eliminates, or at least suppresses the continuity of colonization of MS.[42] This, despite the fact that some organisms such as MS probably have genetic defensive mechanisms to minimize the effects of a low pH.[43]

This phenomenon of a lowering pH resulting in MS being displaced by LB, is seen following irradiation for head and neck cancer, when extensive, multiple, caries lesions develop rapidly because of the destruction of the salivary glands.[44] During the *initial* phases of the developing carious lesions, large numbers of *MS* are involved, only to *decrease* later in number as the LB population *increases*. This is believed to be caused by LB creating a sufficiently low pH to establish a monopoly of the environment.

[f]Gnotobiotic = In this use, the animals were raised in a *sterile* environment.

Adherence

Continuous *adherence* to the solid tooth surface by *S. mutans* is necessary both before and after initial colonization. The first bacteria must establish a foothold on the tooth surface (acquired pellicle) and then maintain their positions while other bacteria continue to colonize in other protected areas offered by the interproximal spaces,

along the gingiva, or in the pits and fissures. Otherwise they would be swept away by the saliva.

Mutans streptococci are able to attach to the tooth surface by either of two mechanisms:[26,27,45] (1) attachment to the acquired pellicle through extracellular proteins (*adhesins*) located on the fimbriae (fuzzy coat) of these organisms; and (2) sucrose-*dependent* mechanisms, in which bacteria *require* the presence of sucrose to produce sticky *extracellular polysaccharides* (*glucans*), that allows attachment and accumulation of additional waves of bacterial colonization.[46]

Sucrose is a disaccharide, consisting of one glucose and one fructose unit (moieties). One of the key enzymes in the conversion of the glucose moiety of sucrose to glucan is *glucosyltransferase*. At times the enzyme may be altered, resulting in the production of a *soluble* glucan that does not support adherence. These mutant strains that form *soluble* glucan are usually noncariogenic.[47]

The effect of sucrose restriction on glucan production is seen in several clinical situations. Children who consume little or no sucrose because of sucrase or fructase enzyme deficiencies have a less cariogenic plaque. Similarly, patients receiving long-term nourishment via stomach tube have less plaque and fewer MS.[48] Individuals restricting their sucrose intake have a decreased proportion of MS in their plaque, but the MS increases when sucrose is reintroduced into the diet.[49] Dietary restriction of sugar has also been shown to reduce the acidogenicity of dental plaque.[50,51]

Ecology of Caries Development

Several studies support the possibility that the initial colonizers can help to determine the eventual pathogenicity of the plaque.[37] Once a species of bacteria has established its *ecologic niche*,[8] other bacteria introduced at a later date appear to have a more difficult task in colonizing. Once established, a niche can be long-lasting. For instance, children with the highest number of MS for deciduous teeth usually experience a higher attack rate for the later permanent teeth.[52]

Mutans streptcoccoi require a *solid surface*—the tooth surface—for successful colonization. During the first year of life *before* eruption of the primary teeth, very few MS are found in the mouth.[53] When teething begins at approximately 8 months, MS often rapidly colonizes the plaque of newly erupting teeth.[54] It has been shown that an important source of infection of infants by MS is from the *caregivers (usually the mother)* by the mouth-to-mouth transmission, such as via kissing, or by sharing a spoon during feeding.[55] Mothers with the highest MS counts often have infants with similarly high caries lesion counts.[56] Since early infection by MS is associated with high decay rates,[57] it has been strongly suggested that an effective means of preventing caries in young children would be to reduce the number of MS in the parents' and siblings' mouths before a child's birth.

Because no entrenched competition from other organisms occurs on eruption, the first bacterial colonizers probably have little difficulty in establishing their ecologic niches on the acquired pellicle and in the saliva. Once the teeth erupt, many of these oral reservoirs of bacteria participate in the formation of the plaque. Each firmly established niche can act as a "seeding" area for other areas of the mouth. Mutans

streptcoocci decrease in number as teeth are lost throughout life and practically disappear following full-mouth extraction.[58] After dentures are inserted, *S. mutans* reappear, only to disappear again when the dentures are removed for an extended period.

[g]An area in the plaque where specific species of bacteria are relatively safe from host protective function of saliva and from other antagonistic bacteria.

Question 2

Which of the following statements, if any, are correct?

A. Streptococcus mutans can be expected in increased numbers at the site of an incipient lesion.

B. Lactobacilli are usually found even earlier than mutans streptococci at the incipient lesion site.

C. Soluble glucans foster better bacterial adherence than insoluble glucans.

D. Caregivers can be a child's worst dental friend.

E. The mutans streptococci require a solid surface on which to colonize.

Coronal Dentin Caries

It is now desirable to revisit the embryology of the tooth,[59] starting at the dentoenamel junction (DEJ) when the ameloblasts and the odontoblasts were lined up at the future DEJ. The objective of the ameloblasts was the future surface of the tooth, while the objective of the odontoblasts was the future border of the dental pulp. During the period of tooth formation, each day the odontoblast laid down a trailing odontoblastic process and a concentric increment layer of *predentin*. Each succeeding day the predentin became a calcified layer of dentin forming a tubule around the odontoblastic process, the lining of which is a *hyper*calcified layer called the *peritubular dentin*. These tubules extended from the DEJ to the dental pulp, in fact a few extended into the enamel as *enamel spindles*.

Between the tubules there is *intertubular* dentin (also called mantle dentin).[60] The tubules contain fluid that originates from the pulp chamber. There is intertubular communication and fluid transport, via *secondary tubules* and smaller sized *canaliculi*. All tubules act as channels for the *convection*[h] flow of fluids that flow *out*ward from the pulp.[61] Dentinal fluid is constantly pumped into tubules by the forces of mastication, with a return of the fluid to the pulp upon release of the pressure.[62] When there are infection products (caries) arriving in the tubules, more fluid is forced into the tubule.[63,64] The pulp fluid also contains important calcium, phosphate and secretory immunoglobulin A.[64,65]

Upon the approach of enamel caries to the DEJ, many of the odontoblastic processes underlying the carious enamel interrod areas of the enamel will lose their vitality. These tubules become *dead tracts* and may begin to partially or wholly calcify The

complete calcification results in a hard calcified group of tubules, called *sclerotic dentin* that acts as a protective barrier to the advancing caries. At the same time, the odontoblasts located on the periphery of the pulp are triggered to begin laying down increments of amorphous *reparative dentin* to further protect the pulp.

In summary, the millions of diffusion and convenction channels in the enamel and dentin respectively, permit a movement of fluid from the tooth surface to the pulp.[66,67] The intertubular secondary canals and the canaliculi provide permeability within the dentin, whilst the DEJ provides the same lateral fluid (acid) mobility that can undermine the enamel and aid in its collapse to form an overt caries lesion. It should be pointed out that even with a visible x-ray lucency that extends into the dentin, if the originating surface zone has not broken down into an overt cavity, the entire pre-caries lesion, even in deep dentin can on the basis of in vitro studies, theoretically (and slowly), be remineralized.[68]

[h]The fluid entering the tooth from the surface is said to *diffuse* inward; fluids arising from the pulp are said to be *convection fluids*.

Root Caries

A general demographic shift is continually occurring in America, with each successive generation living longer. This provides a longer time for more gingival recession and more root caries. In addition, Seniors are consuming an increasing number of medicines that are known to reduce saliva and cause root caries.[69] Katz and colleagues estimated that individuals going into their 30s have about 1 out of 100 surfaces with recession and root caries; when they leave their 50s, about 1 out of 5 exposed surfaces is involved. The roots of the mandibular molars and the mandibular incisors are at the greatest, and the least risk, respectively.[70]

The Third National Health and Nutrition Examination Survey found that the percentage of persons with at least one decayed or filled root surface increased from 20.8% in the 35- to 44-year age group, to 55.9% in those aged 75 years and older.[71] A Canadian study concluded that "the increase in the prevalence of root decay with age may not be due to aging per se, but instead, may be the result of neglect of oral health during the years of growing older." Older adults with continual good oral health still had low rates of root decay.[72] In a study of 5000 subjects in Finland, it was found that men had from 1.1 to 2.5 times more root caries than women. The greatest difference was in the group 60 to 69 years of age.[73]

A number of risk factors have been defined for root caries development, including age, gender, fluoride exposure, systemic illness, medications, oral hygiene, and diet.[74] In terms of the microbiology of root caries, despite early indications of a strong association between Actinomyces species and progressive root lesions,[75,76] more recent studies indicate that plaque and salivary concentrations of the mutans streptococci are correlated positively with the presence of root surface caries.[77,78]

Root caries differs from coronal caries in several aspects. A critical difference is that the tissues affected—enamel vs. cementum—are fundamentally dissimilar. Enamel is much more highly mineralized than cementum or dentin. Because of the *lower mineral* content and *higher organic content* of the cementum-dentin complex, root

caries may progress *both* by *acid demineralization of the inorganic structure* and by *proteolysis of the organic component*.[79] These tissue variations determine the differences in the rate of lesion formation, histologic and visual appearance, as well as in the potential for and rate of remineralization.[80] Clinically, the lesion is initially noncavitated. The carious material is soft and has a yellowish-brown coloration. The lesion can eventually assume any outline and may involve multiple root surfaces (Figure 3-8). When cavitation is evident, lesions tend to spread laterally, have a depth of approximately 0.5 to 1.0 mm, and are of a dark-brown appearance.[81] The lesions appear immediately *below* the cemento-enamel junction, undermining but *not* involving the enamel (Figure 3-8).

Root caries differ from coronal caries in that *bacterial invasion* of cementum and dentin occurs *early*. At times, the invasion features columns of organisms between spikes of relatively intact cementum. At other times, a complete loss of cementum exposes the dentin. Like enamel caries, root caries is amenable to remineralization and/or arrest.[80] Arrested root caries lesions demonstrate three physical characteristics: (1) an *outer* barrier of hypermineralized surface dentin; (2) a sclerotic *inner* barrier between carious and sound dentin; and (3) mineralization occurring *within* the dentinal tubules.[82] Clinically, such remineralized lesions may appear dark and hard; under tactile examination by the explorer, arrested lesions are easily distinguished from active lesions by their smooth, hard, and glassy feel compared to the leathery feel of active root caries.

Prevention of Root Caries

The best prevention for root caries in the elderly population *is the prevention of periodontal disease in middle-age or earlier*. However, since ex post facto[i] remedial treatment is not possible, earlier preventive dentistry care needs to be practiced. The strategies include: (1) daily *mechanical and chemical plaque control* (2) *severe restriction of refined carbohydrates*; and (3) routine professional dental attendance for preventive office identification of risks and counseling on self-care needs. For instance, for those at high risk, a *prescription* dentifrice with a high-fluoride content, Prevident, with 5,000 parts fluoride per million, has been found to significantly increase the electrical resistance[j] of a tooth surface.[83] Frequent professional examinations based on an individual's risk should be routine. All extensive periodontal surgery for pocket elimination should place an individual in a higher root caries risk category for life.[84,85] In later chapters, the use of the antimicrobial mouth rinse, *chlorhexidine*, will be introduced as a very effective mutans streptococci control agent. With professional guidance and patient cooperation, biological repair of a root caries lesion can be achieved in many cases—a desirable option in view of the difficulty and lack of success in restoring root caries via operative procedures.

[i]Ex post facto = After the fact, meaning the it is not possible to correct some events of the past.

[j]Several devices are on the market that are modified versions of the common volt-ohm meter. The patient holds one electrode, while the explorer serves as the second electrode. When the explorer is placed on the suspect area, the resistance of the tooth is measured. A high resistance is associated with no caries, and little resistance is associated with caries probability.

Secondary, or Recurrent, Caries

Secondary caries start with small imperfections or restoration overhangs that exist between the tooth and the margins of a restoration.[86] Also, some tooth-colored fillings have a higher affinity for plaque.[87] Bacteria are able to colonize and multiply at these vulnerable sites, sheltered from the protective effects of saliva and self-care efforts. Eventually, a lesion develops between the cavity margin and the restoration.

The diagnosis of these lesions is difficult.[88] In one study, extracted teeth were cut so that the section included both a clinically *sound amalgam margin* and one defined as "*ditched*." The prevalence of recurrent lesions in both sound and ditched restorations was close to 50%, although it is unknown whether these lesions were truly recurrent or due to residual caries left during a previous cavity preparation.[89] The magnitude of the problem of secondary decay is illustrated by studies indicating that the median survival time of restorations ranges from *5 to 10 years*.[90] Replacement of defective restorations account for inserting several-times-more than needed restorations over a life time.[91] Reducing this problem can best come from preventing the number of primary lesions (primary prevention). Some future relief may be forthcoming from the use of materials that bond directly to the tooth tissue, eliminating the gap between tooth and filling, or from restorative materials that slowly release fluoride, such as glass ionomers and newer fluoride-releasing composites and amalgams.[92,93]

> Figure 3-8 Root caries. The darker staining of the coronal half of the root indicates considerable gingival recession, which is a prerequisite to lesion development.

Question 3

Which of the following statements, if any, are correct?

A. The critical pH for enamel demineralization ranges between 6.0 to 5.5.

B. There cannot be secondary caries, without having had a previous incipient lesion at the same site.

C. Once the incipient lesions become overt, all the lesion zones disappear.

D. Root caries is not necessarily a part of the aging process but is usually a sign of periodontal disease and/or previous periodontal neglect.

E. A dentist usually inserts more restorations as a result of secondary caries than for primary caries.

Measuring Plaque pH, the Stephan Curve

Every time a person eats a food, there is a continuous pH change in the plaque. In many studies, pH microelectrodes have been inserted in bridges and telemonitored to determine these changes. For sugar and sugary snacks, an almost immediate drop in pH occurs, followed by a longer recovery period. This drop and recovery curve has been termed the *Stephan Curve* after Dr. Robert Stephan, an officer in the United

States Public Health Service who first reported on the continuous changes in pH that followed eating and drinking different foods and drinking different beverages.[94] Plaque pH responses to simple sugar rinses by caries-free and caries active individuals exhibited different drops in pH and different lengths of time to return to normal. Thus, different individuals have different capabilities to buffer acid production. (Figure 3-9). Similar pH studies have been accomplished that identify foods that are not hazardous to the teeth, and vice versa, those that are accompanied by a drop past the "critical pH" of pH 5.5 to 5.0. These lists are of considerable value when counseling patients.[95]

The Relationship of Saturation to pH

The concentration of calcium and phosphate ions in the *plaque fluid* bathing the tooth at the plaque-tooth interface is extremely important, because these are the same elements that compose the hydroxyapatite crystal. *If the fluid adjacent to the tooth is supersaturated with calcium and phosphate ions at a given pH, the enamel cannot undergo demineralization*.

The saliva in contact with the teeth is normally supersaturated with respect to the calcium and phosphate in enamel.[15] The bacterial plaque can concentrate these ions to an even greater extent. For instance, both calcium and phosphate are threefold times greater than in the saliva.[96] This increased concentration is of practical importance because calcium and phosphate levels tend to be inversely related to the caries score.[47] It is also of great importance because it is the *plaque fluid that determines the eventual caries status*.

As the pH drops in an acid attack, the level of supersaturation also drops, and the risk of demineralization increases. There is no exact pH at which the demineralization begins, only a general range of 5.5 to 5.0. The range is rather large because demineralization is a function of both pH and duration of exposure of the enamel surface to the acid environment. Different plaques have different initial pHs, buffering potentials, and concentrations of calcium and phosphate in different parts of the mouth. A change in any of these variables results in a different level of supersaturation in the tooth environment.

De- and Remineralization of Teeth, Principles

Throughout this book, there will continue to be many references to de- and remineralization of teeth, both as a pathologic and as a therapeutic process. The demineralization is caused by *plaque acids* causing the dissolution of the tooth minerals making up the basic calcium, phosphate, and hydroxyl crystals of the enamel, dentin and cementum. Remineralization on the other hand, requires the *availability of the same ions, preferably with fluoride as a catalyst* to reconstruct the missing or damaged rods—a process that ten Cate aptly calls, *non-restorative repair*.[97]

There are many calcium and phosphate compounds in the body that vary in formulae and with changes in pH. However, at this time, for the sake of simplicity, the crystals and fluoride compound of most dental interest in the de- and remineralization process are hydroxyapatite (HAP), fluorhydroxyapatite (FHA), and calcium fluoride (CaF_2).

The long-term exposure of teeth to *low concentrations* of fluoride (as found in fluoridated water) results in the gradual incorporation of fluoride into the existing hydroxyapatite (HAP) crystals to form fluorhydroxyapatite (FHA) that is more resistant to acid damage. Conversely, a *higher* concentration of fluoride (as occurs in topical applications, use of fluoride dentifrices, gels, and varnishes, etc.), results in the formation of *surface globules* of calcium fluoride (as seen in electron microscope images). A subsequent coating of these globules by phosphates and proteins of the saliva renders these globules more insoluble.[98] As a matter of terminology, when the fluoride is incorporated into HAP to form FHA, it is said to be firmly bound; whereas, loosely bound fluoride is in the form of calcium fluoride that is *adsorbed onto* the surface of HAP and FHA crystals.[99] See Figure 3-10.

The Relationship Between HAP, FHA and CaF_2

Following an attack by plaque acid(s), the CaF_2 dissolves first, followed in sequence by the HAP, and finally, the FHA (with its fluoride substitutions). As the attack continues, the dissociated ions *increase the saturation level of the immediate fluid sufficiently to slow crystal dissolution, and eventually arrest* further solution of the crystals. As the pH begins to return to normal, crystals begin to *re-form* from the complex *pool* of dissolved ions—some as HAP, some as FHA (with many of the fluoride ions coming from the previous CaF_2, and finally the precipitation of newly adsorbed CaF_2. Any deficiencies are subsequently replaced in time by calcium, phosphate, and fluoride from sources such as the saliva, water, and toothpastes. In observing the above process, one must marvel at the body defense system that in the absence of a cellular or humeral surveillance of the enamel, can use a chemical system to maintain homeostasis—one in which *CaF_2 provides a reservoir for fluoride that is immediately available when and where it is needed*.[100] The only time the system breaks down is when the attacks are too frequent and too prolonged.

Depth of Remineralization

There is little controversy about the success of surface remineralization procedures involving topical procedures, and of using commercial fluoride products such a dentifrices, gels, and varnish to compensate for the daily wear and tear of demineralization (Chapter 9). In the New Zealand School System, they consider x-ray lucencies of incipient lesions that extend midway through the enamel as candidates for remineralization. ten Cate, in an *in vitro* study, found that both the inner enamel and dentin could be remineralized, but very slow. Only the *outer part* of the enamel appeared to be responsive to fluoride diffusion and remineralization.[97] At deeper levels, remineralization could be achieved, but only very slowly. In Scandinavia, the literature reflects the belief that remineralization is a reasonable objective even for lesions reaching to the dentin. The test for remineralization in these cases is that there is no demonstrable caries progress for 2 to 3 years. However, the important fact is that there are no reported studies that indicate whether deep remineralization is or is not successful.

Methods of Varnish Application

In the United States, topical administration of fluorides is usually via cotton applicators, gel trays, and less frequently by using varnish. (Chapter 9). In Europe,

varnishes appear preferable because of the longer exposure to fluoride following application. Since varnishes *do* seal dental tubules involved in hypersensitivity,[101] there is a possibility that they also temporarily seal pores as seen in Figures 3-7A and 3-7B. Once sealed, there could be little or no acid penetration into the "white spot."

At least three commercial varnishes are available in North America—Duraphat (Colgate-Palmolive, NY), Duraflor (Pharmascience, Montreal), and Fluor Protector (Ivoclar, Viyadent, Amherst, NY). The U.S. Food and Drug Administration (FDA) has cleared varnishes for applying fluoride varnish—but only as medical devices to be used as cavity liners and desensitizing agents, not for caries control. Semiannual applications are the most accepted time interval.[102] Supporting this time interval was Seppa's study in Finland where increasing the application interval from two, to four times per year *did not* increase the effectiveness of Duraphat, even in high-risk children.[103]

Varnishes have proved to be effective. One study of 142 2- to 3-year-old children was conducted to determine the anticaries effectiveness of Duraphat. At the end of 9 months, 37.8% of the originally active occlusal, lingual, and buccal lesions of the *control* group became inactive, 3.6% had progressed, and 36.9% did not change. For the *Duraphat group*, 81.2% became inactive, 2.4% progressed, and 8.2% did change (P >. 0001). The author concluded that the use of varnish was easy, safe and efficient; that it was possibly a non-invasive alternative for the treatment of decay in children.[104]

The application of the varnish is preceded by a prophylaxis, flushing, isolating the target teeth, drying, and applying the varnish with a small brush—techniques that are well known and practiced by the *dental hygienist*.

Figure 3-7 A Scanning electron micrograph of dentinal tubules. The tubules are approximately 1.5 um in diameter and surrounded by a highly mineralized collar of peritubular dentin. The matrix between the tubules and peritubular dendentin is the intertubular matrix and consists of bundles of collagen fibrils running in a plane at right-angles to the long axis of the tubules. Mineral crystals are also found aligned along the collagen fibril. (Silverstone, L. M. & Hicks, M. J. The structure and ultrastructure of the carious lesion in the human dentin. *Gerodontics* 1985;1:185-93.)

Figure 3-7 B Scanning electron micrograph showing the zone of demineralization in the body of a lesion in dentin caries. This region is bacteria-free and shows evidence of acid dissolution, especially in the peritubular denin. The sclerosed tubular contents of the translucent zone have also been lost as a result of dissolution. (Silverstone, L. M. & Hicks, M. J. The structure and ultrastructure of the carious lesion in human dentin. *Gerodontics* 1985:1:185-93.)

Figure 3-9 Stephan curves. These curves show the typical plaque pH response to an oral glucose rinse (indicated by the screened area). There is an immediate fall in the pH, followed by a gradual return to resting values after about 40 minutes. Each curve represents the mean of 12 subjects; the pH was measured by sampling method (see Chap. 15) and therefore is an average value for the whole mouth plaque pH. In individual sites away from the salivary buffers, the pH values may fall close to 4.0. The upper curve was obtained from

reconstituted skim milk and the lower one from an apple-flavored drink, showing a large difference in the acidogenicity of these two drinks. (Courtesy of MWJ Dodds, University of Texas Dental School, San Antonio.)

Figure 3-10 Electron micrograph. Loosely bound calcium flouride globules on the surface of the enamel following an application of sodium flouride. These reaction products following the flouride application will be dissolved in two or three weeks into the saliva. Each time an application of flouride dentifrice or mouth rinse occurs, this pattern of globule distribution is repeated, with the extent depending on the specific flouride and concentration. (Courtesy, Dr. M.J. Hicks, Texas Childrens Hospital, Houston, TX. Magnification 5000X)

Question 4

Which of the following questions, if any, are correct?

A. After examining the Stephan Curve recorded for several foods, it is possible to determine which foods and snacks are hazardous to tooth health.

B. As the pH of the plaque fluid falls, it is necessary to have an increasing amount (saturation) of calcium and phosphate in the plaque fluid to prevent the dissolution of the tooth mineral.

C. As the pH drops past the critical pH for enamel dissolution, the dissolving crystals gradually increase the immediate concentration (saturation) of tooth minerals that gradually slow, and possibly arrest the further solution of the rod crystals.

D. It requires a lower critical pH to dissolve a crystal that is in the fluid environment of dissolving CaF_2.

E. The many studies of "deep remineralization" provide adequate scientific (evidence based) verification that it is a valid means to manage incipient lesions where the body of the lesion has progressed past the mid point in the enamel.

Summary

Dental caries is a multifactorial disease involving an interaction of bacteria, diet, host resistance, and time. Cavitation can only occur when demineralization outstrips the body's defensive capability for remineralizations over a period of time. The embryology and histology of the enamel are favorable for either the de- or the remineralization of the enamel. The residual matrix and spacial relationships of rod-to-rod and crystalite-to-crystalite, as well as the less-calcified structures as the incremental lines of Retzius, lamellae, and tufts, allow fluids to diffuse throughout the enamel. Like the wick of an oil lamp, this network is available for the in-and-out movement of tooth-mineral ions and plaque acids. Even when there has been a penetration of the enamel cap by an incipient lesion, this is a pre-caries lesion that can often be remineralized without the need for a restoration. Possibly months or years will elapse before cavitation, or there may even be a natural remineralization that entirely reverses the caries progression. There are several acidogenic bacteria that are causal for caries production, with mutans streptococci and lactobacilli being the most studied. Silverstone opened up the possibility of a new nonrestorative repair era when

he described the de- and remineralizing zones of an incipient lesion. If those in the dental care profession and research can bring remineralization to fruition, millions of teeth can be saved from the dentist's drill. The polarizing and the electron microscopes allow us to see the details of how the plaque acids can easily flow into the body of the lesion and beyond. To increase tooth resistance and, at the same time, the probability of remineralizing any known or unknown incipient lesions, mechanical plaque control strategies consisting of tooth brushing, flossing, and irrigation are used to remove the plaque. Chemical plaque control stratagems involve the use of antimicrobials to kill or suppress the cariogenic bacteria; and fluoride in the forms of water fluoridation, office topical applications, or the use of fluoride rinses or dentifrices are used to improve tooth resistance. There are now the means to greatly reduce the toll of dental caries; yet needed is access to examination and treatment systems based on early identification and treatment of risk factors before they become treatment requirements. Throughout this book, emphasis will be placed on the various strategies now available for preventing or limiting demineralization, or of enhancing remineralization.

Answers and Explanations

1. A, B, and C—correct.

D—incorrect. The interproximal starting point is apical to the contact point; for the pit-and-fissure lesion, it usually begins bilaterally at the orifice of the fissure.

E—incorrect. The dark and the surface zones are the centers for remineralization; the body of the lesion and the translucent zones are centers for demineralization.

2. A, D, and E—correct.

B—incorrect. The MS usually precede the lactobacilli.

C—incorrect. The bacteria-producing soluble glucans often are noncariogenic because of adherence problems; the insoluble glucans are usually produced by the cariogenic bacteria and facilitate adherence.

3. B, D, and E—correct.

A—incorrect. The critical pH for enamel demineralization is from 5.5 to 5.0.

C—incorrect. The same zones are present but are less clearly defined because of the presence of bacteria, plaque, and debris.

4. A, B, C and D—correct.

E—incorrect. No studies to date indicate that "deep remineralization" by use of fluoride therapy is or is not an appropriate method of caries control. There is a theoretical basis, much research, and a plethora of hope and enthusiasm for this approach to nonrestorative "repair" of teeth. (What sugar hath rendered asunder, humankind is now laboring to correct!)

Self-evaluation Questions

1. In 1890, Miller proposed the _____ theory for caries, which is still (with many modifications) a basis for our present concept of the dental caries.

2. The beginning and end-points of a carious lesion are the _____ (initial) lesion, which can be arrested or reversed by remineralization therapy, and the _____ (end point) lesion, which must be restored.

3. The four zones of an incipient lesion seen with the polarizing microscope (starting from the tooth surface) are the _____, _____, _____, and the _____ zones.

4. The zone of the incipient lesion that is the best indicator of remineralization is the _____ zone; the two zones of demineralization are the _____ and the _____ zones.

5. As the pH drops in the environment of the HAP, the sa _____n of calcium and phosphate in the environment must increase in order to protect the crystals. The presence of _____ (element) will also help to protect the crystal at a lower pH.

6. The critical pH for enamel demineralization is within the generally accepted range of pH _____ to _____.

7. The diagramming of the drop and recovery of pH on a graph is often referred to as the _____ curve for the investigator who first published on the phenomenon.

8. Two possible sources of the calcium and phosphate accounting for the hypermineralized surface of root caries are _____ and _____.

9. The four major types (location) of caries are: _____, _____, _____, and _____.

10. Two causes for rampant caries are _____ (dietary "food") and _____ (dry mouth).

11. The pore space in both the translucent and surface zones is 1 percent; dark zone approximately ____ percent, and the body of the lesion ranges up to ____ percent.

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Chapter 5. Toothbrushes and Toothbrushing Methods - *Samuel L. Yankell Ulrich P. Saxer*

Objectives

At the end of this chapter it will be possible to:

1. Give a brief history of the toothbrush, describe its parts in detail, and explain why there is no one "ideal" brush.

2. Compare natural and nylon bristles for their uniformity of length, diameter, and durability.

3. Discuss the wide range of head and handle designs and explain why there are many "new" manual and powered toothbrush products being marketed.

4. Compare and contrast laboratory and clinical evaluations of toothbrush effectiveness.

5. Compare manual and powered toothbrushes for effectiveness and safety.

6. Compare the ADA process for evaluating "standard" and "new" manual toothbrushes.

7. Discuss modifications of toothbrushing methods applicable to special patient care, patients using prostheses, and those under orthodontic care.

8. Discuss interproximal access of different toothbrushes and their possible role in oral-disease treatment and prevention.

Introduction

After teeth have been completely cleaned by the dental professional or by the individual, soft microbial dental plaque continually reforms on the tooth surfaces. With time, plaque is the primary agent in the development of caries, periodontal disease, and calculus—the three conditions for which individuals most often seek professional services. If plaque, particularly at interproximal and gingival areas, is completely removed with home-care procedures, these dental-disease conditions can be prevented. Unfortunately, the majority of the population is unable, uninstructed, or unwilling or does not realize the need to spend the time to remove plaque from all tooth surfaces, and/or the product(s) used are not adequate to remove plaque at critical sites. Plaque deposits can be removed either mechanically or chemically. The focus of this chapter is the mechanical removal of plaque, using toothbrushes and toothbrushing techniques. The following two chapters emphasize the use of products and auxiliary aids with toothbrushes in removing plaque and the maintenance of healthy teeth and gingival tissues.

The Manual Toothbrush

History

Hirschfeld, in his 1939 landmark textbook on the toothbrush and oral care, included an in-depth review of the history of toothbrushing.[1] The exact origin of mechanical devices for cleaning teeth is unknown. Ancient peoples chewed twigs from plants with high aromatic properties. Chewing these twigs freshened the breath and spread out fibers at the tips of the twig for cleaning the tooth and gum surfaces. The Arabs before Islam used a piece of the root of the arak tree because its fibers stood out like bristles; this device was called a siwak. After several uses, the bristle fibers became soft, and a new "brush" was created by stripping off the end and making new bristle fibers. In the seventh-century, Mohammed made rules for oral hygiene, and so it became a religious obligation. To this day the siwak, composed from aromatic types of wood, is still used. Chew sticks not only help to physically clean teeth but also, because they contain antibacterial oils and tannins, may help prevent or remove plaque.[2]

The Chinese are credited for inventing the toothbrush comprising a handle with bristles during the Tang dynasty (618-907 A.D.). They used hog bristles similar to those in some contemporary models. In 1780, in England, William Addis manufactured what was termed "the first modern toothbrush."[3,4] This instrument had a bone handle and holes for placement of natural hog bristles, which were held in place by wire. In the early 1900s, celluloid began to replace the bone handle, a changeover that was hastened by World War I when bone and hog bristles were in short supply. As a result of the blockade of high-quality natural hog bristles from China and Russia during World War II, nylon bristles were used instead. Initially, nylon bristles were copies of natural bristles in length and thickness. They were stiffer than natural bristles of similar diameter. They did not have the hollow stem of natural bristles and, accordingly, did not absorb water. Compared to natural bristles, nylon filaments have the additional advantages that they can be prepared in various uniform diameters and shapes, and can be end-rounded to be more gentle on gingival tissues during the brushing procedure. In 1924, an American dentist reported on 37 different manual toothbrushes with regard to handle shape, head design, bristle type, length, and width. Individual dentists disagreed then, and still do today, on what type of toothbrush was best. The primary toothbrush shapes marketed in the 1940s through 1980s in the United States had flat, multitufted toothbrush-head shapes. Since the 1990s new manual toothbrushes have been introduced with new shapes, sizes, colors, and claimed advantages. By varying the length and the angle of the filaments in the brush head, brushing with these newly designed products has been documented to improve plaque removal since the bristle filaments can be directed into the sulcus or interproximal areas.[3-12] New unconventional toothbrushes with two or more heads or segments of filaments in angular relation- ship have shown improved plaque removal. One new brush with three heads can be used to simultaneously clean the buccal, occlusal, and lingual surfaces.[13-15] The proliferation of brushes can be attributed, in part, to advances in manufacturing, for example, the attachment of bristles into the handle using molding techniques rather than stapling to allow a wider flexibility in toothbrush designs and bristle angulations. In addition, toothbrush bristles are now available in a variety of colors, textures and shapes.

There also has been an increase in both the quality and number of laboratory and

clinical research studies on toothbrushes. The International Association of Dental Research and American Association of Dental Research are major meetings for both academic and industry scientists to present their latest research. In the 1991 and 1992 key-word indexes of the abstracts accepted for presentation at these meetings, toothbrushes were not included as a topic. In 1993 the number of abstracts were ranked as dentifrices > mouthrinses > toothbrushes. Since then, through 2001, the number of dentifrice abstracts has shown marked increases or decreases, with a peak of over 90 abstracts in 1998. Mouthrinse abstracts have shown essentially a leveling-off or a slight decrease in number since 1991. Toothbrush abstracts have continued to demonstrate a consistent increase, and at the 2001 AADR meeting, exceeded dentifrices and mouthrinses.

With the scientific reports about toothbrush contamination after oral or medical bacterial/ viral infections, dental professionals recommend replacing toothbrushes at 3- to 4-month intervals, so repeat purchasing of toothbrush products is done more frequently. The increase in toothbrush sales may be an additional driving force for the marketing of new designs and variety of toothbrushes. Toothbrush pricing has reached new highs with the introduction of "high-tech" manual toothbrush designs and stronger claims, yet the cost per individual product is generally less than the cost for a "family-size" tube of toothpaste or mouthrinse. Toothbrush shipping costs are less, breakage is minimal and the shelf-life (stability) is longer than for other product categories thus, the potential profitability of toothbrushes to the manufacturers may be greater than for dentifrice or mouthrinse products.

Question 1

Which of the following statements, if any, are correct?

A. The toothbrush became commercially available in the United States just before the Civil War; the celluloid handle became popular during World War I; and nylon bristles appeared just before World War I.

B. While toothbrush-head designs have changed considerably in the past decade, toothbrush bristles shapes have remained essentially the same.

C. The cross section of the average toothbrush in the United States, prior to the 1990s, had a flat head and a flat bristle profile.

D. Nylon bristles are more firm or stiffer than natural bristles with the same diameter.

E. In the 1990s toothbrushes have been the subject of a steadily increasing number of laboratory and clinical research studies.

Manual Toothbrush Designs

Manual toothbrushes vary in size, shape, texture, and design more than any other category of dental products.[5] A manual toothbrush consists of a head with bristles and a handle (Figure 5-1). When the bristles are bunched together, they are known as tufts. The head is arbitrarily divided into the toe, which is at the extreme end of the head, and the heel, which is closest to the handle. A constriction, termed the shank,

usually occurs between the handle and the head. Many toothbrushes are manufactured in different sizes—large, medium, and small (or compact)—to adapt better to the oral anatomy of different individuals.[5,7] Toothbrushes also differ in their defined hardness or texture, usually being classified as hard, medium, soft or extra soft. Descriptions and measurements of selected U.S. toothbrushes are shown in Table 5-1.

Much of the early data comparing the efficacy of various toothbrush designs is contradictory because of (1) the lack of quantitative methods used to measure cleaning (plaque removal), (2) the many sizes and shapes of toothbrushes used, and (3) the lack of standardized toothbrushing procedures used in the studies. More recently, toothbrush heads have been altered to vary bristle lengths and placement in attempts to better reach interproximal areas. Handles have also been ergonomically designed to accommodate multiple dexterity levels. As described in the introduction, the change from the old flat toothbrush to multilevel designs was possible because of new bristle technology and manufacturing procedures.

Profiles

When viewed from the side, toothbrushes have four basic lateral profiles: concave, convex, flat, and multileveled (rippled or scalloped). The concave shape can be useful for improved cleaning of facial surfaces, whereas convex shapes appear more useful for improved cleaning of lingual surfaces.[5] Lateral and cross-section profiles and the overhead appearance of selected toothbrushes commercially available in the United States are shown in Figures 5-2, 5-3, and 5-4. In laboratory and clinical studies, toothbrushes with multilevel profiles were consistently more effective than flat toothbrushes, especially when interproximal efficacy was monitored.[6,8,11,16,17]

Bristle Shapes

Recently, new toothbrush bristle shapes and textures have been fabricated, as shown in Figure 5-5. Toothbrush products utilizing these bristles in multiple diameters, textures, and bristle trims have been developed, and laboratory studies have documented improved efficacy of toothbrushes with tapered, feathered and diamond-shaped bristles, compared to toothbrushes with standard round bristles. [18-20]

End-rounding

Originally, individual toothbrush bristles were cut bluntly and often had sharp end configurations. In 1948, Bass reported that these bristle tips could damage the soft tissues and that rounded, tapered, or smooth bristle tips were less abrasive.[21] Although Bass's research was not performed according to strict research protocol, his findings have remained undisputed for more than 40 years. Indeed, advertisers still recommend end-rounded tips for safety and to promote toothbrush sales. When toothbrushes are examined under low magnification, most bristles labeled as "rounded" do in fact appear smooth or end-rounded. However, at higher magnification, as shown in Figure 5-6, many of these "rounded" bristles take on different configurations.[5] During use, bristles become smoother and more end-rounded. With continued use, the bristles of the tuft expand and spread out.[22] Bristle wear has been shown to vary directly with the toothbrushing load and amount of dentifrice and inversely with bristle diameter.[23] In a recent study, there were no significant differences in plaque or gingivitis indices in a

group in which toothbrushes were replaced on a monthly basis compared to the second group using their same toothbrush over the 3 month period. The toothbrushes used for 3 months exhibited a significant increase in the wear index compared to the baseline values.[24] A 1988 scanning-electron microscope study[25] compared end-rounding of bristles from eight marketed types. Based on statistical analysis of 30 toothbrushes of each type, acceptability varied from 22 to 88%, indicating to these authors that some brushes are not sufficiently rounded and are likely to produce gingival damage. In addition, they have abrasive potential on dentin and cementum.

A 1992 study[26] compared a ripple design with a flat-profile brush using a stereoscopic microscope with fiberoptic lighting. Close to 90% of the bristles of the ripple brush were end-rounded, whereas the flat brush had an average of 52% rounded bristles. Apparently, the degree of end-rounding depends on a manufacturer's specifications and not on toothbrush design.

In a study conducted in 2001 on 31 different toothbrushes, only 4 products had more than 50% of the filaments rounded; in 19 products, end-rounding was 12 to 40% and only 0 to 7% in 8 brands. The authors concluded that a large percentage of marketed toothbrushes do not meet acceptable end-rounding criteria.[27] If bristles are cut, frayed, or are hollow they can harbor bacteria, viruses, and other potential periopathogens, especially if no dentifrice is used, and they can transfer these into and around the mouth.[28]

Handle Designs

Many of the new toothbrushes in the United States have a styled-handle design. Modifications, such as triangular extrusions or indentations along the sides for a better grasp, a "thumb position" on the back of the handle for more comfort, and various angle bends to permit better access into and around the mouth, have been introduced. Four toothbrush-handle designs are shown in Figure 5-7. Several brushes have recently been marketed with an "angled" design, stated to be like a dental instrument. As shown in Figure 5-8, these toothbrushes are similar to a dental professional's mirror. Brushes are also available, as depicted in Figure 5-9, with a handle on the same plane as the bristle tips, as are dental instruments used for caries evaluations and prophylaxes. With both the offset and angled-offset designs, points of bristle contact are in line with the longitudinal axis of the handle during brushing. Handle design and length may provide comfort and compliance during toothbrush use and these factors have recently been documented to improve the quality of tooth brushing. This is particularly true of toothbrushes for children, whose dexterity may not be highly developed.[8,9]

Texture

Nylon bristles have a uniform diameter and a wide range of predictable textures. Texture is defined as bristle resistance to pressure and is also referred to as firmness, stiffness, and hardness. The firmness or texture of a bristle is related to its (1) composition, (2) diameter, (3) length, and (4) number of individual bristles per tuft. In the manufacturing process, the diameter of nylon bristles can be well controlled. Because the majority of toothbrushes contain bristles 10- to 12-millimeters long, the diameter of the bristle becomes the critical determinant of texture. The usual range of

diameters for adult toothbrush bristles is from 0.007 to 0.015 inches. Factors such as temperature, uptake of water (hydration), and toothbrush-use frequency affect texture.

Texture labeling is not standardized. Individual manufacturers label their brushes according to their testing criteria. Thus one manufacturer's "soft" grade may be stiffer than another manufacturer's "medium" grade. The International Organization for Standardization (ISO) has formulated testing procedures that permit manufacturers to label their brushes in a consistent manner.[29] The American Dental Association is a member of ISO.

Nylon Versus Natural Bristles

The nylon bristle is superior to the natural (hog) bristle in several aspects. Nylon bristles flex as many as 10 times more often than natural bristles before breaking; they do not split or abrade and are easier to clean. The configurations and hardness of nylon bristles can be standardized within specified and reproducible tolerances. Natural bristle diameters, since they are tapered, vary greatly in each filament. This can lead to wide variations in the resulting texture of the marketed toothbrush. As a result of the advantages of nylon, as well as its ease and economy of production, relatively few natural bristle toothbrushes are marketed.

Actions

Bristle actions caused by different brushing motions are illustrated in a 1992 publication[7] that measured and quantified three-dimensional individual movements during brushing. Data frames were filmed to create a computer-generated reanimation of brushing motions in order to design new toothbrush bristle conformations. These authors concluded that an individual's brushing techniques do not vary and are inadequate; therefore bristle configurations in newly designed toothbrushes could be developed to be adaptable to any brushing style

Figure 5-1 Parts of a toothbrush.

Figure 5-2 Lateral profiles of selected toothbrushes: Aquafresh Flex; Colgate Plus; Colgate Total; Colgate Wave; Crest Complete; Mentadent; Oral-B Advantage; Oral-B P-40; Reach Advanced Design; Reach Plaque Sweeper; Reach Tooth & Gum Care.

Figure 5-3 Cross-sectional profiles of four toothbrushes: Butler GUM; Colgate Total; Oral-B; Reach.

Figure 5-4 Overhead appearance of selected toothbrushes, from left to right: Reach Advanced Design; Aquafresh; Colgate Plus; Crest Complete; Jordan V.

Figure 5-5 New shapes and textures of Tynex nylon toothbrush filaments. (Courtesy of DuPont Filaments.)

Figure 5-6 Toothbrush bristle ends as seen with the scanning electron microscope. A. A coarse-cut toothbrush bristle end, probably the result of an incomplete single-blade cut during the manufacturing process. These sharp projections can reduce the bristles' overall cleaning efficiency and damage oral tissues (SEM 85×). B. A slightly enlarged, bulbous nylon bristle end, resulting from a double-blade or scissor cut during the manufacturing process (SEM 170×). C. A tapered or round-end nylon bristle produced by heat or a

mechanical polishing process (SEM 170×). D. The scrubbing, mechanical action of a toothbrush wear machine has nicely rounded off this bristle removed from a brush that was originally coarse cut. (SEM 170×). (Courtesy of KK Park, BA Matis, AG Christen, Indiana University Dental School.)

Figure 5-7 Four basic shapes of toothbrush handles. *(J Clin Dent.)*

Figure 5-8 Similarity of angled toothbrushes and a dental mirror.

Figure 5-9 Similarity of two dental instruments and a toothbrush with the head on the same plane as the handle.

Powered Toothbrushes

Introduction

Powered toothbrushes were first advertised in *Harper's Weekly* in February 1886,[30] but only became a factor in the U.S. marketplace beginning in the 1960s with the introduction of Broxadent. With the commercial success of this product, battery-powered products were introduced with the advantage of being portable and available at a lower cost. Unfortunately, problems with these battery-powered products included short "working times" and mechanical breakdowns. The enthusiasm for the powered toothbrush declined and was recommended mainly for the handicapped.

In the 1980s, the category of powered toothbrushes was revitalized with the introduction of the InterPlak product. This "second generation" powered toothbrush had a uniquely rotating head and was powered by long-life/ rechargeable batteries. Increased efficacy compared to manual toothbrushes was consistently demonstrated in published studies.[4,8,9, 31-33]

Since then, sonic-powered toothbrushes of a "third" generation have been developed and shown to remove more plaque in comparison to manual toothbrushes, especially in long-term studies. Two primary types of head designs are now used: the rotating, oscillating type with a small, round molar-crown-size brush head and three oscillating brushes with either vibrating or rotational sonic movements.[34-37] Plaque removal by these brushes appears equally effective; periodontal therapeutic effects were demonstrated in pockets of ≤ 5 mm. "Generations" of powered toothbrushes are presented in Table 5-2.

Most recently, powered toothbrushes have been introduced that are battery-powered or disposable after "running down," and are priced, in the United States below $20.00. Published studies have been found on two of these brushes.[38-39]

In most developed countries, the number of powered toothbrush products sold has increased dramatically in recent years. In Switzerland, the regular use of powered toothbrushes increased from 10 to 30% in the last decade. In epidemiological studies, it has been documented that populations are exhibiting increased gingival abrasion and recession. This has been associated with the increased use of oscillating powered toothbrushes. In comparison to these oscillating toothbrushes, sonic toothbrushes have been shown to do little harm to the gingiva. Also sonic brushes of this type can be

used up to 6 or 12 months because the bristles show minimal overt signs of use and do not splay.[40-44]

Bristle/Designs

The heads of most powered or mechanical toothbrushes are smaller than manual toothbrushes and are usually removable to allow for replacements (Figure 5-10). The head follows three basic patterns when the motor is started: (1) reciprocating, a back-and-forth movement; (2) arcuate, an up-and-down movement; and (3) elliptical, a combination of the reciprocating and arcuate motions. Powered toothbrushes are consistently superior to manual toothbrushes in plaque removal and gingivitis efficacy.[9,31,45] Differences are most significant when tested against manual toothbrushes.

Motivation

Motivation to improve oral hygiene appears to be a key factor for patients to purchase powered toothbrushes.[31,46] In a survey by the ADA, of the 139 respondents who owned powered toothbrushes, 21.6% used them regularly, and 25.2% used them occasionally.[47] This survey does not indicate the toothbrushing frequency of the remaining 53%. A published study on the use of powered toothbrushes found that when consumers first purchased the electric brush they increased their frequency of brushing. The effectiveness is especially improved when the users are given instructions and controlled during the first 6-month period. More recently,[48] a survey conducted 6 months after subjects completed a clinical efficacy study indicated that most subjects were not using their powered device twice a day. With the development of the second and third generation of powered toothbrushes, it appears that long-term use is increasing; however, recent publications on this have not been definitive.

Weinstein et al.[49] analyzed the failures of motivation. One of the important aspects is to accept each patient as an individual, and the dental hygienist and dentist should be able to listen to the patient. Oral hygiene can be instructed only when we are informed about a patient's attitudes, and he or she has to demonstrate their oral hygiene. The procedure in brushing for any method used should have a definite sequence. Health professionals should take time and not expect the patient to change more than one thing from session to session. It is important to have a preventive program for each patient, and this starts with the charting. After the first steps we should follow the program to obtain the goals with the patient. The patients' progress should be evaluated from session to session and from year to year. Dental professionals should also accept failures and have an alternative plan to implement in case of failure.

Figure 5-10 Toothbrush heads from four powered toothbrushes: Braun; Interplak; Sonicare; Rota-dent.

Efficiency / Safety Evaluations

Toothbrushing devices have been developed that accurately standardize all of the above factors, in addition to length and number of toothbrushing strokes over simulated anterior or posterior teeth. Published testing methods are now available to evaluate both safety and efficacy of manual and powered toothbrushes (Table 5-3). Differences between products can be determined and, in several areas, are predictive

of clinical results. For example, three laboratory methods have been predictive of clinical plaque removal when plaque assessments focusing on interproximal areas were used. Significant clinical differences between toothbrush designs have also been documented.[8,9,17] Interproximal access efficacy has been directly related to increasing brushing pressures and inversely correlated with bristle texture (the "softer" the texture, the higher the interproximal efficacy).[50,51]

Clinical advantages of various toothbrush-head configurations for removing dental plaque and debris (cleaning efficacy) have been difficult to substantiate. This is attributed to the wide variations among individuals in toothbrushing times, motions, pressures, and in the shape and number of teeth present. Published studies on the clinical superiority of one newly designed manual or powered toothbrush versus another have been inconsistent. It is clear, however, that these new products are more effective than standard manual brushes.[8,9]

The American Dental Association (ADA) Acceptance Program

The American Dental Association (ADA) has established guidelines to enable manufacturers to obtain an acceptable rating and use the ADA Seal of Acceptance. In 1996, the Council on Scientific Affairs of the American Dental Association proposed new guidelines for the Seal of Acceptance.[52] These guidelines require laboratory documentation of acceptable end-roundedness, good manufacturing procedures (GMPs), and equivalency in clinical plaque and gingivitis efficacy compared with a control toothbrush provided by the ADA.

Manual toothbrushes with a standard design, acceptable laboratory data, and GMPs do not require clinical testing. For manual toothbrushes with new designs and for mechanical brushes, the guidelines require only equivalency in plaque and gingivitis reduction compared with a toothbrush provided by the ADA. The clinical protocol is summarized in Table 5-4. The statement to be used in the labeling of products accepted by the ADA is: "(Product Name) is accepted as an effective cleansing device that has been shown to remove plaque and reduce gingivitis when used as directed in a program of good oral hygiene to supplement regular professional care."

As listed on the American Dental Association's website (www.ADA.org), more than 140 manual toothbrushes have been awarded the ADA Seal of Approval (August 2001).

The ADA has developed criteria for acceptance of powered toothbrushes based on both safety and efficacy. These are: (1) laboratory evidence of electric safety, that is, no electric shock hazard; (2) clinical evidence of both hard- and soft-tissue safety under unsupervised conditions; (3) clinical evidence of plaque and gingivitis efficacy compared to a toothbrush already accepted and provided by the ADA; and (4) evidence of proper labeling and advertising claims that may mention plaque reduction but not improvement of any existing oral disease.[52] The required statement for labeling and commercial claims on powered toothbrushes accepted by the ADA is the same as for manual toothbrushes. As of August 2001, 10 powered toothbrushes have been awarded the ADA Seal of Acceptance. Five of these products are distributed by Water Pik Technologies.

Question 2

Which of the following statements, if any, are correct?

A. Using laboratory tests, the relative effectiveness of different toothbrushes can be compared and specific brushes identified as effective for removal of plaque.

B. Interproximal access decreases as the textures of the bristles increases.

C. Interproximal access is better with vertical brushing procedures, compared with a horizontal motion of the brush head.

D. Standard manual toothbrushes can remove plaque as effectively as the newly designed powered toothbrushes.

E. An interproximal plaque index is used to measure interproximal toothbrush cleaning efficiency.

Toothbrushing Methods

The objectives of toothbrushing are to (1) remove plaque and disturb reformation; (2) clean teeth of food, debris, and stain; (3) stimulate the gingival tissues; and (4) apply dentifrice with specific ingredients to address caries, periodontal disease or sensitivity.

During the last 50 years many toothbrushing methods have been introduced, and most are identified by an individual's name, such as Bass, Stillman, Charters, or by a term indicating a primary action to be followed, such as roll or scrub. No one method shows consistently better results in removing plaque than scrubbing. Most studies with manual toothbrushes and the different instructed methods show more gingival abrasion than with powered sonic toothbrushes. Most people brushing with an instructed professional method are not aware that they are brushing in a specific way. Thus it may be more effective to instruct patients to improve their own method. This can be achieved by using a plaque disclosant to stain plaque or identify areas that are missed during tooth brushing. Then the patient can be taught how to clean the sites properly and on the next visit be rechecked. The proposed adaptation has to be recorded in the patient's chart and rechecked at the beginning of the next session, as not all patients can remember all the instructions. Additionally, professionals should never argue with a patient but instead should encourage and help.

Various toothbrushing methods will be briefly described here. For more details see the original papers or this chapter in the previous textbook edition. The toothbrushing methods most emphasized are horizontal scrub, Fones, Leonard, Stillman, Charters, Bass, rolling stroke (press roll), and Smith-Bell. All of these techniques are applicable to the cleaning of the facial, lingual, and to some extent to occlusal surfaces; all are relatively ineffective in cleaning interproximal areas; and only the Bass technique is effective in cleaning the sulcus. The brush motions used in each of these techniques are summarized in Table 5-5.

Natural Methods of Brushing

The most natural brushing methods used by patients are a reciprocating horizontal scrub technique,[53] a rotary motion (Fones's technique),[54] or a simple up-and-down motion over the maxillary and mandibular teeth (Leonard's technique).[55] Patients managing effective toothbrushing with these methods without causing traumatic problems or disease should not alter their brushing methods just for the sake of change.[56]

Stillman's method was originally developed to provide gingival stimulation.[57] The toothbrush is positioned with the bristles inclined at a 45-degree angle to the apex of the tooth, with part of the brush resting on the gingiva and the other part on the tooth (Figure 5-11). A vibratory motion is used with a slight pressure to stimulate the gingiva. The brush is lifted and then replaced in the same area, and pulsing is repeated.

Charters advocated a pressure-vibratory technique to clean interproximal areas.[58] The toothbrush should be placed at a 90-degree angle to the long axis of the teeth so that the bristles are gently forced between the teeth but do not rest on the gums. The brush is moved in several small rotary motions so that the sides of the bristles are in contact with the gum margin. After two or three such motions, the brush is removed and replaced in the same area and the motions are repeated.

It is important to note that the Bass technique was the first to focus on the removal of plaque and debris from the gingival sulcus by the combined use of a soft toothbrush and dental floss. The method is effective for removing plaque adjacent to and directly beneath the gingival margins as part of the self-care regimen for controlling periodontal disease and caries. In the Bass technique, the toothbrush is positioned in the gingival sulcus at a 45-degree angle to the tooth apex. The bristles are then gently pressed to enter the sulcus. A vibratory action, described as a back-and-forth horizontal jiggle, causes a pulsing of the bristles to clean the sulci[59] (Figure 5-12). Ten strokes are advised for each area.

In the rolling-stroke method, the toothbrush bristles are positioned parallel to and against the attached gingiva, with the toothbrush head level with the occlusal plane. The wrist is then turned to flex the toothbrush bristles first against the gingiva and then the facial surface. A sweeping motion is continued until the occlusal or incisal surface is reached (Figure 5-13). The toothbrush bristles are at right angles to the tooth surface as the brush passes over the crown. The press roll action is repeated at least five times before proceeding to the next site.[60]

Modified Brushing Methods

In attempts to enhance brushing of the entire facial and lingual tooth surfaces, the original techniques have been modified. Some modifications like the Bass method may induce a more pronounced gingival trauma with standard brushes.[61] New toothbrush designs such as multilevel and cross-section bristles that have been tested are not only more effective but can be also less harmful.[62]

The following considerations are important when teaching patients a particular toothbrushing technique: (1) the patient's oral health status, including number of teeth,

their alignment, patient's mouth size, presence of removable prostheses, orthodontic appliances, periodontal pockets, and gingival condition; (2) the patient's systemic health status, including muscular and joint diseases, and mental retardation; (3) the patient's age; (4) the patient's interest and motivation; (5) the patient's manual dexterity; and (6) the ease and effectiveness with which the professional can explain and demonstrate proper toothbrushing procedures.

Recommended Powered Toothbrushing Methods

Most powered toothbrush manufacturers do not recommend a specific brushing method, however, the electric brushes should be used in a specified manner. The Swiss Dental Society, in 2001 developed an instruction manual.[63] Instructions for brushes with a sweeping and /or oscillating rotary motion are as follows:

1. The brushes are positioned on the tooth surfaces in a 45- or 90-degree angle to the incisal plane. Only when positioned should the brush be switched to "on." The mouth should be almost closed.

2. The brush should be moved slowly over and around each tooth for 3 to 5 seconds, making sure that the bristles clean the crevices between the teeth.

3. The brush head can be lifted distally and mesially into the interproximal areas to reach the interdental area; the brush always remains on a single tooth.

4. After a period of approximately 5 seconds, the brush is moved to the next tooth surface and repositioned.

5. Experienced individuals can use the brush also in a perpendicular angle to the teeth and gums, but the applied force has to be gentle. In this way, each tooth in the upper and lower arch is cleaned on the buccal and lingual surfaces.

6. It is best to divide the mouth into four quadrants (upper-right, upper-left, lower-right, and lower-left) and to start brushing on a tooth in the upper rear and then clean one surface after the other very systematically.

7. It is an easy way, gives good control for the individual, and does not omit any tooth surface. This method takes more time, because at a single time interval, only one tooth surface can be cleaned.

Toothbrushing Time and Frequency

For many years the dental professional advised patients to brush their teeth after every meal. The ADA has modified this position by use of the statement that patients should brush "regularly." Research has indicated that if plaque is completely removed every other day, there will be no deleterious effects in the oral cavity.[64] On the other hand, because few individuals completely remove plaque, daily brushing is still extremely important to maximize sulcular cleaning as a periodontal disease control measure, as well as to afford an opportunity to use fluoride dentifrices more often in caries control. Where periodontal pockets exist, even more frequent oral hygiene procedures are indicated.

Studies have been conducted in which patients were asked to brush exactly as they did at home and then covertly monitored to determine the length of time of brushing. In the last two decades, the average brushing time was shown to have increased from about 20 to 30 seconds, to 60 seconds,[65,66] and to 80 seconds in a 1995 study.[67] In all of these studies, the individuals claimed that they usually brushed for 2 or 3 minutes. These results demonstrate that people greatly overestimate their efforts or else are telling their professionals what the individuals believe or would like the professionals to hear.

Thorough toothbrushing requires a different amount of time for each individual, depending on such factors as the innate tendency to accumulate plaque and debris; the psychomotor skills; and the adequacy of clearance of foods, bacteria, and debris by the saliva. Only after patients have repeatedly brushed their teeth under the supervision of a dental professional can the adequacy of cleaning in a given time be determined. Often a compromise is made by suggesting 5 to 10 strokes in each area or by advocating the use of a timer. This amount of time, which might be adequate for the average person, may not be sufficient for patients in most need of maximum plaque-control programs. To ensure continued commitment to a personal oral-hygiene program, the benefits of proper oral care must be explained and demonstrated to patients.[9]

Toothbrushing Procedures

Occlusal Surfaces

The occlusal surfaces may be cleaned by either (1) short vibratory strokes, with pressure being maintained to accomplish as deep a penetration of the pits and fissures as possible; or (2) a rapid back-and-forth vibrating motion to force the bristles into the pits and fissures, followed by a sweeping motion to expel the dislodged debris. Long, sweeping, horizontal strokes are contraindicated, because the toothbrush bristles have minimum contact in the deeper and more critical fissures (Figure 5-14). The orifices of the pits and fissures are too narrow for bristle penetration and, whatever the technique, are inaccessible for adequate cleaning. This helps explain why more than 60% of all carious lesions in the mouth are found on the occlusal surface, even though most individuals attempt to brush this surface.

The Anterior Lingual Areas

Access to the lingual surfaces of the mandible and maxilla is difficult. Brushing in these areas can be facilitated by cutting off all tufts on a brush, except the first four or five rows in the toe. This modified brush has unimpeded access to the gingival sulci and lingual fossae areas (Figure 5-15). In the lower arch, the heel of the brush can be used for the same purpose.

Brushing Sequence

A routine brushing pattern should be established to avoid exclusion of any area. One systematic pattern is to teach children to begin by cleaning the occlusal surfaces of the maxillary arches, starting with the molars, and then the same on the mandibular

arches. For children it is most important to brush the pit and fissures. The use of a three-dimensional brush can be recommended as long as children are not able to brush the more difficult buccal and lingual surfaces.[68] Such toothbrushes that hug the teeth and clean the buccal, lingual, and occlusal surfaces simultaneously are easier for children to use, as the brush guides itself from tooth to tooth. Studies show that children favor such toothbrushes.

Adult patients are taught to begin with the distal surface of the most posterior tooth and to continue brushing the occlusal and incisal surfaces around the arch until the last molar on the other side of the arch has been reached. The lower arch is then brushed in a similar manner.

Patients tend to apportion more time and effort on the facial areas of the anterior teeth.[69] Often, right-handed people do not brush the right side of the arch as well as the left side; left-handed people similarly neglect the left side over the right side.

Clinical Assessments of Toothbrushing

Whatever techniques are recommended, the main purpose of tooth brushing is to remove dental plaque from the teeth, including the gingival crevice, with the minimum amount of damage to the teeth and surrounding structures. Disclosing agents provide the means of evaluating the thoroughness of cleaning the teeth.[56,70] The most widely marketed red disclosing products contain FD&C Red #28.

Disclosing agents may be in either a liquid or tablet form. The chewable tablet or the liquid disclosant should be swished around in the mouth for 15 to 30 seconds and then expectorated. Home use of disclosants by the patient should be encouraged to permit self-evaluation of the effectiveness of plaque-control programs. Clinical assessments should be made for evidence of improper tooth brushing. Minor damage that may be noted includes abrasion to the soft tissues (scuffing, bruising, and punctate lesions) or damage to the tooth surface.

Toothbrush abrasion, or the wearing away of tooth substances, occurs from the use of highly abrasive dentifices, too-firm brush bristles, incorrect brushing methods, and excessive pressure during brushing. Common abrasion locations are on the surfaces of the teeth displaced facially and on the cervical areas of exposed root surfaces. Because enamel is harder than cementum, tooth damage usually occurs as a V-shaped, horizontal notch immediately apical to the cementoenamel junction. Further progress of the abrasion can be minimized by use of soft-bristle brushes, changes in brush angulation, pulsing instead of stroking, the use of less abrasive dentifrices, and less pressure during brushing.

Toothbrush Replacement

Toothbrush wear (splayed, bent, or broken bristles) is influenced more by brushing methods than by the length of time or number of brushings per day.[71] The average "life" of a manual toothbrush is approximately 3 months. This estimate can vary greatly, however, because of differences in brushing habits. It is also sound advice for patients to have several toothbrushes and to rotate their daily use, to assure drying between brushings. If toothbrushes need to be replaced more frequently than every

three months, the patient's brushing technique should be checked. Even if the brushing technique is acceptable or has been corrected, toothbrushes should still be replaced frequently. Indeed, after every oral or contagious medical illness, it is imperative that patients be made aware of the importance of having a new toothbrush.

Figure 5-11 Stillman technique seen diagrammatically.

Figure 5-12 Bass technique: A. graphically; B. pictorially.

Figure 5-13 Rolling stroke technique.

Figure 5-14 Occlusal brushing dislodges debris in the pits and fissures of posterior teeth (commonest site of caries) as well as in interproximal incisal areas.

Figure 5-15 Vertical position of the toothbrush for the often constricted lingual area.

Special Needs

Tongue Brushing

Malodor from the mouth most often has its origin on the tongue. Therefore, for persons expiring mouth odor, tongue brushing is important. Tongue cleaning is also indicated for patients harboring a coated tongue. A coated tongue is a bacterial reservoir but could also be a locus for intraoral transmission of organisms during toothbrushing, through infection or reinfection of periodontally treated pockets. This is another reason that Quirynen et al. introduced the full-mouth disinfection concept in periodontal patients to prevent recolonization of bacteria.[28]

The brushing of the tongue and palate helps reduce the debris, plaque, and number of oral microorganisms. The papillae on the tongue provide an area especially conducive to bacterial and debris retention. Tongue cleansing can be accomplished by placing the side of the toothbrush near the middle of the tongue, with the bristles pointed toward the throat. The brush is swept forward, and this motion is repeated six to eight times in each area. The palate should also be cleansed with a sweeping motion. A dentifrice should be used with this brushing of soft tissues to improve cleansing action.[72]

Abutment Teeth and Orthodontic Appliances

Abutment teeth, implants, fixed bridgework and fixed orthodontic appliances require special emphasis on sulcular brushing to prevent gingivitis. Thorough cleansing between orthodontic appliances and gingiva will prevent dental caries. A pre-teen or teenager, as well as patients with extensive reconstruction bridgework, are prone to dental diseases but are also more motivated; therefore, a rigid, preventive program is required. A powered brush and auxiliary aids are suggested.

The effectiveness of a new toothbrush design in orthodontic patients has been documented in different publications. At the end of a 4-month study, a three-sided manual toothbrush significantly decreased gingivitis and was more effective in plaque

removal compared to a flat multitufted toothbrush.[73] Powered toothbrushes have been documented to provide superior efficacy in orthodontic patients compared to results in patients using manual toothbrushes.[9]

Dentures and Removable Orthodontic Appliances

Patients with full dentures can meet their oral hygiene needs with a soft nylon brush for the oral tissues and a denture brush that cleans all areas of the denture. The denture brush with a nonabrasive cleaner should reach into the recessed alveolar ridge area of the denture to ensure maximum cleansing. The oral tissues should be brushed at least once a day using a gentle vibration and long, straight strokes from the posterior to anterior mouth regions.[70]

Patients with removable partial dentures and removable orthodontic appliances need at least three toothbrushes, one for the natural teeth, another for the appliance, and a third for clasps. Brushing clasps, wires, and other metal parts can wear out a regular toothbrush. A clasp brush—2 or 3 inches long, narrow, and tapered—can be obtained as a third brush. Special care is needed to carefully clean all plaque from the clasps as a preventive measure for the supporting teeth.

Handicapped Patients

Some handicapped patients are able to brush their own teeth and can often do so with support and encouragement from dental personnel and the use of special toothbrushes. A manual brush with an enlarged handle, elastic cuff, or small strap attached to the brush or a long-handled holder for patients who cannot raise their arms or do not have hands, permits the patient to brush.[74] The elastic cuff is fitted around the hand and holds the toothbrush in the patient's palm. Patients who are unable to reach their mouths for brushing can, at times, attach the brush in a stationary upright position by using a clamp.[75] The patients bend over to position the brush in the mouth. The National Foundation of Dentistry for the Handicapped is developing a preventive program to encourage toothbrushing to the beat of music. A brush wheel, which can be used in between the teeth and moved through the dentiton without using the hands might be helpful for tetraplegics. The results are almost comparable with handbrushes.[76,77] Mentally retarded patients can often brush using a soft toothbrush with the plastic handle bent for better grasping. A horizontal scrub is often the best that these patients can manage. A three-headed toothbrush or a powered toothbrush assisted by a caregiver can be useful.[37]

Special Uses for Powered Toothbrushes

Powered toothbrushes can be beneficial for parental brushing of children's teeth; for children and adults who are physically handicapped, mentally retarded, aged, arthritic, or otherwise with poor dexterity; and for those patients who are poorly motivated. These brushes are especially recommended for patients who require a larger handle, because powered models are easier to grasp.

Question 3

Which of the following statements, if any, are correct?

A. A meticulous, once-every-other-day program may be as effective as daily morning and evening brushings.

B. The high incidence of caries that occurs on the occlusal surface is usually traceable to inadequate brushing.

C. Toothbrush replacement every 3 months is as important as proper brushing techniques.

D. For a person with a partial denture, the toothbrush used for the natural teeth is not adequate for cleaning the clasps.

E. It is necessary for handicapped persons to have others aid in brushing their teeth.

Summary

Toothbrushing alone cleans buccal and lingual tooth surfaces. No single toothbrushing technique adequately cleans occlusal pits and fissures. No toothbrushing procedure removes all interproximal and subgingival plaque, especially around malposed teeth and fixed prostheses. Interproximal cleaning aids are necessary to complete the tooth-cleaning process. No one toothbrush design has been demonstrated to be most effective for all patients in long-term studies. Dental professionals should be familiar with various toothbrush products, primarily from their own use experience, and have examples of toothbrushes demonstrating various degrees of splaying or bending. These should be demonstrated when prevention methods are being discussed with their patients.

Although manufacturers are advertising variations in bristle shape, bristle size, and number of filaments, no accepted criteria exist for product labeling. The American Dental Association does not yet consider one toothbrush design superior to another but is developing clinical-testing guidelines associated with both plaque and gingivitis reductions. Thoroughness and frequency of brushing are probably more important than a specific toothbrushing method and toothbrushing products. Any method that is taught should be effective, not damaging to the hard or soft tissues, routinely used and should not cause excessive tooth wear. In initiating effective toothbrushing, it is necessary to (1) select the appropriate toothbrush(es) suitable for the patient; (2) instill in individuals the goals of toothbrushing and the need for good oral physiotherapy; (3) teach a technique or combination of brushing methods needed to meet special needs; and (4) assess thorough and effective toothbrushing as a part of the total oral hygiene program

Answers and Explanations

1. A, C, D, and E—correct.

B—incorrect. Toothbrush bristles have also undergone major changes in shape and design.

2. A, B, C, and E—correct.

D—incorrect. The standard manual toothbrush is not as effective as the new powered toothbrushes.

3. C and D—correct.

A—incorrect. It is true that one good cleaning would do the job, but so few people do a good job that several cleanings might be equal to one good try. However, either answer could be correct.

B—incorrect. No matter how well the occlusal surface is brushed, the deep pits and fissures cannot be adequately cleaned with a brush.

E—incorrect. Handicapped persons can often manage brushing with slightly modified oral-hygiene aids or with specially developed brushing devices.

Self-evaluation Questions

1. Three general reasons that people do not spend adequate time for personal oral-health care are _____, _____, and _____.

2. Wadsworth introduced the toothbrush into the United States just before the _____ War.

3. The constricted part of the toothbrush between the handle and the head is the _____. The end of the head is arbitrarily termed the _____; the part closest to the handle is called the _____.

4. Four lateral profiles of brushes sold in the United States are _____, _____, _____, and _____.

5. The American Dental Association Council on _____ (name) continually accomplishes scientific evaluations of devices used in dentistry. To support standardization of professional devices, the ADA is a member of the International _____, which has as its objective the establishment of consistency of labeling.

6. Two synonyms for hardness of bristles and toothbrushes are _____, and _____. Firmness of bristles is caused by three general characteristics of bristles; they are _____, _____, and _____. A medium-texture bristle has a diameter of approximately _____ in.

7. Gingival abrasion can occur with manual toothbrushes because of _____, _____, or _____.

8. Three basic motions of electric toothbrush heads are _____, _____, and _____.

9. Three groups of people who can especially benefit from use of electric toothbrushes are _____, _____, and _____.

10. Four objectives of toothbrushing are _____, _____, _____, and

_____. The three natural methods of toothbrushing are _____,_____, and _____. The motion of the brush in blank no. 1 in the previous sentence is_____; in blank no. 2 is _____; and in blank no. 3 is _____.

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Chapter 6. Dentifrices, Mouthrinses, and Chewing Gums - *Stuart L. Fischman Samuel L. Yankell*

Objectives

At the end of this chapter, it will be possible to:

1. Differentiate between a *cosmetic* and a *therapeutic* dentifrice, mouthrinse, or chewing gum.

2. Explain the three phases of research necessary when applying to investigate a new drug (IND)—the process that precedes receiving a new drug application (NDA), which is necessary to market a new product with a therapeutic claim.

3. Discuss how approval or nonapproval of a new product by the Food and Drug Administration (FDA) differs from acceptance or rejection by the American Dental Association (ADA).

4. Explain the various reasons that the same abrasive material in toothpaste can cause differing levels of abrasion on tooth structure.

5. Name the usual dentifrice ingredients and their percentages in a dentifrice.

6. Name the agents used in dentifrices to produce anticaries, anticalculus, whitening,

and antihypersensitivity effects.

7. Name the active ingredients in typical antiplaque and antigingivitis mouth rinses: one sold over the counter, the other as a prescription item.

Introduction

Dentifrices and mouthrinses are the major products for routinely administering effective *cosmetic* and *therapeutic* agents in the mouth. These products are the most widely used by consumers, generating the largest sales of all dental products. Chewing gums are a new category of products with cosmetic claims and the ability to deliver therapeutic compounds.

Dentifrices and mouthrinses differ considerably. Dentifrices are complex and difficult to formulate. Tremendous innovations have occurred in the past 20 years in the appearance and packaging of dentifrices. The contemporary consumer is faced with many alternatives in *appearance* (pastes, clear gels, stripes) and *packaging* (conventional tubes, stand-up tubes, pumps), as well as products marketed specifically for children. In addition, numerous claims are made for dentifrices. They are said to *prevent* calculus and caries, to whiten teeth, to eliminate hypersensitivity, and to reduce plaque and gingivitis.

Because the public routinely uses them one to three times per day, dentifrices are the most beneficial dental products. Some of this benefit may be lost if a person rinses immediately after brushing because rinsing *decreases* the concentration or reservoir of the active agent(s) in the oral cavity.

Mouthrinses are available in liquid form, the traditional method for stabilizing and delivering many pharmaceutically active agents. Mouthrinses are considered by consumers to have primarily cosmetic benefits (i.e., breath fresheners) and are therefore not used as frequently or routinely as dentifrices in the daily oral-hygiene regimen. Two categories of mouthrinses have been recognized by the American Dental Association (ADA) as effective against *plaque* and *gingivitis* (see ADA website www.ada.org). One category contains the *essential oils* as the active ingredients. Products in this category include Listerine and its generic equivalents containing the original *essential oils.* To date, more than 200 generic versions have been reviewed and accepted by the ADA. These products are sold over the counter. The other category of products contains *chlorhexidine* as the active agent. Currently marketed products are Peridex and Prevident. The Food and Drug Administration (FDA), has approved chlorhexidine-containing products *only* as prescription products.

Chewing gums have the potential to be used by the consumer for periods of 5 to 20 minutes several times a day, until the flavor of the product dissipates. This would enable delivery of a cosmetic or therapeutic agent for a *longer time* than dentifrices or mouthrinses. In addition to prolonged delivery of an agent, chewing gums *stimulate salivary flow,* which can provide a buffer effect and also ensure removal of debris from occlusal and interproximal sites. To assure safety and avoid harmful gastrointestinal effects, active agents delivered by chewing gums must be *safe for swallowing* at the dose delivered in one use or contained in the entire product sold in one package.

Monitoring the Safety and Effectiveness of Therapeutic Dental Products

Caution is needed before introducing a new therapeutic product to the market. Some of the concerns surrounding new products are: Will the active agent disrupt the "normal" bacterial balance of the mouth? Should the search for an ideal agent focus on depressing or eliminating specific disease-related organisms or a broad spectrum of organisms? Should a product be used to preserve a disease-free state while risking the possibility of developing drug resistance? Regardless of the apparent effectiveness of any new product in the laboratory or in controlled clinical studies, public *safety* with unsupervised, widespread availability and use by consumers *is paramount.*

The process by which oral-care agents are evaluated and regulated in the United States has been reviewed by Trummel.[1] Safety and efficacy standards apply not only to prescription medications but *also* to over-the-counter (OTC) drugs. There are three levels of regulation of oral chemotherapeutic agents. The government level includes the *Food and Drug Administration (FDA)* and the *United States Pharmacopoeia Convention.* The *professional or voluntary level* includes the Council on Scientific Affairs (CSA) of the *American Dental Association (ADA).* The *third* level of review includes *consumer advocacy organizations,* advertising standards review panels, and the Federal Trade Commission. In addition, each of the major television networks has its own in-house review committee.

The FDA conducts an ongoing review of all OTC products. One aim of regulation is to protect the patient-consumer from *useless* or *harmful* products. All approval or disapproval decisions by the FDA have the *force of law.*

The stages of FDA approval include preclinical research and development (animal testing, laboratory testing, and toxicity evaluation) followed by clinical research conducted with an approved *investigational new drug (IND)* application. The IND usually includes *three* phases: In phase 1, the study *is limited in scope* and uses only a few subjects to determine the safe dose for humans. For dental products, this usually involves ingestion or exaggerated (three or four times per day) topical applications or both. Phase 2 *involves more subjects* to demonstrate the initial clinical efficacy of the drug and define a dose range for both safety and efficacy. Phase 3 generally includes *double-blind, controlled trials* with "final" formulas to demonstrate long-term safety and efficacy. These range from 3 to 6 months for plaque and gingivitis studies to 2 or 3 years for caries studies. After the company receives an *approved new drug application* (NDA), marketing may begin, but post-marketing surveillance of the product is *mandatory.*

Over the years, the FDA has requested manufacturers of OTC products to submit a *listing* of the active and inactive ingredients in their products as a basis for helping to codify regulations governing OTC sales. Among the many recommendations of the FDA advisory panel[2] that provide for better control of OTC oral therapeutic products is the stipulation that all inactive ingredients be listed on the label by quantity in descending order. Active ingredients, as well as inactive agents, should be in no higher concentrations than necessary for the intended purpose. The panel also recommended that the indicated objective of the active agent(s) must be on the label and that the inclusion of the name of an active agent(s) without stating the proposed

benefits would be considered misleading. Proof must exist to substantiate any claim for a specific therapeutic benefit. For example, dentifrices that have not been subjected to laboratory or clinical trials and do not have the ADA Seal of Acceptance, but only list the inclusion of "decay-fighting fluorides" in their products, *cannot claim* that the dentifrice is anticariogenic, *only* that it contains fluoride. It is possible that the fluoride in the untested dentifrice might not be compatible with other dentifrice ingredients, or the fluoride may not be released in active ionic form and therefore be totally ineffective.

Recommendations also apply to *packaging* and *labeling* guidelines to regulate advertising. For example, the recommendations suggest that all containers for OTC therapeutic dentifrices, rinses, and gels containing fluoride have a label to identify the product, e.g., "anticaries dentifrice"; its use, e.g., "aids in the prevention of dental caries"; a *warning*, e.g., "Do not swallow. Developing teeth of children under 6 years of age may become permanently discolored if excessive amounts of fluoride are repeatedly swallowed"; and directions for use, e.g., "Adults and children 6 years of age or older should brush teeth thoroughly at least twice daily, or as directed by a dentist or physician."

In April 1997, the FDA issued a labeling requirement for fluoride toothpaste. This states, "Keep out of reach of children under 6 years of age. If you accidentally swallow more than used for brushing, seek professional help or contact a poison control center immediately." This recommendation may be an exaggerated response, as most experts believe that neither an adult nor a child could absorb enough fluoride to cause a serious problem.

After years of ignoring claims of antigingivitis efficacy for various OTC dentifrices and rinses, in 1988 the FDA advised manufacturers of such products that they must either cease making such claims or substantiate them. In 1990, the FDA published its call for data stating:

The Food and Drug Administration is announcing a call for data for ingredients containing products bearing antiplaque and antiplaque related *claims*, such as "for the reduction or prevention of plaque, tartar, calculus, film, sticky deposits, bacterial buildup and gingivitis." The agency will review the submitted data to determine whether these products are generally regarded as safe and effective and not misbranded for the label uses. This notice also describes the Attorney General's enforcement of policy governing the marketing of over-the-counter (OTC) drug products bearing antiplaque and antiplaque related claims during the pendency of this review. This request is part of the ongoing review of OTC drug products conducted by the FDA.[3]

In addition to the FDA's regulation of OTC products, the American Dental Association's Council on Scientific Affairs (CSA) continually reviews dental products. The Council is directed to study, evaluate, and disseminate information with regard to dental therapeutic agents, their adjuncts, and dental cosmetic agents that are offered to the public or to the profession.[1] The most important activity of the CSA in meeting this charge is its acceptance program. Unlike the IND process, submission by a manufacturer to the ADA program is *voluntary*. Also, unlike the FDA review process, the primary review responsibilities are conducted by consulting dental

professionals who are appointed by the CSA but are not employees of the ADA. If the product is *safe* and *effective,* the *Seal of Acceptance* is granted and *can be used by the manufacturer* in marketing the product. The Seal provides *assurance* to dental professionals and to the public. In addition to the traditional "print media," this information is available at the ADA website, www.ada.org.

The council recognized that plaque control might best be demonstrated by clinically significant reduction of gingivitis. In 1986, the council issued "Guidelines for Acceptance of Chemotherapeutic Products for the Control of Supragingival Dental Plaque and Gingivitis."[4] The Guidelines for Acceptance are presented in Table 6-1.

The purpose of the separate and independent actions of the FDA and the ADA is to ensure the effectiveness and safety of OTC products and to prevent mislabeling and thus misleading information. *The ADA and FDA differ in their acceptance criteria, which places the responsibility for selecting an effective product on the dental professional.*

Question 1

Which of the following statements, if any, are correct?

A. The Food and Drug Administration (FDA) and the American Dental Association (ADA) have both recognized essential oils and chlorhexidine as antiplaque/antigingivitis agents.

B. The decisions of both the Food and Drug Administration and of the American Dental Association to approve a product have the force of law.

C. To receive the ADA's Seal of Acceptance an antiplaque product must prevent or reduce the severity of some disease caused by the plaque.

D. The sales of both therapeutic and over-the-counter dental products are regulated by the FDA.

E. A manufacturer must secure the ADA's Seal of Acceptance before marketing a dental product.

Dentifrices

According to the dictionary, the term dentifrice is derived from dens (tooth) and fricare (to rub). A simple, contemporary definition of a dentifrice is a mixture used on the tooth in conjunction with a toothbrush. The historic aspects of dentifrice use has been reviewed by Fischman.[5]

Dentifrices are marketed as toothpowders, toothpastes, and gels. All are sold as either cosmetic or therapeutic products. If the purpose of a dentifrice is therapeutic, it must reduce some disease-related process in the mouth. Usually the actual or alleged therapeutic effect is to reduce caries incidence, gingivitis, or tooth sensitivity. The sales appeal of a product, however, is strongly linked to its flavor and foaming action.

In 1970, the dentifrice market amounted to an estimated $355 million; by 1988 it had increased to $1 billion; in 1996 to $1.5 billion; in 2000 to $1.9 billion; and the estimated market for 2005 is $2.2 billion (Figure 6-1).

Packaging

The development of the toothbrush in *1857* provided the stimulus to market commercial dentifrices. Toothpowders were popular because boxes and cans from which they could be dispensed already existed. The formulas consisted of little more than water, soap, and flavor.

Toothpastes began to appear on the market following the development of lead tubes for packaging. The change to plastic packaging during World War II simultaneously:

- Eliminated the possibility of the user ingesting lead,
- Reduced the possibility of incompatibility of the tube and paste components,
- Aided the expelling of the paste by squeezing,
- Permitted an easier and more economic production of tubes, and
- Provided a good surface for the printing of decorative designs and information.

A drawback with the initial use of plastic tubes was the permeability and subsequent loss of flavors though the packaging. This has been resolved with the use of new plastic materials and the use of laminated or layered packaging materials.

In 1984, Colgate introduced the pump dispenser to the market. Separate color compartments used to dispense "striped" products were introduced in Stripe dentifrice by Lever Brothers and are now used in Aquafresh (Smith, Kline, Beecham) and Colgate Total Stripe (Colgate-Palmolive Co.). Cheseborough-Pond's introduced a dual-chamber pump dispenser to keep the peroxide and baking soda components of their dentifrice, Mentadent, separate until immediately prior to use when they are delivered together on the toothbrush.

Dentifrice Ingredients

Dentifrices were originally developed to provide a cosmetic effect and deliver a pleasant taste. They are effective in removing *extrinsic* stains, those that occur on the surface of the tooth. These stains, which are often the end-products of bacterial metabolism, range in color from green to yellow to black. Stains may also result from foods, coffee, tea, cola-containing drinks, and red wines. OTC dentifrices do not remove *intrinsic* stains, which are a result of altered amelogenesis, such as the white-to-brown color changes seen in *fluorosis* or the grayish-blue appearance of enamel following *administration of tetracycline.* Dentifrices, and other OTC products, are also ineffective in altering the yellowing color of teeth seen with *physiologic aging* and in altering the hues of tooth color produced by differing shades of dentin. Therefore, they can only claim to "make your teeth their whitest or brightest"; they *cannot* state, "Makes your teeth whiter or tooth color lighter."

Toothpastes contain several or all of the ingredients listed in Table 6-2. Gel dentifrices are also marketed. Gels contain the *same* components as toothpastes, except that gels have a *higher proportion* of the thickening agents. Both tooth gels

and toothpastes are *equally* effective in plaque removal and in delivering active ingredients.

Abrasives

The degree of dentifrice abrasiveness depends on the *inherent hardness* of the abrasive, *size* of the abrasive particle, and the *shape* of the particle. Several other variables can affect the abrasive potential of the dentifrice: the brushing technique, the pressure on the brush, the hardness of the bristles, the direction of the strokes, and the number of strokes. The abrasive *tested alone* can differ from the same abrasive *tested as part* of a dentifrice formula. The salivary characteristics of individuals may also affect dentifrice abrasiveness.

Calcium carbonate and calcium phosphates were previously the most common abrasives used. These agents often reacted adversely with fluorides. Chalk (calcium carbonate) and baking soda (sodium bicarbonate) are also common dentifrice abrasives. New silicas, silicon oxides, and aluminum oxides are being introduced into dentifrice formulas, with additional efficacy claims.[6-9]

Abrasiveness Testing

Standard laboratory testing uses a machine with several brushes. The *length* of the reciprocating stroke, *number* of strokes, and *pressure* of the brush can be adjusted. Depending on the experimental objectives, enamel, dentin, or cementum is brushed, and the amount of calcium or phosphorus in the resultant slurry is analyzed. A more accurate method has been developed in which extracted teeth are irradiated to activate some of the tooth phosphorus to *radioactive phosphorus.* After brushing root surfaces of sound canines and molars, the amount of radioactive phosphorus removed may be more accurately assessed than with classical chemical analyses. Results are referenced against the amount of tooth substance removed by the use of the control-abrasive, calcium pyrophosphate.[10]

Abrasives usually do not damage enamel, but they may dull the tooth luster. To compensate for this, *polishing agents* are added to the dentifrice formulation. These polishing agents are usually small-sized particles of aluminum, calcium, tin, magnesium, or zirconium compounds. Typically, the manufacturer *blends the abrasives* and the *polishing agents* to form an *abrasive system.* Agents, such as chalk or silica, may have both polishing and abrasive effects. *Smaller* particles (1 mm) have a polishing action, and *larger* particles (20 mm) have an abrasive action.[11]

In selecting a dentifrice, the abrasiveness and polishing characteristics should meet individual needs. For instance, up to 20% of the population does not accumulate visible stain when engaged in their own style of personal oral hygiene.[12] For these individuals, a dentifrice with high polishing and low abrasion should be recommended. For the average individual, an additional amount of abrasive is needed to control accumulating stain. The stains on neglected teeth may be green, orange, or black stains of chromogenic bacterial origin, or yellow and brown stains from smoking. As the abrasive level increases, greater care must be taken to perfect brushing techniques that do not cause *self-inflicted injury* to the teeth or soft tissues. Such injuries can result from excessive pressure, hard bristles, and prolonged

brushing.

When toothbrushing is done without toothpaste, there is little possibility of abrasion. When damage does occur, it usually appears as a V-shaped notch in the *cementum immediately below the cementoenamel junction* (Figure 6-2). This area is vulnerable, because enamel is about *20 times harder* than dentin or cementum. More serious defects usually occur in older individuals who maintain a very high level of oral hygiene.

Humectants

Toothpaste consisting only of a toothpowder and water results in a product with several undesirable properties. Over time, the solids in the paste tend to settle out of solution and the water evaporates. This may result in caking of the remaining dentifrice. Until the 1930s, most toothpaste had a short shelf-life because of this problem. Once the tube was opened, the first expelled paste was too liquid, but the last paste in the tube was either impossible to expel or too hard to use. To solve this problem, *humectants were added to maintain the moisture.* Commonly used humectants are sorbitol, mannitol, and propylene glycol. These humectants are nontoxic, but mold or bacterial growth can occur in their presence. For this reason, *preservatives* such as sodium benzoate are added.

Humectants help maintain the consistency of toothpaste, but despite their presence, the solids tend to settle out of the paste. To *counteract* this, *thickening or binding agents* are added to the formula. Gums, such as gum tragacanth, were first used. These were followed by colloids derived from seaweed, such as carrageenan. These, in turn, were replaced by synthetic celluloses. These celluloses in *low* concentrations are also often used as humectants; in *higher* concentrations they function as gelling agents in the formulation of gel dentifrices. At *high* concentrations (> 40%), humectants also act as preservatives.

Soaps and Detergents

Because toothpastes were originally manufactured to keep the teeth clean, soap was the logical cleansing agent. As the toothbrush bristles dislodge food debris and plaque, the foaming or sudsing action of the soap aids in the removal of the loosened material. Soaps have several disadvantages: they can be irritating to the mucous membrane, their flavor is difficult to mask and often causes nausea, and many times soaps are incompatible with other ingredients, such as calcium.

When detergents appeared on the market, soaps largely disappeared from dentifrices. Today, *sodium lauryl sulfate (SLS) is the most widely used detergent.* It is *stable,* possesses some *antibacterial* properties, and has a *low surface tension,* which facilitates the flow of the dentifrice over the teeth. SLS is active at a neutral pH, has a flavor that is easy to mask, and is compatible with the current dentifrice ingredients. Barkvoll has suggested that patients who suffer from various oral mucosal diseases should avoid the use of dentifrices containing SLS.[13] Low SLS dentifrices have been marketed, which claim to be associated with a lower incidence of oral ulcers.

Flavoring and Sweetening Agents

Flavor, along with *smell, color,* and *consistency* of a product, are important characteristics that lead to *public acceptance of a dentifrice.* If dentifrices did not possess these characteristics, they would probably be poorly accepted. For taste acceptance, the flavor must be pleasant, provide an immediate taste sensation, and be relatively long-lasting. Usually synthetic flavors are blended to provide the desired taste. Spearmint, peppermint, wintergreen, cinnamon, and other flavors give toothpaste a pleasant taste, aroma, and refreshing aftertaste. Some manufacturers use essential oils such as thymol, menthol, etc., which may provide a "medicinal" taste to the product. In addition, these oils may impart antibacterial effects, as will be discussed later in this chapter. It is difficult to formulate a flavor that is universally acceptable, because people have different color and taste preferences.

Figure 6-1 Showing the rapid sevenfold increase in toothpaste sales over the past 30 years, and projected increase till 2005.

Figure 6-2 V-Shaped notches in central incisors resulting from use of a dentifrice with a harsh abrasive system. (Courtesy of Dr. B Baker, University of Texas Dental School, San Antonio.)

Question 2

Which of the following statements, if any, are correct?

A. Gel dentifrices are the same as regular dentifrices, except that they contain a greater proportion of thickening agent.

B. The abrasiveness of an abrasive agent depends on the inherent hardness of an abrasive, the size of the abrasive particles, and the shape of the abrasive particles.

C. The V-shaped damage to the tooth from using an excessively abrasive dentifrice occurs apical to the cementoenamel junction.

D. Synthetic celluloses are now used as thickening agents for toothpastes and gels.

E. If sodium lauryl sulfate is added to the dentifrice formula, foaming can be expected when brushing.

Sweetening Agents

In early toothpaste formulations, sugar, honey, and other sweeteners were used. Because these materials can be broken down in the mouth to produce acids and lower plaque pH, they may increase caries. They have been replaced with saccharin, cyclamate, sorbitol, and mannitol as primary *noncariogenic* sweetening agents. Sorbitol and mannitol serve a *dual role* as sweetening agents and humectants. Glycerin, which also serves as a humectant, adds to the sweet taste. A new sweetener in some dentifrices is *xylitol.* In laboratory studies, it is not metabolized by bacteria to produce acid. In human studies, where it was placed in chewing gums and food, xylitol was noncariogenic. In addition, it demonstrated an anticaries capability by *facilitating* the *remineralization* of incipient carious lesions.

Baking-Soda Dentifrices

Baking soda (sodium bicarbonate) has had a *long history of use as an oral-hygiene aid.* Church and Dwight, a manufacturer of baking soda, and also the manufacturer of the original baking-soda toothpaste, has stated "two out of three dentists and hygienists recommend brushing with baking soda for healthier teeth and gums." In a series of papers published in 1998, antiplaque, gingivitis reduction, stain-removal, and odor-reducing efficacy were *documented* for sodium bicarbonate-containing dentifrices.[6]

Some dentists have also suggested the mixture of baking soda with peroxide as an alternative to the use of commercial dentifrices. Anecdotally, many patients attribute benefit to the routine use of these products. It was inevitable that these products would be incorporated into toothpastes. Selected examples of currently marketed baking soda dentifrices are presented in Appendix 6-1. All contain hydrated silica that is compatible with fluoride. No dentifrice containing baking soda as the *sole* active ingredient has received the ADA Seal of Acceptance. It was only *after fluoride* was added to the formulations, and after required laboratory, animal, and clinical studies were completed, that several baking soda-fluoride dentifrices were accepted as effective in caries control. These baking-soda dentifrices actually contain only a small amount of baking soda, in addition to the standard fluoride-compatible abrasives.

Essential-Oil Dentifrices

The essential-oil ingredients found in Listerine mouthrinse (see below) are also available in a dentifrice formulation. The clinical and laboratory data suggest a benefit to gingival health and plaque reduction.[14,15] This product does *not* carry the ADA Seal of Acceptance.

Therapeutic Dentifrices

The most commonly used therapeutic agent added to dentifrices is *fluoride,* which aids in the control of caries. In 1960, the Council on Dental Therapeutics of the American Dental Association, based on several studies that indicated its effectiveness, classified Crest toothpaste with stannous fluoride as a *caries prophylactic dentifrice.* For the first time, a therapeutic dentifrice was awarded the Seal of *Provisional* Acceptance. In 1964, on the basis of further new and favorable data,[16] the classification was upgraded to *full* acceptance.

The original level of fluoride in OTC dentifrices and gels was restricted to 1,000 to 1,100 ppm fluoride and a total of no more than 120 mg of fluoride in the tube, with a requirement that the package include a safety closure. Therapeutic toothpastes, dispensed on prescription, could contain up to 260 mg of fluoride in a tube.

The following fluorides are generally recognized as effective and safe for OTC sales: 0.22% sodium fluoride (NaF) at a level of 1,100 ppm, 0.76% sodium monofluorophosphate (MFP) at a level of 1,000 ppm, and 0.4% stannous fluoride (SnF_2) at a level of 1,000 ppm. Fluoride levels were increased to 1,500 ppm sodium monofluorophosphate in "Extra Strength Aim," marketed OTC. In published studies,[17,18] this product was 10% more effective than an 1,100 ppm NaF dentifrice. A recently introduced *prescription dentifrice,* Colgate Prevident 5,000, contains 5,000-

ppm fluoride.

One baking soda-peroxide-fluoride dentifrice (Mentadent) is unique in its packaging. Mentadent contains a combination of 0.75% stable *peroxide gel* in conjunction with *baking soda,* and *1,100 ppm sodium fluoride.* The materials are packaged in a two-chamber pump to permit the baking soda and peroxide components to be mixed with the fluoride at the time of delivery. The product has been demonstrated to be safe,[19] and the low level of hydrogen peroxide does not present problems alleged to result from higher levels of peroxide in early animal studies.[20]

Multiple clinical studies of fluoride dentifrices containing NaF, MFP, or SnF_2 in the presence of compatible abrasives and stable formulations have been submitted to and been accepted by the ADA. The Association, therefore, awards the Seal of Acceptance to fluoride dentifrices based solely on laboratory data if they comply with previously submitted clinical data.[21]

The fluoride dentifrices currently accepted by the Council on Scientific Affairs of the American Dental Association are available at the ADA web site, www.ada.org. *Not all fluoride-containing dentifrices have demonstrated anticaries activity.* The level of active fluoride must be adequate and must be maintained over the shelf-life of the dentifrice. The Seal of Acceptance of the American Dental Association is one assurance of an active product. Fluoride dentifrices are discussed more extensively in Chapter 9.

Control of Plaque and Gingivitis

Most intriguing is the concept of *chemical plaque control,* in which chemical compounds are used to *supplement* the usual brushing, flossing, and use of auxiliary aids employed in mechanical plaque control. Antiplaque agents can act directly on the plaque bacteria or can disrupt different components of plaque to permit easier and more complete removal during toothbrushing and flossing. This opportunity to use chemistry to enhance oral hygiene procedures is important because manual plaque-control methods are difficult to teach and monitor, tedious to perform, time-consuming, impossible to accomplish by some physically and mentally handicapped persons, and not used by nonmotivated individuals.

The present chemical plaque-control agents should *not* be considered a panacea because they have not been proven to be a total substitute for routine oral-hygiene measures. *Excessive emphasis on chemical control may encourage some patients to deemphasize proven oral-hygiene methods.*

At the present time, an agent (or agents) analogous to fluoride in controlling caries is being sought to control plaque and gingivitis and to prevent periodontitis. The properties of an ideal form of such an agent are listed in Table 6-3. Although many OTC products are being marketed with plaque-gingivitis claims, only *two* dentifrices are currently marketed with *ADA-accepted* claims (see ADA website, www.ada.org). Both contain *triclosan* and will be discussed further in this chapter.

Stannous Salts

Stannous fluoride (SnF_2), specifically the stannous ion, has reported activity against caries, plaque, and gingivitis.[22] While SnF_2 has a long record as an anticaries agent, long-term stability in dentifrices and mouthrinses has been questioned since clinical antimicrobial activity has only been demonstrated in anhydrous products.[22] The development and subsequent laboratory and clinical efficacy of a stabilized SnF_2 dentifrice has been reported.[23,24] This dentifrice has been marketed in the United States by Procter & Gamble as Crest Gum Care. Clinical studies have been performed versus essential-oil mouthrinses, baking-soda/peroxide dentifrices, and triclosan-containing dentifrices with pyrophosphate/copolymer/zinc citrate. Superior efficacy has been shown for Crest Gum Care in antimicrobial, plaque acidogenicity, gingivitis or gingival bleeding, and calculus control.[25,26] However, long-term studies with this SnF_2 dentifrice demonstrated an increase in extrinsic stain attributed to the stannous ion.

Triclosan

Triclosan is a broad-spectrum antibacterial agent, marketed by its manufacturer, Ciba-Geigy, for use in oral products under the trade name Irgacare. It is effective against a wide variety of bacteria and is widely used as an antibacterial agent in OTC consumer products in the United States, including deodorant soaps and antibacterial skin scrubs. It has also been shown to be a useful antibacterial agent in oral products. A review of the available pharmacological and toxicological information concluded, "Triclosan can be considered safe for use in dentifrice and mouth rinse products."[27]

Many dentifrices and mouthrinses containing triclosan are marketed in Europe. In the United States, one dentifrice, developed by Colgate-Palmolive, contains triclosan, a patented copolymer, 'Gantrez', and fluoride. This product, Colgate Total, has undergone extensive safety[27] and clinical-efficacy testing.[28,29,30] and was approved in 1997 by the FDA as the first dentifrice to help prevent *gingivitis, plaque, and caries.* Colgate Total also has received the American Dental Association's Seal of Acceptance as an "effective decay preventive dentifrice, and to help prevent and reduce gingivitis when used as directed in a conscientiously applied program of oral hygiene and regular professional care. It has also been shown to help reduce the formation of plaque and tartar above the gum line. Its effect on periodontitis has not been determined." Two other "Colgate Totals" have been accepted; "Fresh Stripe" and a new formula making all of the above claims plus whitening. Recently, a 2-year study on Total has documented long-term anticaries efficacy.[31] Research on a triclosan-pyrophosphate combination in a dentifrice has demonstrated plaque *regrowth inhibition* and *anticalculus activity.*[32,33]

A Unilever product containing zinc citrate and triclosan has also received attention. Clinical evaluation has shown this to be effective in reducing plaque formation and in preventing gingivitis. A summary of the zinc citrate-triclosan studies has been published.[34] This product is not currently marketed in the United States.

Anticalculus Dentifrices

Calculus-control dentifrice formulations are designed to *interrupt the process of mineralization of plaque to calculus.* Plaque has a bacterial matrix that mineralizes due to the *super saturation* of saliva with calcium and phosphate ions. Crystal growth

inhibitors may be added to dentifrices to provide a reduction in calculus formation.

In the late 1970s, anticalculus dentifrices began to appear on the market without any evidence of effectiveness.[35] In 1985, Procter & Gamble supplemented their existent Crest anticariogenic toothpaste with a similar anticaries formula that also contained a combination of *tetra sodium phosphate and disodium dihydrogen pyrophosphate.* The soluble pyrophosphates are crystal growth inhibitors, which retard the formation of calculus.[36] This combination has been demonstrated in clinical studies to significantly reduce the amount of calculus formed, compared with a control dentifrice. The dentifrice is marketed as Crest Tartar Control. The formula received the American Dental Association's Seal of Acceptance, but only as a caries control product and only because of its fluoride content. Other similar anticalculus products that are now on the market all contain NaF.

A recent addition to the list of available products is a dentifrice with both a whitening and an anticalculus claim.[37] The product, "Colgate Tartar Control Plus Whitening Fluoride Toothpaste" contains tetrasodium pyrophosphate, sodium tripolyphosphate, a copolymer, and NaF.

Rolla and Saxegaard[38] have noted the possibility of "crystal poisons," such as pyrophosphates and phosphonates, inhibiting remineralization. Such inhibition might adversely affect the anticaries effect of the fluoride in this type of calculus control dentifrice. Zinc citrate trihydrate is used to inhibit calculus formation in the tartar control versions of both Aim and Close-Up. Clinical studies[39] have shown that *zinc citrate does not affect the caries inhibition of fluoride.*

Despite favorable anticalculus data, the ADA seal *has not been awarded* to products with only an anticalculus claim, because the ADA considers calculus inhibition as a cosmetic, not a therapeutic effect. With an anticalculus agent, two simultaneous beneficial effects—caries control and calculus inhibition—are available with one brushing operation. The currently marketed ADA accepted anticaries products that also inhibit calculus formation might be found at the ADA web site, www.ada.org (see Appendix 6-2).

Question 3

Which of the following statements, if any, are true?

A. Baking soda, peroxide, and fluoride are incompatible in a dentifrice.

B. The present consensus is that chemical-plaque control is only a supplement to mechanical-plaque control.

C. Stannous fluoride is both an anticaries and an antigingivitis agent.

D. Triclosan is an effective antibacterial agent.

E. Crest Tartar Control toothpaste has received the American Dental Association's Seal of Acceptance as a therapeutic anticalculus product.

Antihypersensitivity Products

Many people experience pain when exposed areas of the root, especially at the cemento-enamel junction, are *subjected to heat or cold.* To address this issue the ADA has formed the Ad Hoc Committee on Dentinal Sensitivity. Several OTC dentifrices have been accepted with the active agents such as *potassium nitrate, strontium chloride,* and *sodium citrate.*[40] Currently accepted products may be found on the ADA web site, www.ada.org (see Appendix 6-2). Potassium citrate has also been accepted by the British Department of Health.

The American Dental Association's Council on Dental Therapeutics has approved a dentifrice (Sensodyne F) with a combination of active ingredients, demonstrating both antihypersensitivity and caries-preventive benefits. This is another example of a therapeutic dentifrice directed simultaneously at solving two problems, caries and hypersensitivity, with the same brushing operation.

Whiteners

Considerable controversy surrounds the use of stain removers and tooth whiteners. Products are being marketed for professional use or for use by the patient at home. Many claims for efficacy and safety are under review by agencies and government panels. The ADA website should be consulted for a list of currently accepted products. Cosmetic benefits of dentifrices remain important to patients.

Surveys reveal a growing U.S. market share for dentifrices claiming "whitening" or "stain control." These dentifrices control stain via *physical methods* (abrasives) and *chemical mechanisms* (surface active agents or bleaching/oxidizing agents). Although the public perceives these as more abrasive than ordinary toothpastes, their abrasiveness is usually intermediate among the products tested.

Dentifrices marketed with tooth-whitener claims are available as a toothpaste or gel, or are used in a two- or three-step treatment "process." These products usually contain *hydrogen peroxide* or *carbamide peroxide* as their bleaching or whitening ingredient. Carbamide peroxide breaks down to form urea and hydrogen peroxide. Hydrogen peroxide, in turn, forms a free radical containing *oxygen,* which is the active bleaching molecule. Home-bleaching products may contain other chemicals to aid in the delivery of the *bleaching* agent. Glycerin or propylene glycol is commonly added to thicken the solution and prolong contact with the tooth surface. In the two- or three-step products, agents can be delivered to teeth via a custom-made tray or by toothbrushing.

There is concern that regular use of the peroxides or their breakdown products may enable overgrowth of undesirable organisms, including yeasts, possibly leading to "black hairy tongue." In addition, peroxides may damage the pulp or the soft tissues of the mouth. Delayed wound healing is also a concern, as is the possible mutagenic effect of strong oxygenating agents. The Food and Drug Administration has sent a regulatory letter to producers of those commercial tooth-whitening agents containing peroxides to inform them that these products are classified as drugs. The letter asked for information about possible side effects, such as delayed wound healing, periodontal harm, and mutagenic potential. The American Dental Association's

Council on Scientific Affairs has issued "Guidelines for the Acceptance of Peroxide Containing Oral Hygiene Products."[41]

A dentifrice (Crest Extra Whitening) has been introduced which uses a stain specific soft tissue technology.[42] This product, which contains NaF, claims calculus control activity as well as stain removal and an anticaries benefit.

Mouthrinses

Freshening bad breath has been the traditional purpose of mouthrinses. The 1992 market for such products was estimated at $635 million. Sales increased to $739 million in 2000. In addition to the traditional cosmetic use, therapeutic mouthrinses are now available.

The claimed active ingredients of mouthrinses include *quaternary ammonium compounds, boric and benzoic acids, and phenolic compounds.* As with dentifrices, commercial sales of cosmetic rinses have been related to taste, color, smell, and the pleasant sensation that follows use. The pleasant sensation is often enhanced by the addition of astringents. Commonly used astringents are alum, zinc stearate, zinc citrate, and acetic or citric acids. Zinc sulfate has been added to mouth rinses as a claimed antiplaque ingredient.

Alcohol in mouthrinses is used as a *solvent, a taste enhancer*, and an agent providing an *aftertaste.* The alcohol content of commercial rinses, ranging *up to 27%*, may constitute a danger for children, especially those from 2 to 3 years of age. According to the National Poison Center Network, 5 to 10 ounces of a mouthrinse containing *alcohol can be lethal for a child* weighing 26 pounds. Between 1987 and 1991, the nation's poison-control centers logged more than 10,000 reports of children younger than 6 years old drinking mouthrinses containing alcohol; *3 died* and another 40 had life-threatening conditions or suffered permanent injuries.[43] The American Academy of Pediatrics has recommended that OTC liquid preparations be *limited to 5% ethanol*, that safety closures be required, and that the packaged volume be kept to a "reasonable minimum to prevent the potential for lethal ingestion."[44]

The Council on Scientific Affairs of the American Dental Association requires child-resistant caps on all alcohol-containing mouth rinses that bear the Seal of Acceptance.[43] The council also requires manufacturers of ADA-accepted mouthrinses that contain more than 5% alcohol to include the following statement on the label: "Warning: Keep out of reach of children. Do not swallow. Contains alcohol. Use only as directed." The attorneys general of 29 states have petitioned the U.S. Consumer Products Safety Commission to require child safety caps on bottles of mouthwash that contain more than 5% alcohol.

Research from the National Cancer Institute (NCI) has *linked* alcohol and mouthwash to *mouth and throat cancers.*[45] After taking into account participants' smoking and drinking habits, it was found that cancer patients were more likely than the control group to have rinsed regularly with a high-alcohol (25% or more) mouthwash. The researchers concluded that alcohol may or may not cause cancer in and of itself but may promote the disease by dissolving and dispersing other cancer-causing substances within the mouth and throat. The ADA has stated, "According to a

statement from the NCI, it is premature to make recommendations about any alcohol-containing mouthwashes. In the meantime, the Association suggests that patients continue to use the therapeutic mouthrinses accepted by the ADA . . . and recommended by their dentists."[46]

Cosmetic Mouthrinses

Halitosis

Oral malodor has been a neglected research area. Indeed, the first scientific symposium on halitosis research was not held until 1991. Further research and education is needed in this important area because many practicing dental professionals still believe that bad breath usually comes from the stomach. Identifying the cause of halitosis and developing an appropriate treatment plan can be difficult.[47] Published studies by Spouge[48] and by Tonzetich[49] have demonstrated that oral malodor *usually derives from the mouth* itself and may be reduced following oral hygiene. To motivate improvement in oral hygiene, dental professionals should advise patients that bad breath might result from microbial putrefaction within the mouth. Rosenberg[50] notes, "Bad breath is a cause of concern, embarrassment, and frustration on the part of the general public. Oral malodor, whether real or perceived, can lead to social isolation, divorce proceedings, and even 'contemplation of suicide.' "

A body of science currently exists to permit the *quantitative assessment of bad breath*, which should be able to verify product claims for treating this important symptom. Many rinses have breath-freshening claims. Many claim breath freshening, but the effect is caused by flavors and has no effect after 3 to 5 hours. In diagnosing and treating complaints of bad breath, the clinician should consider psychological as well as physical factors.[51]

Xerostomia Mouth Rinses

Many people experience dry mouth (*xerostomia*) traceable to *several possible causes*, such as damage to the salivary glands following radiation therapy for head and neck cancer, Sjogren's syndrome, and use of tranquilizing drugs, especially the tricyclic antidepressants. In such cases the mucous membrane is continually dry and uncomfortable. To ameliorate the dryness, artificial salivas have been developed, which are used *ad libitum* by the patient to moisten the mucous membrane.

Because xerostomia is correlated with an *increased caries incidence*, the rinses usually contain *fluoride* as well as chemical compounds in concentrations that closely parallel those of saliva. The rinses that contain fluoride may, in reality, be *remineralizing solutions*. Several artificial salivas have been accepted by the ADA; among which are Salivart and Xero-Lube. For a current listing, consult the ADA website, www.ada.org. Several moisturizing agents are also available to xerostomia patients.[52]

Therapeutic Mouth Rinse Agents

Chlorhexidine

The FDA has approved prescription plaque-control rinses containing 0.12% chlorhexidine. Peridex (Omni Oral Pharmaceuticals) has received the ADA seal and Prevident (Colgate) is also marketed. Directions call for a twice daily, 30-second rinse with 1 oz of such solutions.

Chlorhexidine has proved to be one of the most effective antiplaque agents to date.[53] Chlorhexidine is a cationic compound that binds to the hydroxyapatite of tooth enamel, to the pellicle, to plaque bacteria, to the extracellular polysaccharide of the plaque,[54] and especially to the mucous membrane.[55] The chlorhexidine adsorbed to the hydroxyapatite is believed to inhibit bacterial colonization.[56] After binding, the agent is *slowly released in active form over 12 to 24 hours*.[57] This ability of the oral tissues to adsorb an active agent and to permit its slow release in active form over a prolonged period is known as *substantivity.* As the substantivity of an antiplaque agent decreases, the frequency of use needs to be increased.

Chlorhexidine has not proved beneficial as the *sole* method of treating periodontitis with deep pockets. Following root planing, prophylaxis, or periodontal surgery, chlorhexidine irrigation may be effective in helping to control inflammation and subgingival plaque.[58]

In some countries, such as the United States, chlorhexidine products are available only by prescription. In others, such as the United Kingdom, they are available over the counter. Although chlorhexidine is quite effective, it is not active against all relevant anaerobes. A high minimal concentration is necessary for efficacy. Some side effects are associated with chlorhexidine use, of which stain is the most common.[59] Occasionally *altered taste* sensation is reported.[60] *Increased calculus* formation,[61] *superficial desquamation of tissue,* and *hypersensitivity* have also been noted.[62,63] Chlorhexidine is inactivated by most dentifrice surfactants and, therefore it is not included in dentifrices. Also, because of this inactivation, it is *critical for dental professionals* to alert patients not to use chlorhexidine mouthrinses within 30 minutes before or after regular toothbrushing.

Although chlorhexidine may be more effective than any other current antiplaque agent and has a definite role in preventive and control dental procedures, it is not a "magic bullet." Its *side effects* and inadequate activity range somewhat limit its use.

Essential Oils

Listerine antiseptic was the first OTC antiplaque and antigingivitis mouth rinse to be approved by the ADA.[64] Patients are advised to rinse twice daily with 20 mL of Listerine for 30 seconds, in addition to their usual oral-hygiene regimen. Listerine has been used as a mouthrinse for more than 110 years. The active ingredients are *thymol, menthol, eucalyptol, and methyl salicylate,* termed *essential oils.* The original formula contains *26.9% alcohol.* A flavor variation of the product, Cool Mint Listerine Antiseptic, which also has received the ADA seal, contains 21.6% alcohol. Microorganisms *do not* develop a resistance to the antibacterial effects of essential oils, such as clove oil (eugenol) and thyme oil (thymol).[65] In long-term clinical trials, Listerine has been shown to *reduce* both plaque accumulation and severity of gingivitis by up to 34%.[66] Microbial sampling of plaque in these trials has demonstrated no undesirable shifts in the composition of the microbial flora.

Based on laboratory testing, more than 200 generic versions of original Listerine have also been granted the ADA seal and are marketed under numerous trade names. These may be found at the ADA website, www.ada.org.

A recent study evaluated the comparative efficacy of Listerine and an antiplaque/antigingivitis dentifrice (Colgate Total).[67] When used in conjunction with a fluoride dentifrice and usual oral hygiene, Listerine was reported to provide a greater benefit in reducing plaque than did Colgate Total.

As with chlorhexidine, rinsing with an essential oil mouth rinse *per se* is unlikely to be effective in treating periodontitis because the solution does not reach the depths of the periodontal pockets. Irrigation studies, using irrigator tips designed to deliver solutions subgingivally, suggest that Listerine and Peridex may have some value as adjuncts to mechanical therapy.

For the dental professional, it *may be important* for patients to use a mouthrinse prior to aerosol-generating procedures. Unless an effective dry-field technique is used, the bacterial aerosol generated by a high-speed turbine in a 30-second period is roughly equivalent to the patient sneezing in the dentist's face.[68] A study by Wyler and coworkers[69] found that even a preliminary water rinse temporarily reduced the bacterial aerosol population by 61%, brushing alone by 85%, and an antibacterial mouthrinse by 97%. Fine and coworkers,[70] using a simulated office visit model, showed that *preprocedural* use of an antimicrobial mouth rinse (Listerine) resulted in a 93.6% reduction in the number of viable bacteria in a dental aerosol produced by ultrasonic scaling. The effect of this reduction on actual disease transmission has not been determined.[71]

Fluoride Rinses

The active agents in over-the-counter fluoride mouthrinse products are sodium fluoride (NaF) or acidulated phosphofluorides (APF) at concentrations of 0.05 and 0.44%, respectively. The dose directions are 10 ml of product to be used *once daily*.

Published long-term clinical studies have consistently shown anticaries effectiveness *equal or superior* to fluoride dentifrices. The ADA website, www.ada.org, currently lists seven nonprescription fluoride-containing mouthrinses that have received the Seal of Acceptance for anticaries effectiveness.

Question 4

Which of the following statements, if any, are correct?

A. Xersostomia mouthrinses are usually formulated to prevent demineralization and promote remineralization.

B. The ADA has awarded its Seal of Acceptance to a whitener-containing carbamide peroxide.

C. The agent in cosmetic mouthrinses that poses the greatest danger to 2- to 4-year-

old children is alcohol.

D. Chlorhexidine mouthrinses are most effective when used immediately after brushing.

E. Fluoride-containing mouthrinses are not as effective as fluoride-containing dentifrices for anticaries activity.

Chewing Gum

Because gum chewing is pleasurable, people normally chew for longer periods of time than they spend brushing their teeth. Likewise, gum may complement toothbrushing by reaching many of the tooth surfaces commonly missed during brushing. The average American fails to contact approximately 40% of tooth surfaces during toothbrushing, especially the posterior teeth and lingual surfaces. Regular toothbrushing removes only about 35 to 40% of dental plaque present on tooth surfaces. In addition, chewing gum is especially advantageous during the course of the day when toothbrushing is not possible or convenient.

Beneficial effects of gum chewing include *increased saliva* production resulting in the mechanical removal of dental plaque and debris. Studies have shown that chewing sugared or sugar-free gum is an effective means of reducing plaque accumulation and that gum chewing can also effectively reduce established plaque on many tooth surfaces (see Appendix 6-3).

Since 1997, three major review articles devoted solely to chewing gum and potential oral health benefits have been published.[72-74] The interest of researchers in effective gum additives coupled with the acceptance and use of chewing-gum products by the general public makes this a new and potentially important category to be considered by dental professionals. In 1999, the worldwide chewing-gum market was estimated to be 560,000 tons per year, or approximately 5 billion U.S. dollars.[74]

During gum chewing, salivary flow rates increase, especially in the first few minutes, because of *both mechanical and gustatory stimulation.* Increased salivary stimulation can continue for periods of *5 to 20 minutes,* usually until the flavor(s) in the product dissipates. However, even with unflavored chewing gum, saliva flow, as evidenced by swallowing rates, increase over baseline.[75] The beneficial effects of additional saliva in the mouth include increased *buffer capacity* and *mineral super saturation,* both of which help regulate or increase plaque pH, and increase plaque calcium levels (pCa).[76] In addition, increased saliva flow can assist in loosening and removing debris from occlusal or interproximal sites, and can be beneficial to xerostomia patients.

The focus of chewing gum research to date has been on "sugar-free" products,[77] which contain polyol sweeteners such *as sorbitol or xylitol.* These sweeteners are not broken down by plaque or oral microorganisms to produce acid. Plaque pH studies have documented reduction of plaque acidity and maintenance of plaque neutrality both during and, with xylitol, for periods of 2 to 3 weeks following, gum chewing.[73] In addition, gums containing xylitol have shown *anticaries* activity in several *long-term studies.*[73] Chewing a *sorbitol-based chewing gum* after meals significantly *reduced dental caries* incidence in a three-year study.[78]

Studies[79] have shown that a commercial chewing gum containing 5% sodium bicarbonate (Arm and Hammer Dental Care) is capable of removing significant amounts of plaque and reducing gingivitis when used as an adjunct to regular toothbrushing. Stain removal is also of interest to the consumer. Studies simulating a realistic situation (twice-daily brushing and unsupervised use of a baking soda chewing gum) demonstrated *reduction in stain* after four weeks.[79]

Consumers have relied on chewing-gum products for "fresh breath." A recent report on reducing volatile sulfur compounds associated with oral malodor and organoleptic scores indicates that the products tested are effective primarily as masking agents (flavor) and for the mechanical role in cleaning tooth surfaces. Reduced malodor levels were obtained during initial use of the products, but decreased to baseline levels at the three-hour assessment times.[80]

Reynolds[81] has proposed the introduction of *casein phosphopeptide* to chewing gum as a mechanism to *remineralize* early carious lesions. In situ studies appear promising.[82] Trident Advantage, with Recaldent, makes use of this technology.

An overview of selected agents added to chewing gums is presented in Appendix 6-3. Compounds such as chlorhexidine and fluorides would appear to be useful when delivered using chewing gum as the vehicle, since there would be a minimum of potentially interfering agents in the gum product (compared to abrasives in dentifrices and water and alcohol vehicles in mouthrinses), as well as a sustained time of release and availability in the oral cavity. In addition, the active agents would be available at occlusal sites, which are prime areas for plaque growth and pit and fissure decay. Neither of these agents is available in the United States. Since chewing-gum products are often in the mouth several times a day, the concentration of ingredients released (especially fluoride) *must be safe for swallowing.*

Question 5

Which of the following statements, if any, are correct?

A. Saliva flow rates increase while using chewing gum.

B. An increased saliva flow would help dilute any acids in the plaque.

C. Xylitol and sorbitol flavoring of chewing gum is ideal because both are anticariogenic.

D. A chewing gum with 5% sodium bicarbonate has been demonstrated to reduce stain and reduce gingivitis.

E. Casein phosphopeptide appears to be a promising agent in chewing gum for enhancing remineralization.

Summary

The self-use of dentifrices and mouthrinses is proving to be an important preventive

dentistry measure.

Dentifrices, mouthrinses, and chewing gums can be categorized as either cosmetic or therapeutic. Cosmetic products have traditionally been used to remove debris, provide a pleasant "mouth feel," and temporarily reduce halitosis. To improve on their marketability, flavors, stripes, sprinkles, and colors have been added to dentifrices and mouthrinses. Recently, other ingredients have also been added to temporarily depress the oral bacterial population or to prevent or moderate some disease process in the mouth.

The widespread use of therapeutic fluoride dentifrices and mouthrinses is credited with helping to reduce the worldwide prevalence of dental decay. All products carrying the ADA Seal also contain fluoride.

Other agents are now being used to target other oral-health problems.

The Food and Drug Administration has developed rigid guidelines for testing the safety and efficacy of products prior to their introduction on the market. Part of the function of the regulatory process is to differentiate between products whose potential risks are sufficiently low to allow them to be sold over the counter and those whose possible hazards justify restriction to prescription use.

While the ADA considers antiplaque, anticalculus, and breath-freshening claims as cosmetic, they will review data and allow manufacturers to make these statements, if coupled with a disease related activity (e.g., prevents gingivitis or caries). Toothpastes containing potassium nitrate, strontium chloride, and sodium citrate have antihypersensitivity properties; other toothpastes with tetrasodium phosphate and disodium dihydrogen pyrophosphate retard the formation of calculus. Chlorhexidine is a highly effective antiplaque, antigingivitis agent, accepted by the ADA, but with significant side effects and may only be dispensed on prescription. Listerine, containing essential oils, has been popular for over a century, and has demonstrated the same properties but without the side effects of chlorhexidine. More than 200 generic versions of Listerine, containing essential oils, have been accepted by the ADA for plaque and gingivitis claims.

Chewing gum products are a new dental category in which manufacturers are making claims for cosmetic and therapeutic effectiveness. At this time, neither the ADA nor the FDA has approved any chewing gum products for dental therapeutic claims.

Answers and Explanations

1. A, C, and D—Correct

B—incorrect. The rulings by the FDA have the force of law; the rulings by the ADA are advisory to the profession and public.

E—incorrect. All actions to secure the Seal of Approval are voluntary. The manufacturer is not committed to apply for it, or to use it in advertising. However, to use it for marketing purposes is to the advantage of any manufacturer having received it.

2. A, B, C, D, and E—correct.

3. B, C, and D—correct.

A—incorrect. Under some circumstances, it is possible they are incompatible. However, the fact that there is now a dentifrice called Mentadent is proof that peroxide, baking soda, and fluoride can be compatible.

E—incorrect. Remember, the ADA awards the Seal of Acceptance for therapeutic products, and not for cosmetic agents. Calculus is considered a cosmetic blight.

4. A, C—correct.

B—incorrect. This is another example of the ADA policy of not awarding the Seal of Acceptance for a cosmetic product.

D—incorrect. Since chlorhexidine is inactivated by most dentifrice surfactants, a period of about 30 minutes should elapse between toothbrushing and chlorhexidine mouthrinsing.

E—incorrect. The fluoride mouthrinses are probably better than fluoride dentifrices, possibly because they allow better access to caries-prone interproximal locations.

5. A, B, C, D, and E—correct.

Self-evaluation Questions

1. Name at least four tooth and gum conditions for which formulas have been developed to help prevent or control: _____, _____, _____ and _____.

2. The _____ is the name of the award given by the ADA to dental manufacturers who have prepared a therapeutic product that is safe and efficient.

3. There are three levels of concern for the safety and efficiency of prescription and over-the-counter dental products. The government level includes the Food and Drug administration and the _____; the second level is voluntary professional oversight assumed voluntarily by the _____, while the third level is by _____ advocates.

4. The two mouthrinses granted the Seal of Acceptance by the ADA as antiplaque and antigingivitis are _____ and _____.

5. Two factors that can decrease or enhance the abrasiveness of a toothpaste are: _____ and _____.

6. The difference between an abrasive and a polishing agent is _____; when the two are mixed together, they constitute an _____.

7. The three fluoride compounds most used in fluoride dentifrices are _____, _____, and _____.

8. Three properties of a dentifrice or a mouthrinse that do not contribute to the therapeutic or cosmetic effects, but must be considered because of marketing necessities are _____, _____, and _____.

9. The agent added to a toothpaste formula to preserve the moisture is called a _____.

10. A toothpaste formula where the effective agent is solely baking soda is a (cosmetic)(therapeutic) dentifrice.

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Chapter 7. Oral Health Self-Care Supplemental Measures to Complement Toothbrushing - *Terri S. I. Tilliss Janis G. Keating*

Objectives

At the end of this chapter, it will be possible to

1. Explain the reasons why supplemental oral health self-care is needed to complement toothbrushing.

2. Identify factors, in addition to oral conditions, that influence selection of supplemental oral hygiene devices and techniques.

3. State the purposes, indications, contraindications, techniques, advantages, and limitations of the following oral hygiene devices:

- Dental floss,
- Dental floss holder,
- Dental floss threader,
- Wooden or plastic triangular stick,
- Toothpicks and holder,
- Interproximal brush or devices,
- Tongue cleaners,
- Others: yarn, rubber or plastic tip, gauze, automated interproximal devices.

4. Justify the purpose and explain techniques for the use of mouthrinses and oral irrigators.

5. Describe proper oral hygiene self-care for dental implants.

6. Explain proper oral hygiene self-care for removable partial and full dentures.

Introduction

Supplemental plaque removal measures beyond toothbrushing are necessary in order to thoroughly remove plaque.[1,2,3] Although toothbrushing can be effective at removing the plaque residing on buccal and lingual aspects of teeth, it is generally ineffective for interproximal surfaces.[4,5] There are numerous intraoral sites and conditions better served by plaque removal methods and devices other than toothbrushing. Examples of these sites include, fixed prostheses, crown margins, furcations of multirooted teeth, orthodontic appliances, the tongue, implants, and dentures.

Interproximal aspects of teeth are not very accessible for the removal of plaque by the toothbrush.[1] These sites have consistently been shown to harbor high amounts of plaque.[6-8] Regular interproximal plaque removal is recommended for the following reasons:

- Incomplete plaque removal can increase the rate and growth of new plaque.[9]
- Allowing plaque to remain on some tooth surfaces can facilitate development of a complex microflora on other cleaned surfaces.[10,11]
- Individuals who clean interproximally on a daily basis have less plaque and calculus.[12]
- Interproximal plaque removal is beneficial for preventing gingival and periodontal infections as well as for reducing or eliminating diseases in these soft tissues.[1,13,14]
- There is interproximal site predilection for gingivitis, periodontitis, and caries.[1,15,16]
- Prevention of dental caries can be facilitated by effective daily interproximal plaque removal.[17]

The dentition or periodontal tissues can be altered as a result of disease, repair, or from architectural tissue changes following therapy. When this occurs, a device and/or technique must be introduced to accommodate these changes. It has been shown that supragingival plaque removal influences subgingival plaque composition, however, plaque removal efforts should extend as far subgingivally as possible.[10,11,18]

To determine the most appropriate products and practices for interproximal plaque removal the *Process of Care Model* is useful (Figure 7-1). This treatment model is described in detail in the textbook, *Dental Hygiene Theory and Practice*.[19] The oral health professional must carefully assess numerous oral health and disease risk factors. These include current oral self-care practices, and past and current oral health status. The importance of this *assessment* phase cannot be overemphasized. During the *diagnosis* and *planning* phases, the risk factors and the appropriate oral self-care products and procedures to address these risks are jointly determined with the individual. The oral health professional can then apply theory-based educational and motivational strategies during the *implementation* phase to facilitate behavioral change. Ensuring that such behavioral changes are consistent with the lifestyle of the individual will increase the potential for long-term compliance of oral self-care practices. The *evaluation* phase focuses upon outcomes to determine whether modifications to the oral hygiene strategies are indicated. Continuation or change of an oral hygiene regimen is based upon the evaluation of tissue health. The evaluation process is continuous over the lifespan as the dentition and soft tissues may become altered with time.

A personalized oral hygiene regimen will best meet the needs of the individual. When employing an interproximal cleaning technique, a systematic approach following prescribed techniques will enhance plaque removal without causing soft tissue damage. The oral hygiene self-care recommendations, which have been agreed upon by the individual and the oral health care provider, are documented in the dental chart and modified as necessary at subsequent re-care visits. This type of documentation allows for continuity of care.

Figure 7-1 Phases of the Process of Care Model

Oral Health Self-Care

Self-care includes all activities and decisions of an individual to prevent, diagnose, or treat personal ill health. This concept as applied to care of the oral cavity is referred to as oral self-care or oral health self-care, replacing earlier terms such as personal plaque control, home care, and oral physiotherapy. One primary purpose of oral health self-care is to prevent or arrest periodontal disease and caries by reducing plaque accumulation.[5] Less than optimal oral self-care is regarded as a major risk factor for periodontal disease. In order to determine the most appropriate self-care practices for each individual, a variety of factors must be assessed:

- Presence of gingival inflammation and bleeding,
- Alterations of the interdental gingival architecture caused by tooth alignment, spacing, recession, and lack of attached tissue,
- Malalignment of teeth and tooth morphology,
- Configuration of embrasure spaces,
- Extent and location of plaque and calculus accumulation,
- Caries experience and susceptibility,
- Evidence and risk factors for periodontal diseases,
- Trauma from improper use of oral-hygiene devices,
- Current oral self-care practices and level of manual dexterity/mental capacity,
- Compliance potential, and
- Presence, configuration, and condition of restorations.

There is no universally accepted oral hygiene device. The appropriate oral hygiene regimen is determined according to the dictates of the oral condition, personal preferences, dexterity, and lifestyle.[16] Adequate instruction in the use of any recommended device must be provided.

Additional Considerations: Plaque and Caries

Utilizing an *evidence-based* approach to understand oral disease, several conclusions can be drawn. Little data supports the theory that interproximal plaque removal alone reduces the incidence of caries. One reason for this is that the ubiquitous use of fluoride makes it difficult to separate out the benefits of fluoride from that of interproximal plaque removal. One study did demonstrate that interproximal caries could be prevented when daily interproximal flossing is performed by an oral health professional.[17] However, other studies of supervised self-performed interproximal

cleaning were unable to demonstrate a caries reduction.[20-23] There are several studies documenting the correlation between the general level of mechanical plaque removal and the incidence of caries.[24-26] It appears that only a very high level of personal mechanical plaque removal impacts the caries rate. This level is difficult for the average person to sustain.[27] Consequently, fluorides and dietary carbohydrate control should be emphasized, in addition to interproximal plaque removal for optimal effect on the caries rate.

Plaque and Gingivitis

It has been shown that removing plaque once every 48 hours is sufficient to reduce microbial plaque accumulations that are mature enough to induce gingival inflammation.[6,28] For those with existing inflammation or periodontitis, every 48 hours is not frequent enough.[1] Under these conditions it has been shown that colonization and maturation of plaque occurs more rapidly in the presence of, than in the absence of inflammation.[4,6,29,30]

Frequency of Plaque Removal

The preceding information describing the relationship between plaque and caries and plaque and gingivitis, suggests that the optimal frequency for mechanical plaque removal is not precisely known. Based on the one study correlating caries with daily flossing,[17] it seems advisable to remove interproximal plaque at least once every 24 hours for caries prevention. Likewise, the evidence-based approach suggests that those with an existing gingivitis or periodontitis should remove interproximal plaque on a daily basis. However, those with healthy gingiva may be able to practice interproximal plaque removal only once every 48 hours. Therefore, with more daily attempts at plaque removal, it is more likely that the additive efforts will maximize the removal of plaque. Since the ideal frequency of interproximal plaque removal has not been shown, individual factors such as the amount of inflammation, caries susceptibility, plaque removal efficiency, and accumulation and virulence of plaque must be considered in the recommendation.

Interestingly, although 94% of oral health professionals and researchers attending a symposium on mechanical plaque removal believed interproximal cleaning was an essential component of a successful oral self-care program, only 51% believed it was needed on a daily basis. Only 77% percent felt that interproximal cleaning should be advised for the whole dentate population rather than just for those deemed susceptible to periodontal disease and caries.[31] Determining the risk factors that increase one's susceptibility to caries and periodontal diseases could identify which individuals are in need of consistent interproximal cleaning, and at what frequency.

Supervised oral self-care practice sessions promote proper utilization of oral hygiene devices by providing an opportunity to monitor technique. Adjustment of technique can maximize plaque removal while minimizing tissue damage. After such instruction and reinforcement, success in oral health measures ultimately rests with the individual. Principles of learning and motivation should be applied to encourage compliance (see Chapter 16).

Question 1

Which of the following statements, if any, are correct?

A. Supplemental plaque removal is useful for individuals with orthodontic appliances.

B. Plaque allowed to remain on interproximal surfaces will not impact plaque accumulations on clean surfaces.

C. The Process of Care Model begins with the evaluation phase.

D. Self-care as it relates to health includes all activities and decisions individuals make about their health.

E. A large body of research supports the theory that interproximal plaque removal alone reduces the incidence of caries.

Dental Floss

Dental floss is best indicated for plaque and debris removal from Type I embrasures where the papilla fills the interproximal space and the teeth are in contact. For Type II and III embrasures, devices other than floss may be more effective in removing plaque[32,33] (Figure 7-2). Effective use of dental floss accomplishes the following objectives.

1. Removes plaque and debris that adheres to the teeth, restorations, orthodontic appliances,[34] fixed prostheses and pontics,[35] gingiva in the interproximal embrasures,[36] and around implants.[37,38]

2. Aids the clinician in identifying the presence of interproximal calculus deposits, overhanging restorations, or interproximal carious lesions.

3. May arrest or prevent interproximal carious lesions.[17]

4. Reduces gingival bleeding.[1,39]

5. May be used as a vehicle for the application of polishing or chemotherapeutic agents to interproximal and subgingival areas.[39]

Not all interproximal contact areas, whether natural or restored, have the same configuration. Consequently, several types of floss are available to accommodate these differences. These vary from thin unwaxed varieties, to thicker waxed tapes and include variable thickness floss (Figure 7-3). Clinical trials have shown no significant differences in the cleansing ability between waxed and unwaxed floss.[1] Wax residue has not been found on tooth surfaces cleaned with waxed floss.[40]

Unwaxed floss is frequently recommended because it is thin and slips easily through tight contact areas. However, unwaxed floss can fray and tear when contacting rotated teeth, heavy calculus deposits, or defective and overhanging restorations. Frequent floss breakage may discourage continued use. For these conditions, waxed, lightly waxed, or shred-resistant floss are recommended.

Waxed dental tape, unlike round dental floss, is broad and flat, and may be effective in an interproximal space without tight contact points. Additional types of floss, such as those made of polytetrafluoroethylene (PTFE, teflon-like), are stronger and more shred-resistant. They have been shown to be preferred by those who have tight contacts or rough proximal tooth surfaces.[41] Other varieties, such as tufted floss increments alternated with standard floss, and floss which stretches for insertion are alternatives.

Some brands of dental floss and tape are colored and flavored. In addition to increased appeal, color provides a visual contrast to plaque and oral debris, thus enabling one to see what is being removed, possibly increasing the motivation to floss. One study indicated a user preference for waxed over unwaxed floss and mint-flavored waxed floss over plain waxed floss.[42] Flosses impregnated with a variety of agents have been introduced; examples of these include floss treated with baking soda, fluoride, herbal extracts, antimicrobial agents, or abrasives for whitening. Fluoride-impregnated floss has been *marketed* but lacks efficacy data for affecting the caries rate.

One type of variable-thickness floss has a stiff end to allow for threading under bridges, beneath tight contact areas, under pontics, through exposed furcations, and around orthodontic wires (Figure 7-4). This floss combines a section of unwaxed floss with an area of thicker nylon meshwork to clean larger surface areas. Variable thickness floss may be recommended for use in cleaning implant abutments, areas with open contacts, wide embrasures, or sites where recession and bone loss permit access to furcations. It can also be used to remove plaque from the distal aspect of the most distal tooth in all quadrants.

When recommending a type of floss, the specific oral conditions, patient preference, and ability are all factors that need to be considered. A limitation of flossing is the inability to conform to a concave interproximal surface such as the mesial of maxillary premolars. Other interproximal devices will clean those surfaces more effectively (Figure 7-5).

Dental Flossing Methods

Two frequently used flossing methods are the spool method and the circle, or loop, method. Both facilitate control of the floss and ease of handling. The spool method is particularly suited for teenagers and adults who have acquired the necessary neuromuscular coordination required to use floss. The loop method is suited for children as well as adults with less nimble hands or physical limitations caused by conditions such as poor muscular coordination or arthritis. Flossing is a complex skill, so until children develop adequate dexterity, usually around the age of 10 to 12 years, an adult should perform flossing on the child. Younger children whose teeth still exhibit primate spaces (no interproximal contact) will not require flossing.

When using the spool method, a piece of floss approximately 18 inches long is utilized. The bulk of the floss is lightly wound around the middle finger. Space should be left between wraps to avoid impairing circulation to the fingers (Figure 7-6 A). The rest of the floss is similarly wound around the same finger of the opposite hand. This

finger can wind, or "take up," the floss as it becomes soiled or frayed to permit access to an unused portion. The last three fingers are clenched and the hands are moved apart, pulling the floss taut, thus leaving the thumb and index finger of each hand free (Figure 7-6 B). The floss is then secured with the index finger and thumb of each hand by grasping a section three quarters to 1 inch long between the hands (Figure 7-6 C).

For the loop method, the ends of the 18-inch piece of floss are tied in a knot. All of the fingers, but not the thumbs of the two hands are placed close to one another within the loop (Figure 7-7). Whether using the spool or the loop method of flossing, the same basic procedures are followed. The thumb and index finger of each hand are used in various combinations to guide the floss between the teeth.

When inserting, floss, it is gently eased between the teeth with a seesaw motion at the contact point. The gentle seesaw motion flattens the floss, making it possible to ease through the contact point and prevent snapping it through, thus avoiding trauma to the sulcular gingiva (Figure 7-8 A). Once past the contact point, the floss is adapted to each interproximal surface by creating a C-shape. The floss is then directed apically into the sulcus and back to the contact area (up-and-down against the side of the tooth) several times or until the tooth surface is clean (Figure 7-8 B). The procedure is repeated on the adjacent tooth in the proximal area, using care to prevent damage to the papilla while readapting to the adjacent tooth. A clean, unused portion should be used for each interproximal area.

In general, flossing is best performed by cleaning each tooth in succession, including the distal surface of the last tooth in each quadrant. The individual should be assisted with problem areas and encouraged to utilize whichever method produces the best results.

Criteria for evaluation are based on the efficacy of plaque removal and safety of the flossing method. Incorrect flossing can often be detected through clinical observation of the gingiva and the technique (Figure 7-9). Signs that suggest incorrect use of dental floss include gingival cuts, soft tissue clefting, and cervical wear on interproximal root surfaces. (Figures 7-10, 7-11) If flossing trauma is evident, further instruction should be given until the individual has become adept. Proper instruction and practice allows most motivated adults to master either the spool or loop method of flossing. In certain circumstances, the use of a floss holder, floss threader, variable-thickness floss, or pre-cut floss strands with a stiff end may be more effective.

It is important to note that a flossing habit has traditionally been difficult for people to embrace. In reality, only a very small proportion of individuals practice daily flossing. Findings have ranged from 10 to 21% of population.[43-48] Floss may be superior to other interproximal cleaning methods, but for those who have not or will not adopt a flossing behavior another interproximal device may be more effective than no interproximal cleaning.[49] A less effective device used on a regular basis is superior to sporadic use of a more effective device. Participation of the individual in selecting an interproximal cleaning device and regimen is crucial to improving and/or enhancing compliance. Sometimes individuals agree to adopt a behavior because this is what the clinician wants to hear. Axelsson has referred to this as "a hasty affirmative in a moment of suddenly inspired courage."[43]

Dental Floss Holder

The floss holder is a device that eliminates the need for placing fingers in the mouth. It is recommended for individuals with:

- Physical disabilities,
- Poor manual dexterity,[50]
- Large hands,
- Limited mouth opening,
- A strong gag reflex, and/or
- Low motivation for traditional flossing.[50]

The floss holder may also be helpful when one person is assisting another with flossing. Limited scientific data comparing finger-manipulated flossing to the use of a floss holder shows no difference in plaque removal.[47] Studies have found that, when compared, a significant majority of individuals preferred the floss holder over finger-manipulated flossing.[47,51,52] It should be emphasized that effective initial education and reinforcement are necessary for proper use of the floss holder. Use of the floss holder may aid in developing a flossing habit and should be considered when individuals experience difficulty with manual flossing.[53]

A variety of different floss-holder designs are available (Figure 7-12). Most commonly, they consist of a yoke-like device with a 3/4- to 1-inch space between the two prongs of the yoke. The floss is secured tightly between the two prongs and the handle is grasped to guide the floss during use. The width and length of the handle are important features to consider when recommending the use of a floss holder to those with limited gripping abilities.[54] Most floss holders require that floss be strung around various parts of the holder prior to each use. This assembly mechanism allows for re-threading of the floss whenever the working portion becomes soiled or begins to fray. Some devices have a floss reservoir in the handle. This improvement allows for ease of threading and advancing the floss while maintaining the proper tautness. Several brands of pre-threaded, one-time-use floss holders are available; they require minimal dexterity, a factor that may help improve compliance.

When using a holder, the floss is inserted interproximally, using the same technique employed for finger-manipulated flossing. Once through the contact point, the floss and holder are pushed distally to clean the mesial surface of a tooth or pulled mesially to clean the distal surface (Figure 7-13). This pulling or pushing motion creates conformity to the tooth convexities, thus allowing the floss to slide apically into the sulcus. The floss is then activated in the same manner as with finger-manipulated flossing, by moving the floss in the direction of the long axis of the tooth.

Strict attention should be given to achieving the desired floss tension when assembling the floss holder. To ensure tautness, the prongs can be forced together while securing the floss. The most persistent problems with the yoke-like devices are the difficulties in loading and threading the floss, maintaining tension of the floss between the prongs and decreased ability to adapt the floss into a C-shape around the proximal surface. Any device recommended should allow for ease of threading, maintenance of proper tautness, and easy manipulation by the user.

Automated floss holders have been introduced but have not shown an advantage over manual flossing.[55] However, those with large diameter handles may be especially helpful for patients with limited manual dexterity and inability to grip a smaller diameter (Figure 7-14).

Dental Floss Threader

A floss threader is a plastic loop into which a length of floss is inserted, similar to threading a needle. The threader is used to carry the floss interproximally in the following circumstances:

- Through embrasure areas under contact points that are too tight for floss insertion,
- Between the proximal surface and gingiva of abutment teeth of fixed prostheses,
- Under pontics,
- Around orthodontic appliances, and
- Under teeth that are splinted together.

Care should be taken to prevent trauma by *not* forcing the stiff end of the floss threader into the gingival tissues. When cleaning under a fixed partial denture, the floss threader is inserted from the facial and pulled completely through to the lingual aspect until the floss is against the abutment or pontic (Figure 7-15 A). The floss may then be disengaged from the threader. The floss is adapted to one abutment tooth surface in the area of the embrasure (Figure 7-15 B) and moved in the direction of the long axis of the tooth to remove plaque from the proximal surface. It is important to glide the floss through the space between the pontic and the gingiva in order to clean the underside of the pontic (Figure 7-15 C). After cleaning the underside of the pontic, it is necessary to slide the floss to the opposite proximal surface (Figure 7-15 D). Removal of the floss from between the abutment and pontic is accomplished by pulling it out from the facial aspect.

Figure 7-2 Embrasure Types. a. Type I—papilla fills interproximal space. B. Type II—slight to moderate recession of papilla. C. Type III—extensive recession or complete loss of papilla.

Figure 7-3 Varieties of Floss. a. Dental tape. B. Waxed. C. Unwaxed. D. Variable thickness floss.

Figure 7-4 Variable thickness floss with stiff end used to clean under the pontic and at interproximals of abutments of a fixed partial denture. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Figure 7-5 Dental floss is not as effective in cleaning teeth with interproximal root concavities since it does not contact the surfaces of the concavity. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Figure 7-6 Spool method for dental flossing. A. Floss is lightly wound and spaced around the middle finger of each hand. B. The last three fingers are clenched, pulling the floss taut and leaving the index finger and thumb of each hand free. C. The floss is held with the index finger and thumb of each hand by grasping a section three quarters to 1 in. long between the hands. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Figure 7-7 Loop method of dental flossing. A. All fingers except the thumbs are placed within the loop for easy maneuverability. B. For the mandibular teeth, the floss is guided with the two index fingers. C. For the maxillary teeth, the floss is guided with two thumbs or one thumb and one index finger. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Figure 7-8 A. To insert, the floss is gently eased between the teeth, while sawing it back and forth at the contact point. B. the floss is directed into the sulcus and back to the contact area several times or until the tooth surface is clean. (Courtesy of John O. Butler Co., Chicago, IL.)

Figure 7-9 Improper dental floss technique with potential for gingival "floss-cuts." (Floss should adapt to interproximal surface in a C-shape.)

Figure 7-10 Gingival "floss-cuts" created by failure to adapt floss to interproximal surface in a C-shape.

Figure 7-11 Groove created on mesial aspect of tooth (arrow) caused by movement of floss in bucco-lingual (horizontal) rather than apical-occlusal (vertical) direction.

Figure 7-12 A variety of floss holders. The first three depicted in photograph are pre-threaded for one-time use. The last floss holder exhibits a floss-reservoir in handle.

Figure 7-13 Correct use of floss holder on mesial aspect of tooth. Note that floss is taut, pulled mesially to adapt in C-shape, and extended subgingivally.

Figure 7-14 Automated floss holder. Note large diameter handle for easier grasping.

Figure 7-15 Use of a dental floss threader. A. Floss threader inserted under pontic. B. Floss adaption to mesial of abutment. C. Floss underneath pontic. D. Floss adaption to distal of abutment. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Question 2

Which of the following statements, if any, are correct?

A. Waxed dental floss is better for removing interproximal dental plaque than unwaxed.

B. A major problem encountered with the dental floss holder is maintaining tautness of the floss.

C. The spool method of using interdental floss is the preferable technique for introducing children to flossing.

D. Individuals indicate a preference for the floss holder, but it is not as effective as flossing with the spool or loop methods.

E. Only about 70% (plus or minus 5%) of the people who routinely brush, floss.

Wooden or Plastic Triangular Sticks

Interproximal cleaning can be facilitated using sticks made of wood or plastic (Figure 7-16). Balsa and birchwood are most common since they are pliable. A reduction in inflammation and bleeding sites has been demonstrated utilizing wooden or plastic sticks to reduce plaque accumulations.[14,56] They can be used for Type I, II, or III embrasures, but are best suited where the papilla does not completely fill the embrasure space.[43] These sticks are triangular in cross section to slide easily between teeth and to reduce potential tissue trauma. The stick is inserted interproximally from the buccal aspect with the flat surface, the base of the triangle, resting on the gingiva. The tip of the stick is angled coronally and is moved in a bucco-lingual direction (Figure 7-17). Wooden sticks have an advantage over plastic in that the pointed end can be softened in the mouth by moistening it with saliva. A softer stick can be more easily adapted to the interproximal surface. The stick should be discarded if the wood becomes splayed as splinters could be forced into the gingival tissues. Plastic sticks can be thoroughly washed and reused.

Triangular wood sticks have been shown to reduce bleeding sites and to do so better than rinsing with chlorhexidine.[56] In oral hygiene studies of participants exhibiting bleeding papillae, it was found that plaque removal with wooden triangular sticks effectively reduced inflammation more in the coronal regions of the interproximal pocket than in the apical regions.[56] It has also been suggested that by depressing the papilla, wood sticks can extend 2 to 3 millimeters into the sulcus, thus enhancing subgingival plaque removal.[43] A resurgence in the popularity of wood sticks has prompted recent marketing of design variations from the traditional triangular woodsticks. One such model is a fabric coated or "flocked" plastic stick (Figure 7-16).

Toothpicks

A comprehensive history of toothpick use suggests that toothpicks are one of the earliest and most persistent "tools" used to "pick teeth." The toothpick may date back to the days of the cavepeople, who probably used sticks to pick food from between the teeth.[57,58] The nobility and the affluent used elaborate toothpick kits of metal, ivory, and carved wood; the less affluent whittled sticks for the same purpose.

Toothpicks are utilized along the sulcus and in the interproximal surface to dislodge food debris and plaque. Consistent use of the toothpick can result in firm, resilient tissue. Outcomes are generally similar to triangular wood sticks, although one study showed superior benefits of the triangular wood stick.[59] Toothpicks are generally considered easier to manipulate than floss and indeed are used more than floss for oral hygiene.[60] A drawback with toothpick utilization is the possibility of contributing to recession, blunting the papillae, or causing even more severe damage with improper use. Recommended use of the toothpick is described with the toothpick holder and is an important aspect of oral hygiene self-care instruction.

Toothpick Holder

Although a toothpick may be manipulated by hand, the toothpick holder is a handle designed to increase effective application of the traditional toothpick by holding it securely at the proper angle. It also serves as an extension of the fingers in hard-to-reach areas. In particular, toothpicks in a handle have been suggested for cleaning the lingual embrasures of the posterior teeth.[61]

Plaque removal is achieved by tracing the gingival margin around each tooth or furcation area, and in each interproximal area with moderate pressure. Interproximally, the toothpick is moved back and forth between the buccal and lingual aspects to remove plaque and stimulate gingival tissues (Figure 7-18).

A variety of toothpick holders are available commercially (Figure 7-19). The toothpick is inserted into an adjustable plastic contra-angled handle, with the excess wood end broken off by snapping the toothpick in a downward direction. This leaves a stem to prevent the tip from disengaging from the holder. The toothpick can be positioned acutely on one end to access lingual surfaces and obtusely at the other end to adapt to buccal surfaces. The use of the toothpick holder is indicated in the following circumstances:

- Plaque removal along the gingival margin and within the gingival sulci or periodontal pockets,
- Cleaning of concave proximal surfaces (Figure 7-5),
- Cleaning of accessible furcation areas,
- Cleaning around orthodontic appliances and fixed prostheses,
- Application of chemotherapeutic agents (such as burnishing fluoride into the tooth to treat hypersensitivity or delivering chlorhexidine into the gingival sulcus).

When using a toothpick to remove plaque, it may be pre-moistened with saliva to soften the wood, just as with the wood stick. When applied to the gingival margin, the blunt tip is placed perpendicular to the long axis of the teeth. As previously mentioned, care should be taken to avoid subgingival insertion or vigorous interproximal use because of potential damage to the gingiva or teeth.

Interproximal and Uni-tufted Brushes

Small interproximal brushes which are attached to a handle come in a variety of designs. Some of the designs have a nonreplaceable brush; the entire device is discarded when the brush is worn (Figure 7-20). Interproximal brushes can be utilized to clean spaces between teeth and around furcations, orthodontic bands, and fixed prosthetic appliances with spaces that are large enough to easily receive the device (Figures 7-21, 7-22). They may also be used to apply chemotherapeutic agents into interproximal areas as well as furcations. Foam tips initially developed for use with implants are an ideal mechanism for delivery of medicaments interproximally or at furcations. Interproximal brushes are preferable to the use of dental floss for cleaning between teeth where the papilla does not fill the embrasure space or where root concavities are present[2,32] (Figure 7-5). The brushes are tapered or cylindrical in shape and are available in a variety of sizes (Figure 7-23). The core of the brush that holds the bristles is made of plastic, wire, or nylon-coated wire.

When determining the appropriate size of interproximal brushes, the diameter of the bristles should be slightly larger than the space to be cleaned. The brush can be moistened, and then inserted into the area at an angle approximating the normal gingival contour (Figure 7-24). A bucco-lingual movement is used to remove plaque and debris. Caution should be exercised to prevent damage to the tooth or soft tissues from the firm wire or plastic core of the brush. Implant abutments are easily cleaned

with interdental brushes however, extreme caution should be exercised to prevent scratching of the titanium surface.[38] Only plastic coated wires are recommended. Foam brushes can also be utilized for this purpose.

The uni-tufted brush, also known as the single-tufted brush, is efficient for removing plaque in numerous sites (Figure 7-25). These include the following:

- Mesial and distal surfaces of teeth adjacent to edentulous spaces, including the distal of the last molar in each quadrant,
- Furcations and fluted root surfaces (mesial aspect of maxillary first premolars and mandibular first molars) that have been exposed because of gingival recession or periodontal surgery,
- Wide open embrasures where papillae have been lost,
- Around dental appliances, including implants and orthodontic wires and brackets.

One study demonstrated the advantages of combining the use of the uni-tufted brush with the toothpick.[62] The end of the tuft is directed into the interproximal area by combining a rotating motion with intermittent pressure. The uni-tuft brush has also been suggested for application of chemotherapeutic agents. By softening in very hot or boiling water, handles of some uni-tufted brushes can be bent in order to allow easier access to the posterior buccal and lingual interproximal areas.

Other Interproximal Devices

Rubber or Plastic Tip

These devices consist of a conical, flexible rubber or plastic tip attached to a handle or to the end of a toothbrush (Figure 7-26). Primarily utilized for gingival massage, they can be used to remove plaque and debris from exposed furcation areas, open embrasures, and along the gingival margin. In one study, no differences were found in plaque or gingival index scores when comparing the rubber tip, dental floss, and interproximal brushes.[63] However, practitioners do not generally view rubber or plastic tips as effective plaque removal devices. Additional research is warranted. The lack of consistent evidence underscores the value of selecting a device that the educator is enthusiastic about in order to enhance compliance and have the greatest likelihood of success for an individual.

The tip is placed at a 90-degree angle to the long axis of the tooth and traced with moderate pressure along the gingival margin (Figure 7-27). In an open embrasure area, the tip is moved in and out in a bucco-lingual direction. The use of a tip attached to an angled shank rather than a toothbrush handle may allow for greater ease of access and adaptation. To prevent damage to the soft tissues, care should be taken to avoid inserting the tip subgingivally. Another design is made of elastomeric flanges and adds texture to the rubber tip allowing for a potential increase in plaque removal ability (Figure 7-28).

When used to massage the gingiva the rubber tip stimulates the tissue leading to increased keratinization.[64,65] However, since the keratinization occurs in the oral rather than the sulcular gingiva, improved gingival health probably results from removal of bacterial plaque rather than from the stimulation.[66] Some practitioners also

recommend the rubber tip following periodontal surgery to aid in tissue re-contouring.

Knitting Yarn

In areas where the papillae have receded and the embrasure is open, white knitting yarn (no dyes) can be used in place of floss for proximal cleaning. Wool yarns should be avoided because of the possibility of tissue irritation. The rationale for use of yarn is that the increased thickness and texture can enhance plaque removal. When access is limited, a floss threader may be used to insert the yarn into the embrasure (Figure 7-29). Once the yarn has been drawn through the embrasure, the technique is the same as for dental floss, taking care not to traumatize the tissue. The inconvenience of acquiring yarn, which is not a common household item, may affect compliance.

Gauze Strip

A gauze strip can be used for cleaning the proximal surfaces of teeth adjacent to edentulous areas, teeth that are widely spaced, or implant abutments. To prepare the strip, a 2-inch-wide gauze bandage is unfolded and refolded lengthwise. The lengthwise edge of the gauze is positioned with the fold toward the gingiva and the ends folded inward to avoid gingival irritation (Figure 7-30). The gauze is adapted by wrapping it around the exposed proximal surface to the facial and lingual line angles of the tooth. A bucco-lingual "shoeshine" stroke is used to loosen and remove plaque and debris. Gauze strips are recommended to clean the most distal surface of the most distal tooth in the mouth. It is particularly beneficial for the distal abutment tooth for partial dentures, an area where plaque accumulation is frequently abundant and thick.

Automated Interproximal Cleaners

Automated interproximal cleaning devices have been developed to help improve individual compliance with cleaning the proximal surfaces of teeth. One such device is a nylon filament tip that moves at 10,000 linear strokes per minute (Figure 7-31). When compared to manual flossing, research demonstrates that the device is able to reduce plaque levels and bleeding, and improve gingival parameters equal to that achieved with manual flossing.[67]

Several powered toothbrushes have attachments that are designed with a small number of bristles to remove plaque at the interproximal areas, around furcations, prosthetic abutments, and implants. One such device uses a uni-tuft of microfine filaments that move in an orbital motion. Another uses several bristle tufts that are 0.006 millimeters diameter with end-rounded bristles that oscillate when activated. One study found this automated oscillating interproximal brush to be safe and effective in removing plaque from the interproximal area.[68]

Figure 7-16 Variety of wooden and plastic triangular sticks. Plastic—three sticks depicted at left. Note flocked design of first plastic stick. Wooden—three sticks depicted at right. Note balsa wood composition of last wooden stick (others are birch).

Figure 7-17 Placement of balsa wood triangular stick interproximally. (Note: base of triangular stick rests at gingival aspect.)

Figure 7-18 Use of a toothpick holder. A. Tip is placed perpendicular to the long axis of the tooth to clean along gingival margins. B. Tip is placed at less than a 45° angle on the tooth to clean along marginal gingiva. Frayed tip is used to burnish or brush the tooth surface. (Courtesy of Marquis Dental Manufacturing Company, Aurora, CO.)

Figure 7-19 A variety of toothpick holders.

Figure 7-20 A variety of interproximal brush devices.

Figure 7-21 Interproximal brush moved in a bucco-lingual direction between teeth.

Figure 7-22 Interproximal brush directed into furcation.

Figure 7-23 Replaceable interproximal brush inserts in 3 varying sizes.

Figure 7-24 Interproximal brush inserted between abutment and pontic of fixed partial denture at angle approximating the gingival contour. Brush can be inserted under pontic if space allows. (Courtesy of Dr. Linda S. Scheirton, Creighton Universary, Omaha, NB.)

Figure 7-25 A variety of uni-tufted brushes.

Figure 7-26 A variety of rubber or plastic tip devices.

Figure 7-27 Use of interdental tip stimulator to remove plaque. The tip is placed at a 90° angle to the long axis of the tooth and traced along the gingival margin or moved in a buccolingual direction in an open embrasure area. (Courtesy of John O. Butler Co., Chicago, IL.)

Figure 7-28 Rubber tip device with textured elastomeric flanges.

Figure 7-29 Use of knitting yarn in wide open embrasure. A. Yarn is looped through dental floss and inserted through the contact point. B. Yarn is drawn through the embrasure. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Figure 7-30 A 6-in. length of 1-in. gauze bandage folded in half with the folded edge adjacent to the gingiva for adaptation. (Courtesy of Dr. Linda S. Scheirton, Creighton University, Omaha, NB.)

Figure 7-31 Automated interproximal cleaner.

Question 3

Which of the following statements, if any, are correct?

A. Utilization of wood or plastic sticks does reduce plaque accumulations and does clear food debris from the mouth.

B. Incorrect use of a toothpick may cause gingival recession or damage to the papillary tissues.

C. The diameter of an uni-tufted brush should be slightly smaller than the embrasure space to be cleaned.

D. When utilizing a gauze strip to clean in interproximal surface the fold should be positioned toward the gingival margin.

E. A rubber tip aids in removing oral debris and plaque from the gingival sulcus, but the keritinization it stimulates, is on the oral side of the gingival margin.

Tongue Cleaners

Tongue cleaning has been practiced since antiquity.[69] Studies on tongue debridement have renewed interest in this supplemental measure to further reduce bacterial plaque beyond toothbrushing and interproximal cleaning.[70,71] The large papillary surface area of the tongue dorsum favors the accumulation of oral microorganisms and oral debris. Anatomically, the shorter fungiform papillae and the longer filiform papillae create elevations and depressions that may entrap debris and harbor microorganisms, making the tongue an ideal location for bacterial growth. Oral debris from these sites may contribute to plaque formation in other areas of the mouth.[72] Reduction of this debris by mechanical tongue debridement can affect plaque accumulation, and oral malodor.[73]

Various designs of tongue cleaners are available (Figure 7-32). A soft-bristled toothbrush can also be used after the standard toothbrushing regimen. When using a tongue cleaner, the device is placed on the dorsal surface of the tongue close to the base of the tongue and pulled forward, pressing lightly against the surface of the tongue (Figures 7-33, 7-34). This process is repeated to cover the entire surface area of the tongue. Smokers or those with coated, deeply fissured tongues, or with elongated papillae (hairy tongue) will find that tongue debridement is especially beneficial in reducing oral bacteria.

Oral Malodor and the Tongue

Oral malodor, another term for bad breath or halitosis, can have a systemic origin or may originate in the oral cavity. A thorough physical examination can rule out a systemic disorder. Usually odors in the oral cavity occur when sulfur-containing proteins and peptides are hydrolyzed by Gram-negative bacteria in an alkaline environment.[74] Odiferous sulfur-containing end products created by this process include hydrogen sulfide, methylmercaptans, and dimethyl sulfide. A shift from a predominantly gram positive *to* a gram negative and anaerobic bacterial population is associated with odor production. Local factors such as reduced salivary flow, and/or a rise in oral pH may affect this shift. Inconsistent or ineffective interproximal plaque removal can provide a niche for gram negative bacteria to degrade sulfur-containing amino acids resulting in malodor.[72] The presence of periodontal disease may also be a contributing factor since the inflammatory process creates substrates that stimulate bacterial growth.[75] Also, the putrefaction process and its concomitant odor occurs more rapidly when bacterial accumulations on the tongue are high. Active periodontal disease, exhibiting deeper pockets, contributes to malodor. Thus, for some people, management of periodontal disease is an important aspect in the control of malodor.

Treatment considerations can include instruction in consistent tongue brushing or scraping since the dorsoposterior surface of the tongue seems to be a common site for production of volatile sulfur compounds. Additionally, the intraoral use of chlorine dioxide rinse, which oxidizes the malodorous sulfides has been shown to effectively reduce malodor.[72]

Figure 7-32 A variety of tongue cleaners.

Figure 7-33 Plastic tongue cleaning device used by pressing against tongue in an arc. Note serrations that provide a scraping action.

Figure 7-34 Plastic tongue cleaning device used by pressing in a posterior to interior direction.

Rinsing

Vigorous rinsing of the mouth will aid in the removal of food debris and loosely adherent plaque. Although water rinsing does not remove attached plaque, it may help return the mouth to a neutral pH following the acid production that results from ingesting fermentable carbohydrates. Rinsing or use of an irrigator is also helpful for individuals with orthodontic appliances.

For maximum effectiveness, a technique should be adopted whereby fluid is forced through the interproximal areas of clenched teeth with as much pressure as possible in order to loosen and clear debris. Use of the lip, tongue, and cheek muscles aids in forcing the fluid back and forth between the teeth prior to expectoration.

Rinsing has a limited impact supragingivally and is not efficient in subgingival penetration. It has no impact in reducing clinical parameters associated with gingival inflammation. However, the use of a therapeutic agent enhances the effect of rinsing. Antimicrobial rinsing has been utilized as part of a full mouth disinfection approach to improve oral tissue health.[76,77] See Chapter 6 for a detailed discussion of the impact of chemotherapeutics.

Irrigation Devices

Irrigation devices are a means of irrigating specific areas of the mouth whereas rinsing is a means of flushing the entire mouth. A home-irrigation device that is used for self-care provides a steady or pulsating stream of fluid, although the pulsating stream is preferable (Figure 7-35). Irrigation can result in the disruption of loosely attached or unattached supra- and subgingival plaque. The action is twofold. Loosely attached microflora are disrupted when the pulsating fluid makes initial contact. There is a secondary flushing action as the irrigant is deflected from the tooth surface. The microflora is disrupted both qualitatively and quantitatively.[78]

It has been demonstrated that irrigation with water or a nontherapeutic placebo can improve gingivitis or early periodontitis, when combined with toothbrushing.[79-83] Supragingival water irrigation alone, without toothbrushing, is not effective and is inferior to toothbrushing.[84-87] Home irrigation is not indicated for those who brush effectively and have no gingival inflammation. Individuals with inconsistent or

ineffective interproximal cleaning, fixed orthodontic appliances, crowns, fixed partial dentures, and implants however, may benefit from a home irrigation self-care regimen.[86,88] Oral irrigation may also be helpful for individuals who have jaws temporarily wired together for stabilization following surgery or head and neck trauma. Irrigation has been shown to reduce proinflammatory cytokines involved in the bone destructive process when periodontal diseases are present.[89]

The standard tip is designed for supragingival use. The tip is directed perpendicular to the tooth at or near the gingival margin. The cannula-type tip is directed into the gingival sulcus and allows a focused lavage adding to the depth of penetration.[90] Rubber-tipped cannulas can be angled into the sulcus about 2 millimeters. Home subgingival irrigation has been used to deliver medicaments further into the gingival sulcus.[81,91] Several studies demonstrated additional reductions in gingivitis and bleeding when using an antimicrobial agent in an oral irrigator with a cannula-type tip.[82, 92-94] Use of the cannula-type tip should be limited to individuals with adequate skill and dexterity.

Antimicrobial agents used as the irrigant have shown clinical and microbiologic improvements in those with gingivitis.[83,95,96] The failure to reach the base of the pocket may explain why supragingival irrigation is more effective against gingivitis than periodontitis. Investigations have compared supragingival irrigation with water to rinsing with chlorhexidine. Some studies show no difference[97] while others found chlorhexidine rinse more effect-ive in plaque removal than water irrigation alone.[82,86]

The potential for supragingival irrigation to induce bacteremias has been studied, but does not appear hazardous to healthy patients[98] since toothbrushing,[99] creates a similar level of bacteremia. Oral irrigators used inappropriately by those with poor oral hygiene have induced bacteremias, but the relationship to bacterial endocarditis is unclear.[98] There is less risk of bacterial endocarditis from irrigation of a healthy mouth than when irrigating an inflamed mouth because of differences in microbial load.[98, 99]

Figure 7-35 Oral irrigator for home use. Note standard tip and additional hygiene accessories. (Courtesy of Waterpik Technologies.)

Implant Maintenance

Meticulous oral hygiene self-care is essential in maintaining dental implants. Plaque and calculus accumulate more rapidly, in larger amounts and adhere more easily to the implant abutment than to natural teeth.[100] The epithel- ial barrier and connective tissue attachment mechanism is not as strong around an implant when compared to a natural tooth. This weaker attachment allows for a more rapid bacterial invasion of the biologic seal which can contribute to the destruction of osseous integration. Effective plaque removal is a critical factor in the maintenance of a healthy biologic seal and to prevent implant failure.[101] There is a positive correlation between the amount of plaque and subsequent gingivitis and bone loss around implants.[102]

The loss of natural teeth resulting in the placement of implants is often caused by a history of poor oral hygiene resulting in dental disease. A commitment to meticulous daily oral hygiene self-care is critical for those with implants. Cleaning the abutment posts, bars, and prosthetic superstructures, presents a challenge that can be even more

demanding than cleaning natural teeth. As with natural teeth, a combination of devices is usually needed to remove plaque from all surfaces. The goal of implant maintenance is to regularly remove soft deposits without altering the surface of the implants. Damage to titanium implants can increase corrosion and affect the molecular interaction between the implant surface and host tissue.[103] A scratched surface may lead to increased plaque accumulation.[104] The subsequent bacterial invasion can progress rapidly to peri-implantitis and potential implant failure.

An effective brushing technique should be the first component of an implant oral hygiene self-care regimen. A soft, manual toothbrush can be used. A sonic powered toothbrush has been shown to be better than a manual toothbrush in reducing plaque and bleeding scores around implants.[105] Some individuals may prefer a powered rotary brush with a tapered brushhead design. Neither type of powered brush was found to damage the implant surface and both were effective in areas where access is difficult.[105,106] Whatever brush is used, a demonstration of the adaptation of the brush to the abutment posts and pontics should be provided. The dentifrice used should meet American Dental Association standards to ensure that it is not abrasive.

To aid in plaque removal from abutment posts there are a variety of other devices that can be utilized (Figure 7-36). A tapered or cylindrical shaped interproximal brush or uni-tufted brush can be used with an in-and-out motion to clean the abutment posts. (Figure 7-36 B, 7-36 C) The interproximal brush must have a nylon-coated wire rather than the standard metal wire to prevent scratching the implant with the tip of the interproximal brush. Foam tips are an alternative choice for cleaning the interproximal surface of an implant. To help control bacteria, the foam tip, interproximal brush, or either of the powered brushes may be dipped into an antimicrobial solution such as chlorhexidine gluconate (0.12%). Alternately, a cotton swab can be used to apply the agent.

Any type of floss, tape, or yarn can be used for circumferential plaque removal around abutment posts. In some cases, traditional floss with a floss threader, variable thickness floss or gauze can be placed in a 360-degree loop around the abutment post and moved with a shoeshine motion in the direction of the long axis of the tooth. Alternately, floss products designed specifically for use with implants can be used (Figure 7-36 A). Ribbon floss is a wide, woven, sometimes braided, gauze-like version of floss, which provides increased texture to enhance plaque removal. One product has a hook on the end of the floss ribbon to allow for wrapping the floss around an entire post by inserting from the facial aspect, thus eliminating insertion from both facial and lingual surfaces. Yarn and shoelaces can also be used. Placing a small amount of nonabrasive toothpaste on the flossing product can polish the posts.

Oral irrigators can be used for cleaning around abutments, however, the water spray should be used on the lowest setting and should not be directed subgingivally. Daily subgingival irrigation with 0.06% chlorhexidine has shown beneficial effects on gingival, plaque, bleeding, and calculus indices while rinsing with 0.12% chlorhexidine affected gingival and bleeding indices only.[107] The substantivity effect is not as strong for implants as for natural teeth but would be better facilitated by subgingival irrigation than rinsing.

A critical factor in successful implant maintenance as with all oral health self-care is

to recommend only the minimal number of cleaning devices needed for effective plaque removal. With proper instruction, the motivated individual can successfully maintain implants.

Figure 7-36 Cleaning implants: A. Circumferential placement of Postcare braided nylon cord. B. Interproximal brush. C. End-tuft brush. (Courtesy of John O. Butler Co., Chicago, IL.)

Denture Maintenance

Instructions should be provided for the proper care and cleaning of both the dentures and the underlying tissues. According to one survey, only 40% of dentures worn by the elderly are adequately cleaned.[108]

Care of the soft tissues on which a denture rests includes removing the denture overnight or for a substantial time each day, cleaning and massaging the tissues under the denture daily,[109,110] and performing regular oral self-examinations to observe and report any irritation or chronic changes in appearance of the tissues. Failure to remove the denture may result in oral malodor, excessive alveolar ridge resorption, diseased or irritated oral tissues, or the development of epulis fissuratum.

Cleaning and massaging of the soft tissues can be performed simultaneously by brushing with a soft-bristled toothbrush or by massaging with the thumb or forefinger wrapped in a clean facecloth. Deposits that form on dentures include pellicle, plaque, calculus, oral debris (e.g., desquamated epithelial cells), stain and food debris. The microscopic porous surface of a denture attracts dental deposits.

Consistent, effective cleaning of dentures not only serves to enhance the sense of oral cleanliness, but also serves to prevent oral malodor, denture stomatitis, and other tissue irritations. Mucosal irritation may impair eating, which can have a negative nutritional impact on a frail, elderly individual. The incidence of denture stomatitis varies from 20 to 40% of the denture population and occurs most commonly in females. Frequently, denture wearers are only aware of the aesthetic benefits to be derived from maintaining cleanliness. It is incumbent upon the oral health professional to stress the numerous health benefits of denture cleaning.

Bacterial and fungal organisms can colonize the porous denture surface. For candidial infections the denture should be soaked in a nystatin antifungal suspension while simultaneously treating the oral tissues with the same medication. Daily thorough cleansing of the denture is recommended because dentures harbor the bacteria involved in the creation of the volatile sulfur compounds that contribute to oral malodor.[110,111] Commonly practiced cleaning methods include immersion, brushing, or a combination of both.

Immersion Cleaners

Immersing the denture in a cleaning solution has the advantage of reaching all parts of a denture, while with brushing, areas of the denture may be missed. Consequently a combination may result in a more thoroughly cleaned denture. When selecting an immersion cleaner, the type of denture material must be considered. Alcohol or essential oils found in commercial mouthwashes are not compatible with denture

acrylic, which may become dry or lose color from prolonged contact with these substances.

Hypochlorite solutions diluted 1:10 with tap water act as antifungal and antibacterial agents.[112] Adding a teaspoon of calcium-chelating dishwasher detergent (e.g., Calgonite®) may help to control calculus or stains. Care must be taken to not immerse appliances with metallic components in hypochlorite solutions since the metallic surface may corrode.[112] It is imperative that individuals be instructed to thoroughly rinse the bleach off before placement on the oral tissues. Acetic acid (vinegar) can be used for immersion, will kill some organisms, and is less caustic to soft tissues if not thoroughly rinsed.

Commercial alkaline peroxide powders and tablets are available. These typically contain an alkaline for oxidizing, perborate or carbonate for effervescing, and a chelating agent (EDTA).[106] When dissolved in water, these agents decompose and release oxygen bubbles, which mechanically loosen plaque debris on the denture surface. The alkaline substances and detergent enhance the mechanical effect of the bubbles. A 99% bacterial kill has been reported with these commercial products, and their effects are enhanced at 122° F.[113] Enzyme proteolytic agents have been used but appear inferior to alkaline peroxides.[114]

Cleaning the Denture

Brushing in conjunction with an abrasive agent or brushing a denture before and after it has soaked in an immersion cleaner, can be utilized to aid in the removal of deposits. Incorrect use of an abrasive agent (poor technique and/or too much pressure) can damage the denture. A brush with medium or soft end-rounded bristles, if used properly, should not abrade denture materials. A denture brush provides access to all surfaces of a denture (Figure 7-37). The dental professional should assess the level of manual dexterity when providing instruction in denture brushing.

Nonabrasive agents such as soap or baking soda, or a commercial dentifrice may be safely used in conjunction with a brush. Other agents may be harmful to denture materials. The denture delivery appointment is an excellent time to explain and demonstrate how to care for the new denture.

Ultrasonic or sonic devices are available for home denture cleaning. They utilize a cleaning solution in conjunction with agitation produced by ultrasonic (inaudible, high frequency) or sonic (audible) sound waves to remove debris and stains. Studies verify the efficacy of the ultrasonic cleaner; they are more effective than brushing with water.[115,116] Use of these devices may be particularly helpful for individuals with limited dexterity or for the personal-care staff at long-term care facilities. Whichever method is used, the denture should be thoroughly rinsed under running, tepid water before reinsertion into the mouth in order to remove any substances that could irritate soft tissue.

Instruction in the recommended method of self-care of their denture and of the tissues upon which it rests is critical to successful denture maintenance. It is the responsibility of the dental professional to ensure an understanding of both the "why" and "how" of denture maintenance and the potential consequences of poor denture

self-care. Explaining the procedure, demonstrating the correct method, and then requesting a return demonstration are all instructional methods to improve compliance. Written instructions and recommendations should be provided for easy reference and referral.

Figure 7-37 Brushing the alveolar surface of a full denture with a denture brush. Note the firm hold to prevent the denture slipping out of the hand.

Question 4

Which of the following statements, if any, are correct?

A. Implants accumulate dental plaque and thus can contribute to the development of periodontal disease.

B. Plaque removal from an implant can best be accomplished with a pipe cleaner.

C. The stream of solution from an irrigating device should be directed apically to clean the sulcus around implants.

D. Immersion cleaning of dentures is usually more effective than brushing because immersion ensures the cleaning agent reaches all areas of the denture.

E. After providing education on auxiliary methods of oral hygiene, instructions should be given on the use of several methods to solve the patient's problem.

Summary

In addition to oral conditions, several factors affect the appropriate selection and use of supplemental oral hygiene devices. The dexterity and motivation for performing oral hygiene procedures, and the preferences for specific devices should be assessed when recommending supplemental oral hygiene devices and techniques. When a device is introduced, it is essential that the proper application in all areas of the mouth be demonstrated and that the potential for damage with improper use is understood.

Despite adequate dexterity and ability, attainment of optimal oral health requires motivation and daily compliance in performing oral care. To enhance compliance and skill development, the number of recommended oral hygiene devices should be limited. Studies examining compliance and effectiveness indicate that development of proper skills and a willingness to use supplemental oral hygiene devices is facilitated when the number of devices is limited to no more than two.[117,118] Personal preferences for particular oral hygiene devices should also be considered. Although a specific device may be favored by the oral health professional, it will be ineffective if not used. If an individual has shown a preference for a specific device, its use should be encouraged. For example, if an individual uses a toothpick but presents with inadequate oral hygiene as evidenced by disclosed plaque and/or tissue inflammation the oral health professional might consider one of the following:

- Instruction to enhance the effectiveness with the toothpick,
- Introduction of a toothpick holder to facilitate access and manipulation of the toothpick,

• Use of the wooden or triangular interdental stick because of its similarity to the toothpick.

A wide variety of interproximal plaque removal devices are available. The oral health professional will need to stay informed of the research describing new devices, as it becomes available. Devices with evidenced based significance should be considered. Clinical experience and expertise should not be discounted, however, since these are also important components of evidence-based decision making.[119,120] It is incumbent upon the oral health professional to consistently investigate evidence and apply clinical judgment.

Answers and Explanations

1. A and D—correct.

B—incorrect. Studies show incomplete plaque removal increases rate and growth of new plaque.

C—incorrect. Motivational factors are considered during the planning phase. The evaluation phase focus is on patient outcomes and whether the oral hygiene self-care regimen needs to be adjusted.

E—Incorrect. Only when rigorous interproximal cleaning was performed by an oral hygiene professional was there a reduction in caries incidence; there is very little evidence to support that theory.

2. B and E—correct.

A—incorrect. Waxed and unwaxed floss have both been shown to be equally effective in removing plaque from the interproximal surface, without leaving a waxy residue. There is no evidence to indicate that one type of floss is better than the other.

C—incorrect. The circle (or loop) method is best for children who do not yet have the dexterity needed for the spool method.

D—incorrect. There is no study to date that shows one method is more effective than the other. Patient preference on the other hand, favors the use of floss holders.

3. A, B, D, and E—correct

C—incorrect. It should be slightly larger so as to effectively scrub against the surface disrupting and removing bacterial plaque.

4. A and D—correct.

B—incorrect. Circumferential plaque removal from an implant is best accomplished with a soft material that can be wrapped around its circumference: floss, tape, or yarn. Metal wire in a pipe cleaner could scratch the implant.

C—Incorrect. The stream of solution from an irrigating device should be at a right angle to the long axis of the tooth; otherwise bacteria can be forced into the blood supply to the area.

E—Incorrect. It is best to restrict the recommendation to one or two options, which will enhance compliance potential.

Self-evaluation Questions

1. The tooth surface least accessible to the toothbrush is the (interproximal) (buccal) (lingual) surface.

2. The (waxed) (unwaxed) floss frays and breaks more frequently on contact with calculus and restoration overhangs. The spool method of flossing requires (more) (less) psychomotor coordination than is required for the circle method. When using floss for the loop method, approximately _____ inches are needed, of which only about _____ inch(es) is/are held between the fingers to insert the floss between the teeth. A new segment of floss (is) (is not) used to clean each interdental space. If floss is forced too deeply into the sulcus, it can cause _____ in the gingiva, whereas if it is whipsawed buccolingually with too much force, it causes _____ of the cementum. If a periodontal condition exists, there is/are usually (one best) (several satisfactory) device(s) for plaque removal from areas with difficult access.

3. Four indications for the use of a dental floss holder in lieu of regular finger flossing are _____, _____, _____, and _____.

4. Three indications for the use of a floss threader are _____, _____, and _____.

5. Research (has) (has not) proved the value of the toothpick in maintaining oral health.

6. Irrigation devices have been used (successfully) (unsuccessfuly) to deliver medicaments further into the gingival sulcus.

7. Scratching the titanium implant while removing plaque can cause a more rapid buildup of _____ and hence pose a greater risk of gingivitis and periodontitis.

8. The wrapping of floss around an implant post for plaque removal is accomplished using a _____ _____ motion.

9. One study indicates that as few as _____% of the dentures worn by the elderly are adequately cleaned. Failure to maintain clean dentures can result in denture _____ _____ (overgrowth of tissue), a condition which is seen in 60 to 70% of denture wearers.

10. Four objectives that may be attained by proper use of dental floss are: _____, _____, _____, and _____.

11. Two auxiliary cleaning aids that can be used to safely and effectively clean under

a fixed partial denture are _____ and _____.

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Chapter 8. Water Fluoridation - *M. Elaine Neenan Mike Easley Michael Ruiz, Research Assistant*

Objectives

At the end of this chapter, it will be possible to

1. Define water fluoridation and the rationale for using water systems to provide for primary prevention of dental caries.

2. List and describe the four historical periods in the evolution and development of community water fluoridation.

3. Discuss the benefits and efficacy/effectiveness of water fluoridation.

4. Describe the cariostatic mechanisms of fluoride, including the pre- and post-

eruptive effects.

5. Define the impact of multiple sources of fluoride on the decline of dental caries and the role of water fluoridation.

6. Discuss fluorosis, fluoride supplementation, and the need to monitor exposure to fluoride.

7. Describe the effect on caries prevalence when water fluoridation is discontinued in a community.

8. Describe the economic aspects of water fluoridation.

9. State the optimal fluoride concentration range, in parts per million (ppm), for maximum caries protection with minimal risk of fluorosis.

10. List the chemicals used for water fluoridation and briefly describe the technical aspects of fluoridation, including monitoring and surveillance of water fluoridation in the United States.

11. Discuss the Safe Drinking Water Act and the EPA standards for natural fluoride levels.

12. Discuss the safety of fluoridation in terms of impact on health.

13. Define the role of dental-health professionals in continuing to educate the public about water fluoridation.

14. Discuss the mechanisms in which community water fluoridation may be enacted in the United States.

15. Summarize the readiness assessment factors for initiating a fluoridation campaign.

16. Discuss Sandman's principles of risk perception, the principles of risk communication and the myths related to risk communication.

17. Summarize the techniques used by opponents of water fluoridation and elaborate on the means to overcome these objections.

18. Summarize the current status of water fluoridation as it relates to Healthy People 2010, the National Health Objectives.

Introduction

Community water fluoridation (hereafter known as fluoridation) is defined as the upward *adjustment of the natural fluoride level* in a community's water supply to prevent dental caries. It is a population-based method of primary prevention that uses piped water systems to deliver low-dose fluoride over frequent intervals. Through water fluoridation, the preventive benefits accrue to consumers, *regardless of age or socioeconomic status.* Fluoridation has been cited as one of the *top-ten* public-health

achievements of the century. Extensive research over the past half-century has consistently confirmed the efficacy, safety, and cost-effectiveness of fluoridation. Fluoridation, a major contributor to the documented decline in dental caries in the 1950s to 1980s, has continued to be efficacious in caries reduction during the past 20 years in which *multiple sources of fluoride* (especially fluoride-containing dentifrices) have played a role in caries reduction. Continued monitoring of fluoride exposure, especially from adjunctive sources like fluoride-containing dentifrices, is important in achieving the appropriate balance between *maximum caries preventive benefit and minimal risk of fluorosis.* Enactment of fluoridation can occur at the state level but more often has been implemented at the local level through *administrative action* or a *vote* of the electorate. Initiating a fluoridation campaign requires that an assessment be done to determine community readiness. External forces, including public opinion, the political climate, role of the media, voter turnout, knowledge, skills, and savvy of campaign committee, etc., impact the ability to garner majority support for this issue. While fluoridation is sound public policy, the practice of fluoridating community water supplies has been challenged by vocal opponents since its inception. Consequently, communities often become embroiled in major campaigns attracting significant media attention. Dental-health professionals need to remain informed about fluoridation and keep abreast of the literature and latest research. They also need to provide accurate information to their patients and remain prepared to address any concerns and/or fears. Equally as important, they need to be able to assess the forces that affect public attitudes, evaluate the policy process, and understand the strategies employed by the opposition. This chapter reviews the history, as well as the efficacy, cariostatic mechanisms of action, safety, cost-effectiveness, and engineering aspects of fluoridation. Additionally, strategies used by opponents of fluoridation are discussed along with the principles of risk communication.

Definition and Background

The American Dental Association officially defines water fluoridation as "the adjustment of the natural fluoride concentration of fluoride-deficient water supplies *to the recommended level* for optimal dental health."[1] For all practical purposes, fluoridation can be considered a 20th century adaptation of a naturally occurring process since *virtually all sources of community drinking water in the United States* contain some natural fluoride.[1] Fluoridation is classified as a primary public-health intervention for dental-disease prevention because everyone benefits just by drinking fluoridated water.

Fluoridation can also be thought of as a form of *nutritional supplementation* in which fluoride is added to the drinking water. Nutritional supplementation is frequently used to prevent diseases with the addition of: vitamin C to fruit juices to prevent scurvy; vitamin D to milk and breads to prevent rickets; iodine to table salt to prevent goiter; folic acid to grains, cereals, and pastas to prevent birth defects (including spina bifida); and other vitamins and minerals to breakfast cereals to promote normal growth and development.[2-3]

The treatment of water for public consumption is a primary public-health activity that has been used by public-health agencies to prevent diseases since the 1840s. Water treatment prevents diseases such as: amoebic dysentery, cholera, enteropathogenic diarrhea (*E. coli*), giardiasis, hepatitis A, leptospirosis, paratyphoid fever,

schistosomiasis, typhoid fever, and many other diseases including dental caries.[2-3]

Fluoridation is an example of an ideal public-health intervention in that: (a) it is socially equitable and *does not discriminate* against any group; (b) consumers receive *continuous protection* with no conscious effort on their part to participate when they drink optimally fluoridated water; (c) it works without requiring individuals to gather in a central location as with other disease-prevention programs, such as immunizations; (d) it does *not require the costly services of health professionals* to deliver; (e) there are *no daily-dosage schedules* to remember; (f) there are no bad-tasting oral medications to be taken; and (g) *no painful inoculations* have to be endured in order to receive the benefits.[2-3]

Extensive scientific documentation over the past half-century, including several comprehensive reviews has established and consistently reaffirmed the *safety and efficacy* of community-water fluoridation. Based on the preponderance of scientific evidence, every U.S. Surgeon General since 1950 has advocated the adoption of water fluoridation by communities. Dr. Luther Terry, U.S. Surgeon General, 1961 to 1965, described water fluoridation as one of the four great advances in public health, calling it one of the *"four horsemen of public health,"* along with *chlorination, pasteurization,* and *immunization.* Dr. C Everett Koop, U.S. Surgeon General, 1981 to 1989 stated the following: "Fluoridation is the single most important commitment that a community can make to the oral health of its children and to future generations."[4] In 1992, Dr. Antonia Novello, the U.S. Surgeon General at that time, stated that "the optimum standard for the success of any prevention strategy should be measured by its ability to *prevent or minimize disease, ease of implementation, high benefit-to-cost ratio,* and *safety.* Community water fluoridation to prevent tooth decay clearly meets this standard."[5] Most recently, U.S. Surgeon General David Satcher stated: "community water fluoridation remains one of the great achievements of public health in the twentieth century" and "an inexpensive means of improving oral health that *benefits all residents* of a community, young and old, rich and poor alike."[6] In the first-ever report released in May 2000, on oral health in the United States, "Oral Health in America: A Report of the Surgeon General," Dr. Satcher noted that ". . .. one of my highest priorities as Surgeon General is *reducing the disparities in health* that persist among our various populations. Fluoridation holds great potential to contribute toward elimination of these disparities."[7]

Fluoridation is a population-based method of primary prevention designed to serve as the *cornerstone* for the prevention of dental caries, one of the most prevalent childhood diseases. It was initiated on *January 25, 1945* when Grand Rapids, Michigan fluoridated its public-water supply. Since then, more than 14,300 community-water systems serving nearly 10,500 American communities have fluoridated their water systems.[8] This includes *47 of the 50 largest U S. cities* where fluoridation is either actively practiced or where it is in the process of being implemented following approval by governmental/legislative bodies or voters. See companion website table.[a]

[a]On Feb. 11, 2003, California's largest water agency—Metro Water District of Sonata, California—approved a measure to add fluoride to its water supply. This involves 26 cities and water districts, the largest being San Diego.

History of Community Water Fluoridation

The history of community water fluoridation in the United States can be traced back to the early years of the 20th century and may be categorized into four separate periods or phases.[3,9-12] The *four* periods are: (1) clinical discovery phase; (2) epidemiological phase; (3) demonstration phase; and (4) technology transfer phase.

The first period, the *clinical discovery phase*, 1901 to 1933, was characterized by the pursuit of knowledge relative to the cause of developmental enamel defects present in dental enamel of people living in certain western areas of the United States. *Dr. Frederick McKay,* a Colorado Springs, Colorado, dentist, noticed that some of his patients presented with an enamel defect that occurred during tooth formation and appeared to be undermineralized or *hypomineralized.*[13] Local dental practitioners noted that the defects, which became known by local residents as *"Colorado Brown Stain,"* varied in degree of *hypomineralization* of the teeth with the most severe form consisting of a brown stain and pitting (*mottling)* of the enamel.[13] Dr. McKay notified the dental profession about his findings through publication of his observations in *Dental Cosmos,* the premier national dental journal of the times. After reporting his findings, Dr. McKay sought the consultation of Dr. G. V. Black, a noted researcher, and subsequently began to examine children in various nearby communities in order to determine the extent of the condition in the population. Not only was McKay able to demonstrate that what he now termed "mottled enamel" was confined to specific geographic areas, but he also hypothesized that it was directly related to s*omething in* the drinking water in these areas.[3,13]

Around the same time period (early 1930s), *H. V. Churchill*, a *chemist* with the Aluminum Company of America (ALCOA), demonstrated an association between high levels of naturally occurring fluoride in the drinking water and mottled teeth.[13-14] Subsequently, researchers Smith and Smith submitted a report, demonstrating a *causal relationship* between fluoride and mottling which was identified in the scientific literature as *dental fluorosis.*[13-14]

Drs. McKay and Black also observed a corollary finding: *People who had dental fluorosis also experienced less dental decay.* The search for additional information about the role of fluoride in the cause of dental fluorosis and the prevention of dental caries led to what is known as the second period, the *epidemiological phase* (1933 to 1945). During this phase, *a major epidemiological study*, known as "Dean's 21-City Study," was conducted by *Dr. H. Trendley Dean* with assistance from colleagues at the U. S. Public Health Service's National Institutes of Health. In this study, teams of researchers examined the teeth of children who lived in 21 different communities with varying levels of naturally occurring fluoride in the drinking water.[13-14] Dean and his team documented *the number of carious lesions and fluorosed teeth* observed in each of the 21 communities and *compared the findings with the fluoride concentration* in the respective water supplies. The findings from "Dean's 21-City Study," showed that: (1) the more fluoride in the water, the fewer dental caries in children, constituting an *inverse relationship* between the level of natural fluoride in the water and the prevalence of dental caries; and (2) higher levels of fluoride were associated with fluorosis of the teeth, meaning that there was a *direct relationship* between the level of natural fluoride in the water and the prevalence of dental fluorosis.[13] Dean's results showed that both a decreased risk of dental caries and a decreased risk of dental

fluorosis were attained with water fluoride levels of approximately *1 part per million (ppm) of fluoride.*[13] At this level, substantial reductions *(up to 60%) in dental caries* were exhibited with approximately 10% of the population exhibiting *very mild* dental fluorosis, which the investigators considered to be acceptable and cosmetically inconsequential.[13] The unattractive form of *fluorosis* (often called *mottling*) that was associated with higher levels of fluoride did not occur at the 1 ppm level. Consequently, 1 ppm became the benchmark level used by the U.S. Public Health Service in establishing the optimal range, *0.7 to 1.2 ppm* required to maximize the benefits of dental caries reduction and minimize the risk for dental fluorosis.[15-16] (Optimal levels discussed in subsequent section) (see Figure 8-1, Dean's Index/caries/fluorosis curve).

The third period, known as the *demonstration phase,* began in January 1945, and was characterized by a series of clinical trials that compared the dental and medical results following the deliberate addition of fluoride to the drinking water in four cities.[13-14] These four cities were also paired with four "control cities," in which the same study criteria were observed in communities with negligible levels of naturally occurring fluoride. Fluoride was added to the public water supply of *Grand Rapids, Michigan,* in order to test the hypothesis that an upward adjustment of the natural fluoride level to a concentration of 1.0 ppm would prevent dental caries in the population. Grand Rapids was the *first city* in the world to fluoridate its drinking water as a dental health promotion/disease prevention measure; after *13 to 14 years, a 55% reduction* in the rates of decayed, missing, and filled teeth (dmft) for children 12 to 14 years of age was observed. Three other experimental cities, Evanston, Illinois; Newburgh, New York; and Brantford, Ontario participated in similar controlled fluoridation studies, achieving similar reductions in dental caries rates (48 to 70%) after 13 to 15 years[7,13-14] (Table 8-1, Demonstration Phase).

The *demonstration phase* lasted until about 1954 when the benefits of the optimal adjustment of fluoride levels in drinking water became so apparent that many U.S. cities began fluoridation programs for their citizens. Thus the *demonstration phase* overlapped slightly with the fourth period in the history of community water fluoridation, the *technology transfer phase.*

The *technology transfer phase* began about 1950 when planning for the implementation of fluoridation began in earnest in many large U.S. Cities. Continuing to this day, the technology transfer phase is characterized by the establishment of a set of national health goals, which includes fluoridation. The *Year 2010 Health Objectives for the Nation* call for the implementation of water fluoridation in *all American communities that have communal water sources where implementation is technologically feasible. The target goal for fluoridation is: 75% of the population on community-water systems should live in communities with fluoridated water by the year 2010.*[17]

In 1992, when the last *Fluoridation Census* was published, approximately *135 million Americans* were consuming fluoridated water while an *additional 10 million* were drinking water with optimal levels of *naturally occurring fluoride,* equating to 57% of the entire population or *62%* of those who are served by *centralized piped-water systems.*[7-8] (see Table 8-2.) As of 2000, the percentage of the population receiving optimally fluoridated water through public water systems has risen to 65.8% and 26

states achieved the Healthy People 2000 goal of 75% of the population served by community water fluoridation[8] (see Figure 8-2). From 1992 to 2000, 28 cities adopted fluoridation, with an estimated 8,295,552 million people added to the Fluoridation Census.[18] In the *November 2000* presidential election, 23 U.S cities/counties voted on fluoridation ordinances/as either referenda or initiatives.[19] Of the 23 cities, 9 cities with a total population of *3,957,079 approved fluoridation* while 14 cities with a total population of *366,347 rejected fluoridation* at the polls. While the actual numbers of cities rejecting fluoridation exceeded those approving the measure during this election, the population voting to benefit from fluoridation exceeded the population denying themselves the benefits by ten-fold.

The *technology transfer phase* has extended fluoridation *worldwide*, with Singapore implementing fluoridation in 1958, serving 100% of the population.[20] The Republic of Ireland became the first country to actually legislate mandatory nationwide fluoridation in 1960. Israel initiated its mandatory universal fluoridation program in 1981. Currently, a national fluoridation effort is underway in Chile in conjunction with the Pan American Health Organization. Advocated by the World Health Organization, fluoridation benefits over 360 million people in 60 countries worldwide.[1,21]

Because of its 56-year history of effectiveness in reducing the prevalence of dental caries in the United States, water fluoridation was recently cited as one of the top-10 public-health achievements of the 20th century by the U. S. Centers for Disease Control and Prevention.[22]

Figure 8-1 As the fluoride content of water increases beyond 1 ppm, the index of fluorosis escalates more rapidly than the decayed, missing, filled permanent (DMF) decreases. (From Horowitz HS. *An Update for Dental Practice.* New York: American Academy of Pedodontics, MedCom, Inc., 1976.)

Figure 8-2 Percentage of U.S. Population by State Served by Fluoridated Public Water Supply, 2000. (*Source:* CDC, 2000; *MMWR 2002;* 51(07); 144-7.)

Question 1

Which if any of the following statements, is/are correct?

A. Virtually all sources of water for community water systems in the United States contain some natural fluoride.

B. Fluoridation does not involve adding anything to the water supply that is not already there.

C. The history of the water fluoridation story in the United States began with the investigation of "Colorado brown stain" by Dr. Frederick McKay and Dr. G. V. Black

D. H. Trendley Dean of the U.S. Public Health Service established the relationship between fluoride and dental fluorosis.

E. The optimal concentration of fluoride in drinking water for dental therapeutic purposes, is between 2.0 and 4.0 ppm.

Benefits and Efficacy/Effectiveness of Fluoridation

Over the past 56 years, numerous studies have been conducted on the *effectiveness of fluorides* and fluoridation in *preventing dental caries* and *decreasing caries rates.* When Grand Rapids, Michigan, decided to fluoridate its water supply in 1945, a long-term study of schoolchildren was initiated to determine the effectiveness of fluoridation in decreasing dental caries rates; the study found that after 11 years of fluoridation, dental caries rates declined by *50 to 63%*.[1,13,23] Corroborative studies in the same era conducted in New York (Newburgh-Kingston) and Illinois (Evanston-Oak Park) reported reductions in caries rates from 57 to 70%.[1,13] "Of 73 studies published between 1956 and 1979, the most frequently reported caries reduction was 50 to 60% and it was generally acknowledged that fluoridating a community's water supply would *reduce dental decay by half.*"[13]

While children and adolescents are the major beneficiaries of fluoridation, adults can also benefit. The impact of fluoride on the teeth of adults has become more important as adults are retaining their teeth longer than in previous decades because of improved dental-health practices and availability of preventive interventions, especially fluoridation. With aging, teeth remain susceptible to coronal caries, and more of the root surfaces become exposed to the oral environment, resulting in increased susceptibility to root caries. Research indicates that *"root caries* manifests as a significant dental problem as early as ages *35 to 44, doubling in the 45- to 54-year* age group, and *redoubling in the 55- to 66-year* group."[13,24] Results of a national survey of root caries found that 67% of men and 61% of women between ages 65 and 84+ had root-surface lesions.[13,25]

Studies in adults have consistently reported less coronal and root caries in the teeth of adults residing in communities with higher levels of water-borne fluoride.[9] Results of one study of young adults aged 20 to 34 years, showed 25% less coronal caries (decayed, filled surfaces) in those who resided in fluoridated (adjusted or natural) communities compared with those who had no exposure to fluoridated water.[13,26] Similar findings were noted in a study of older adults, mean ages 40 and 43 where residents of communities with 1.6 ppm fluoride in the water had 28% fewer coronal caries and 17% fewer root caries than residents of communities with 0.2 ppm fluoride.[13,24] Newbrun has estimated that the reduction in caries attributable to water fluoridation for adults, *aged 20 to 44,* is between *20 to 30%* for *coronal caries* and *20 to 40%* for *root caries.*[13,27]

During the early periods of fluoridation, the primary source of fluoride was the drinking water; consequently, the reductions in dental caries rates attributed to water fluoridation were significant. Decades later, an epidemiological study of more than 39,000 children aged 5 thru 17 years was conducted in 1986/1987 by the National Institute of Dental Research (NIDR).[13,28,29] This study determined that younger children who had lived all their lives in optimally fluoridated communities experienced 39% fewer carious lesions and fillings when compared with those children who had lived in communities that were not fluoridated.[13,28,29] Other reports showed similar levels of reduction in decay rates.

In 1992, Newbrun estimated that fluoridation prevents *30 to 39%* of dental caries in the *primary* dentition, 11 to 38% in the *mixed* dentition, and *35%* in the *permanent*

dentition.[27] The decline in percentages of caries reduction has been found in both fluoridated and nonfluoridated communities, with children who had always been exposed to community water fluoridation demonstrating mean DMFS scores ranging from 18 to 40% lower than those who had never lived in fluoridated communities.[18,27,30,31] Water fluoridation has played a dominant role in the decline in caries even though the absolute differences in caries prevalence that once were observed between fluoridated and nonfluoridated communities appears to be diminishing.[28] The recently released York review (published in 2000) of 26 studies on fluoridation effectiveness, found that fluoridation of drinking water supplies reduces caries prevalence in variable ranges with a median of 14.6% reduction in rates as measured by the change in dmft/DMFT scores and the proportion of caries-free children.[32] The report also attempted to address the *impact of fluoride-containing toothpaste* on the effectiveness of fluoridation.[33] The authors of the review acknowledged that the effectiveness estimates could be biased because of inadequate adjustment for the impact of potential confounding variables.[32] Dental scientists contend that the failure to adjust for confounding variables in the review made it difficult to interpret the findings. The use of a median range of reduction in caries by the authors was considered misleading and inappropriate for establishing fluoridation effectiveness. Nonetheless, it is clear from studies cited previously that there has been a worldwide decline in dental-caries rates even though certain population groups are still disproportionately affected by dental caries. The decline in dental-caries rates has been attributed to the widespread use of multiple fluorides from various sources: community *water supplies, supplements, fluoride rinses gels,* and *varnishes,* and *dentifrices.*[13,34-36]

The reduction in the absolute measurable benefits of water fluoridation has been attributed to the *dilution* and *diffusion* effects.[13] *Dilution* results from the increased availability of fluoride from multiple sources, *diluting the impact of any one source of fluoride,* including water.[9,29,36-37] According to Ripa, *"dilution is the apparent reduction in the measurable water fluoridation benefits resulting from the ubiquitous availability of fluoride from other sources in both the fluoridated and the fluoride deficient comparison community."*[13] Today, the most universally available source of fluoride in the United States is fluoride-containing dentifrice (toothpaste).[13,35] All *fluoride-containing dentifrices* have *very high levels of fluoride (1,100 to 1,500 ppm)* and are a significant source of fluoride overexposure and fluorosis. Moreover, they are not meant to be swallowed, especially during the years when the crowns of teeth are forming. Nearly four decades ago, the American Dental Association (ADA) gave its seal of approval to Crest toothpaste, the only fluoride dentifrice available at that time. However, by 1980, *98% of the available dentifrices contained fluoride.*[13,38]

In separate studies, Brunelle and Carlos in 1990 and Murray in 1992 found greater percentages of caries-free children and lower caries-prevalence rates in fluoridated communities where other sources of fluoride were also available. After adjusting for other sources of fluoride, they found a 25% difference in dental-caries prevalence. The findings of these two studies led the researchers to conclude that *water fluoridation remains an important contributor* to caries prevention.[9,29]

The other major modifying factor regarding the effectiveness of fluoridation, the *diffusion* effect, results from the consumption of commercial foods and beverages that were processed in a fluoridated community and transported to fluoride-deficient

communities,[13,39] making fluoride available to consumers in the fluoride-deficient community.[13,39] Ripa described *diffusion* as *"the extension of benefits of community water fluoridation to residents of fluoride-deficient communities."*[13] *Diffusion* has also been called the *"halo effect."* The differences in caries prevalence rates between fluoridated and nonfluoridated communities are diminishing.[13,40] According to Ripa, "the weaker association reported by contemporary studies between exposure to fluoridated drinking water and caries experience, therefore, is not due to a lessening of the effects of waterborne fluoride, but is actually caused by the extension of those effects, through a process of '*diffusion*,' of fluoride into fluoride-deficient areas."[13] Increased traveling to fluoridated communities impacts the effect of diffusion as well. Also, residents who live in a fluoride-deficient community and work on a *military base* in the same community may be exposed to fluoridated water since most military bases are fluoridated.

As described above, the decline in dental-caries rates was greatest (up to 65 to 70%) in the earlier years (1940s, 1950s, 1960s) of fluoridation when water was the primary source of fluoride and the availability of other sources of fluoride was limited. The caries-inhibition effectiveness of fluoride in water resulted in a parallel rush to develop other sources of fluorides: (1) adjunctive *systemic fluorides,* such as tablets, drops, lozenges, and vitamins with fluoride, which are meant to be swallowed and are prescription items intended to be dispensed to the public by licensed health professionals; and (2) topical fluorides, which are intended only for topical application and are not meant to be swallowed.[13,34] Some *topical fluorides* are used by professionals in the dental office while others are used in public-health programs and in schools. Additionally, over-the-counter (OTC) products are used by consumers.[13,34] As more cities adopted fluoridation and the ingestion of dietary fluoride supplements increased in fluoride-deficient communities, consumer use of fluoride-containing products such as toothpastes, mouthrinses, and gels also increased. As a result, exposure to fluoride from numerous sources has become more widespread, with benefits accruing at varying levels. At the same time, it is becoming more *difficult to accurately determine the level of reduction* in caries rates attributed to fluoridated water alone versus other sources. Most researchers now believe that the *"dilution" and "diffusion"* effects are responsible for the decline in dental caries rates in nonfluoridated, and to a lesser degree, in fluoridated communities.[9]

Mechanisms of Action

Systemic fluorides are beneficial in decay prevention in that they are ingested and incorporated directly into the hydroxyapatite crystalline structure of the developing tooth. The smaller fluoride ions replace hydroxyl ions in the crystalline structure of the tooth, producing a less-soluble apatite crystal[13,41] Over the past several decades, the caries-preventive properties of fluoride have been attributed primarily to its *pre-eruptive effects* on the developing teeth. But systemic fluorides also provide a topical effect resulting in marked *post-eruptive benefits.* Saliva, which contains fluoride from ingestion, is continually available at the tooth surface and becomes concentrated in dental plaque where it inhibits acid-producing cariogenic bacteria from demineralized tooth enamel. Fluoride accomplishes this by interfering with the enzymatic activity of the bacteria and by controlling intracellular pH, thus reducing bacterial acid production and thereby reducing dissolution of tooth enamel.[1,22,42-48] According to Bowen, fluoride concentration in the plaque is 50 to 100 times higher than in the

whole saliva.[13,49]

Fluoride also interacts with calcium and phosphate ions from saliva and adsorbs to the tooth surface, thereby enhancing remineralization.[50] Recent research shows that remineralization represents the primary mechanism by which fluoride works, occurring after tooth eruption, and making the topical effect important in caries reduction for people of all ages.

In summary, systemic fluoride has been found to reduce dental decay by three mechanisms: (1) the conversion of hydroxyapatite into fluorapatite which reduces the solubility of tooth enamel in acid and makes it more resistant to decay; (2) reduction of acid production by dental-plaque organisms; and (3) the remineralization of tooth enamel that has been demineralized by acids produced by decay causing bacteria.[1]

Question 2

Which of the following statements, if any, is/are correct?

A. Fluoridation prevents an estimated 20 to 30% of coronal caries in adults and an estimated 20 to 40% of root caries.

B. *Dilution* is the "apparent reduction in measurable water fluoridation benefits resulting from the ubiquitous availability of fluoride from other sources" such as fluoride-containing dentifrice, fluoride mouthrinses, and professionally-applied topical fluorides.

C. *Diffusion* is the "extension of the benefits of community water fluoridation to residents of fluoride-deficient communities" such as occurs when residents of nonfluoridated communities work in fluoridated communities, when, children from nonfluoridated communities attend school in fluoridated communities, and when people from nonfluoridated communities consume certain foods processed in fluoridated communities.

D. All fluoride-containing dentifrices have very low levels of fluoride and do not contribute to fluoride overexposure and fluorosis.

E. Fluoride reduces (1) acid production in the plaque which (2) reduces the amount of demineralization, which (3) allows the tooth to more easily "repair" itself by remineralization.

Dental Fluorosis

Dental fluorosis has been described as a series of conditions occurring in those teeth that have been exposed to excessive sources of fluoride ingested during enamel formation. Dental fluorosis can present in a number of ways, from a barely discernable white lacy appearance to a more severe form that could be classified as a developmental defect of the enamel.[13] Fluorosis, regardless of severity, *cannot occur once enamel formation is complete and the teeth have erupted; therefore older children and adults are not at risk for dental fluorosis.*[1,51-52] Dental fluorosis occurs when children consume excessive levels of fluoride in various ways, such as when

drinking water from private wells or community-water systems with higher-than-optimum levels of naturally occurring fluoride. However, the greatest likelihood of exposure to excess fluoride in children results from: (1) *inadvertent ingestion of toothpaste* containing very high concentrations of fluoride and (2) taking of inappropriately prescribed dietary fluoride supplements.[1,53] The degree of fluorosis depends on the total dose of fluoride, as well as on the timing and duration of fluoride exposure.[54]

In 1942, H. Trendley Dean developed a *system of classification for dental fluorosis.* He established a series of categories that ranged from questionable (white flecks or spots or "snowcapping"), to very mild (small opaque paper-white areas or streaks known as veining, covering less than 25% of the tooth surface, to mild (opaque white areas covering less than 25% of the tooth surface), to moderate (marked wear on occlusal/incisal surfaces, may include brown stains), to severe (mottling and brown staining affecting all tooth surfaces).[13] Dean's fluorosis index continues to be widely used today.[1,55] However, not all enamel opacities are caused by fluorosis; some are caused by other chemical agents such as *strontium* or pharmaceutical agents such as *tetracycline.* Idiopathic opacities also exist for which the cause(s) is/are currently unknown.[13,56] According to a survey conducted by the National Institute of Dental Research in 1986 to 1987, the majority of fluorosis cases identified *were classified as being very mild or mild.* The minor forms of fluorosis (questionable, very mild, or mild fluorosis) are not considered to be abnormal, nor are they considered to constitute an adverse health effect. However, both researchers and practitioners should continue to monitor and assess the risk of dental fluorosis in order to ensure that the more severe forms of fluorosis do not occur. In 1936, Trendley Dean estimated that approximately *10%* of children who drank optimally fluoridated water would develop *very mild dental fluorosis.*[57] More recent studies have shown that dental fluorosis attributed to fluoridation is around *13%.*[1,58]

As previously mentioned, questionable, very mild, and mild fluorosis usually result from very young children swallowing too much fluoride-containing toothpaste or from the inappropriate supplementation with prescription fluoride products such as (1) when physicians and dentists independently prescribe fluoride supplements, or (2) when physicians and dentists prescribe fluoride supplements without checking the fluoride content of the child's water supply so that, in either case, a child gets a "double" dose of fluoride on a daily basis. Monitoring total fluoride intake is complicated considering the availability of multiple sources of fluoride. Also, fluoride from tablets/drops is ingested and absorbed at one time of day as opposed to fluoride in water where the ingestion and absorption of low-dose fluoride is distributed throughout the day. These factors have been con- sidered in the establishment of fluoride dosage schedules where in recent years, the dosages have been lowered, particularly in the first 6 months of life. The *Dietary Fluoride Supplement Schedule* approved by the American Dental Association, the American Academy of Pediatrics, and the American Academy of Pediatric Dentistry should be followed when prescribing fluoride supplements[7] (see Table 8-3, fluoride supplements). Fluoride ingestion should be reduced during the ages of tooth development, particularly under the age of three. Parents need to assist in attainment of this goal *by supervising small children* during toothbrushing to ensure that their children do not swallow the toothpaste.

Antifluoridation groups frequently and inappropriately exhibit photographs of children and/or adults having severe *fluorosis* in which pitting or mottling of the enamel and brown stains are evident and attribute these manifestations directly to water fluoridation, often describing dental fluorosis as a major risk factor for people of all ages. In making dental-health decisions, patients depend upon the dental professional team to assist them in evaluating the risks versus the benefits of a given procedure or public health measure. To do this, dentists and dental hygienists need to stay current regarding the scientific literature and to use this knowledge as a basis for educating themselves and their patients. The risk of developing *very mild fluorosis versus* the benefit of decreased dental caries and attendant treatment costs should be communicated to patients who express concern.

Discontinuation of Water Fluoridation

Opposition to fluoridation, as well as governmental action, has resulted in the discontinuance of this public-health measure in various locales. Interestingly enough, the *cessation* or *discontinuation* of fluoridation has resulted in the implementation of several studies to determine the impact of such reversals on dental health. Results have consistently shown that dental-caries rates *increase dramatically* when fluoridation is discontinued. In 1960, the city of Antigo, Wisconsin, discontinued fluoridation after having fluoridation for 11 years. Six years later, when Antigo elementary school children were found to have substantial increases in caries rates, ranging from *41 to 70 percent, fluoridation was reinstated.*[13,59] Similar findings occurred in Scotland upon cessation of fluoridation where in the town of Wick, caries rates increased by *40% in primary teeth and by 27% in permanent teeth.* This dramatic increase in dental-caries rates occurred *despite* fluoride toothpaste being readily available and national-caries rates in Scotland continuing to decline.[13,60] Moreover, 5 years after fluoridation was discontinued in the town of Stranraer, caries rates increased to levels approaching those found in the nonfluoridated town of Annan. In Stranraer, restorative dental treatment costs for decay alone rose by *115%.*[1,13,61]

Similar results can occur if a city changes its water source from one that is optimally fluoridated to one that is fluoride-deficient. The impact would be equivalent to discontinuation of fluoridation. In a Public Health Service Report on risks and benefits of fluoride, it stated that "one way to demonstrate the effectiveness of a therapeutic agent, such as fluoride, is to observe if the benefits are lost when the agent is removed."[13,62] Clearly, these studies serve to demonstrate that the discontinuation of community water fluoridation has resulted in the significant loss of dental caries preventive benefits.[1,63]

Cost of Water Fluoridation

Water fluoridation provides significant cost savings for a community and has been described as "one of the most cost-effective preventive dental programs available."[13,64] Estimates of the cost of water fluoridation will vary depending upon the factors included in the calculations.[13,65] The size and complexity of the water system, including the number of systems, the number of wells, whether or not the systems use a dry feeder or solution (wet) feeder system, purchase of equipment and installation, purchase of fluoride, labor, and maintenance, as well as the number of

service connections and size of the population all factor into the cost of fluoridation.[13,64-66]

"The cost of water fluoridation is usually expressed as the annual cost per person of the population being served."[13] An inverse relationship exists between the cost per person and the population of a community. Consequently, the cost per person is lower in larger communities and higher in smaller communities where the actual cost of fluoridation may approach that of other methods of caries prevention.[65] Fluoridation also eliminates or diminishes additional costs incurred through other forms of fluoride administration, such as costs incurred when accessing professionals in order to obtain provider-prescribed fluoride products, compliance irregularities, and the lower effectiveness of other forms of fluoride distribution. Fluoridation is the most cost-efficient and cost-effective method of dental caries prevention for almost all communities[7] (see Table 8-4, cost/benefit).

Another way to look at cost-saving benefits is to determine the beneficiaries of dental-treatment cost savings. Employers who pay prepaid dental-care fringe benefits for their employees save on costs. Hidden production or service costs caused by dentally-related missed workdays by employees are also minimized through fluoridation. Taxpayers who support public programs would also benefit from dental-treatment cost savings. In fact, skyrocketing dental Medicaid expenditures in California (a state with a low percentage of the population having access to fluoridation) provided the impetus for the enactment of a statewide mandatory fluoridation bill there in 1995.[67-68] As will be discussed later, recent studies comparing dental Medicaid expenditures in Louisiana and Texas also demonstrated that treatment costs were significantly higher in nonfluoridated communities than they were in fluoridated communities. Patients can also be expected to benefit from lower health-care bills, lower dental-care costs, and lower insurance premiums because of lower costs incurred by providers for uncompensated care.[2]

With the availability of baseline levels of dental-caries rates and treatment costs, two different types of analyses can be done to determine (1) the *effectiveness* of fluoridation as a *dental-caries preventive measure (cost-effectiveness analysis)* as well as (2) associated cost savings (a *cost-benefit analysis.*)[13] Ripa has stated that "the *greater the initial caries prevalence* and treatment costs, the *greater the potential benefits* to be realized by the introduction of fluoridation."[13] The national average recurring cost of water fluoridation has been estimated at $0.50 per person per year while the national average cost of one simple restoration is $62.[7,66] If one were to multiply the approximate average life expectancy of a U.S. resident (75 years) by the annual per capita cost of fluoridation ($0.50), it appears evident that the *$37.50* total for a *lifetime of protection* through fluoridation would more than offset the cost of just one simple restoration for one tooth.[3,13,69] Additionally, for every carious lesion initially prevented, the need for repeated restorations and treatment of *recurrent* carious lesions is reduced over a lifetime.[13,70] Different studies have shown that the replacement rates for amalgam restorations caused by recurrent decay varies between 38 and 50%; the savings to be realized from prevention are substantial.[13,71,72] The national average benefit-to-cost ratio for fluoridation is: 80:1; (MMWR/ CDC) on average, *for every $1 dollar spent on fluoridation, $80 is saved in treatment costs.*[3,66]

Three recent studies further demonstrate the substantial cost-benefits generated

through community water fluoridation. Brown and colleagues, in a comprehensive study for the Texas Department of Health were able to demonstrate cost-savings for the publicly funded Texas Health Steps Program (Texas EPSDT-Medicaid Program) when comparing program costs for clients from fluoridated communities with those from non-fluoridated communities.[73] Similarly, Barsley and colleagues were able to demonstrate that the costs for hospital-based treatment of acute dental conditions to Louisiana's publicly funded Medicaid Program were much less for residents of fluoridated communities than for residents of nonfluoridated communities.[74,75] Finally, Wright and colleagues established conclusively that fluoridation remains an extremely cost-effective public-health program in New Zealand in their comprehensive 1999 report for the New Zealand Ministry of Health.[76]

Question 3

Which of the following statements, if any is/are correct?

A. Dental fluorosis occurs when an excessive amount of fluoride is ingested during the period of enamel formation *only.*

B. Dental fluorosis does not occur once enamel formation is complete and the teeth have erupted, therefore older children and adults are not at risk for dental fluorosis.

C. Discontinuation of fluoridation has no impact on dental caries rates.

D. The national cost-benefit ratio attributed to water fluoridation is 95:1.

E. The national average cost to fluoridate a public water system in the U.S. is approximately $0.50 per person per year.

Optimal Fluoride Levels

The U.S. Public Health Service established an optimal standard for fluoride in the drinking water in the United States based upon the *annual average of maximum daily air temperature.* As a result, the optimal level is actually a range, 0.7 to 1.2 ppm (see Table 8-5, optimal levels), which assumes greater water consumption in hotter climates and less water consumption in colder climates.[14] Consequently, *the higher the average temperature in a community, the lower the recommended water fluoride level.*

However, determining daily fluoride intake is impacted by other factors such as consumer use of home distillation and reverse-osmosis water-treatment systems which can remove significant amounts of fluoride from the water supply.[13,77-80] The consumption of bottled water and other beverages, such as soft drinks and fruit juices, complicate the matter since fluoride levels in these beverages varies greatly.[13,78,80-81] Additionally, determination of fluoride intake may be altered by the widespread use of air conditioning in homes, automobiles, and workplaces such that some people from warmer climates no longer require the higher volumes of liquid. For these reasons, Burt has cautioned that temperature guidelines used in establishing the optimal range need to be periodically monitored in order to determine the need for revision.[14] Hong Kong has adjusted its water fluoride level since initiating

fluoridation to achieve its goal of maximizing the benefits of caries reduction while minimizing the risk of fluorosis in an environment of increased fluoride exposure.[82] In a 1994 report issued by the World Health Organization, it was recommended that some regions, especially tropical and subtropical areas, revise the optimal range to establish appropriate higher and lower limits.[82]

Safety of Water Fluoridation

The issue of safety relative to adding fluoride to the water supply has often been raised in fluoridation campaigns by opponents who attribute nearly every disease known to mankind to fluoridation. It is common practice for fluoridation opponents to distribute pictures of children alleged to have crippling skeletal fluorosis and attribute the malady to fluoridation. Skeletal fluorosis occurs in India and other areas that have *extremely high natural fluoride levels,* ranging *from 20 to 80 ppm.*[83] Decades ago, five documented cases of skeletal fluorosis were found in the United States, (in rural areas with well water in which the natural fluoride level was found to be very high).[1,83] At the time, public health authorities resolved the problem by installing *defluoridation* equipment and since then, there has not been a documented case of skeletal fluorosis in the United States other than about 25 cases occurring from occupational exposures.[1] Because of fluoride's affinity for bones and teeth, its impact on bone health is often called into question by anti-fluoridationists. By implementing water fluoridation at the recommended level (0.7 to 1.2 ppm), there is no evidence of anyone developing skeletal fluorosis.[1] Currently, in the United States, naturally occurring fluoride levels vary widely from less than 0.1 ppm to greater than 13 ppm. The variation in fluoride levels in public water systems is amenable to adjustment: (a) upward to achieve dentally therapeutic levels in community water supplies that are fluoride deficient through fluoridation or (b) downward to attain the maximum concentration of fluoride allowable by *defluoridation.*[13] However, instances of *defluoridation* are rare (discussed in later section).

In general, safety concerns about fluoridation relate to a number of factors, including: toxicity of fluoride, total fluoride intake, fluoride absorption, the impact on human health, the effect on the environment, water quality, and the engineering aspects. Over the past 56 years, numerous studies have been conducted in communities where the natural fluoride level is either higher than or equivalent to the recommended level for dental-caries prevention, as well as in communities where the fluoride level has been adjusted to the optimal level; results have repeatedly and convincingly confirmed the safety of fluoride in the water supply.[84-86]

Since the late 1970s, allegations against fluoridation have focused on cancer. The possibility of a cancer risk associated with fluoridation of public-water supplies was raised in a 1977 self-published paper about 20 U.S. cities.[87-88] The analyses purported to show that fluoridation of drinking water in 10 of the cities caused a 10% increase in cancer mortality in those cities compared with 10 other cities that did not have fluoridation. According to the authors, before fluoridation, the average cancer death rates were increasing in a similar manner in both groups of cities but immediately after the start of fluoridation, the rates diverged with higher cancer mortality rates seen in the fluoridated group vs. the nonfluoridated group.[87-88] The immediate divergence in rates failed to factor in the latency period for cancer (usually over 5 years, and for some cancers, as long as 20 to 30 years).[89-90] Manifestation of a

divergence in cancer mortality rates would require 5 to 10 years between exposure and death from cancer. Since the divergence in crude cancer mortality occurred at the exact same time that fluoridation was introduced, it is inaccurate and disingenuous to attribute the divergence to fluoridation.[89-90] Also, average crude death rates were used in the study, ignoring the differences and changes in age, race, and sex composition, widely recognized risk factors known to affect the death rates from specific types of cancer.[89-90] Subsequent analyses of the same set of data by National Cancer Institute investigators, using internationally accepted epidemiological methods and controlling for confounding variables, concluded that there was *no evidence* that fluoridation caused cancer in the 10 fluoridated cities.[91] Follow-up studies, including studies in populations with high levels of naturally occurring fluoride in water in the U.S. and various other countries also failed to show a positive relationship between fluoridation and cancer.[92-98] Results of studies of fluoridation and cancer were reported by the Royal College of Physicians in Britain in 1976 in which they concluded: "there is no evidence that fluoride increases the incidence or mortality of cancer in any organ." Subsequently, British researchers, Drs. Doll and Kinlen, reported in the *Lancet* that "none of the evidence provided any reason to support that fluoridation is associated with an increase in cancer mortality, let alone causes it."[99]

In 1990 two separate studies by the National Toxicology Program (NTP) of the National Institute of Environmental Health Sciences and Procter and Gamble were conducted to assess the carcinogenicity of sodium fluoride in which rats and mice were deliberately fed excessive amounts of fluoride (75 to 125 ppm). One group, male rats, showed "equivocal" evidence of carcinogenicity, where "equivocal" is defined by the NTP as a marginal increase in osteosarcomas that may be chemically related, but in which there is insufficient evidence to prove or disprove that a relationship exists.[1,100-102] Subsequently, the U.S. Public Health Service established a Subcommittee on Fluoride to review the studies. The Subcommittee on Fluoride determined that the two animal studies failed to establish an association between fluoride and cancer. The NTP Report prompted a comprehensive "Review of fluoride: benefits and risks" by the U.S. Public Health Service in 1991 in which it concluded that fluoride in water is not carcinogenic. Other comprehensive reviews came to the same conclusion.[1,84,86,97,103-104]

Additionally, scientists at the National Cancer Institute examined more than 2.2 million death records and 125,000 cancer case records in counties using fluoridated water and concluded that there was no indication of a cancer risk associated with fluoridated drinking water.[1]

In a report prepared in 1993 by the National Academy of Science, National Research Council at the request of Environmental Protection Agency (EPA), a review of the literature focused on toxicity and health risks of fluoride. This report stated that the "currently allowable fluoride levels in drinking water do not pose a risk for health problems such as cancer, kidney failure or bone disease" and that the EPA's primary standard of 4 ppm for naturally occurring fluoride would provide an adequate margin of safety against adverse health effects.[1,86,105-106]

The Safe Drinking Water Act, enacted by Congress in 1986, established *primary* and *secondary standards* for *natural fluoride levels* in public drinking water in the United States. The legislation set the *primary standard* (the maximum concentration of

fluoride allowed in public drinking water systems) at *4.0 ppm* and further stated that *natural* water sources exceeding this level *must* be defluoridated,[13,14,107] although no communities have defluoridated under this provision. A *secondary standard* of 2.0 ppm for a *natural* source was also established as the *recommended* maximum. Under this *secondary standard,* when the water exceeds 2.0 ppm, community residents are *informed* of the greater risk for dental fluorosis.[13,107]

Whereas the EPA is responsible for monitoring public-water systems in the United States, it requires that public-water systems not exceed fluoride levels of 4 ppm; public water systems with natural levels exceeding the limit are expected to *defluoridate* in accordance with the primary standard. *Defluoridation* is infrequently implemented in the United States primarily because of the lack of demand on the part of the public living in high natural fluoride areas of the country.[13] *Defluoridation* also has budgetary implications in that the cost of defluoridation for a community having high natural fluoride levels is approximately *10 times greater* than the cost of fluoridating a water supply in a community with deficient natural fluoride levels.[13] Also, some communities may be forced to find alternative water supplies in the event of forced closure of existing water supplies having natural fluoride levels exceeding the *primary standard.* Finding alternative water supplies poses a major challenge for many communities in the United States. Compliance with EPA standards in geographical areas having high natural fluoride levels may be greatly affected by these factors.[13,14]

When fluoride is ingested, it is rapidly absorbed from the stomach and small intestine into the systemic circulation, where about half becomes bound to the hard tissues (the bones and unerupted teeth) and the rest is eliminated via efficient urinary excretion. Since the major site of fluoride accumulation in the body is the bone, almost no fluoride is present in the soft tissues.[13] As stated by Ripa in the previous edition of this textbook: "fluoride can be deposited in the (1) *adsorbed layer* of the bone, (2) the *crystal structure,* and (3) the *bone matrix.*[13,49,108-109] The fluoride in the adsorbed layer is in equilibrium with the blood and can be rapidly raised or lowered, depending on ingestion patterns and the efficiency of kidney function."[13] It is known that "blood plasma fluoride levels begin to rise about 10 minutes after ingestion and reach *maximal levels within 60 minutes,* subsequently returning to pre-ingestion levels after 11 to 15 hours."[13,110] In crystal formation, the fluoride ion is thought to be involved in an ionic exchange with the hydroxyl ion and is incorporated into the crystals of the bone, where it is more slowly removed, most likely through the osteoclastic action seen in remodeling."[13] Fluoride that is not stored in bone is rapidly excreted through the kidneys, where the rate is highest the first hour, then begins to fall for the next 3 hours, after which there is a low, continuous plateau.[13] With the consumption of fluoridated water, the excretion rate is more constant because of a more continuous intake of fluoride.[13]

Because of the role of the kidneys in fluoride excretion, concerns have been raised by anti-fluoridationists about the safety of fluoridation in patients with impaired kidney function or who have kidney failure requiring dialysis. Impact on kidney function was addressed by the National Research Council in which it concluded that the "ingestion of fluoride at currently recommended concentrations is not likely to produce kidney toxicity in humans."[86] The standard of care relative to the treatment of kidney failure patients on hemodialysis machines who are exposed to large quantities of water, calls

for the removal of all minerals, including fluoride, from water used in dialysis.[1,111-112] This requirement for removal of minerals (including fluoride) *only applies to the dialysate* used during the dialysis process and *does not apply to minerals ingested* through drinking water. In other words, renal dialysis patients and patients with chronic kidney disease can continue to ingest water with optimal fluoride levels. Additionally, numerous studies of people with long-term exposure to drinking water with fluoride concentrations, some as high as 8 ppm, showed no increase in kidney disease.[112]

Concerns about the accumulation of fluoride in the body[113-115] relate primarily to people's concerns about the effect of fluoridation on bone mineral density and whether or not there is increased risk for osteoporosis and fractures. The results of several ecological studies over a 20 year period from 1980-2000, comparing fracture rates in fluoridated and non-fluoridated communities were mixed, from increased rates in hip,[116-118] proximal humerus and distal forearm fractures[119] to no effect on fracture risk[120-123] to decreased risk of hip fracture.[124,125] Since ecological studies use community-wide data, confounding variables associated with rates of fracture including age, sex, estrogen use, smoking, and body weight cannot be controlled. To address these deficiencies and the limitations of ecological study design, a multicenter prospective study on risk fractures for osteoporosis and fractures was done by Phipps et al. in which investigators assessed bone mass, risk factors, development of incident nonspinal fractures, ascertainment of prevalent and incident vertebral fractures, and exposure to fluoridated water in 7,129 women 65 years and older.[126] The conclusions of this study reported in October 2000 were as follows: (1) "long term exposure to fluoridation does not increase the risk of osteoporotic fracture among older women and may reduce the risk of fracture of the hip and vertebrae in older white women" and (2) "our results support the safety of fluoridation as a public health measure for the prevention of dental caries".[126]

Interestingly enough, sodium fluoride has been used to treat established osteoporosis for over 30 years.[127] Data on the use of high-dose sodium fluoride (75 mg daily) for the treatment of vertebral osteoporosis suggests that the incidence of hip fracture may be increased and bone density may be diminished while the use of low-dose sodium fluoride (25-50 mg daily) therapy appears to have a protective effect against spine fractures but no apparent effect on hip or wrist fracture risk.[128]

According to the National Institute of Dental and Craniofacial Research (NIDCR), "no credible scientific evidence supports an association between fluoridated water and conditions such as cancer, bone fracture, Down's syndrome, or heart disease as claimed by some opponents of water fluoridation."[129]

Most recently, the York Review examined studies relative to the safety of fluoridation and concluded that there was no evidence of any adverse health affect caused by community water fluoridation.[32]

Engineering Aspects: Chemicals and Technical Systems Used

Water-treatment chemicals are used for a number of reasons including: disinfection, absorption, algae control, decolorization, oxidation, metal coagulation, water softening, filtra- tion, pH control, iron control, coagulation, corrosion control,

chlorination, and fluoridation.[13,130-131] Primarily, three chemicals are used for water fluoridation in the United States and are required by the states to meet the American Water Works Association (AWWA) standards for the specific chemical: *sodium fluoride, sodium silicofluoride,* and *hydrofluosilicic acid.*[48] Sodium fluoride (granular or powder) and sodium silicofluoride (granular) are used in distribution systems that use "dry" compounds, while hydrofluosilicic acid, a liquid, is used in solution or "wet" systems. Sodium fluoride was the first compound used in controlled water-fluoridation programs and is still widely used in smaller community-water systems (usually those serving fewer than 5,000 people).[13,130-131] *Sodium silicofluoride* is substantially less expensive than *sodium fluoride* and tends to be used in community-water systems serving between 5,000 and 50,000 people. Today, the most frequently used compound for water fluoridation in the United States is *hydrofluosilicic acid,* because of its low cost and ease of handling; it is used primarily in larger communities with water-distribution systems serving 50,000 or more people and represents approximately 57% of all fluoridation systems in the United States.[130,132] Opponents of fluoridation often attempt to distinguish between *sodium fluoride* and the hexafluorosilicates, *sodium silicofluoride* and *hydrofluosilicic acid* in terms of availability of the fluoride ion. Fluoridation opponents disparage the hexafluorosilicates as "junk dumped into the drinking water" that "contaminates the water with a harmful residue." However, according to the Environmental Protection Agency (EPA), no hexafluorosilicate remains in drinking water at equilibrium, which is readily achieved.[133] This means that there is no difference in the source of fluoride ions from the three chemicals used in fluoridation as the detractors would have one believe.[133] In response to anti-fluoridationist claims, Newbrun stated that "the use of fluorosilicates is a good example of successful recycling which benefits both the environment and the consumer."[134]

Determination of the appropriate compound to use in fluoridation depends largely upon the type of distribution system used by the individual water plant. According to Reeves, National Fluoridation Engineer at the U.S. Centers for Disease Control, the most common methods by which fluoride is added to water supplies in the United States are: (1) the *volumetric dry feeder* system which delivers a predetermined quantity of fluoride chemical (either sodium fluoride or sodium silicofluoride) in a given time interval. However, sodium fluoride is not recommended for volumetric dry feeders because of its higher cost which is nearly two and a half times that of sodium silicofluoride; (2) the *acid-feed* system, in which a small metering pump is used to add *hydrofluosilicic acid* to the water-supply system; and (3) *the saturator feed* system, which is unique to water fluoridation, uses an upflow saturator to provide saturated solutions of sodium fluoride in constant strengths of 4% and is pumped into the water system via a small metering pump.[130-131] Additionally, the Venturi fluoridation system is used by the U.S. Indian Health Service in some extremely small rural communities.

Monitoring and Surveillance of Fluoridation

The process of adding fluoride to drinking water supplies to the level recommended for achieving the maximum dental therapeutic benefits is technically simple, uncomplicated, and similar to the processes used when dealing with chlorine and other water-treatment chemicals.[13,130] All three types of fluoride chemicals used in the water fluoridation process are certified as to their purity and safety when used

appropriately. Interestingly, there are 48 additional chemicals approved by the U.S. Environmental Protection Agency and certified as safe for addition to drinking water by the American Water Works Association and NSF International (National Sanitation Foundation). Contrary to popular perception, fluoride does not affect the taste, odor, color, or turbidity of the water at the levels used for water fluoridation.[13,130-131]

In order for fluoridation to be implemented, a number of factors should be taken into consideration. Of prime importance is the *compatibility* of the fluoride chemical to be used with the existing water-treatment and distribution system.[13,130-131] Other factors impacting the technical engineering aspects of fluoridation include: (a) source of water—underground or surface water, (b) size of the water plant; (c) number and types of point sources of water (one treatment plant or many treatment plants with water coming from wells, reservoirs, rivers, aqueducts, or desalination plants); (d) number of injection points (where fluoride is introduced into the water); (e) fluoride chemical costs, including transportation; (f) modification of existing plant vs. construction of a new plant; (g) need for training of water-plant operators; and (h) type of monitoring and surveillance system to be used.[7,13,130-131]

Modern water-plant design ensures that excessive amounts of fluoride are prevented from entering the water supply. Properly designed fluoridation systems prevent the addition of excess fluoride to the water system in several ways: (1) only a limited amount of fluoride is maintained in the hoppers (or day tanks), (2) positive controls have been installed for feeding fluoride from the hoppers into the dissolving tanks, and (3) metering pumps are installed so that they are electrically connected to the water pump in a manner that ensures that if one fails, both stop operating and no fluoride is added to the system.[13,130-131]

Maintaining a constant level of fluoride in the water supply is the responsibility of the water-plant operators. Variation in the ad- justed water fluoride levels has occurred in water plants where the operators are not properly trained and/or the operator turnover is high.[50-56] Variability in water fluoride concentration may also occur if a water plant fails to provide adequate and appropriate storage facilities, if there is malfunctioning of feed equipment, or if proper water-analysis equipment is lacking, all of which are readily avoidable with proper planning and implementation. Most of the variances in fluoride concentrations that have occurred are due to poor monitoring at water treatment facilities and have resulted in fluoride levels *below* the recommended level *(hypofluoridation).*[13,135] For this reason, communities that have implemented fluoridation must continue to monitor the fluoride levels in order to ensure that the full benefits of fluoridation will accrue in a community. *Hyperfluoridation* occurs when an excess amount of fluoride is added to the drinking water over several days, usually secondary to an overfeed from malfunctioning equipment and/or maintenance errors.[13,136-137] Over the past 56 years there have been seven instances of *hyperfluoridation* which resulted in outbreaks of acute fluoride poisoning in the United States, all of which could have easily been prevented.[13,138] Thus, when a community decides to fluoridate its public-water supplies, it also must assume the responsibility for monitoring the equipment, training the water-plant operators, and implementing performance reviews to ensure that the process is in place to protect the public from an overfeed. The Centers for Disease Control and Prevention offers weeklong water-plant operators training programs designed to assist plant operators in

sustaining and monitoring their fluoridation systems.[131]

Question 4

Which of the following statements, if any is/are true?

A. Numerous studies over the past several years have consistently demonstrated the safety of water fluoridation.

B. The National Cancer Institute has reviewed the literature on fluoridation and has concluded that there is substantial credible evidence associating fluoridation with cancer and has recommended that fluoridation be halted worldwide immediately.

C. Sodium fluoride is the most frequently used chemical for water fluoridation in the United States.

D. Monitoring of fluoride levels at water treatment plants is essential to prevent both hypofluoridation and hyperfluoridation.

E. Fluoride does not affect the taste, color, odor, or turbidity of the water at the levels used for fluoridation.

Other Fluoride Vehicles

Many countries without centralized water distribution systems have chosen to add fluoride to table salt, a process known as *"salt fluoridation,"* in order to provide primary dental caries preventive benefits to their populations; approximately 40 million people use fluoridated salt.[82] Using salt as a vehicle of fluoride supplementation is similar to the concept of iodine supplementation and is a relatively inexpensive method of fluoride delivery. Like water fluoridation, *salt fluoridation* results in small amounts of fluoride being released from plasma throughout the day.[139-140] In order to achieve dental-caries reductions at levels comparable to water fluoridation, the level of fluoride supplementation of refined salt should be at least 200 mg F/kg as NaF or Ca CaF_2.[141-142] *Salt fluoridation* requires centralized salt production, as well as monitoring.[82] Since the consumption of high quantities of sodium is a risk factor for hypertension, the use of fluoridated salt is not recommended for those at risk.[82,143] Countries utilizing *salt fluoridation* extensively include Switzerland, France, Costa Rica, Jamaica, and Germany.[144,145] It has also been introduced in Mexico, Spain, Columbia, Brazil, and Hungary where its use has been found to be appropriate.[82,139,142,146-148]

Also, *"milk fluoridation,"* the addition of 5 mg of fluoride to 1 litre of milk has been introduced as a vehicle of school-based fluoride delivery in some countries (Bulgaria, Chile, China, the Russian Federation, and the United Kingdom).[82] While encouraging results have been reported with *milk fluoridation*, no widespread clinical trials have been reported.[82] Additional studies are required to adequately assess *milk fluoridation* as a viable caries prevention strategy. According to the WHO Report, "the distribution of fluoridated milk can be more complicated than that of fluoride supplements (tablets or drops)."[82] As a result, the existence of an established distribution system that includes provisions for pasteurization and refrigeration is a limiting factor in *milk*

fluoridation programs.[82]

Fluoride mouthrinses were developed in the 1960s as a *school-based* public-health measure designed to provide access to fluoride without requiring a visit to the dentist office. *School-based weekly fluoride rinse programs* using 0.2% sodium fluoride have been shown to be effective in preventing coronal caries in school children who are at risk for dental caries. Estimates of dental caries reductions observed prior to the establishment of efficacy, range from 20 to 50%. Since the establishment of efficacy for fluoride mouthrinses, the level of caries reduction appears to be less than originally observed. Additionally, the cost-effectiveness of fluoride mouthrinse programs appears to be diminished because of the declining prevalence of dental caries in general.[7,149]

Implementation requires that children enrolled in the program participate consistently over time to receive maximum benefit. However, many children as they get older (middle/high school years) decline to participate, believing that fluoride rinsing is a program for younger children. Significant coordination and monitoring in the schools, parental consent, tracking children as they move from elementary school to middle and high school, and commitment on the part of school officials is required for caries-reduction outcomes. According to the Centers for Disease Control, 3.25 million schoolchildren were participating in fluoride-rinse programs in 1988.[7]

Enactment of Water Fluoridation as Public Policy

In May 2001, Partnership for Prevention, a nonprofit, nonpartisan organization issued a report, *Priorities in Prevention: Oral Health*, in which it stated that "oral health is not solely dependent on individual behaviors." This report identified prevention opportunities for policy makers and business/community leaders with community water fluoridation topping the list of oral-health strategies that work.[150]

Fluoridation is not legislated at the federal level in the United States. However, legislation may be introduced in state legislatures, although very few of these measures have been enacted at the state level in recent years. Statewide *fluoridation laws* were enacted primarily in the late 1960s and require fluoridation in ten states: California, Connecticut, Delaware, Georgia, Illinois, Minnesota, Nebraska, Nevada, Ohio, and South Dakota. Moreover, the District of Columbia and the U.S. Commonwealth of Puerto Rico also legislated jurisdiction-wide mandatory fluoridation. In addition, Kentucky mandates fluoridation of all public water systems serving 1,500 persons or more by *administrative regulation* under the authority of its state health commissioner (see Table 8-6, statewide fl). Legislation requiring fluoridation failed in two states in 1989 along with anti-fluoridation bills that failed in five states. Anti-fluoridation bills failed in four states in 1990 as well (ADA, 1991).

Successful adoption by legislatures of mandated statewide fluoridation laws in recent history include Nevada and Delaware. California passed legislation in 1995, mandating fluoridation in communities having 10,000 or more service connections, pending availability of funds. Around the same time period, a bill was introduced in Oregon, requiring that communities fluoridate; if they failed to comply, they would be required to reimburse the health department for dental-treatment bills.[151-152] Economics appears to have been the driving force and the common denominator in

these state legislative initiatives. For some states, especially those in which a small percentage of the population has access to fluoridated water and/or those states with high dental-caries rates and high Medicaid costs, mandatory statewide fluoridation laws could be a viable strategy. Equally important, however, is the political will to implement fluoridation at the local level as "mandatory" state laws often have local option provisions.

From a public-policy perspective, fluoridation is more often perceived as a local issue that is enacted either by *governmental administrative action* (ordinance that is voted upon by a city council or city/county commission) or by a vote of the public. Interestingly enough, the local health official often has both the power and authority under city/county charter to order the fluoridation of public water systems but rarely invoke such power. Generally speaking, a vote of the public is referred to as a *voter initiative* if the vote is to implement fluoridation or as a *voter referendum,* if the vote is to confirm, alter, or eliminate an existing mandatory fluoridation law. Frequently, a *voter initiative* is often referred to as an *initiative referendum. Voter initiative* and a *voter referendum* have been used interchangeably. Consequently, it is important to review the city charter to ascertain the correct mechanism to be pursued in a community.

In some cases, public officials seek to avoid controversy by opting to put an ordinance on the ballot; in other cases, a referendum vote can be forced by a signature petition. A forced petition referendum usually requires a percentage of signatures, usually 10 to 20% (varies according to city or county charter provisions or state constitutional requirements) of registered voters who voted in the previous election. In the final analysis, implementation of fluoridation in the United States is now achieved primarily by *governmental administrative decision* or by a *vote of the electorate.*

Fluoridation Actions

In the 1950s to 1960s, "*initiative referenda*" represented the majority of fluoridation actions in the United States; of the 1,009 *initiative referenda,* fluoridation was adopted in 411 communities and defeated in 598 communities.[68] In the 1980s, two out of every three fluoridation *initiative referenda* were defeated, while gains were achieved by 77% (199/258) of communities utilizing the governmental *administrative mechanism.* In the late 1980s and early 1990s: city council/commission *administrative action* authorized fluoridation in 318 communities while 32 *initiative referenda* were held in which 19 were won and 13 were lost, indicating an improvement in *initiative referenda* success compared to previous decades. In 1994, 47 U.S. communities authorized fluoridation: 46 were city council or commission actions and one was an "*initiative referenda*" action.[68,153] Of the 46 communities, authorizing fluoridation by administrative action, 36 had populations less than 10,000 while the remaining 10 communities had populations greater than 10,000. Greater successes in terms of numbers of communities have been achieved through the governmental administrative decision process but they are concentrated in the smaller communities.[154-157]

As previously stated, 23 U.S cities/counties voted on fluoridation ordinances in the November 2000 presidential election. Of the 23 cities, 9 cities with a total population of 3,829,185 approved fluoridation while 14 cities with a total population of 381,888 rejected fluoridation at the polls (see Table 8-7 and Table 8-8). Further analysis of this

data shows that in general, the *initiative/referenda* wins occurred in larger population centers (except North Attleboro, MA, and Leavenworth, KS) while the *initiative/referenda* losses tended to occur in the smaller communities, except Spokane, WA.[19,158]

Readiness Assessment for Initiating a Fluoridation Campaign

In order for the United States to achieve the goal of 75% fluoridation by the year 2010, the obstacles that affect the legal framework in which fluoridation is implemented must be carefully analyzed. An assessment of a number of factors that impact the implementation of fluoridation in the United States is essential to developing targeted educational strategies and defining the fluoridation campaign message. Some of the major factors include: demographic trend data, external forces, public opinion, political climate, media influence, voter turnout/apathy, lack of public awareness of the benefits of fluoridation, perception of benefits vs. risks of fluoridation, and lack of political campaign skills among health professionals.

Demographics

According to projections, the United States will need to add approximately 30 million people, served by more than 1,000 water systems to the *Fluoridation Census* in order to get within striking range of the Year 2010 Goal.[7] Between 1990 and 1998, the greatest population growth occurred in metropolitan areas in the West (mountain and pacific states) and South (south atlantic, east south central, and west south central states) regions of the United States.[158] The 15 largest nonfluoridated cities in the United States have a total population approximating 5 million people; 12 of the largest nonfluoridated cities are located in the West and South regions where metropolitan population gains ranged from 13.1% in the South to 13.8% in the West.[158] At the same time, nonmetropolitan population gains ranged from 7.5% in the South to 16.1% in the West.[158] As previously stated, 7 of the largest nonfluoridated cities are in California where fluoridation legislation passed in 1995. A significant percentage of the needed fluoridation census gains will have to come from our nation's cities which continue to be the population magnets and represent approximately 80% percent of the total population in the United States.[159] Achieving fluoridation, whether by city council/commission action or by *voter initiative or voter referendum*, is more difficult in our urban centers where massive resources and protracted major grass-roots, culturally relevant campaigns are generally required. While suburbanites tend to vote, inner-city residents often tend not to vote. The implications of urbanization/suburbanization will have an impact on efforts to fluoridate many of the nonfluoridated cities.

The diversity of the U.S. population presents a challenge to the preventive health educational and political efforts because each racial and ethnic group has unique attitudes, beliefs, and expectations about preventive health outcomes that need to be considered. While racial and ethnic minorities are not as likely to vote as whites, efforts should be made to provide accurate information to the entire community, as well as to encourage broader participation in the voting process by all voters. Additionally, the ability to communicate in a language other than English may also be important in a local campaign effort. In 1990, nearly 32 million (14% of the nation's population 5 years and over) said that they spoke a language other than English at

home, compared with 23 million (11%) a decade earlier.[68] Over half of those who said they spoke a language other than English at home reported speaking Spanish.[68] America is also aging. More and more people in their 50s and 60s have surviving parents, aunts, and uncles and four-generation families are common. Those aged 65 years and older comprised 17% of the adult population but cast 22% of the ballots while those aged 18 to 24 comprised 14% of the voting age population, but accounted for only 6% of voters.[160] Older populations have higher rates of edentulousness and are less likely to visit the dentist.[66] As noted, the elderly do vote and they also tend to view fluoridation as a benefit primarily directed at children and therefore may be less likely to be supportive. Framing fluoridation solely as a childrens' health issue is problematic for campaign organizers.

The likelihood of voting increases with education as well as age and income, resulting in certain groups making up a disproportionate share of voters. According to the Census Bureau, 84% of all adults, ages 25 and older, had completed high school while only 26% had completed a bachelor's degree.[161] Homeowners were about twice as likely as renters to vote (53% vs. 27%).[162,163] One in three children born in America live in poverty.[164] The poor are also less likely to have dental insurance or to obtain preventive care. And while the economically disadvantaged stand to benefit the most from fluoridation, they often do not vote. Geographic mobility of the population, often related to the job market, can also impact fluoridation success at the polls. People who have lived in fluoridated communities are often surprised to find that their new community is not fluoridated and are even more surprised by the controversy generated when fluoridation is placed on the public agenda. Having lived in a fluoridated area previously, it is speculated that new residents to a nonfluoridated community would generally tend to favor fluoridation.[165]

External Forces/Public Opinion/Political Climate

Over the past two decades, there has been a move towards federal decentralization, that is transferring power, control, and funding from the federal government back to the states to administer programs. States are faced with problems associated with many of the social issues that are likely to have significant budgetary implications. As a result of statutes requiring balanced budgets, governors have been forced to control costs. Rapid increases in Medicaid costs alone have strained many state budgets, causing greater scrutiny of expenditures. Dental Medicaid expenditures were among those examined by the state of California and viewed by some as the impetus for passing fluoridation legislation in 1995.[68]

Fluoridation campaign committees need to research their local city or county charter to ascertain the mechanisms/processes by which their community can fluoridate as well as the provisions and timeframes, taking into account early voting and/or extended voting periods which impact the campaign. Fluoridation campaign committees also must analyze the economic climate, as well as the results of recent local issue elections, including the impact of negative campaigns, in order to assess the mood of the electorate. The opposition, its strength and credibility must also be assessed; underestimating the energy, tenacity, and ingenuity of the opposition are major causes of fluoridation ordinance failures. Research, including an assessment of external forces, such as in a SWOT analysis (strengths, weaknesses, opportunities, and threats) is critical to the development of a strategic campaign plan. Fluoridation

committees also need to determine if community leaders and elected officials have the political will to shepherd a fluoridation measure through the enactment of an ordinance, either by administrative action or a vote of the electorate.

In his book, *Rational Lives: Norms and Values in Politics and Society*, Dennis Chong noted that individuals make decisions across both social and economic realms and that "our preferences inevitably reflect the costs and benefits of the available options and the influence of psychological dispositions formed over the life span."[166] Knowing the public stance on a particular issue is also important. In a 1990 National Health Interview Survey (NHIS) of 41,104 adults regarding public knowledge of the purpose of fluoridation, 62% correctly identified the purpose.[66] Knowledge of the purpose of fluoridation was highest among persons aged 35 to 54 years of age (68 to 70%) while younger (18 to 24) and older (≥ 75) persons had less understanding, at 49% and 40%, respectively.[66] Other findings showed that persons with higher educational attainment levels were more than twice as likely as those with less than a high school education to correctly identify the purpose of fluoridation (76% vs. 36%).[66] When presented with conflicting information regarding benefits and risks of fluoridation, discernment and the ability of the electorate to make informed decisions may be compromised. Additionally, the dynamics inherent in plebiscites (fears, anxieties, discontentment, anger, resistance to authority, and resentment of professionals) can derail the decision-making process.[167] Direct democracy poses a significant challenge for proponents of any issue because they must "settle the public's mind on all aspects of a question and they must bear the burden of restraint."[167] Securing majority support for any issue placed before the voters requires a very high level of initial support in order to achieve a successful result on election day because support erodes over the duration of a campaign.[165,167]

Intensive and ongoing efforts to educate the public about fluoridation should be implemented prior to initiation of a political campaign and sustained through the decision making process and continued thereafter. It took 25 years to enhance public knowledge and change attitudes about smoking in the United States; to sustain the gains, the education must continue. Similarly, just because communicable diseases are rare today, it doesn't follow that immunization programs should receive less emphasis. Public-health professionals have learned the hard way that eliminating public-health programs, such as immunization, is quickly followed by a rapid reappearance of previously rare diseases. They also recognize that once diseases are under control, the most difficult task is to educate the public about the need to continue successful programs in order to prevent return of the disease. Similarly, fluoridation education should continue.

In a telephone survey conducted by Research!America in May 2000, 85% of Americans responded that oral health is *very important* to their overall health.[150,168] A 1998 national Gallup poll of consumers' opinions about water fluoridation showed that 70% supported fluoridation,[1,169,170] however, a local poll may be necessary to provide local elected officials with public opinion data in order for them to enter the fluoridation fray. Public opinion polls may be essential in determining a community's willingness to adopt fluoridation; they can also provide crucial information relative to crafting a clear fluoridation message. Knowing who votes is also important. Additionally, it should be pointed out that not all voters are wealthy, or are better educated suburbanites, and not all nonvoters are poor, less educated inner city

dwellers. According to a national survey, there are 5 different groups of nonvoters: "doers", "unpluggeds", "irritables", "don't knows", and "alienateds."[171] A common strategy used in campaigns is to focus on consistent voters and elderly voters while neglecting the nonvoters, including, in some cases, the inner city voter.[171] Limited resources mean many campaigns limit their focus to groups of expected voters, a practice that is contrary to the principle of inclusiveness, and generally ill-advised, especially with controversial issues.

Perhaps the most crucial parameters for assessing fluoridation success are *timing, readiness,* and *organization.* There are numerous examples of communities throughout the United States where timing, readiness, and organization have played a role in the success or failure of a fluoridation ballot measure; many communities have held more than one fluoridation *initiative/ referendum* to obtain passage. Professional campaign managers, particularly those with experience running issues campaigns, can assist local fluoridation committees in evaluating the TIMING options and in establishing a timeline. Fluoridation campaigns also require financial resources to get the message out. Fundraising can be a major stumbling block for many communities and needs to be considered early on in the campaign.

Public Perception of Risks vs. Benefits of Fluoridation

A mandatory law that passed in both houses of the California state legislature[67] and was signed into law by the governor in 1995, requires communities to fluoridate but it also placed the cost at the local level where it could be interpreted as an *"unfunded mandate."* Many *unfunded mandates* are seen by the electorate to be designed to help the poor at the expense of the working middle class. *Unfunded mandates* generally are viewed as being coercive (not voluntary) and as being controlled by society rather than by the individual, and as such, may be thought to raise the level of public outrage (discussed in later section). According to Sandman (1990), the public's perception of risk is based on the level of outrage felt with respect to a given potential or perceived hazard while the scientific/public health community views risk in terms of the degree of actual hazard. These differing perspectives are exploited by fluoridation opponents who seek to increase the perception of hazard.[172] In Sandman's risk perception analysis of fluoridation (see Table 8-9), 13 variables were examined in which an overall negative score of 7 was assigned.[68,172] Only 4 variables were considered to be positive with respect to mitigating public outrage. On the negative side, fluoridation was summarized as being: "coercive when done by administrative action; industrial or man-made (artificial); dreaded because of alleged association with cancer; unknowable due to the scientific controversy that results when experts appear to disagree; controlled when the public is excluded from the decision making process; not trustworthy with respect to the source of information and mechanisms of accountability; as having closed or secret sources, giving the impression that information is being withheld; and is viewed as being arrogant, as evidenced by contempt for the public's perception."[173] The challenge for fluoridation advocates is to effectively communicate risk/benefit information, using strategies targeted at reducing the outrage towards fluoridation.[172,173] (These strategies are discussed in a later section.)

Question 5

Which of the following statements, if any is/are true?

A. Many countries without centralized water distribution systems use *salt fluoridation,* a process whereby fluoride is added to table salt, in order to provide primary dental caries preventive benefits to their populations.

B. An objective of the U.S. Public Health Service is to have 75% of United States citizens consuming fluoridated water by 2010.

C. The likelihood of voting decreases with education as well as age and income, resulting in certain groups making up a disproportionate share of voters.

D. The public's perception of risk is based on the level of outrage felt with respect to a given potential or perceived hazard while the scientific/public health community views risk in terms of the degree of actual hazard.

E. From a public-policy perspective, fluoridation is more often perceived as a federal or national issue that is mandated by legislation.

Role of Media in Forming Public Opinion and Public Policy

According to a recent report, 93% of persons surveyed said that they regularly watched local news, while 48% reported having watched television network news and 50% reported having read a newspaper, the previous night; 18% listened regularly to talk radio.[174] Over the past decade, there has been increased interest on the part of scholars, political scientists, sociologists, pollsters, politicians, and journalists in tracking the role of mass media on public policy, public opinion, and voting behaviors.[174] Reporting on health issues has not been immune to the confluence of forces where the media often resort to generating controversy in order to increase readership and/or listening/ viewing audience. Also, manufacturing of the news seems to be a trend on the rise in the United States, raising questions of accountability. A 1995 U.S. News poll found that the public appears wary of both the mainstream media and talk radio.[174]

Older, wealthier, and more educated consumers are more likely to read a newspaper. More often than not, fluoridation is explored more thoroughly in the print media, yet readership is declining all over the country, as newspapers merge or shut down operations. While the print media have often editorialized in favor of fluoridation at the time of a public vote, it often comes too late, as doubt has been solidified in the minds of the public. In some cases, however, the media have taken a proactive role in enacting fluoridation[175]; this occurred in Phoenix, Arizona, in 1989 and consequently, 1 million people were added to the fluoridation census.

A busier consumer often turns to the TV for a quick capsule view of what is happening locally, around the state, nationally, and internationally.[176] Consequently, messages are transmitted via 1- to 2-minute sound bites where the negative perspective is more amenable to the world of sound bites than the positive perspective. Converting detailed data to sound bites is a challenge for proponents of a complex public-policy issue, who are held to a veracity standard. In other words, it is far easier to convey opposition to public policy than it is to convey support.

Additionally, the visual impact of this medium can be used either for or against a public policy issue and must be considered in a media campaign strategy. In a network's attempt to be fair and get the viewers attention, mixed messages are often conveyed through use of a news clip that portrays a highly negative visual image coupled with a verbal message that is positive and educational. Mixed messages can create doubt and sometimes apathy and/or cynicism.

Radio, and especially talk radio is not a mass medium to the extent that television is. Over the past decade, the number of radio talk shows has escalated dramatically, with nearly 10% of radio stations having a talk format, serving as a vehicle for the listening public to participate in the political debate.[174] Radio talk shows or "talk radio" are a powerful force in U.S. politics today, in part because of the end of the Fairness Doctrine as well as to changing technology.[174] In 1985, the Federal Communications Commission (FCC) ruled that the Fairness Doctrine, requiring that broadcasters provide a reasonable opportunity for the presentation of opposing views on controversial public issues, was no longer needed.[174] The FCC's ruling was upheld by a Federal Appeals court in 1989.[174] With the end of the Fairness Doctrine, neither radio program hosts nor stations have an obligation to provide balance or present competing views.[174] Consequently, talk radio is an important vehicle for the public to obtain information on a given topic, including fluoridation. If only one viewpoint is expressed on fluoridation, the impact on public opinion could be significant given that talk radio listeners tend to be more politically active.

The use of the satellite dish has enabled stations to receive broadcast quality from anywhere in the country at a relatively low cost; it has also fostered national syndication, allowing local hosts to have access to an instant network.[174] This has been demonstrated in various local efforts to bring fluoridation to the forefront of various communities' agendas. Talk radio hosts connect the listeners with spokespersons from around the country, sometimes establishing a platform for anti-science perspectives that are repeated over and over, yet, have not been substantiated or subjected to evidence-based review. It is not unheard of to encounter talk radio in which a majority of the callers tend to be the solid "aginer voting block" that votes against any issue brought forward by local, state, or federal government. National syndication however, cannot guarantee listenership in today's high-speed world where competition for large audiences in major markets is fierce.[174] Radio is an important medium for disseminating information and should be factored into educational and political media strategies.

Talk radio can be considered an intimate medium in which the caller is usually anonymous and the discussion is spontaneous.[174] According to a report released in 1996 by the Annenberg Public Policy Center: (a) 18% of adults in the United States listen to at least one political call-in radio show twice a week or more; and (b) political talk-show listeners are more likely to consume all news media (other than TV news) and to be more politically savvy and involved, regardless of ideology.[174]

According to a recent nationwide poll of talk radio programs, three-fourths of the talk radio audience is younger than age 60 and listeners tend to have higher incomes and be better educated, with 39% holding college degrees, compared with 21% of Americans overall. Also, 9 of every 10 political talk-radio listeners are registered to vote, compared with 6 of 10 Americans in general.[174]

As public-policy issues, including fluoridation, surface in a given community, they are subjected to media review. The role of mass media in framing critical issues and influencing public policy as well as their impact on how public opinion and values are formed is important to American democracy. Understanding and working with the media in educating the public about public policy issues, including fluoridation is critical to an informed electorate.

Recent advances in technology, such as the information superhighway, have provided yet another powerful communication tool—the *World Wide Web (internet).* The proliferation of websites has exploded in the past few years and with it, the instantaneous dissemination of information and opinions on every topic imaginable. The internet has been embraced by the public as a means of ready access to information. There are a number of health-related websites, including those dealing with water fluoridation. If one searches the internet using various search engines, a significant quantity of information can be found. One search engine turned up 24,100 matches while another had 2,500 matches for "fluoridation." The downside of internet use as a source of valid information is that much of the health information available on the web is opinion-based and has not gone through a rigorous scientific review process, putting the onus on the public for discerning truth from fiction regarding the information presented. Another problem associated with fluoridation information on the internet is the paucity of information from credible research-based entities and recognized professional organizations. Unfortunately, the public is subjected via the internet to predominately negative information from biased opposition groups, rather than being provided objective, science-based information about the safety, efficacy, and cost-effectiveness of fluoridation.

Voter Turnout/Voter Apathy

Knowledge of voter participation is important because *fluoridation is the only public-health issue that is regularly voted on in a community.* Many Americans have opted out of the political decision-making process because of business/time constraints, apathy, and other factors.[177] In the November 2000 presidential election, only 51.21% of eligible voters (voting age population—all persons of age 18 or over) actually cast votes compared to 63.06% in 1960. Historically, voter turnout has been higher in presidential election years than in federal election "off years" where voter turnout went from 47.27% in 1962 to 36.4% in 1998 (see Figure 8-3). Since 1990, nearly three-quarters of the growth in the voting-age population occurred in the 45- to 64-year-old age group, representing approximately 3 of 10 of the voting-age population.[178] According to the Census Bureau, the most likely voters tend to be: white, women, older, married, those with more education, those having higher incomes, those who are employed, and those who are homeowners and/or longtime residents. Interestingly enough, people living in the West are the least likely to register to vote but those who do register to vote are most likely to vote (see Figure 8-4, map/voting/US).

Voter turnout at the local level has followed the same pattern as seen at the state and national levels, with percentages dipping into the teens.[68] Low voter turnouts and special elections have traditionally spelled disaster for fluoridation, especially in larger cities where the "aginer factor" (a constant block of voters who vote against any

government initiative or government involved proposal) is sure to vote in an *initiative/referendum.* Supporters of fluoridation, seeking a vote of the electorate, should consider placing an ordinance for fluoridation on a regularly called ballot that is expected to have higher voter turnouts. Mayoral or county commissioners elections (or even gubernatorial or presidential elections if election laws allow for local issues) generally have voter-turnout rates that are greater than those observed in a special election. In cases where local governing authorities decide to call a public vote on fluoridation in order to let the voters decide, they often opt to have a special election or vote to place it on any other election other than the mayoral or county commissioners election, in order to avoid taking a position on fluoridation that might affect their election or reelection status.[68]

Because of low voter turnout, the prevailing wisdom among proponents of fluoridation suggests that referenda be avoided, but this is contrary to Sandman's (1990) warning that decisions that are perceived as being coercive or as not allowing for individual control/freedom of choice, are more likely to increase the level of outrage, and therefore decrease acceptability. In view of the skepticism of the electorate and taking Sandman's risk perception principles into consideration, it may be necessary to consider the full range of options for implementing fluoridation in communities where city council/commission action are not possible.[68] Perhaps the initiative process or changes in the jurisdictional charter should be considered as part of a comprehensive political strategy. Some jurisdictions, however, may not allow citizens the right to petition. Additionally, in many U.S. communities today, voting periods have been extended. The impact of these factors on an *initiative/referenda* campaign can be substantial in terms of organization, resources, and sustaining get-out-the-vote (GOTV) efforts.[68]

Negative campaigning has also contributed to public disillusionment with policy issues as well as to voter apathy. Political advertisements have not been subject to the same scrutiny as other forms of campaigning, although some networks are taking on the challenge of analyzing such advertisements. Engendering fear and anger among the electorate is the basis of negative campaigning. Fluoridation is one of those issues that lends itself very well to negative campaigning because people's fears and/or anger are exploited by the opposition. Unfortunately, the electorate doesn't necessarily have to agree with those opposed to fluoridation for the issue to fail at the polls. Instead, the voters often react to the vicious negative campaigning and tune out altogether, opting out by voting the issue down or failing to vote at all (no-shows) in hopes that the "controversy" will go away. Even if the voters question the claims of the anti-fluoridation faction, they may be encouraged to vote against fluoridation just because they have a scintilla of doubt about which side is right and opt to "wait and see" until all "questions" have been answered.

Mistrust of government and erosion of public confidence in the government's ability to solve problems, anger at being left out of the process, anger at tax increases, fear of the future because of loss of economic security, fear of employment layoffs, anger and fear caused by declining incomes and benefits, fear of crime, fear of loss of individual rights and freedom, and a sense that the government is ineffective, have had the effect of simultaneously immobilizing a large sector of the population while mobilizing a negatively motivated electorate.[68] Anger and fear are the two most powerful factors motivating people to act and both take front-and-center stage in a

fluoridation campaign. They are also the emotions capitalized upon by the anti-fluoridationists to defeat fluoridation and they are the basis of Sandman's risk perception principles.

Skepticism on the part of the public resulting from programmatic failures in public health (e.g., swine-flu vaccine) combined with a distrust of established scientific methods and confusion over recommended health practices that seem to change weekly, tend to fuel the flames, angering and frustrating the public.[179] Public knowledge and attitudes toward fluorida- tion demonstrate the need for health profess- ionals to continually advocate for fluoridation in order to preserve the successes already achieved.[176,180-183] Fluoridation campaigns that are not sufficiently broad-based with multiple constituencies involved in the decision-making process are likely to encounter problems when political decisions have to be made.[154,184-188] Fluoridation committees composed of health professionals alone are not enough to realize a fluoridation win on a ballot measure, or for that matter, in governmental body actions.

Political Skills and Knowledge of Fluoridation Committees

Even though fluoridation is the only health issue that is often voted on (plebiscite), it is taught as a public-health intervention, not as a political issue. Health professionals are frequently called upon to be involved in fluoridation efforts, yet they often lack political experience, skills in mediation and conflict resolu- tion, or media expertise to deal with the issues raised in a campaign. Knowledge and experience in managing an actual political campaign, including marketing the pro-fluoride message, fundraising, organizing phone banks and get-out-the-vote (GOTV) efforts, such as blockwalking, are not normally taught in the curricula of the health disciplines, and certainly not in U.S. dental schools.[68] Physicians and dentists are often co-opted into reacting negatively, by withdrawing and disappearing and by being defensive and condescending when confronted with a potential voter who disagrees with the premise that fluoridation is the best caries preventive method that a community can adopt.[68] Sometimes, health professionals fail to get involved in fluoridation efforts because they don't feel confident that they can cite evidence-based rebuttals to the broad, all-inclusive laundry list of allegations and objections to fluoridation.[2,189-192] Familiarity with Sandman's risk perception principles (see Table 8-9) as they relate to fluoridation and knowing how to decrease the level of outrage are critical skills that every health professional should have but are not taught. Health professional students are taught to deliver care to individuals not to communities. Even in public health programs, students do not learn these skills, creating a leadership gap as it relates to advocating for fluoridation. In the final analysis, the desired outcome is more dependent upon political skills than on knowledge of fluoridation.[68]

Figure 8-3 Voter Turnout Presidential & Congressional Elections: 1960-2000. (*Source:* Census Bureau, 2000.)

Figure 8-4 Voting Percentages in the United States by State, 1998. (*Source:* U.S. Census Bureau, *Current Population Survey*, P20-523RV, August 2000.)

Question 6

Which of the following statements, if any is/are true?

A. Newspapers usually are helpful in screening misinformation contained in letters to the editor and by columnists

B. The Federal Communications Commission (FCC) ruled in 1985 that the Fairness Doctrine is necessary because it requires that broadcasters provide a reasonable opportunity for the presentation of opposing views on controversial public issues.

C. Talk-radio programs are good forums for serious debates on controversial subjects because they present all sides of an issue equally.

D. Converting detailed data to sound bites is a challenge for proponents of a complex public-policy issue because they are held to a veracity standard.

E. Internet use is a good source of valid health information because the internet site guarantees that all the information provided has gone through a rigorous scientific review process and therefore the public need not be concerned about discerning truth from fiction regarding the information presented.

Why Dentists and Dental Hygienists Should Be Involved in Promoting Community-Water Fluoridation

The third of the five major ethical principles included in the American Dental Association's *Principles of Ethics and Code of Professional Conduct* is the *Principle of Beneficence* that expressly states ". . . that professionals have the duty to act for the benefit of others." Directly related to this specific ethical principle is a designated Code of Professional Responsibility regarding Community Service, that further states that ". . . dentists have an obligation to use their skills, knowledge, and experience for the improvement of the dental health of the public and they are encouraged to be leaders in their community . . .. "[193] Furthermore, the fourth principle is the Principle of Justice emphasizing that "the dentist has a duty to treat people fairly." In concert with this principle is the designated *Code of Professional Responsibility* that states, "in its broadest sense, this principle expresses the concept that the dental profession should actively seek allies throughout society on specific activities that will help improve access to care for all," *[including access to known preventive measures like community-water fluoridation]*.[b] The American Dental Association's ethical standards are but one reason that community-water fluoridation, clearly the gold standard of community-based programs for the prevention of dental caries, should be actively supported by practicing dentists and dental hygienists. Similarly, the *American Dental Hygienists Association's Code of Ethics* states that "ethics compel us to engage in health promotion/disease prevention activities"[194]—advocating for water fluoridation meets this criteria.

The obligation to promote scientifically justified community-based programs, such as community-water fluoridation, also stems from a dentist's/dental hygienist's obligation to serve the community in exchange for the community's contribution to the dentist's/dental hygienist's privilege of practicing dentistry/dental hygiene. First, the privilege of practicing dentistry/dental hygiene has been granted to dentists/dental hygienists by the public, through their state legislators, in the form of a statewide dental practice act managed by a state dental-licensing board. The privilege of practicing dentistry/dental hygiene has been allocated to dentists/dental hygienists

because of society's recognition of the need for having qualified practitioners to serve the public's oral-health needs. Society retains the right to maintain, modify, or cancel these privileges at any point that they feel that the dentists/dental hygienists of their state are not meeting society's needs. By assisting communities to authorize and implement community-water fluoridation, dentists/dental hygienists demonstrate in one small way, their desire to meet their many obligations to society.

[b]Bracketed phrase added. Brackets enclose a phrase added by authors to further illustrate the relationship of the dentist's obligation to promote community-water fluoridation to the ADA's Code of Ethics and Code of Professional Conduct.

Opposition to Community-Water Fluoridation

While fluoridation is generally not considered controversial among the scientific community, it persists as a political lightening rod, an issue to be avoided at all costs by the political community. Pasteurization, immunization, and chlorination all faced opposition initially which subsequently subsided.[195] However, since Grand Rapids, Michigan, began fluoridating its public-water supply in January 1945, there has been a relentless effort by a small but determined opposition to undermine the efforts of health professionals and civic leaders to implement fluoridation around the world. Interestingly enough, "pure-water committees" seem to spring up wherever there appears to be a fluoridation measure on the public agenda. Such committees define pure water as fluoride-free; forty-eight other chemicals frequently used in water treatment are excluded from their definition of pure water. Invoking "the pure water/fluoride-free zone" is a clever tactic designed to push emotional buttons against fluoridation. On the other hand, fluoridation proponents assume that the misinformation dissem- inated by the opponents will be easily refuted and/or readily dismissed since the facts favoring fluoridation are mistakenly thought to be clear and convincing. However, such a miscalculation may be the basis for underestimating the power of the highly motivated opposition who believe that fluoridation is a prime example of government intrusion.[68]

Techniques Employed by Opponents of Fluoridation

Bernhardt & Sprague compiled a list of techniques frequently used by the opposition in attempting to stop the process of fluoridation.[196] Several additional techniques have been categorized and included in the following summary of various techniques employed by opponents of fluoridation.

1. *Neutralizing Politicians*: Once the fluoridation legislation/ordinance has been introduced, opponents attempt to convince state and local elected officials to remain neutral, rather than make the appropriate health policy decision to fluoridate the water supply. Opponents often attempt to give the impression that there is "scientific" legitimacy for their positions by quoting "alternative medicine" websites or using pseudo-scientific spokespersons in hearings, correspondence, and distributed propaganda. Anti-fluoridationists try to convince the elected officials to refer the issue to public vote, usually, a special election where there is inadequate time for proponents to organize effectively, rather than to decide the issue through the normal legislative/administrative process. This strategy is favorable to fluoridation opponents who are often more adept at running a scare campaign focused on negative claims

about fluoridation than they are at convincing skeptical legislators/city/county officials to agree with their views without verification.

The opposition often resorts to massive letter-writing and phone-calling campaigns designed to give the impression that "everyone is against fluoridation," when in fact the vast majority of citizens may very well be supportive of fluoridation. The strategy also involves bombarding the print media with letters-to-the-editors to foster the notion that there is widespread disapproval of fluoridation. Swamping legislators and/or city leaders with reams of propaganda falsely claiming "evidence of harm," without substantiation or of interference with their "freedom of choice," even though legitimate research results universally refute claims of harm and the courts have repeatedly held that fluoridation interferes with no ones' constitutional freedoms, is part of the overall strategy.

Co-opting legislators and/or city leaders is another strategy designed to neutralize public officials. Overestimating the extent of the opposition often results in leaders taking the path of least resistance and concluding that it is safer to have the electorate make the decision rather than enter the fray. By arousing serious doubts about safety, antifluoride zealots give local elected officials an expedient excuse to delay favorable administrative action. Thus, not only has the legislative/city/county official been neutralized, the antifluoridationists have gained more time to inundate the public with negative propaganda designed to create fear and doubt among the public and alter public perception of fluoridation.

2. *Use of the Big Lie:* Anti-fluoridationists repeatedly allege that fluoride causes cancer, kidney disease, heart disease, genetic damage, osteoporosis, Down's syndrome, AIDS, Alzhei- mer's disease, nymphomania, violent behavior, crime, and practically every other malady known to man—a veritable laundry list of unproved allegations. Nonetheless, these laundry lists are repeated so frequently in anti-fluoride pamphlets, letters-to-the-editor, and phone calls to talk-radio shows, that the public may actually begin to believe the unsubstantiated claims. In order to lend some aura of legitimacy to the unproved claims, pseudo-scientists frequently appear as the authors of such letters. In the fall of 2000, a leading anti-fluoridationist spokesman announced to radio listeners in San Antonio, Texas, that fluoridation was directly responsible for 35,000 deaths each year in the United States. If there were any truth to this big lie, fluoridation would cease simultaneously all over the United States and around the world. Yet, while this big lie is clearly off the charts, some questioned why anyone would make such a serious allegation if it weren't true, or possibly true. Public skepticism often results from such a scenario.

The appearance of an allegation in print (such as in the letters to the editor section of local newspapers) is often believed by the public to be evidence of the allegation's validity. The public incorrectly assumes that the "authorities" (in this case print media editors) would not allow allegations to be printed if they were untrue. Thus, the media often become unwitting pawns of the anti-fluoridationists, unless the newspapers are large enough and sophisticated enough to have employed qualified and responsible science editors to eliminate from publication those letters that are scientifically unsound and which constitute a potential for harm to the public.

3. The use of *Half-Truths,* where an out-of-context statement is used to imply a cause-

and-effect relationship with some negative result alleged to have been caused by fluoridation: For example, fluoridation opponents claim that "fluoride is poison, so don't let them put it in our water." This statement ignores the principle that toxicity is related to dose of a substance and not to mere exposure to the substance itself. Chlorine, vitamin D, table salt, iodine, antibiotics, even water, serve as excellent examples of substances that are harmful in the wrong amounts but beneficial in the correct amounts.

Another example is: "fluoride causes dental fluorosis or mottling." By itself, this claim fails to take into account either the source of the fluoride, the amount of fluoride, the mechanism of fluoride exposure, or the time of exposure as related to the dental age of the person exposed. When this claim is made, opponents disingenuously equate fluoridation with the severe form of fluorosis, not the milder forms, even though they know their information is a misrepresentation of the facts. Community-water fluoridation is not responsible for causing the severe dental fluorosis depicted in the photos displayed by the anti-fluoridationists. As stated previously, approximately 13% of children who drink optimally fluoridated water will develop very mild fluorosis. Dental fluorosis as seen in the United States manifests primarily as the milder forms and has been mostly attributed to the inappropriate ingestion of large amounts of fluoride-containing dentifrice by young children who were not properly supervised during toothbrushing and to improper supplementation of fluoride through careless prescriptive practices. Anti-fluoridationists frequently adopt the intellectually dishonest practice of showing photographs of teeth with tetracycline staining or of extremely rare cases of severe dental fluorosis that have occurred in other countries because of extensive industrial pollution or long-term ingestion of extremely high naturally-occurring fluoride levels from noncommunal water sources. They then falsely claim that this will be the result for anyone, including adults, who might drink fluoridated water. As stated previously, adults are not at risk for dental fluorosis.

Another half-truth espoused frequently in the 1980s was: "The majority of AIDS victims come from fluoridated cities." This half-truth was frequently made in misguided attempts to persuade San Francisco's public into stopping fluoridation in that city. This claim continued to be made, even after the discovery of the virus that causes AIDS. While most AIDS patients coincidentally reside in major metropolitan areas and most major metropolitan areas are fluoridated (47 of the 50 largest cities in the United States), the anti-fluoridationists logic never did explain the high incidence of AIDS in Los Angeles, San Diego, or Newark (New Jersey), all not fluoridated at the time of the claim. This same anti-fluoridationist apparently changed his mind and claimed during his unsuccessful 1992 third-party campaign for the U.S. presidency, that AIDS is caused by the AIDS drug AZT, implying that there is a plot by medical professionals, drug companies, and the government to infect certain groups with AIDS.

4. Utilization of *Innuendo:* A frequently used fluorophobic tome is, that "while one glass of fluoridated water will not kill anyone, it is the glass after glass of fluoridated water, as with cigarette after cigarette, that takes its toll in human health and life." This technique uses a guilt-by-association ploy, attempting to link the known health risks of cigarette smoking (for which there is substantial scientific evidence) to alleged risks from drinking fluoridated water (for which there is no scientific evidence).

Another oft-used claim by fluoridation opponents is that "insufficient research has been carried out to prove absolute safety, and therefore consumers and government officials are urged to wait until all doubt about the safety of fluoridation has been 'scientifically' resolved." This argument could be used indefinitely in that it is impossible to ever prove absolute safety for all time for anything. Unqualified acceptance of this argument would mean that literally all technologic advancements achieved in the age of science would have to be eliminated. Thousands of studies and untold risk-benefit analyses have shown that fluoridation is safe and effective for the entire population.

5. *Quoting of Inaccurate Statements and the Use of Statements Taken Out of Context:* The best way to illustrate this common technique of anti-fluoridationists is to refer to two frequently used anti-fluoridation publications, the *Lifesavers* (Sic) *Guide to Fluoridation* (a pamphlet)[197] and *Fluoride: the Aging Factor.*[198] (a book). Both use essentially the same "scientific references," both are distributed frequently in campaigns, opposing fluoridation, and both documents were marketed by their author as "scientific documents." The one-sheet pamphlet claimed over 250 references. Subsequently, a group of 20 scientists and public health officials from around the United States did a systematic review, tracking down the original references in order to evaluate their validity as used by the author. The project took two years and resulted in the production of a 184-page monograph, entitled *Abuse of the Scientific Literature in an Anti-fluoridation Pamphlet.*[199] In the monograph, the 20 scientists documented that the information in the *Lifesavers (Sic) Guide to Fluoridation*[197] pamphlet was primarily fabricated pseudo-science for which no scientific evidence was available. Some of the findings of this protracted review included: (a) of the 250 references, only—48 were from reputable scientific journals; (b) 116 of the 250 references had no relevance to community water fluoridation whatsoever; (c) many of the references actually supported fluoridation with the works of respected scientists selectively quoted, misquoted, and misrepresented in order to make them appear to discourage the use of fluorides.[198]

6. *Quoting of Experts:* Some of the quoted experts have legitimate academic or professional credentials, although not necessarily in disciplines qualified to serve as experts in health research specific to fluorides. Moreover, anti-fluoridationists occasionally find a credentialed individual to speak against mainstream science. The statements by these marginalized individuals, while of questionable authority, are often exploited by the opposition.

Some nationally known figures who may have opposed fluoridation early in their professional life prior to the accumulation of overwhelming scientific evidence in its favor, often have their earlier statements quoted by anti-fluoridationists despite having publicly changed their position to one of support for fluoridation. As an example, the opposition repeatedly claim that Nobel Laureate and physician Hugo Theorell "condemns" fluoridation when, in fact, he publicly changed his position to one of support as far back as 1967. The public is further confused when anti-fluoride zealots utilize the services of "alternative medicine" spokespersons to "prove" that the medical community is divided in its position on fluoridation. Unable to discriminate between legitimate scientists and purveyors of unproven therapies, some in the public see the dispute as a conflict between competing health care philosophies and

ideologies.

7. *The Conspiracy Gambit:*[196] Because alleged conspiracies are difficult to disprove, they are a favorite of the health-conspiracy theorists. The alleged "conspirators" often include the American Medical Association, the American Dental Association, the American Council on Science and Health, the equipment and chemical supply companies, the Communist Party, both the aluminum and phosphate fertilizer industries, toothpaste manufacturers, or any other organization appearing to be threatening to the anti-fluoridationists. Highest on their list of conspirators is the "government" (including the Public Health Service, the Environmental Protection Agency, the prestigious National Institutes of Health, the world-renowned Centers for Disease Control, and the Food & Drug Administration). Conspiracies generate a tremendous amount of anger among those susceptible to conspiracy propaganda, a factor that negatively impacts fluoridation efforts.

8. *The use of Scare Words:*[196] Anti-fluoridationists frequently play on the current phobias and concerns of the public by describing fluoridation in ecologically-linked or environmentally-loaded terms or phrases such as, "pollutant, toxic waste product, chemical by-product, dumped in the water, or forced down our throats." Fluoride is also frequently linked by fluorophobics with words like "poison, genetic damage, cancer, AIDS, or artificial"—words that certainly conjure up fear by the public when linked to something to which they think they will be unwittingly exposed. Fear is a major factor that negatively impacts fluoridation efforts.

9. *The Debate Ploy:*[196,200] The opponents of fluoridation often try to entice unsuspecting media commentators, government officials, or program planners into holding a debate on the "pros and cons" of fluoridation. Proponents of fluoride are then often trapped into consenting to public debates. Jarvis has published a list of reasons for not debating the anti-science health viewpoint: (a) the purpose of the debate is to win the audience, not to discover truth. Science is not decided by debating in a public forum, but by careful experimentation, confirmation of findings through independently conducted experiments, submission of all findings to qualified colleagues and peers for critical analysis, and publication of findings in reputable peer-reviewed journals. In a debate, even though the proponents may win the debate, they are just as likely to lose the audience. (b) In media circles, there is a saying that "everyone is the same size on television." In other words, debates give the illusion that a scientific controversy exists when, in reality, this is not the case. Public debates also promote the illusion that there are equal numbers of "scientists" on each side of the issue. The vision of "dueling PhD's or dueling doctors" encourages the public to reject fluoridation until the "experts on both sides can agree." (c) An opponent of fluoridation, utilizing the laundry list approach, can present more misinformation in 5 minutes than can be refuted in 5 hours, thus fostering confusion on the part of the public. Proponents are never provided enough time to adequately refute the opponents' charges, because complete refutations, by their nature, take much longer than the sound-bite length charges of the anti-fluoridationists. (d) Public exposure favors the opponent, enabling him or her to gain name recognition for the viewpoint they are promoting. By sharing the platform with respected scientists who are there to defend fluoridation, equal status and credibility is granted. Anti-fluoride groups often "attach" themselves to other organizations' events in order to draw attention to their cause. (e) It is difficult to compete in a debate without appearing to discredit the

opponents personally. When a fluoridation proponent is refuting a negative statement made by the anti-fluoridation spokesperson who is spreading misinformation, the proponent has to be able to separate the anti-science message from the anti-science messenger, an extremely difficult task. Moreover, the debate format often favors the maverick viewpoint as the perceived underdog, as in a David vs. Goliath showdown, generating sympathy for the anti-fluoridation perspective. One of the strategies used by anti-fluoridationists in a debate setting is to intimidate the proponent(s) by threatening to file a lawsuit for defamation of character. This strategy is very unsettling for an untrained proponent who often loses focus, confidence, and becomes ineffective as a debater. When such a threat is made in a debate, it is hard to remember that few lawsuits have actually been filed and none of these lawsuits have been successfully prosecuted by any of the anti-fluoridationists.

Five additional anti-fluoridationist techniques not cited by Bernhardt & Sprague in their paper because they have appeared since its publication include the use of contrived organizations, subversion of the media, commandeering established organizations, misuse of electronic publishing, and commandeering meetings.[2]

(1) The *use of contrived organizations* is most disturbing:[2] The opponents often form their own pseudo-scientific organizations having names that sound like legitimate scientific entities, but which are in reality, front organizations for the anti-fluoridationist movement.

(2) *Subversion of the media*:[2] The job of the media is to present all sides of an issue. Often the media appear to be more interested in publicizing a controversy than in accurately representing an issue.[2] Many campaign committees have encountered a popular media philosophy "if it bleeds, it leads". Moreover, it is often more profitable for the media to do a story on the "dangers" of fluoridation which can be sensationalized than to do one on the many scientifically sound, but emotionally unexciting, reasons for supporting fluoridation. Also, the anti-establishment and anti-science viewpoint tends to be more flamboyant and interesting to a media seeking to portray readily understandable examples of John Q. Public fighting city hall. It is important to remember that scientific rebuttals to flamboyant anti-fluoridation claims are often, by their nature, dry, unemotional, complex, difficult to explain in lay terms, hard for the public to grasp conceptually, and difficult for the media to interpret and report.

(3) *Commandeering Established Organizations:*[2] In several instances, anti-fluoridationists have commandeered established organizations in an attempt to gain access to the organization's credibility for the anti-fluoride cause.[2] Two recent examples involve the Pennsylvania Sierra Club and one of the collective bargaining units for the U.S. Environmental Protection Agency. In August 1997, a member of the Pennsylvania Chapter of the Sierra Club held a press conference in Harrisburg in which she claimed that the Sierra Club called for a ban on fluoridation in that state.[2,201] Within a few days, the officers of the Chapter issued an official statement rebuking the member's action and stating that the press conference was held "without the knowledge or authorization of any Pennsylvania Sierra Club officer."[2,202]

The U.S. Environmental Protection Agency (EPA) has over 18,000 employees represented mostly by four collective bargaining units. The smallest of these

bargaining units was Local 2050 of the National Federation of Federal Employees (NFFE), a union that variously claimed to represent about 900, 1000, 1100, and finally 1550 EPA employees, but whose dues-paying membership was apparently much less.[2] About 20 dissident members of the union held a meeting on July 2, 1997, where a minority of them voted to oppose California's mandatory fluoridation law.[2] A subsequent press conference falsely claimed that all union members unanimously approved the resolution and subsequent mass mailings of propaganda leaflets from two of the union's anti-fluoride activists falsely implied that the USEPA opposed fluoridation.[2,203]

(4) *Misuse of Electronic Publishing*:[2] Numerous anti-fluoridation websites have been established in order to promote the anti-fluoride political agenda and to recruit converts to their movement.[2] In addition, many "alternative medicine" websites have included anti-fluoridation sections as part of their marketing effort, along with information that: opposes traditional scientific medical practice, attacks orthodox medical practice, including such widely accepted public-health practices as immunization programs. Many of these anti-fluoridation web sites contain "articles," letters, endorsements, or references to purveyors of "alternative" or "complementary medicine." Some also contain links to websites operated by practitioners and marketers of non-scientific therapies.

(5) *Commandeering Meetings*:[2] Often, anti-fluoridation spokespersons attempt to insert themselves into the agenda of scheduled meetings or hearings in order to gain a forum from which to disseminate their anti-fluoridation message.[2] It is not uncommon for anti-fluoridationists to attempt to utilize question and answer periods in public meetings to espouse the "pitfalls" of fluoridation rather than to ask questions of scheduled speakers. Town meetings, allegedly scheduled to provide opportunities for proponents and opponents to present their cases, often serve as a convenient forum from which anti-fluoridation spokespersons try to dominate the available time.

Question 7

Which of the following statements is correct, if any?

A. Pseudo-science can prevail over science when the electorate is not familiar with either side of the subject being debated.

B. Fear is often used by fluoridation opponents to create doubt about the safety and benefits of fluoridation.

C. Support by practicing dentists and dental hygienists for community water fluoridation is incongruent with American Dental Association's ethical standards.

D. The use of half-truths to imply a cause-and-effect relationship such as "fluoride is a poison, so don't let them put it in our water" is a common technique employed by fluoridation opponents.

E. In a debate, one speaker's truth is no more authoritative than the second speaker's misinformation in a highly technical discussion.

Risk Communication

Community water fluoridation, while long accepted by qualified scientists and credible professional organizations as a safe, effective, efficient, economic, socially equitable, and envi- ronmentally sound public-health activity, has endured attacks from a small, but highly vocal, group of tenacious antagonists throughout its 56-year history. These attacks have served to raise questions among some members of the public, have sometimes served as a convenient excuse for elected officials to avoid making decisions to fluoridate individual community water systems, and have accommodated some in the media where the issue is often exploited so as to appear to be a rift among "experts" on both sides of the issue, creating doubt among the public about the issue. Most communities successfully work through the "controversy," usually a result of hard work by health professionals and sustained objectivity on the part of community leaders and elected officials. Often misinformation is broadly disseminated, adversely influencing community sentiment such that other measures become necessary to counter the mass phobia sometimes generated during the legislative, campaign or administrative process.

Risk communication is a recent addition to the armamentarium of health professionals promoting fluoridation in their communities. It serves as a mechanism by which to counter some of the negative community sentiment generated during attempts to fluoridate communities. Sandman has classified this intense negative feeling about what is perceived by some to be a health risk as *outrage*.[172] According to Sandman, the public defines risk in terms of "levels of outrage". The scientific and health community, who define risk in terms of "hazard, are often too slow to recognize the disparity between actual risk (hazard) as calculated by the scientific community and perceived risk (outrage) as echoed by the public.[172] According to Sandman, the public pays far too little attention to hazard, while most experts pay absolutely no attention to outrage.[172] A public whose level of outrage has been heightened by a well-orchestrated anti-fluoridation campaign, will be less receptive to educational campaigns by proponents of fluoride until the level of outrage is reduced. Pertinent risk information cannot be communicated when the level of outrage is high because the intended recipients of the information cannot collate the complex explanations while frightened by the fluoridation message and/or angry at the fluoridation messenger.

Scholars of risk perception have defined more than 20 factors that affect the public's level of outrage. A few of Sandman's favorite factors are presented as follows (see Sand-man/Table 8-9).

- Voluntariness: A voluntary risk is much more acceptable to people than a risk felt by the public to have been coerced because a voluntary risk generates little or no outrage.[204] Voluntariness helps explain why antifluoridation propagandists will offer organized voluntary fluoride supplement programs as an acceptable (to them) alternative to "coerced" community water fluoridation.[204]
- Control: When disease prevention and exposure mitigation are in the hands of individuals (fluoride supplements), the risk (though not the hazard) is perceived by them to be much lower than when the same programs are controlled by a government agency (municipal water system and health department).
- Fairness: People who feel that they are enduring greater risks than their neighbors,

especially if they feel that they are without access to greater benefits, are naturally outraged, more so if the rationale for increasing their risk appears to have been decided through the political process rather than through science.[204] Even though fluoridation benefits people of all ages, older Americans often assume that it only benefits children and frequently complain that they are being put at risk without accruing any benefits themselves.

- Process: Sometimes the process by which fluoridation is approved becomes the principle focus of the public's outrage, particularly when the agency or group promoting fluoridation portrays itself as arrogant rather than concerned, dishonest rather than trustworthy, and manipulative rather than collaborative.[204]
- Morality: American society has evolved in its thinking about pollution to feel that it is not just harmful, it is morally evil.[204] Fluoridation opponents often attempt to portray fluoridation as a form of pollution and claim that fluoride chemicals are products marketed by the chemical industry as beneficial (fluoridation) in order to avoid paying the costs to dispose of these chemicals. When fluoridation proponents start talking about cost-risk tradeoffs in this kind of political climate, they often appear to be callously advocating a morally relevant risk.
- Familiarity: Exotic, high-tech facilities and processes (computer-monitored water treatment plants that add fluoride and other chemicals) provoke more outrage than do familiar risks (fluoride-containing toothpaste as part of home dental care).[204]
- Memorability: A memorable accident (especially one involving chemicals or radiation, like Love Canal (New York), Bhopal (India), Times Beach (Missouri), Three-Mile Island (Pennsylvania), or Chernobyl (Ukraine), makes the potential risk easier to imagine and therefore, perceived to be more risky.[204] A strategy used by fluoridation opponents is to attempt to engender fear among the public by emphasizing the statistically minute potential of overfluoridation or hyperfluoridation as if it were a likely catastrophic event.
- Dread: Illnesses like cancer, AIDS, Alzheimer's disease, or end stage renal disease are more dreaded than dental caries.[204] Fluoridation opponents help incite fear among the public by falsely claiming that fluoridation causes these dreaded diseases or makes them incurable, while at the same time attempting to minimize fluoridation's strong preventive effect on dental caries.
- Diffusion in Time and Space: Hazard-A (rampant dental caries) ultimately could result in the deaths of 50 or more anonymous people a year across the country, while Hazard-B (a poorly monitored and poorly operated fluoridation system) resulted in one very well publicized death recently (despite 56 years of safe, effective fluoridation efforts that daily benefited tens of millions of people).[204]

Myths and Actions Related to Risk Communication

Some of those involved in community organization for fluoridation promotion fail to properly consider the role of outrage in the community decision-making process. They assume that the public will trust them and that by merely presenting the scientific data, the public will be "won over." By ignoring the role of outrage, they miss the opportunity to succeed through use of a collaborative effort in community education and community decision-making. Chess and others have categorized a number of *myths* and actions *related to risk communication.*[205] Ten of them include:

- Myth 1: Because the fluoridation referendum is so close, we don't have enough time and resources to have a risk communication program.[205]

- Action 1: Train fluoridation proponents to communicate more effectively. Plan projects such that there is time to involve the public in priority setting and decision-making.[205]
- Myth 2: Telling the public about a potential risk related to fluoridation is more likely to unduly alarm people than keeping quiet.[205]
- Action 2: Fluoridation proponents can decrease the potential for alarm by giving the public a chance to express their concerns and by appropriately responding to these concerns.[205]
- Myth 3: Communication is less important than education. If people knew the true risks related to fluoridation, they would accept them.[205]
- Action 3: Pay as much attention to your process for dealing with people and their fears of fluoridation as you do to explaining the scientific data.[205]
- Myth 4: We shouldn't go to the public until we can provide answers or solutions to all their perceived fears about fluoridation.[205]
- Action 4: Provide information about fluoridation and discuss concerns about risk management options. Involve the community in the development of strategies for which they have a stake.[205]
- Myth 5: These issues and this scientific data regarding fluoridation are too difficult for the public to understand.[205]
- Action 5: Separate public disagreement with your fluoridation promotion practices from misunderstanding of the highly technical issues related to fluoridation.[205]
- Myth 6: One of the easiest myths for dental professionals to embrace is that technical decisions should be left in the hands of technical people.[205]
- Action 6: Provide the public with information about fluoridation. Listen to community concerns about fluoridation. Involve people with diverse backgrounds on the fluoridation committee so that much thought and discussion goes into developing fluoridation policies and strategies.[205]
- Myth 7: I am just a dentist/dental hygienist, risk communication is not my job.[205]
- Action 7: As a public servant, whether the fluoridation promoter works for a health department or has a private dental/dental hygiene practice, you have a responsibility to the public. Learn to integrate risk communication into your efforts and help others from the fluoridation committee do the same.[205]
- Myth 8: If we give them an inch, they will take a mile.[205]
- Action 8: If you listen to people when they are asking for inches, they are less likely to demand miles. Avoid the battleground that could result from attempts to stifle discussion about all aspects of fluoridation. Do not attempt to stifle discussion of issues about which fluoridation proponents are uncomfortable. Involve the public early and often.[205]
- Myth 9: If we listen to the public complain about risks from fluoridation, we will devote scarce resources to issues that are not really a great threat to the public's health.[205]
- Action 9: Listen carefully and early to avoid controversy and the potential for disproportionate attention to lesser issues.[205]
- Myth 10: Activist anti-fluoride groups are responsible for stirring up unwarranted concerns.[205]
- Action 10: Anti-fluoride activists help to focus public anger. Work hard to gain the public's trust early, so that you can work with responsible public groups to promote the adoption of responsible public policy regarding fluoridation.[205]

Covello and Allen[206,207] have developed a list of *Ten Deadly Sins of Communication.* They are fairly self-explanatory and follow: (1) appearing unprepared; (2) handling questions improperly; (3) apologizing for yourself or your organization; (4) not knowing knowable infor- mation; (5) unprofessional use of audiovisual aids; (6) seeming to be off schedule; (7) not involving participants; (8) not establishing rapport; (9) appearing disorganized; and (10) providing the wrong content.

It remains obvious that the mere dissemination of information to the public, without any attempts to communicate the complexities and uncertainties of risk, does not necessarily ensure that the public will understand or accept community water fluoridation. Well-managed risk-communication efforts will help ensure that the public is provided with messages that are constructively formulated, transmitted, and received, and that they will be more likely to result in positive thoughts and an acceptance of fluoridation. In the words of Baruch Fischhoff, "If we have not gotten our message across, then we ought to assume that the fault is not with our receivers."[208]

Principles of Risk Communication

The *principles of risk communication,* if practiced universally, can go a long way towards increasing the speed with which the public accepts community water fluoridation as a local policy option. Covello and Allen[208] have developed *Seven Cardinal Principles of Risk Communication,* all designed to help fluoridation promoters accomplish their goals.

1. Accept and involve the public as a partner. Your goal is to produce a public informed about the advantages of fluoridation, not to defuse public concerns or replace actions.

2. Plan carefully and evaluate your efforts. Different goals, audiences, and media require different approaches and different actions.

3. Listen to the public's specific concerns. People often care more about trust, credibility, competence, fairness, and empathy than about statistics and details.

4. Be honest, frank, and open. Trust and credibility are difficult to obtain and, once lost, are almost impossible to regain.

5. Work with other credible sources. Conflicts and disagreements among organizations make communication with the public much more difficult.

6. Meet the needs of the media. The media are usually more interested in controversy than risk, simplicity than complexity, danger than safety. Help them understand the differences.

7. Speak clearly and with compassion. Never let your efforts prevent your acknowledging the tragedy of an illness, injury, or death, or even their potential. Acknowledge and empathize with people's fears. People can understand risk information, but they may still not agree with you. Some people will never be satisfied with your answers.

Summary

Water fluoridation is the prime example of community-based caries prevention where the benefits accrue to all individuals consuming drinking water that is optimally fluoridated without regard to socioeconomic status. Fluoridation remains a safe, effective, efficient, economical, environmentally sound, and socially equitable public health measure to prevent dental caries.[2] It also fulfills all of the requirements of an excellent public policy.[2] Based upon extensive scientific evidence, regarding the safety and effectiveness of fluoridation, numerous national and international organizations and agencies have advocated for the adoption of fluoridation as a means of reducing dental caries in a community. Despite some minor opposition that sporadically delayed fluoridation's implementation in some locales, substantial progress has been made toward achieving the long-term goal of universal fluoridation in the United States. While the opposition to fluoridation has been fairly disorganized and generally not too effective, recent opposition from "alternative medicine" zealots and purveyors of unproved health modalities suggests that the public, elected officials, and the media may be both confused and unduly influenced in the future by such open support of anti-fluoridation efforts. Open opposition to fluoridation gives the "alternative medicine" promoters a convenient public forum with which to stress their "philosophical" differences from traditional science-based health care. The dental profession should educate the public, using risk-communication principles as a standard business practice to assist patients in obtaining accurate information about fluoridation.

What needs to be kept in perspective is the tremendous success that health professionals have had in bringing community water fluoridation, one of the greatest public-health achievements of the 20th century, to more and more Americans each year. The Surgeon General of the United States has included a fluoridation objective in his Year 2010 Health Objectives for the Nation.[7,209] By the year 2010, 75% of the population on community-water systems should live in communities with fluoridated water according to one of the document's goals. Given the substantial growth in population in the U.S. since the last official fluoridation census was published in 1992 where over 62% of the population on community-water systems were benefiting from fluoridation, and the addition of millions of people because of recent successful fluoridation campaigns in Los Angeles, San Diego, Las Vegas, San Antonio, and Salt Lake City, the gap between those who have access to fluoridated water and those who do not have access to this public-health measure is beginning to be acknowledged by policy makers, community leaders, and health professionals (see Figure 8-5, Fluoridation Growth).

As health professionals, we must realize "oral health is not solely dependent on individual behaviors," that attainment of optimal oral health requires a partnership between the dental health profession and both the patient and their community. "Universal access to fluoride requires a community's commitment to water fluoridation" and the dental profession must provide the leadership, technical expertise and guidance to policy makers as well as to the public to bring fluoridation to fruition. Every dental office, dental health department, community dental health program, and dental school should have copies of the American Dental Association's

monograph, Fluoridation Facts in its libraries in order to be adequately briefed on the issue.

Figure 8-5 Fluoridation Growth, by Population, United States, 1945-2000. (*Source:* Centers for Disease Control, 2000; U.S. Census Bureau; *MMWR 2002*, 51(07);144-7.)

Answers and Explanations

1. A, B, C, D—correct.

E—incorrect. The recommended optimal range of fluoride concentrations in drinking water is. 7 to 1.2 ppm depending on mean annual temperature.

2. A, B, C, E—correct.

D—incorrect. All fluoride-containing dentifrices have very high levels of fluoride (1,100 to 1,500 ppm) and are a significant source of fluoride overexposure and fluorosis.

3. A, B, E—correct.

C—incorrect. Discontinuation of fluoridation has a significant deleterious impact on dental-caries rates; several studies have consistently shown that dental-caries rates increase dramatically when fluoridation is discontinued.

D—incorrect. The national cost-benefit ratio has been demonstrated to be 80:1 where for every $1 dollar spent on fluoridation, $80 is saved in treatment costs.

4. A, D, E—correct.

B—incorrect. No credible scientific evidence has associated water fluoridation with cancer, and the National Cancer Institute has not recommended the cessation of fluoridation.

C—incorrect. Hydrofluosilicic acid is the most frequently used chemical for community water fluoridation.

5. A, B, D—correct.

C—incorrect. The likelihood of voting *increases* with education as well as age and income, resulting in certain groups making up a disproportionate share of voters.

E—incorrect. From a public-policy perspective, fluoridation is more often perceived as a *local issue* that is enacted either by *governmental administrative action* (ordinance that is voted upon by a city council or city/county commission) or by a vote of the public.

6. D—correct.

A—incorrect. First, newspapers usually do not have experts in all technical areas; secondly, they try to publish an equal number from each side of an issue to be fair, to encourage debate—and to promote the sale of the paper.

B—incorrect. In 1985, the Federal Communications Commission (FCC) ruled that the Fairness Doctrine, requiring that broadcasters provide a reasonable opportunity for the presentation of opposing views on controversial public issues is *no longer needed.* With the end of the Fairness Doctrine, neither radio program hosts nor stations have an obligation to provide balance or present competing views.

C—incorrect for the same reasons as A.

E—incorrect. The downside of internet use as a source of valid information is that much of the health information available on the web is opinion-based and has not gone through a rigorous scientific review process, putting the onus on the public for discerning truth from fiction regarding the information presented.

7. A, B, D, E—correct.

C—incorrect. The third of the five major ethical principles included in the American Dental Association's *Principles of Ethics and Code of Professional Conduct* is the *Principle of Beneficence* that expressly states ". . . that professionals have the duty to act for the benefit of others." Directly related to this specific ethical principle is a designated Code of Professional Responsibility regarding Community Service, that further states that ". . . dentists have an obligation to use their skills, knowledge, and experience for the improvement of the dental health of the public and they are encouraged to be leaders in their community. Support by practicing dentists and dental hygienists for community water fluoridation *is congruent* with the American Dental Association's ethical standards.

Self-Evaluation Questions

1. The first person to demonstrate the relationship between dental caries and water fluoride levels in the United States was _____ (U.S. Public Health Officer).

2. The apparent reduction in measurable water fluoridation benefits resulting from the ubiquitous availability of fluoride from other sources in both fluoridated and fluoride-deficient comparison communities is known as _____.

3. The extension of the benefits of community water fluoridation to residents of fluoride-deficient communities due to the transport of food and beverages commercially prepared with fluoridated water is known as _____; it is called the _____ effect.

4. A _____ analysis is used to relate the dollar cost of water fluoridation to the treatment costs saved.

5. The major posteruptive effect of fluoride from community water supplies is its reduction of _____ and enhancement of _____.

6. Other community-based methods of providing fluoride to communities where water fluoridation is not feasible include _____ and _____.

7. _____communication is a new tool to be used by dental and public health professionals in order to assist communities in choosing to fluoridate their community water systems.

8. Jarvis recommends that dental and public health professionals not get involved in a _____ with antifluoridationists because the primary goal of such an activity is to win the audience rather than to discover scientific truths.

9. _____has stated that the use of sodium silicofluoride and hydrofluosilicic acid for fluoridating community water systems serves as an excellent example of beneficial recycling, where both industry and the public benefit.

10. _____ state(s) (plus the District of Columbia and Puerto Rico) currently mandate(s) statewide fluoridation through legislation, while state(s) have/has mandated statewide fluoridation through administrative regulation.

11. Every recent _____ of the U.S. Public Health Service has recognized the value of community water fluoridation and has promoted its adoption as good public policy.

12. The level of risk as perceived by the public (perceived risk) is often defined by community organizers as _____ while the level of risk as determined by science-based experts (actual risk) is often defined by community organizers as _____

13. A _____ is an election which has been designed to affirm, change, or cancel previously established legislation, while a voter _____ is an election that was petitioned by voters to establish a law requiring something (for example requiring fluoridation or banning fluoridation).

14. In the final analysis, winning a fluoridation campaign depends more upon _____ than on knowledge of fluoridation.

15. Radio talk shows or "talk radio" are a powerful force in U.S. politics today, in part because of the end of the _____ as well as changing technology.

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Chapter 9. Topical Fluoride Therapy - *Kevin J. Donly George K. Stookey*

Objectives

At the end of this chapter, it will be possible to

1. Indicate the only three fluoride compounds accepted for professional applications to control caries and indicate their relative effectiveness.

2. Discuss the possible chemical reactions associated with the topical application of sodium fluoride (NaF), stannous fluoride (SnF_2), and acidulated phosphate fluoride (APF).

3. Relate what percentages of NaF and SnF_2 are available for office and home use (as solutions or as gels).

4. Describe how a liquid or gel topical application of fluoride is applied to the teeth. Emphasize particularly those parts of the technique that are especially important with regard to safety and efficacy.

5. Nearly all dentifrices on the market contain fluoride. Indicate why the early dentifrices did not produce the expected caries decrements.

6. State the expected decrease in caries formation following use of dentifrices and mouthrinses containing fluoride.

7. Describe fluoridated varnishes and fluoride-releasing dental restorative materials and the potential of these materials to inhibit demineralization and enhance remineralization.

Introduction

When communal-water supplies are available, water fluoridation clearly represents the most *effective, efficient,* and *economical* of all known measures for the prevention of dental caries although similar results have been observed with fluoridated salt in many countries. Unfortunately, fluoridated water is available to only about two-thirds of the population. Thus it is obvious that additional measures are needed for the dental profession to provide greater protection against caries to as many segments of the population as possible.

The term *topical fluoride therapy* refers to the use of systems containing relatively large concentrations of fluoride that are applied locally, or topically, to erupted tooth surfaces to prevent the formation of dental caries. Thus this term encompasses the use of fluoride rinses, dentifrices, pastes, gels, and solutions that are applied in various manners.

Mechanism of Action

Studies of the use of professional topical fluoride applications for the control of dental caries began in the early 1940s. Since that time, it has been generally accepted that the fluoride content of enamel is *inversely* related to the prevalence of dental caries.

Using *in vivo* enamel-sampling techniques and improved analytic methods investigators have been better able to quantitate this relationship. For example, Keene and coworkers[1] explored this relationship in young naval recruits 17 to 22 years of age; their observations are summarized in Table 9-1. These data suggest that the presence of *elevated* levels of fluoride in surface enamel is associated with minimal caries experience.

A much more extensive investigation of this relationship was reported by DePaola and coworkers.[2] These investigators similarly examined 1,447 subjects, 12 to 16 years of age, who were lifetime residents of selected fluoridated and nonfluoridated communities; again the inverse relationship between enamel fluoride content and caries prevalence is apparent.

At the time of tooth eruption, the enamel is not yet completely calcified and undergoes a *post-eruptive period*, approximately *2* years in length, during which enamel calcification continues. Throughout this period, called the period of enamel *maturation*, fluoride, as well as other elements, continues to accumulate in the more superficial portions of enamel. This fluoride is derived from the *saliva* as well as from the exposure of the teeth to fluoride-containing *water* and *food*. Following the period of enamel maturation, relatively little additional fluoride is incorporated from such sources into the enamel surface.[3] Thus, most of the fluoride that is incorporated into the developing enamel occurs during the *pre-eruptive* period of enamel formation and the *post-eruptive* period of enamel maturation.

The continued deposition of fluoride into enamel during the later stages of enamel formation, and especially during the period of enamel maturation, results in a concentration gradient of fluoride in enamel. Invariably the *highest* concentration of fluoride occurs at the very outermost portion of the enamel surface, with the fluoride content *decreasing* as one progresses inward *toward the dentin*.[4,5] This decrease in fluoride concentration is extremely rapid in the outermost *5 to 10 microns* of enamel and is much less pronounced thereafter. This characteristic fluoride concentration gradient has been observed in unerupted teeth as well as in erupted teeth and in both the permanent and deciduous dentition, regardless of the amount of previous exposure to fluoride.

The presence of elevated concentrations of fluoride in surface enamel serves to make the tooth surface more resistant to the development of dental caries. Fluoride ions,

when substituted into the *hydroxyapatite crystal*, fit more perfectly into the crystal than do *hydroxyl ions*. This fact coupled with the greater bonding potential of fluoride serves to make the apatite crystals more compact and more stable. Such crystals are thereby *more resistant* to the *acid dissolution*[6,7] that occurs during caries initiation. This effect is even more apparent as *the pH* of the enamel environment *decreases* due to the momentary loss of minute quantities of fluoride from the dissolving enamel and its nearly simultaneous *reprecipitation* as a fluorhydroxyapatite.[8]

Most of the initial studies concerning topical fluoride applications were conducted with sodium fluoride. It was recognized at that time that prolonged exposure of the teeth to low concentrations of fluoride in the dental office was not practical. To overcome this problem, two approaches were explored: *increasing the fluoride concentration and decreasing the pH of the application solution*.

Although the ability of sodium fluoride to increase the resistance of enamel to acid dissolution had been reported on several occasions, it had also been reported that lowering the pH of the sodium fluoride solution greatly increased its protection against enamel decalcification. Five clinical caries studies were conducted to evaluate the effectiveness of *acidulated sodium fluoride* topical solutions. The fluoride solutions were acidulated in various manners (e.g., acetic acid, acid phthalate) and used with varying conditions, but in no instance was a statistically significant caries-preventive effect observed. Thus, the use of acidulated sodium fluoride systems was abandoned, at least *temporarily*.

On the other hand, the observed results of increasing concentrations of fluoride were very encouraging, particularly when multiple applications were used. Although it was initially postulated that the effectiveness of topically applied sodium fluoride was due to the formation of a fluorhydroxyapatite,[9,10] subsequent investigations indicated that the primary reaction product involved the transformation of surface hydroxyapatite *to calcium fluoride*.[11-16]

Question 1

Which of the following statements, if any, are correct?

A. The maturation of enamel is an occurrence that continues at a linear rate from eruption into adulthood.

B. The fluoride content is highest at the outer surface of the enamel and decreases at a linear rate toward the dentin.

C. As a result of acid-induced demineralization followed by remineralization in the presence of fluoride, hydroxyapatite can become fluorhydroxyapatite.

D. The enamel is relatively more protected by neutral pH fluoride solutions than acidulated solutions.

E. With higher concentrations of fluoride, the main reaction product is fluorhydroxyapatite.

The preceding reaction involves the breakdown of the apatite crystal into its components followed by the reaction of fluoride and calcium ions to form calcium fluoride with a net *loss* of phosphate ions from treated enamel. Newer fluoride systems incorporate a means of preventing such phosphate loss.

The early investigators of the reaction between soluble fluoride and enamel observed that the nature of the reaction products was markedly influenced by a number of factors, including fluoride *concentration*, the *pH* of the solution, and the length of *exposure*. For example, the use of *acidic fluoride* solutions greatly favored the formation of *calcium fluoride*.[11] *Neutral sodium fluoride* solutions with fluoride concentrations of *100 ppm or less* resulted primarily in the formation of *fluorapatite*, whereas higher fluoride concentrations resulted in the formation of *calcium fluoride*.[15] Because topical applications of sodium fluoride involve the use of 2.0% solutions (slightly over 9,000 ppm), it follows that the use of these solutions essentially involves the formation of *calcium fluoride*.[14]

The second fluoride compound developed[17,18] for topical use in the dental office during the 1950s was *stannous fluoride* (SnF_2). Compared with that of sodium fluoride, the reaction of SnF_2 with enamel is unique in that *both* the cation (stannous) and the anion (fluoride) react chemically with enamel components. This reaction is commonly depicted as follows:

Note from the equation that the formation of *stannous fluorophosphate* prevents, at least temporarily, the *phosphate* loss typical of sodium fluoride applications. Incidentally, the exact nature of the tin-containing reaction products varies depending on reaction conditions, including pH, concentration, and length of exposure (or reaction time).[19,20]

A third topical fluoride system for professional use was developed during the 1960s and is widely known as APF, *acidulated phosphate fluoride*. This system was developed by Brudevold and coworkers[21,22] in an effort to achieve greater amounts of fluorhydroxyapatite and lesser amounts of calcium fluoride formation. These investigators reviewed the various chemical reactions of fluoride with enamel (hydroxyapatite) and concluded that (1) if the pH of the fluoride system was made acidic to enhance the rate of reaction of fluoride with hydroxyapatite and (2) if phosphoric acid was used as the acidulant to increase the concentration of phosphate present at the reaction site, it should be possible to obtain greater amounts of fluoride deposited in surface enamel as *fluorhydroxyapatite* with minimal formation of *calcium fluoride* and minimal loss of *enamel phosphate*. On the basis of this chemical reasoning, APF systems were developed and shown to be effective for caries prevention.

Subsequent independent studies of the reactions of APF with enamel indicated, however, that the original chemical objectives were only partially achieved. The

major reaction product of APF with enamel is also *calcium fluoride*,[12,23,24] although a greater amount of fluorhydroxyapatite is formed than with the previous topical fluoride systems. The chemical reaction of APF with enamel may be written as follows:

It is obvious from the preceding discussion that the primary chemical reaction product with *all* three types of topical fluoride systems (i.e., NaF, SnF_2, and APF) is the formation of *calcium fluoride* on the enamel surface.

The initial deposition of calcium fluoride on the treated tooth surfaces is by *no means* permanent; a relatively rapid loss of fluoride occurs within the first *24 hours*,[25] with some continued loss occurring during the *next 15 days*.[26-29] The rate of loss varies between patients and is influenced by the nature of the fluoride treatment.[30,31] Nevertheless, it is known that *each* individual professionally-applied fluoride treatment results in an *increase* in the permanently-bound fluoride content of the outermost layers of the enamel with a subsequent decrease in the susceptibility of the enamel for caries initiation and progression.

The role of the calcium fluoride deposits on the enamel surface following professional fluoride applications in providing the observed cariostatic benefits has been the subject of numerous investigations. It is known that the *most desirable form of fluoride* in enamel for caries prevention is *fluorhydroxyapatite* and that the most efficient means of forming this reaction product occurs with *prolonged exposure* of the enamel to *low* concentrations of fluoride. It is also known that *calcium fluoride* may serve as a fluoride *source for enamel remineralization*,[32,33] and that calcium fluoride dissolves much more slowly in the oral environment than in an aqueous solution due to the presence of a phosphate or protein-rich coating of the globular deposits of calcium fluoride on the enamel surface.[34] As a result of this continued research, there is a growing body of convincing evidence suggesting that the deposits of calcium fluoride serve as an important *fluoride reservoir* and that these phosphate-coated globules are dissolved in the presence of plaque acids providing an available *source of both fluoride and phosphate* to facilitate the remineralization of decalcified areas.[35]

Regardless of the mechanism of action of professionally applied topical fluoride treatments, the results of clinical trials clearly indicate that the *benefits are related to the number of treatments*. Table 9-2 summarizes a clinical study[36] in which schoolchildren were given a dental prophylaxis and a topical application of 8 percent SnF_2 at 6-month intervals throughout a 3-year period. Dental-caries examinations were performed initially and each year thereafter. It is apparent from these data that the caries-preventive benefits *increased* in relation to the number of treatments. Similar observations have been noted with the other two fluoride systems used for professional applications. The original sodium fluoride topical application procedure developed by Knutson[37] specified a series of four treatment during a 2-week period. Mellberg[38] and coworkers[39] have also indicated the need for repeated topical applications of APF to obtain maximal benefits. Thus, it follows that maximal patient benefits can *only* be obtained with repeated topical applications regardless of the

nature of the fluoride system used.

It was noted earlier that the reaction of SnF_2 with enamel resulted in the formation of tin-containing compounds. Although much less is known regarding the precise nature and ultimate fate of these compounds, it appears that they contribute significantly to the cariostatic activity of SnF_2. The tin reaction products formed on sound enamel surfaces appear to be leached from the enamel in a manner similar to that for calcium fluoride.[40] The greatest accumulation of stannous complexes *occurs in circumscribed areas of enamel defects*; typically such areas are *hypo*mineralized and are frequently the result of decalcification associated with the initiation of the caries process. Extremely high concentrations of tin, about 20,000 ppm, have been reported in these locations.[41] Clinically, these areas, which have been described as frank carious areas, *become pigmented* (presumably because of the presence of the tin complexes) and appear to be more calcified following the application of SnF_2. This pigmentation has thus been suggested as being indicative of the arrest of carious lesions and is typically retained for 6 to 12 months or longer, implying that these stannous reaction products are of considerably *greater* significance than those formed on sound enamel.

At reduced concentrations of *0.10 to 0.15% fluoride*, all of the foregoing fluoride compounds have also been approved for use in dentifrices and gels intended for personal use, and sodium fluoride at a concentration of *0.05%* has also been approved for use in *mouthrinses sold over-the-counter*. In general, it is recognized that the mechanism of action of these fluoride compounds is *similar* at all the concentrations utilized for both professional and home-use products.

One additional fluoride compound, *sodium monofluorophosphate,* has been approved for use in dentifrices; this compound has the empirical formula Na_2PO_3F and is commonly known as MFP. Though evaluated in one study as an agent for topical fluoride application in the dental office, its use in this manner has received little consideration. Although the mechanism of action of MFP is thought to involve a chemical reaction with surface enamel, the precise nature of this reaction is *poorly understood*. Some investigators have suggested that the fluorophosphate moiety, PO_3F^5, may undergo an exchange reaction with phosphate ions in the apatite structure but the presence of PO_3F^5 in enamel has never been demonstrated, and such a reaction mechanism appears unlikely. Others have suggested that the PO_3F^5 complex is enzymatically dissociated by phosphatases present in saliva and dental plaque into PO_3^2 and F^2, with the ionic fluoride reacting with hydroxyapatite in a manner similar to that described earlier. The fact that the treatment of enamel with MFP results in less fluoride deposition and less protection against enamel decalcification than is observed with simple inorganic fluoride compounds such as sodium fluoride, while yet imparting nearly comparable cariostatic activity, is indicative of a more complex mechanism of action.

For the most part, the foregoing discussion of the chemical reactions of concentrated fluoride solutions with enamel suggests that the reactions occur on the *outer enamel surface* and serve to make that surface more resistant to demineralization. It is apparent that this process is particularly predominant in newly erupted teeth that are undergoing continued enamel maturation (calcification) for the first 2 years following eruption into the oral cavity. In such instances, some of the applied fluoride readily penetrates the relatively permeable enamel surface to *depths of 20 to 30 millimeters*

and readily reacts with the calcifying apatite to form a fluorhydroxyapatite. Furthermore, the dissolution of the calcium fluoride deposited on the enamel surface provides additional fluoride ions, which become incorporated in maturing enamel.

It has become increasingly apparent, however, during the last decade that very little fluoride deposition *lasting more than 24 hours* occurs when fluoride is applied to sound, *fully maturated enamel*. This situation apparently occurs regardless of the nature of the fluoride compound, the concentration of fluoride, or the manner of application. Thus, there appears to be *no* preventive benefits from the application of fluoride to maturated, sound enamel.

As noted in Chapter 3, the caries process begins with a demineralization of the *apatite adjacent to the crystal sheaths*. This permits the diffusion of weak acids into the subsurface enamel, and because the subsurface enamel has a lower fluoride content and is less resistant to acid demineralization, it *is preferentially* dissolved, forming an *incipient, subsurface lesion*. As this process continues, it becomes clinically apparent as a so-called "*white spot*" that, in reality, is a rather extensive subsurface lesion covered by a relatively intact enamel surface. Thus, enamel surfaces that clinically appear to be sound or free of demineralization frequently have areas that have been slightly decalcified with *minute subsurface lesions* that are not yet detectable clinically. This situation is particularly likely to exist in patients with clinical evidence of *caries activity on other teeth*.

It now appears that the predominant mechanism of action of fluoride involves its ability to *facilitate the remineralization* of these demineralized areas. Topically applied fluoride clearly *diffuses* into these demineralized areas and reacts with calcium and phosphate to form fluorhydroxyapatite in the remineralization process. It is also noteworthy that such remineralized enamel is *more resistant* to subsequent demineralization than was the original enamel. This process has been shown to occur with all forms and concentrations of fluoride, including concentrations as low as 1 ppm such as is found in optimally fluoridated drinking water. Studies conducted in our laboratories, however, have clearly shown that the amount of fluoride deposition in subsurface lesions following a topical fluoride application is much greater than that occurring following the use of lesser concentrations of fluoride provided by fluoride rinses or dentifrices. As a result, topical fluoride applications appear to be an *effective means of inducing the remineralization of incipient lesions.*

Question 2

Which of the following statements, if any, are correct?

A. On demineralization, more phosphate is lost from the hydroxyapatite crystal in the presence of sodium fluoride than when stannous fluoride is present.

B. Calcium fluoride deposits on the tooth surface serve as a fluoride reservoir.

C. As the number of treatments with topical fluoride increases, so does the caries-preventive benefit.

D. Stannous fluoride is deposited in greatest concentration where the enamel is least

perfectly mineralized.

E. The use of MFP results in less cariostatic action than other neutral fluorides, even though a higher concentration is usually found on the tooth surface.

Effects of Fluoride on Plaque and Bacterial Metabolism

Thus far, we have assumed that the cariostatic effects of fluoride are mediated through a chemical reaction between this ion and the outermost portion of the enamel surface. The preponderance of data supports this view. A growing body of information suggests, however, that the caries-preventive action of fluoride may also include an *inhibitory effect on the oral flora involved in the initiation of caries*. The ability of fluoride to *inhibit glycolysis* by interfering with the enzyme *enolase* has long been known; concentrations of fluoride as low as 50 ppm have been shown to interfere with bacterial metabolism. Moreover, fluoride may *accumulate in dental plaque in concentrations above 100 ppm*. Although the fluoride normally present in plaque is largely bound (and thus unavailable for antibacterial action), it *dissociates* to ionic fluoride when the pH of plaque decreases (i.e., when acids are formed). Thus, when the carious process starts and acids are formed, plaque fluoride in ionic form may serve to *interfere* with further acid production by plaque microorganisms. In addition, it may react with the underlying layer of dissolving enamel, *promoting its remineralization as fluorhydroxyapatite*. The end result of this process is a "physiologic" *restoration* of the initial lesion (by remineralization of enamel) and the formation of a *more resistant* enamel surface. The ability of fluoride to promote the reprecipitation of calcium phosphate solutions in apatitic forms has been repeatedly demonstrated.

In addition to these possible effects of fluoride, several investigators have reported that the presence of tin, especially as provided by stannous fluoride, is associated with significant *antibacterial activity*, which has been reported to *decrease both* the amount of *dental plaque* and *gingivitis* in both animals[42] and adult humans.[43] Existing evidence suggests that these antibacterial effects of fluoride and tin may also contribute to the observed cariostatic activity of topically applied fluorides.

Topical Fluoride Applications

The use of concentrated fluoride solutions applied topically to the dentition for the prevention of dental caries has been studied extensively during the past 50 years, although few studies have been conducted since the 1970s. This procedure results in a significant *increase* in the resistance of the exposed tooth surfaces to the development of dental caries and, as a result, has become a standard procedure in most dental offices.

At present, three different fluoride systems have been adequately evaluated and approved for use in this manner in the United States. These three systems are *2% sodium fluoride, 8% stannous fluoride, and acidulated phosphate fluoride systems containing 1.23% fluoride.*

Available Forms

When topical fluoride applications became available to the profession, the fluoride compounds (sodium fluoride and stannous fluoride) were obtained in powder or crystalline form, and aqueous solutions were prepared immediately prior to use. Subsequently it was realized that sodium fluoride solutions were stable if stored in plastic containers, and this compound became available in liquid and gel, as well as powder, form. With continued research of different types of agents and recognition by the dental profession of their inherent disadvantages with regard to patient acceptance and stability, as well as the need to use professional time more efficiently, the trend has been toward the use of ready-to-use, stable, flavored preparations in gel form.

Sodium Fluoride (NaF)

This material is available in powder, gel, and liquid form. The compound is recommended for use in a 2% concentration, which may be *prepared* by dissolving 0.2 g of powder in 10 mL of distilled water. The prepared solution or gel has a basic pH and is stable if stored in plastic containers. Ready-to-use 2% solutions and gels of NaF are commercially available; because of the relative *absence of taste considerations* with this compound, these solutions generally contain little flavoring or sweetening agents.

Stannous Fluoride (SnF_2)

This compound is available in powder form either in bulk containers or preweighed capsules. The recommended and approved concentration is *8%*, which is obtained by dissolving 0.8 g of the powder in 10 mL of distilled water. Stannous fluoride solutions are quite acidic, with a pH of about 2.4 to 2.8. Aqueous solutions of SnF_2 are not stable because of the formation of stannous hydroxide and, subsequently, stannic oxide, which is visible as a white precipitate. As a result, solutions of this compound must be prepared immediately prior to use. As will be noted later, SnF_2 solutions have a bitter, metallic taste. To eliminate the need to prepare this solution from the powder and to improve patient acceptance, a stable, flavored solution can be prepared with glycerine and sorbitol to retard hydrolysis of the SnF_2 and with any of a variety of compatible flavoring agents. Ready-to-use solutions or gels with the proper SnF_2 concentration, however, are *not* commercially available.

Acidulated Phosphate Fluoride (APF)

This treatment system is available as either a *solution or gel*, both of which are stable and ready to use. Both forms contain 1.23% fluoride, generally obtained by the use of 2.0% sodium fluoride and 0.34% hydrofluoric acid. Phosphate is usually provided as orthophosphoric acid in a concentration of 0.98%. The pH of true APF systems should be about *3.5*. Gel preparations feature a greater variation in composition, particularly with regard to the source and concentration of phosphate. In addition, the gel preparations generally contain thickening (binders), flavoring, and coloring agents.

Another form of acidulated phosphate fluoride for topical applications, namely *thixotropic* gels, is also available. The term thixotropic denotes a solution that sets in a gel-like state but is not a true gel. On the application of pressure, thixotropic gels behave like solutions; it has been suggested that these preparations are more easily

forced into the interproximal spaces than conventional gels. The active fluoride system in thixotropic gels is *identical* to conventional APF solutions. Although the initial thixotropic gels exhibited somewhat poorer biologic activity in in vitro studies, subsequent formulations were at least equivalent to conventional APF systems. Even though few clinical efficacy studies have been reported,[44] the collective data were considered adequate evidence of activity; these preparations have been *approved by the American Dental Association.*

Within the past few years, a foam form of APF has become available. Laboratory studies indicate that the amount of fluoride uptake in enamel following applications using the foam is *comparable* to that observed with conventional APF gels and solutions. The primary advantage of foam preparations is that appreciably less material is used for a treatment and therefore lesser amounts are likely to be inadvertently swallowed by young children during the professional application.

Application Procedure

In essence, two procedures are available for administering topical fluoride treatments. One procedure, in brief, involves the isolation of teeth and continuously painting the solution onto the tooth surfaces. The second, and *currently* more popular, procedure involves the use of fluoride gels applied with a *disposable tray*.

Until recently it was assumed that it was necessary to administer a thorough dental prophylaxis prior to the topical application of fluoride. This hypothesis was supported by the results of an early study that suggested that topically applied sodium fluoride was more effective if a prophylaxis preceded the treatment.[45] The results of four clinical trials,[46-49] have indicated that a prophylaxis immediately prior to the topical application of fluoride is *not necessary*. In these studies, the children were given topical applications of APF in the conventional manner except that three different procedures were used to clean the teeth immediately prior to each treatment; these procedures were either a dental prophylaxis, toothbrushing and flossing, or no cleaning procedure. The results indicated that the cariostatic activity of the APF treatment was *not* influenced by the different preapplication procedures. Thus, the administration of a dental prophylaxis prior to the topical application of fluoride must be considered *optional*; it should be performed if there is a *general need* for a prophylaxis, but it need not be performed as a prerequisite for topical fluoride applications.

, 9-2, 9-3, 9-4, 9-5, and 9-6 illustrate the major steps recommended for Figures 9-1 applying topical fluoride solutions. The essential armamentarium for the application of concentrated fluoride solutions consists of cut cotton rolls, suitable cotton-roll holders, cotton applicators, and treatment solution. If a prophylaxis is performed, the patient is allowed to rinse thoroughly, and then the cotton rolls and holders are positioned so as to isolate the area to be treated. It is a common practice when using fluoride solutions to isolate *both* right or left quadrants at one time so as to be able to treat one-half of the mouth simultaneously. The isolated teeth are then dried with compressed air, and the fluoride solution is applied using cotton applicators. Care should be taken to be certain that all tooth surfaces are treated. The application is performed by merely swabbing or "painting" the various tooth surfaces with a cotton applicator thoroughly moistened with the fluoride solution. The swabbing procedure

is repeated *continuously and methodically* with repeated "loading" of the cotton applicator so as to keep the tooth surfaces moist throughout the treatment period. At the conclusion of this period, the cotton rolls and holders are removed, the patient is allowed to expectorate, and the process is repeated for the remaining quadrants.

It should be stressed that various precautions should be routinely taken to *minimize* the amount of fluoride that is inadvertently swallowed by the patient during the application procedure. A number of reports[50-56] have shown that 10 to 30 mg of fluoride may be *inadvertently* swallowed during the application procedure, and it has been suggested that the ingestion of these quantities of fluoride by young children may contribute to the development of dental fluorosis in those teeth that are unerupted and in the developmental stage. Precautions that should be undertaken include (1) using only the *required* amount of the fluoride solution or gel to perform the treatment adequately; (2) positioning the patient in an *upright* position; (3) using efficient *saliva aspiration* or suctioning apparatus; and (4) requiring the patient *to expectorate thoroughly on completion* of the fluoride application. The use of these procedures has been shown to reduce the amount of inadvertently swallowed fluoride to *less than 2 mg*, which may be expected to be of little consequence.[57]

After the topical application is completed, the patient is advised not to rinse, drink, or eat for *30* minutes. The necessity of the latter procedure has not been substantiated; the fact that it has been followed in most of the prior clinical studies serves as the primary basis for this recommendation. This recommendation is also supported, however, by a 1986 study[58] that measured the amount of fluoride deposition in incipient lesions (subsurface enamel demineralization) in patients who either were, or were not, permitted to rinse, eat, or drink during this 30-minute posttreatment period. It was found that *significantly greater fluoride deposition occurred when the patients were not permitted to rinse, eat, or drink following the fluoride treatment.*

Whichever fluoride system is used for topical fluoride applications, the teeth should be exposed to the fluoride for *4 minutes* for maximal cariostatic benefits. This treatment time has consistently been recommended for both sodium fluoride and APF. Some confusion has arisen, however, with regard to stannous fluoride, because shorter application periods of 15 to 30 seconds with stannous fluoride have been reported to result in significant cariostatic benefits. Nevertheless, the collective results of these and subsequent clinical investigations indicate that maximum caries protection is achieved only with the use of the *longer* exposure period. Thus, although reduced exposure periods of 30 to 60 seconds might be appropriate as a fluoride maintenance or preventive measure in patients with very little caries activity, the use of the longer, 4-minute application should be *required* for patients with existing or potential caries activity.

Application Procedure—Fluoride Gels

A slightly different technique is commonly suggested for providing treatments with fluoride gels. Although these preparations may be applied by using the same basic procedure as described for solutions, the use of *plastic trays* has been suggested as a more convenient procedure. As with the use of topical fluoride solutions, the treatment may be preceded by a prophylaxis if indicated by existing oral conditions. With the so-called tray application technique, the armamentarium consists simply of a

suitable tray and the fluoride gel.

Many different types of trays are available; selection of a tray adequate for the individual patient is an important part of the technique. Most manufacturers of trays offer sizes to fit patients of different ages. An adequate tray should *cover all the patient's dentition*; it should also have enough depth to reach beyond the neck of the teeth and contact the alveolar mucosa to prevent saliva from diluting the fluoride gel. Some of the trays used in the past did not meet these requirements. Some were made of vinyl and frequently either did not reach the mucosa or impinged on the tissue, thus forcing the dentist to cut the flanges of the tray. Currently, disposable *soft styrofoam trays* are available and seem to be adequate. These trays can be bent to insert in the mouth and are soft enough to produce no discomfort when they reach the soft tissues. With these trays, as well as with some of the previous types of trays, it is possible to *treat both arches simultaneously*.

If a prophylaxis is given, the patient is permitted to rinse, and the teeth of the arch to be treated are dried with compressed air. A ribbon of gel is placed in the trough portion of the tray and the tray seated over the entire arch. The method used must ensure that the gel reaches *all* of the teeth and flows interproximally. If, for instance, a soft pliable tray is used, the tray is pressed or molded against the tooth surfaces, and the patient may also be instructed to bite gently against the tray. Some of the early trays contained a sponge-like material that "squeezed" the gel against the teeth when the patient was asked to bite lightly or simulate a chewing motion after the trays were inserted. It is recommended that the trays be kept in place for a *4-minute treatment period* for optimal fluoride uptake, although some systems recommend a 1-minute application time. As noted previously, the patient is advised not to eat, drink, or rinse for *30* minutes following the treatment.[58] Figure 9-7 illustrates the tray technique of fluoride gel application.

Figure 9-1 It is advisible to seat the patient in an upright position to help minimize the flow of topical solution down the child's throat.

Figure 9-2 If desired, the topical application may be preceded by a thorough prophylaxis. The smooth tooth surfaces are cleaned with a prophylactic paste applied with a prophy cup, A, following the gross removal of heavy exogenous deposits (calculus) with hand instruments. A prophy brush is similarly used on the occlusal surfaces, B, while unwaxed dental floss is used to draw the paste interproximally to clean the proximal surfaces, C.

Figure 9-3 A 6-inch and 4-inch roll are placed in a Garmer holder in such a manner that, A, the lingual roll extends across the midline to isolate an area beyond the central incisors, and B, the long buccal roll is bent so as to isolate both the upper and lower vestibules.

Figure 9-4 The cotton-roll holder is placed in the mouth, thereby isolating both an upper and lower quadrant from the retromolar to a point beyond the central incisors.

Figure 9-5 The isolated teeth are, A, dried with an air syringe in a systematic manner, B, so as to avoid missing any tooth surface.

Figure 9-6 Using the same application pattern as in Figure 9-5B, fluoride solution is applied with a cotton applicator, with continual reapplications to maintain all tooth surfaces moist with the solution for a 4-minute period.

Figure 9-7 Appropriate sized soft styrofoam trays are used to avoid pinching the soft tissues. A ribbon of gel is dispensed into the trough of the tray. Enough gel should be used to cover all tooth surfaces, but care should be used to avoid an excess which will flow into the mouth. (Experience will teach the operator how much gel to use.) The patient is shown the loaded mandibular tray, A, which is ready for insertion, B. The maxillary tray is inserted after the mandibular is in place. The patient is then asked to bite together so as to be more comfortable and, at the same time, to force the gel against the teeth. The use of thixotropic gels facilitates the wetting of all tooth surfaces. The trays should be maintained in place for 4 minutes.

Question 3

Which of the following statements, if any, are correct?

A. Assuming that less than 1 ppm of fluoride is in the saliva (true), the dental plaque may have 100 times this level.

B. Stannous fluoride is quite stable when stored in aqueous phase.

C. A thixotropic gel looks like a gel, acts like a gel, and is a gel.

D. After a topical application of fluoride, a patient should not eat or drink for a half-hour.

E. A liquid topical solution should be maintained on the teeth for the same time as a gel tray treatment: 4 minutes.

Application Frequency

As previously mentioned, although a single, topical application is accepted as not being able to impart maximal caries protection, considerable confusion has arisen regarding the preferred frequency for administering topical fluoride treatments. Much of this confusion is caused by the absence of controlled, clinical evaluations of this variable, particularly with the most commonly used agent, acidulated phosphate fluoride.

The original Knutson technique[37] for the topical application of sodium fluoride consisted of a series of four applications provided at approximately 1-week intervals with only the first application preceded by a prophylaxis. It was further suggested that this series of applications be administered at ages 3, 7, 10, and 13 years, with these ages selected, or varied, in accordance with the eruption pattern of the teeth.[59] The objective of the timing was to provide protective benefits to "the permanent teeth during the period of changing dentition." Because this treatment sequence did not coincide with the common patient-recall pattern in the dental office, Galagan and Knutson[60] explored the possible use of longer intervals of 3 or 6 months between the individual applications constituting each treatment series. The results of their work indicated that although significant benefits were obtained with single applications provided at 3- or 6-month intervals, maximal benefits were obtained only with a series of treatments. Nevertheless, the administration of single applications of sodium

fluoride at 3- to 6-month intervals became a common practice, because these intervals were more convenient to the dentist and his or her normal recall system.

When stannous fluoride and acidulated phosphate fluoride were subsequently developed and evaluated, apparently little if any attempt was made to determine the optimal treatment frequency. Instead, the treatments were administered as single applications provided at 6- or 12-month intervals, which were convenient to the normal office schedules. Because these treatment intervals resulted in significant cariostatic benefits, the procedure that was ultimately approved and recommended involved this application frequency.

In view of this background, it seems that the frequency of topical applications should be *dictated by the conditions and needs presented by each patient and not by the convenience of the dental office.* This conclusion is supported by the data cited earlier that a series of applications is required to impart maximal caries resistance to the tooth surface.

Thus, it is recommended that new patients, regardless of age, with active caries be given an initial series of four topical fluoride applications within a period of 2 to 4 weeks. If desired, the initial application may be preceded by a thorough prophylaxis, the remaining three applications constituting the initial treatment series should be *preceded by toothbrushing* to remove plaque and oral debris. It should be obvious that this series of treatments may be very conveniently combined with the plaque control, dietary counseling, and initial restorative programs that the dentist has devised for these patients. Following this initial series of treatments, the patient should be given single, topical applications at intervals of 3, 6, or 12 months, *depending on his or her caries status.* Patients with *little* evidence of existing or anticipated caries should be given single applications every 12 months as a preventive measure.

Special effort should be made by the dentist to schedule topical fluoride applications so as to provide the treatment to newly erupted teeth within 12 months after eruption, and preferably *as close to eruption as possible*. As noted earlier, an approximate 2-year enamel maturation period occurs immediately following tooth eruption. As illustrated in Table 9-3, the preventive benefits of fluoride are invariably much greater on *newly erupted teeth* than on previously erupted teeth. This finding is apparent regardless of the fluoride system used and is presumably due to the greater reactivity, permeability, and ease of formation of fluorhydroxyapatite in enamel still undergoing calcification (or maturation).

Although it is important to expose newly erupted teeth to topical fluoride, it may be more appropriate to utilize a fluoride varnish for newly erupted primary teeth. Children at this young age may swallow too much of a topical fluoride gel and may have precooperative behavior making it difficult to use typical topical fluoride gels or foams.

Efficacy of Topical Fluoride Therapy

Well over 100 human clinical studies demonstrate that topical fluoride therapy contributes significantly to the partial control of dental caries. Unfortunately, the practitioner is frequently concerned, and sometimes confused, about which procedure

or agent should be employed in a given situation to provide a maximal degree of dental caries protection for the patient. Such concern and confusion is understandable when it is realized that dental caries investigators themselves frequently do not agree on these matters.

The results of the numerous clinical investigations with various topical fluoride agents and treatment procedures have been the subject of several reviews.[64-76] Therefore no attempt is made to repeat these reviews here.

As noted earlier, three different types of fluoride systems (i.e., NaF, SnF_2, and APF) have been evaluated and approved as safe and effective for topical fluoride applications *by both the American Dental Association (ADA)*[77] *and the Food and Drug Administration (FDA).*[78] To determine which of these systems may be the most effective, it would be desirable to compare the results of independent clinical studies in which all three systems have been tested when used in the recommended manner. Unfortunately, such data are not available, and alternative procedures must be sought.

Different approaches have been taken to estimate the magnitude of the cariostatic benefits that may be expected from topical applications of the different approved fluoride systems. One approach is simply to list all of the pertinent clinical trials and then determine the arithmetic mean of the reported caries reduction. This approach has been utilized by several investigators,[69-71] and the results observed for children residing in a nonfluoridated community are summarized in Table 9-4. Another approach is to utilize an empirically based procedure with existing clinical data to predict the efficacy of different systems;[79] these data are also shown in Table 9-4. Whatever the approach, *study designs varied in a number of ways,* such as the number and frequency of topical applications and the study duration. These variations serve to confound estimates of cariostatic efficacy. Nevertheless, it is apparent from Table 9-4 that all three types of topical fluoride systems result in *appreciable cariostatic benefits* of comparable magnitude with percentage reductions ranging from 27 to 36%. Furthermore, the data suggest that fluoride applied in gel form may be slightly less effective than solutions.

Considerably less information is available to document the efficacy of topical fluoride applications in adults. A total of 14 clinical trials were conducted in adults during the period 1944 through 1974, but the studies utilized a wide variety of experimental conditions, including the type of topical fluoride system, frequency of applications, and duration of the test period.[80-93] Although most of the methods resulted in a significant cariostatic benefit, the magnitude of this effect varied considerably, as might be expected. Furthermore, none of these studies used the application frequency suggested earlier for children.

It is generally recognized by dental scientists, however, that the dental caries process is fundamentally the *same in both children and adults,* although the rate of progression in young and middle-aged adults is frequently much slower because of a variety of factors, including more efficient oral hygiene and fewer between-meal snacks. Conversely, in older adults the rate of progression may increase because of medications that *reduce salivary flow.* It is commonly assumed, therefore, that topical fluoride applications are effective for coronal caries prevention *regardless of the age of the patient.* Root caries will be discussed later. Once again the frequency of

application should be dictated by the needs of the patient; in the presence of frank or incipient caries activity, an initial series of applications should be given followed by *maintenance applications* at 3, 6, or 12 months, depending on patient needs (i.e., evidence and extent of caries activity). Similarly, the choice of the fluoride system (NaF, APF, SnF_2) may be at the discretion of the dentist because there appears to be little, if any, difference in their efficacy.

On occasion it has been suggested that present topical fluoride treatment systems involve the use of excessive concentrations of fluoride. For example, some have suggested that the use of 0.4% rather than 8% stannous fluoride is adequate to obtain maximal benefits from topical applications of this compound. The basis for such suggestions invariably rests with the results of *in vitro* studies, quite commonly enamel-solubility studies, in which maximal effects are achieved with lesser concentrations of fluoride. Unfortunately, *in vitro* data do not necessarily predict clinical effects, and the results of a clinical investigation[94] clearly contradict these suggestions. As shown in Table 9-5, the use of lower concentrations of stannous fluoride resulted in smaller caries-preventive benefits in children. Thus, until considerably more clinical data to the contrary become available, there is no legitimate basis for using concentrations of fluoride for topical applications *other than those that have been adequately evaluated clinically* and approved by review groups.

The relative superiority of acidulated phosphate fluoride gel or solution systems is a frequent topic for research. Five clinical trials directly investigated this question, and the results are summarized in Table 9-6. Four of these studies[63,95-97] involved single annual applications; another one[44] involved semiannual treatments. These data suggest that the two forms are quite comparable, particularly when applied semiannually. In practice the gels are greatly preferred due to their ease of application and reduced chair time when trays are used.

Question 4

Which of the following statements, if any, are correct?

A. The semiannual application of fluoride to the teeth has proved to be the most effective time interval to reduce caries incidence.

B. There are no documented studies in which sodium fluoride, stannous fluoride, and acidulated phosphate fluoride have been tested in the same study.

C. Stannous fluoride is better than acidulated fluoride and sodium fluoride in nonfluoridated areas.

D. Both stannous fluoride and acidulated fluoride are effective on children, but neither is effective on adults.

E. A liquid topical of a 0.4% solution of stannous fluoride is as effective as an 8% solution.

Root Surface Caries

As noted elsewhere, the increased retention of the teeth during adulthood because of various caries-preventive measures and the increase in life expectancy in many countries has resulted in an increased prevalence of root surface caries in adults. According to the 1985 to 1986 United States Public Health Service (USPHS) survey of adults,[98] about one-half of U.S. adults are afflicted with root surface caries by age 50, with an average prevalence of about three lesions by age 70. Interestingly, a study conducted at the University of Iowa[99] has indicated that adults over age 65 can expect an incidence of about 0.9 newly decayed, missing, or filled (DMF) coronal surfaces per year as well as about 0.6 new DMF root surfaces per year. Thus, this form of caries has received increased attention of dental scientists during the past decade, with investigations covering both its *cause* and *measures for prevention*.

Quite clearly, fluoride is very effective for the prevention of root surface caries as evidenced by a limited number of clinical trials and numerous *in vitro* as well as *in situ* studies. For example, the results of several epidemiologic studies have demonstrated that the presence of *fluoridated drinking water* throughout the lifetime of an individual prevents the development of root surface caries.[100-103] The magnitude of this effect is consistently *greater than 50%*. Furthermore, it has been observed[104] that the use of a NaF dentifrice results in a significant decrease in root surface caries of *more than 65%*.

Much less information is available, however, to document the effect of topical fluoride applications on the prevention of root caries and particularly the relative efficacy of different fluoride systems. Nyvad and Fejerskov[105] reported the arrestment of root surface caries following the topical application of 2% NaF and the daily use of a fluoride dentifrice. Wallace and coworkers[106] reported a *70%* reduction in the incidence of root surface caries following semiannual applications of an APF gel during a 4-year study period. To obtain some perspective on the potential efficacy of different topical fluoride systems[107,108] we have utilized an established animal root caries model. The results of this investigation are summarized in Table 9-7. From these data it is apparent that all three approved topical fluoride systems decreased the formation of root caries by *63 to 76%* in this preclinical model. In the absence of the results of similar clinical data and with the recognition that the application of 8% SnF_2 imparts a brown pigmentation to exposed dentin, it seems appropriate to recommend the topical use of *2% NaF* for the prevention of root caries.

Recommendations—Topical Fluoride Treatments

On the basis of the foregoing discussion, it is apparent that although periodic topical applications of any of the three approved agents provide protection against dental caries, maximal patient benefits may be expected only through the use of selected procedures. These recommended procedures include the following:

- Accepting the relative inefficiency of single, topical applications of fluoride solutions, patients with existing evidence of caries activity, whatever their age, should be given an *initial series of topical fluoride treatments followed by quarterly, semiannual, or annual treatments* as required to maintain cariostasis. The initial series of treatments should consist of four applications administered during a 2- to 4-week period, with the first treatment preceded by a thorough prophylaxis if indicated.
- Whatever fluoride system is selected, the application period (i.e., the time the teeth

are kept in contact with the fluoride system) should be 4 minutes in all patients with existing caries activity. Shorter treatment periods may be permissible in the performance of treatments to *maintain* cariostasis.

Fluoride Varnishes

Fluoride-containing varnishes have more recently become available in the United States and are being recommended for providing topical fluoride treatments, particularly for very young children. Most varnishes contain *5.0% sodium fluoride* (2.26% fluoride) and a typical application requires only 0.3 to 0.5 mL of the varnish, which contains 3 to 6 mg of fluoride. The application procedure involves cleaning the tooth surfaces by toothbrushing, painting the varnish on the teeth, and drying. The *varnish is retained for 24 to 48 hours* during which time fluoride is released for reaction with the underlying enamel. It is recommended that the applications be repeated at 4- to 6-month intervals.

The efficacy of fluoride varnishes for caries prevention has been repeatedly demonstrated in Europe, where they have been in common use for many years, and the results of these studies have been summarized in recent reviews.[76,109,110] These studies have consistently demonstrated a significant reduction in the incidence of dental caries and also have indicated that the magnitude of the benefit is related to the frequency of application, particularly in children at high risk for caries. Promising research has been conducted in the United States, specifically aimed at using fluoride varnishes as a preventive agent for children *at high risk for early childhood caries*.[111]

Little information is available to compare the effectiveness of fluoride varnishes with professionally applied topical fluoride solutions or gels. The results of a clinical study[112] conducted in children in India comparing the efficacy of a fluoride varnish with topical applications of an APF gel indicated that while both treatments resulted in a significant reduction in caries, the *fluoride varnish was more effective.* Seppa and coworkers[113] recently reported the results of a clinical trial comparing semiannual applications of the sodium fluoride varnish with similar applications of an APF gel in 12- and 13-year-old children with high past caries experience, and observed *no* significant differences between the two treatment regimens. In the absence of additional clinical data it appears that these *two treatment procedures are at least equivalent.*

Initiation of Therapy

Practitioners frequently wonder when they should recommend and initiate a topical fluoride application program. All too frequently the tendency is to defer such treatments until the child is 8 to 10 years of age and a majority of the permanent dentition has already erupted.

As discussed earlier, it is well established that the enamel surface of a newly erupted tooth is not completely calcified and therefore that the period when the tooth is most susceptible to carious attack is the first few months after eruption. Furthermore, it has been shown that topical fluoride treatments are effective for both the deciduous and permanent dentitions. Thus, it follows that topical fluoride therapy should be initiated *when the child reaches about 2 years of age,* when most of the deciduous dentition

should have erupted. The treatment regimen should be maintained at least on a semiannual basis throughout the period of increased caries susceptibility, which *persists for about 2 years after eruption* of the permanent second molars (i.e., until the child is about 15 years of age).

It should be added that the susceptibility of the dentition to dental caries *does not end at age 15*. It is probable, however, that the gradual *decrease in caries susceptibility with increasing age* will permit a less frequent topical application program to maintain cariostasis in many patients, and annual fluoride treatments may suffice.

Problems and Disadvantages

Some clinical situations may alter the selection of the treatment agent. For example, the use of stannous fluoride may be contraindicated for aesthetic reasons in specific instances. The reaction of tin ions with enamel, particularly carious enamel, results in the formation of *tin phosphates*, some of which are brown in color. Thus, the use of this agent produces a temporary brownish pigmentation of carious tooth structure. This stain may exaggerate existing aesthetic problems when the patient has carious lesions in the anterior teeth that will not be restored. Stannous fluoride, however, has not been found to discolor composite restorative materials.

Another problem frequently raised, particularly by pedodontists, concerns the strong, unpleasant, metallic taste of stannous fluoride. Although experienced practitioners can handle this problem, there is no question that flavored, APF preparations are much *better accepted by children.* Experimental, flavored, stannous fluoride preparations that diminish, but by no means eliminate, the taste problem have been evaluated clinically but are not yet commercially available.[36] Until the taste problem of stannous fluoride is solved, most pedodontists agree that the *agent of choice* for children is APF.

Acidulated phosphate fluoride systems have the disadvantage of possibly etching ceramic or porcelain surfaces. As a result, porcelain veneer facings and similar restorations should be *protected with cocoa butter, vaseline, or isolation* prior to applying APF. Alternatively, sodium fluoride may be used instead of APF.

Without doubt, the tendency in many dental offices is to use a specific topical fluoride system and treatment regimen for every patient. It should be emphasized, however, that the specific needs of the patient should be ascertained initially and a specific treatment program developed to fulfill those needs. For example, the use of a series of four or more topical fluoride applications within a 4-week period followed by repeated single applications at 3- to 6-month intervals should be considered for a patient with a severe caries problem. Likewise, a reduced topical application time of 30 seconds as opposed to 4 minutes may be adequate to maintain a patient with little or no current caries activity. In other words, the practitioner should be familiar with the indications and contraindications for using various approaches and select the treatment system and conditions that best meet the needs of the patient.

Fluoride-Containing Prophylactic Pastes

Fluoride-containing prophylactic pastes have been available and widely used in dental

offices for more than 40 years to clean and polish accessible tooth surfaces and restorations. Because these pastes contain abrasives, which are harder than enamel, in order to clean and polish efficiently, inevitably a *small amount of the enamel surface will be removed by abrasion during the prophylaxis*. The actual amount of enamel removed during a prophylaxis is very small and has been shown[114,115] to involve the loss of surface enamel to a depth of about 0.1 to 1.0 microns during a 10-second polishing. Since it has been noted that the greatest concentrations of fluoride in enamel occur in the outermost surface layers, it follows that the loss of even this small amount of surface enamel during a prophylaxis results in the exposure of an enamel surface having a *lower concentration* of fluoride than was present prior to the prophylaxis.[116,117]

When fluoride-containing prophylactic pastes first became available in the 1950s it was thought that the use of the preparations to perform a routine dental prophylaxis would result in a significant reduction in the subsequent development of dental caries and a number of clinical trials were conducted to determine the magnitude of this benefit. The results of these investigations, considered collectively, indicated that the use of these pastes resulted in a very modest increase in the resistance of the tooth surfaces to the development of dental caries, but the magnitude of this effect was not statistically significant. As a result, fluoride-containing prophylactic pastes have *never been accepted* as therapeutic agents by review agencies such as the ADA or the FDA. However, they are *commonly recommended* for use during a prophylaxis in order to at least replace the fluoride lost from the enamel surface by abrasion during the procedure. Should the clinician use fluoride-containing phophylaxis pastes? In summary, the following recommendations are proposed:

- When a simple prophylaxis is administered, which will not be followed by a topical fluoride application, fluoride-containing prophylactic pastes should be used to replenish the fluoride lost during the procedure.
- When a topical fluoride application is given to a caries-susceptible patient and a prophylaxis is deemed to be necessary, it is advisable to administer the preceding prophylaxis with a fluoride-containing paste. Although no definitive proof of the additive benefits of both procedures exists as yet, an increased benefit has been shown in some studies. Even when doubt exists, it is preferable to give the patient the possible benefit of any increased protection.

Question 5

Which of the following statements, if any, are correct?

A. The first active programs for topical fluoride therapy should be initiated following the eruption of the first permanent molar.

B. After age 15, the need for topical applications of fluoride ceases.

C. A prophylaxis increases the effectiveness of the topical application of fluorides.

D. From 0.1 to 1.0 mm of enamel is removed during a prophylaxis.

E. The use of a fluoride-containing prophylactic paste results in a significant

reduction in caries formation.

Dentifrices

Through the years dentifrices have been defined as preparations intended for use with a toothbrush to clean the accessible tooth surfaces. They have been prepared in a variety of forms, including pastes, powders, and liquids. The history of dentifrices dates back several centuries. The earliest writings concerning measures to achieve oral cleanliness refer to the use of toothpicks, chewsticks, and sponges; suggested dentifrice ingredients were dried animal parts, herbs, honey, and minerals.

Materials actually detrimental to oral health were used for many years; these materials included excessively abrasive materials, lead ores, and sulfuric and acetic acids. With the appreciation for the need of safe and efficient dentifrices came the research and development that have led to the dentifrices available today and the development of a major industry. In the United States alone, dentifrice sales approached *$1.5 billion during 1*995, and major manufacturers have invested millions of dollars, particularly during the past three decades, to improve these products further and to expand their capacities to promote oral health. Without question the dental profession and the scientific community, as well as the general population, have profited immeasurably, both directly and indirectly, from the efforts of the dentifrice industry.

As a result, the functions of present-day dentifrices have been considerably expanded to include the following: (1) cleaning accessible tooth surfaces, (2) polishing accessible tooth surfaces, (3) decreasing the incidence of dental caries, (4) promoting gingival health, and (5) providing a sensation of oral cleanliness, including the control of mouth odors. These functions should be accomplished in a safe manner without undue abrasion to the oral hard tissue, particularly dentin, and without irritation to the oral soft tissues.

This chapter makes no attempt to review the components and functions of dentifrices. For information on these topics, as well as a review of the early attempts to develop therapeutic dentifrices using agents other than fluoride, the reader is referred to several excellent reviews[118-123] (see also Chapter 6).

Fluoride Dentifrices

At present fluoride is by far the *most effective dentifrice* additive for caries prevention. The initial studies with fluoride-containing dentifrices were only modestly encouraging. The results of two clinical trials indicated a significant beneficial effect with formulations containing fluorapatites and rock phosphates. However, studies using products containing sodium fluoride at concentrations of 0.01 to 0.15 percent failed to indicate any beneficial effect.[118] In retrospect, these and later failures were probably largely caused by the use of an *incompatible abrasive system* (i.e., calcium carbonate) in the formulations. In the 40-year period since the initial clinical trials with fluoride-containing dentifrices, the results of more than 140 controlled clinical studies have been published. For a review of many of these studies, the reader is referred to a recent report.[123]

In 1954, the first report was published concerning the use of a dentifrice containing

stannous fluoride (0.4%); this study indicated a significant beneficial effect attributable to this agent.[124] Since then the results of more than 60 clinical investigations with stannous fluoride-containing dentifrices have been reported; the vast majority of this work has been performed with formulations containing *calcium pyrophosphate* as the abrasive agent, although insoluble sodium metaphosphate and hydrated silica have also been used.

Without doubt, the most extensive documentation of the cariostatic benefits of fluoride dentifrices was generated with the original stannous fluoride-calcium pyrophosphate formulation (i.e., Crest) with supportive information using this fluoride compound with other abrasive systems. The results of these investigations not only indicated that the normal home use of the stannous fluoride dentifrice resulted in a significant decrease in the incidence of dental caries in children but that *similar benefits were derived by adults*. Furthermore, these effects were found to be additive to those provided by communal fluoridation and by topical fluoride treatments. As a result of these findings, the Council on Dental Therapeutics of the ADA first awarded complete acceptance to a fluoride dentifrice, specifically the stannous fluoride-calcium pyrophosphate formulation *(Crest)*, in 1964.

Primarily because of the identification of a formal mechanism by the ADA to recognize dentifrices on the basis of clinical documentation of a cariostatic activity, much effort was devoted by dentifrice manufacturers to the development and documentation of effective formulations during the past four decades. A number of effective products subsequently became available.

During the 1960s, a number of reports of clinical trials indicated that the use of *sodium monofluorophosphate* (MFP), Na_2PO_3F, in a dentifrice likewise contributed significantly to the control of dental caries. The first product, *Colgate MFP*, used insoluble sodium metaphosphate as the abrasive system, and this formulation was shown to reduce the incidence of caries in children by as much as 34%. On the basis of these studies, this dentifrice was *approved as safe and effective by the FDA in 1967 and accepted by the ADA in 1969*.

An interesting and unique characteristic of MFP is its compatibility with a *wide variety of dentifrice abrasive systems*. In contrast to other fluoride compounds, such as sodium fluoride and stannous fluoride, which are almost completely dissociated in aqueous solution to yield fluoride ions that readily react with available cations, the fluoride in MFP remains largely complexed as PO_3F^5 in solution. This fluoride complex is compatible with a wide variety of abrasive systems and therefore may be readily incorporated into a variety of different dentifrice formulations while continuing to provide cariostatic activity.

Additional studies with dentifrices containing sodium fluoride have indicated that this agent also effectively contributes to the control of dental caries in children. The first of the sodium fluoride dentifrices to be substantiated in this regard included sodium metaphosphate as the abrasive agent. On the basis of three favorable clinical trials, this product (Durenamel) was given *provisional acceptance* by the ADA Council on Dental Therapeutics; however, this product is *no longer available*. Another sodium fluoride dentifrice has been the subject of several clinical trials. This formulation (Gleem) contains calcium pyrophosphate as the abrasive agent and has been found to

exert a significant beneficial effect on the incidence of dental caries in children in both low-fluoride and optimal fluoride areas.

In 1981, the formulation of Crest was changed by replacing stannous fluoride with sodium fluoride and using hydrated silica in place of calcium pyrophosphate as the abrasive system. Interestingly, these changes and the resultant increase in the amount of available and biologically active fluoride in the product resulted in the revised formulation being significantly more effective than the formulation originally approved.[125,126] Since that time, many additional products containing sodium fluoride have been approved by the ADA. One reason for the increased use of sodium fluoride is that it is the preferred agent for use *in tartar- control formulations containing soluble pyrophosphates*. Furthermore, as noted later in this section, there is evidence that sodium fluoride formulated in a highly compatible abrasive system may be a superior anticaries agent. In terms of fluoride concentration, most dentifrices currently marketed in the United States contain *1,000 ppm fluoride, with two exceptions*. The Crest family of products contain a hydrated silica abrasive system which is less dense than other conventionally used abrasive systems. Because dentifrice is dispensed by the consumer on the basis of volume, not weight, the concentration of fluoride *was increased to 1,100 ppm* so that the dose delivered would be comparable to nonsilica-based products. On the other hand, *Extra Strength Aim, which contains 1,500 ppm* fluoride from sodium monofluorophosphate, has an elevated fluoride content in an attempt to enhance its anticaries efficacy. Several controlled clinical trials have shown that, in dentifrices employing silica-abrasive systems and sodium monofluorophosphate as the fluoride source, 1,500 ppm of fluoride is *statistically significantly more effective than 1,000 ppm*, with a margin of superiority of about 15%.[127-129]

The potential role of fluoride dentifrices in the etiology of dental fluorosis needs to be mentioned. Several reports have indicated an *increasing prevalence of fluorosis* in the United States.[130-132] A national survey of U.S. children conducted between 1986 and 1987 indicated that about 22% displayed some evidence of dental fluorosis; however, it is important to note that, in terms of severity, *a majority had either very mild or mild fluorosis*, and only about *1% were classified as moderate or severe*.[133,134] Certainly a number of sources of fluoride may be responsible, individually or collectively, for causing fluorosis. In evaluating the role fluoride toothpastes may play, however, the practitioner should consider several important factors. First, for fluorosis to occur, excessive levels of fluoride must be ingested *during the time of enamel formation*.[135] For practical purposes, the *anterior teeth* are of most concern aesthetically, and these are *only susceptible to becoming fluorotic during the first 3 years of life*[136] *and particularly during the period of 15 to 20 months of age*.[137] Of course, the risk of toothpaste ingestion is increased in younger children, and some studies have shown that very young children may ingest enough toothpaste to be at risk of dental fluorosis.[138] In fact, one study found that children who brush with a fluoride toothpaste *before 2 years of age have an elevenfold greater risk of developing fluorosis than children who begin brushing later*.[139] These considerations have prompted the ADA to recommend that *children under age 3 should be advised to use only a "pea-sized" quantity of a fluoride dentifrice for brushing and that this quantity be gradually increased with age so that not until age 6 is the child using a "full-strip" of dentifrice on the brush head*. In making recommendations, the practitioner must consider what other sources of fluoride the child may be ingesting, such as fluoride or

fluoride-vitamin supplements, fluoridated communal-water supplies, and infant formula prepared with fluoridated water.[139]

Not infrequently, the practitioner is asked if all fluoride dentifrices provide the same amount of caries-preventive benefits. In an attempt to answer this question, a review[123] examined the results of all published studies involving fluoride dentifrices. It was concluded on the basis of a considerable body of information that the use of *sodium fluoride with highly compatible abrasive systems, such as hydrated silica or acrylic particles, is the most effective dentifrice system for caries prevention at this time*.

Subsequent literature reviews compared all available clinical data regarding the relative efficacy of compatible sodium fluoride dentifrices and those containing sodium monofluorophosphate.[140-142] Using different statistical procedures, these reviews concluded that sodium fluoride was significantly more effective than sodium monofluorophosphate. Based on a meta-analysis, Johnson[143] concluded that the magnitude of this difference was 7%. It is interesting that many fluoride dentifrices marketed outside the United States contain mixtures of sodium fluoride and sodium monofluorophosphate with a total fluoride content of 1,500 ppm. Clinical efficacy data of these latter systems have also been reviewed recently[140,141] with the conclusion that they are numerically less effective than an equivalent concentration of bioavailable sodium fluoride.

It is significant that the extensive research with fluoride dentifrices has resulted in the regular use of these products by a major segment of our population. In terms of total dentifrice sales, nearly *95%* consists of the *accepted or approved formulations*. For an updated listing of fluoride-containing dentifrices receiving acceptance by the ADA, *the reader is referred to the ADA website at http://www.ada.org*. The widespread acceptance and use of these products by the general public has been considered one of the primary factors contributing to the apparent decrease in the prevalence of dental caries observed in the United States.[144,145]

In general, it should be apparent from this brief review that the use of approved fluoride dentifrices results in a significant decrease in the incidence of dental caries. In view of this, the use of such preparations should be routinely recommended.

Question 6

Which of the following statements, if any, are correct?

A. The use of fluoride dentifrices have yielded equivocal results as caries control agents when used as the sole method of fluoride application.

B. Fluoride dentifrices appear to reduce caries in a range of approximately 20 to 45%.

C. The safety of a new dentifrice containing new fluoride compounds must be approved by the FDA before it is accepted by the ADA.

D. In 1981, Crest changed the fluoride in its formula from sodium fluoride to stannous fluoride.

E. Despite the fact that several dentifrices contain fluoride, approximately 40% of the population prefers nonfluoride-containing brands.

Multiple Fluoride Therapy

From the prior discussions of various measures to apply fluoride to erupted teeth, it is apparent that *no* single fluoride treatment provides *total protection* against dental caries. Recognition of this fact led early investigators to evaluate the use of combinations of fluoride measures.

Multiple fluoride therapy is a term that has been used to describe these fluoride combination programs. As originally developed, this program included the application of fluoride in the dental office in the form of both a fluoride-containing prophylactic paste and a topically applied fluoride solution and the home use of an approved fluoride dentifrice. In addition, some form of systemic fluoride ingestion, preferably communal-water fluoridation, was included.

The only published reports of clinical investigations that attempted to assess the total effect of this type of multiple fluoride therapy on dental caries involved the use of stannous fluoride topical systems.[146-151] In each of these studies, the topical fluoride treatments were administered semiannually; the results are summarized in Table 9-8. The results of these investigations indicate that the combination of *topical fluoride applications and home use of a fluoride dentifrice resulted in about 59% fewer carious lesions*.

The fact that the magnitude of this benefit is somewhat less than that of the components evaluated individually indicates that the caries-protection effects of the individual components (i.e., prophylactic paste, topical solution, and dentifrice) are only partially additive. Nevertheless, it is important to note that the combination of stannous fluoride treatments not only reduced the incidence of caries by more than 50% in both children and young adults but did so in both the *presence and absence of communal fluoridation.* If one accepts a 50% caries reduction attributable to water fluoridation and another 50% reduction of the remaining caries from the use of multiple fluoride treatments, it is apparent that the use of multiple fluoride therapy, including communal fluoridation, results in an *overall reduction in caries of about 75%*.

During the past few years, clinical investigators have explored combinations of fluoride treatments using agents other than stannous fluoride with variable success. For example, Beiswanger and coworkers[152] reported that additive benefits were observed with topical applications of acidulated phosphate fluoride and the home use of a stannous fluoride dentifrice. Neither Downer and associates[153] nor Mainwaring and Naylor,[154] however, were able to demonstrate additive benefits from the combined use of a sodium monofluorophosphate dentifrice and topical application of acidulated phosphate fluoride.

The available data relating to multiple fluoride therapy thus suggest additive benefits from the use of either stannous fluoride or acidulated phosphate fluoride in the dental office and the home use of dentifrices containing fluoride. This does not necessarily

mean that other combinations of fluoride treatments may not provide additive benefits but merely that they have not yet been evaluated; hopefully the results of future investigations will clarify this matter. In the meantime, the dental practitioner is strongly advised to use *combinations of fluoride treatments to provide maximal caries protection for patients*.

Fluoride Rinses

In 1960, reports began to appear indicating that the regular use of neutral sodium fluoride solutions decreased the incidence of caries. In an attempt to identify topical fluoride measures especially appropriate for use *in dental public health programs*, this approach was studied extensively during the subsequent 15 years. Whereas these studies employed a wide variety of experimental conditions, a number of investigations involved either the daily use of solutions containing 200 to 225 ppm or the weekly use of solutions containing about 900 ppm fluoride. The majority of these studies were conducted *in schools* with supervised use of the rinse throughout the school year.

The results of these investigations have been summarized on several occasions and will not be repeated here.[77,121,155-158] In general, both types of fluoride rinses resulted in significant caries reduction of about *30 to 35%*. On the basis of these findings, the simplicity of administration, and the lack of need for professional dental supervision, weekly fluoride-rinse programs in schools are becoming increasingly popular and are being aggressively promoted by dental public-health agencies. Fluoride rinses were approved as safe and effective by the FDA in *1974*[78] and by the Council of Dental Therapeutics of the ADA in *1975*.[157] A "Guide to the Use of Fluoride" was published in the September 1986 issue of the *Journal of the American Dental Association*. The composition and recommended use of approved products is shown in Table 9-9.

Nearly all of the early investigations using fluoride rinses involved children residing in areas in which the drinking water was deficient in fluoride. As a result, the approvals given to fluoride rinses were related to their use in nonfluoridated communities. Reports[61,122,159,160] indicated, however, significant benefits from fluoride rinses used in the presence of an optimal concentration of fluoride in the drinking water. Three additional reports have appeared relative to the use of fluoride rinses in children residing in fluoridated communities. The results of all three studies indicate that *cariostatic benefits provided by fluoride rinses are additive to those derived from communal fluoridation*.[161-163] In view of these collective observations there appears to be *no reason* to restrict the use of fluoride rinses to nonfluoridated communities.

The approval of fluoride rinses by the FDA and the ADA's Council on Dental Therapeutics for use in public health programs opened the door for the home use of these products as a component of multiple fluoride preventive programs. Although the approved preparations were intended to be available strictly by prescription, a *0.05% neutral sodium fluoride* rinse (Fluorigard) was subsequently introduced for over-the-counter (OTC) sale. Ultimately, approval was given to fluoride rinses distributed OTC for home use, although some restrictions were required. These restrictions included the distribution of quantities containing *no more than 300 mg fluoride in a single container, a cautionary label to avoid swallowing, and an indication that the preparations should not be used by children younger than 6 years of a*ge. At present

there are several fluoride rinses distributed in this manner; these products contain about 225 ppm fluoride and are intended *for daily usage*.

The question of additivity of the effects of fluoride rinses to those obtained using fluoride with other vehicles has received contradictory answers. Ashley and associates[164] found a modest additivity of benefits from the supervised daily rinsing in school with an acidulated phosphate fluoride rinse coupled with supervised brushing in school plus normal home use of a sodium monofluorophosphate dentifrice. A similar observation was reported by Triol and coworkers.[165] On the other hand, Blinkhorn and coworkers[166] failed to observe any indication of additive caries protection between the similar supervised daily use of a neutral 0.05% sodium fluoride and the home use of this same dentifrice. Likewise, Ringelberg and associates[167] failed to find additivity between a daily sodium fluoride rinse and home use of a stannous fluoride dentifrice. Similarly, Horowitz and coworkers,[168] in a study involving the supervised weekly use of a sodium fluoride rinse and daily fluoride tablets plus the home use of approved fluoride dentifrices, observed a caries reduction comparable in magnitude to that reported earlier by these investigators with fluoride tablets or rinses used individually.

Additive effects can also be inferred from the numerous school fluoride rinse studies in which caries reductions from 30 to 35% were observed. Because the majority of these children in both the control and experimental groups used fluoride-containing dentifrices, it follows that the benefits observed in those studies were obtained above those provided by the fluoride dentifrices. The same conclusion can be reached from the data reported by Birkeland and coworkers[155] in Norway, a country where *over 90% of the children use fluoride dentifrices.* After 10 years of a mouthrinsing program, these authors found a caries reduction of *over 50% and reduction in the need for restoration of more than 70%*.

It can thus be concluded that fluoride rinses have a place as a component of a preventive program *along with*, but not as substitutes for, other modalities of fluoride use. Their main use is for patients with a high risk of contracting caries. Although existing evidence may lead some to doubt whether additional benefits for the patients accrue from the use of rinses, it is preferable in these instances to give the patients the benefit of the doubt. Examples of patients for whom fluoride rinses should be recommended include:

- Patients who, because of the use of medication, surgery, radiotherapy, and so on, have reduced salivation and increased caries formation.
- Patients with orthodontic appliances or removable prostheses, which act as traps for plaque accumulation.
- Patients unable to achieve acceptable oral hygiene.
- Patients with extensive oral rehabilitation and multiple restorative margins, which represent sites of high caries risk.
- Patients needing fluoride in their home care but cannot tolerate a custom-fitted tray.
- Patients with gingival recession and susceptibility to root caries.
- Patients with rampant caries, at least as long as the high caries activity persists.

As a general rule, *daily rinses* should be recommended rather than a weekly regimen; not only does the daily procedure appear to be slightly more effective, but, as a

practical consideration, it is easier for patients to remember and comply with a daily procedure. In all these instances, it is important to remember that the rinses should *not be used in place of any of the other modalities* of fluoride use but as part of a comprehensive, preventive program that should also comprise plaque control, frequent fluoride topical applications, the home use of a fluoride dentifrice, diet control, and testing to determine if and when the oral environment is no longer conducive to caries. For children living in nonfluoridated areas, the prescription of fluoride supplements may also be considered.

Fluoride Gels for Home Use

During the past 15 years, a number of fluoride gels have become available as additional measures that may be used to help achieve caries control. These procedures contain 0.4% stannous fluoride *(1,000 ppm fluoride*) or 1.0% sodium fluoride *(5,000 ppm*) and are formulated in a nonaqueous gel base that *does not contain an abrasive system*. Their recommended manner of usage involves toothbrushing with gel (similar to using a dentifrice), allowing the gel to remain in the oral cavity for 1 minute, and then expectorating thoroughly.

Even though no controlled clinical trials have been conducted of these products used in this manner, a number of them have been approved by the ADA's Council on Dental Therapeutics as an additional caries-preventive measure for use in patients with rampant caries. The basis for the approval of these products has been the numerous prior clinical caries studies using dentifrices containing the same amount of stannous fluoride coupled with analytic data demonstrating the stability of these preparations.

From a practical point of view, the recommended use of fluoride gels is generally similar to that cited earlier for fluoride rinses. In other words, they may be considered as an *alternative* to the use of fluoride rinses and an *adjunct* to the use of professional, topical fluoride applications and fluoride dentifrices as a collective means of achieving caries control in patients who are especially prone to caries formation. Like fluoride rinses, the use of these gels is generally restricted to the period required to achieve caries control. Compared with fluoride rinses, however, fluoride gels appear to have an *advantage in terms of patient compliance*. Because these preparations are only distributed to patients by their dentists, it is commonly thought that patients are more likely to use them in compliance with the recommendations of their dentist.

It should be stressed that fluoride gels should *not be used in place of fluoride dentifrices*. Because the gels contain *no abrasive system* to control the deposition of pellicle, their use in place of a dentifrice results in the accumulation of stained pellicle in the majority of patients within a few weeks. Nevertheless, the proper use of these preparations in combination with professional topical fluoride applications and the home use of fluoride dentifrices may be expected to help achieve caries control in caries-active patients.

Question 7

Which of the following statements, if any, are correct?

A. If a 20% reduction in caries occurs from water fluoridation and then another 25% from topical fluoride therapy, the total reduction is 45%.

B. Fluoride rinses are of little value in fluoridated areas.

C. A fluoride-rinse container should not contain more than 300 mg of fluoride.

D. A school rinse program can be expected to produce caries reduction on the order of 30%.

E. It is more practical to have people use a daily rinse than a weekly rinse.

Fluoride-Releasing Dental Restorative Materials

Fluoride-releasing dental restorative materials may provide an additional benefit in preventive dentistry. Although not currently available in the United States, a fluoride-releasing amalgam has demonstrated *recurrent caries inhibition* at enamel and dentin restoration margins.[169] Likewise, both chemical-cured and light-cured *glass ionomer cements* have demonstrated caries inhibition at enamel and dentin restoration margins.[170-173] Fluoride-releasing *resin composites* have also consistently demonstrated recurrent caries inhibition at enamel margins, yet there are *conflicting results whether caries inhibition occurs at dentin margins*.[170,171,173,174] Preliminary studies indicate that glass ionomer cement and fluoride-releasing resin composite have synergistic effects with fluoride rinses and fluoridated dentifrices, in the *remineralization of incipient enamel caries*.[175-178] The materials may act as a fluoride delivery system. Upon exposure to additional external fluoride, the material surface *undergoes an increase in fluoride*. This fluoride is subsequently released and has demonstrated demineralization inhibition and even remineralization at adjacent tooth structure. Further clinical research to evaluate these fluoride-releasing restorative materials may provide more information for clinical recommendations.

Question 8

Which of the following statements is incorrect?

A. Fluoride-releasing dental restorative materials can effectively inhibit adjacent enamel demineralization.

B. Fluoride-releasing dental restorative materials have been shown to effectively inhibit enamel demineralization on adjacent interproximal tooth surfaces.

C. Glass ionomer cements and fluoride-releasing resin composites have similar effectiveness of adjacent demineralization inhibition.

D. Glass ionomer cements and resin composites can uptake fluoride at the surface, following exposure to topical fluorides, and subsequently release the fluoride.

Toxicology of Fluoride

The handling of fluorides is carefully regulated in industry by *occupational safety*

health legislation and in the marketplace by *the FDA*. Commercial dental fluoride products and professional practices can be toxic and even lethal when used inappropriately. The lethal dose for an adult is somewhere between 2.5 and 10 g, with the *average lethal dose being 4 to 5 g*. The use of the "average lethal dose" is a very imprecise designation that makes it difficult to predict the outcome of an accidental swallowing of an excess of fluoride. To correct this problem, a body-weight based, *probable toxic dose* (PTD) standard has been recommended as a more practical approach to making treatment decisions. With it, the urgency for first aid and more definitive emergency treatment can be determined rapidly. The PTD approach, first reported by Bayless and Tinanoff, bases the level and urgency of treatment on the *number of multiples of 5 mg/kg* of fluoride ingested (Table 9-10).

If the amount ingested is *less than 5 mg/kg*, the office use of available calcium, aluminum, or magnesium products as first aid antidotes should suffice. If the amount is *over 5 mg/kg*, first aid measures should be *expeditiously* applied, *followed by hospital observation* for possible further care. Finally, if the amount of fluoride ingested approaches or *exceeds 15 mg/kg*, the immediate first aid treatment should be followed by a *most urgent action to move the patient swiftly into a hospital emergency room where cardiac monitoring, electrolyte evaluation, and shock support is available. Ingestion of 15 mg/kg fluoride can be lethal*.

Fluoride Toxicity

Fluoride acts in four general ways: (1) when a concentrated fluoride salt contacts moist skin or mucous membrane, hydrofluoric acid forms, causing a *chemical burn*; (2) it is a general *protoplasmic poison* that acts to inhibit enzyme systems; (3) it *binds calcium* needed for nerve action; and (4) *hyperkalemia occurs, contributing to cardiotoxicity*.

When dry fluoride powder contacts the mucous membrane or the moist skin, a reddened lesion occurs, and later the area becomes swollen and pale; still later, ulceration and necrosis may occur. In past years, skin burns of this type were common for many water engineers who emptied drums of fluoride agents into the hoppers feeding water supplies. Federal and state occupational safety acts have greatly reduced this danger.

Following excessive ingestion of fluoride, nausea and vomiting can occur. The vomiting is usually caused by the formation of hydrofluoric acid in the acid environment of the stomach, *causing damage to the lining cells of the stomach wall*. Local or general signs of *muscle tetany* ensue caused by the drop in blood calcium. This can be accompanied by abdominal *cramping* and pain. Finally, as the hypocalcemia and hyperkalemia intensify, the severity of the condition *becomes ominous with the onset of the three C's that can portend death—coma, convulsions, and cardiac arrhythmias*. Generally, death from ingestion of excessive fluoride occurs within 4 hours; *if the individual survives for 4 hours, the prognosis is guarded to good*.

Emergency Treatment

Four actions are salient in treating fluoride poisoning: (1) *immediate treatment*, (2)

induced vomiting, (3) protection of the stomach by binding fluoride with *orally administered calcium or aluminum preparations,* and (4) maintenance of blood calcium levels *with intravenous calcium*. Urgent and decisive treatment is mandatory once the PTD of 15 mg/kg has been approached or exceeded. The speed of initiating proper treatment can be critical to a person's chance for survival.[179] The blood level reaches its maximum from 0.5 to 1 hour after the fluoride is ingested. *By that time it can be too late*.

If an excessive amount of sodium fluoride is ingested, *first aid treatment can be initiated*. Milk, or better yet, milk and eggs should be given, for two reasons: (1) As demulcents, they help protect the mucous membrane of the upper-GI tract from chemical burns; and (2) they provide the calcium that acts as a binder for the fluoride. Lime water (calcium hydroxide) or Maalox (an aluminum preparation), can be drunk to accomplish the same purpose. Plenty of fluid, *preferably milk*[a] should be ingested to help dilute the fluoride compound in the stomach. *Vomiting is beneficial* and often occurs spontaneously; it also can be induced by *digital stimulus* to the base of the tongue or with syrup of ipecac, if available. When vomiting does occur, the majority of the ingested fluoride is often expelled. Preferably, the patient should be taken *directly to the emergency room of a hospital.* Otherwise the closest emergency medical service unit or physician capable of dealing with fluoride toxicity is the alternative. Once in a well-equipped medical facility, several options are possible, such as *gastric lavage, blood dialysis, or oral intravenous calcium gluconate to maintain the blood calcium levels. Every effort should be made to rid the body rapidly of the fluoride or to negate its toxicity before a refractory hyperkalemia and cardiac fibrillation become a greater problem than the fluoride intoxication*.[180]

U.S. population, and alternative methods for the provision of systemic fluoride leave much to be desired. Thus, additional measures are obviously needed for providing greater protection against caries to as many segments of the population as possible.

The term topical fluoride therapy refers to the use of systems containing relatively large concentrations of fluoride that are applied locally, or topically, to erupted tooth surfaces to prevent the formation of dental caries. This term encompasses the use of fluoride rinses, dentifrices, pastes, gels, and solutions that are applied in various manners. Among dental practitioners, however, this term is generally considered to refer to professional topical fluoride treatments performed in the dental office.

Chronic Fluoride Exposure

At high levels of industrial fluoride exposure, as experienced by cryolite and bauxite workers prior to the era of occupational safety regulations, the combined intake of fluoride through inhalation, ingestion, and water consumption often resulted in a daily dose of over 20 mg. This exceedingly high level of continual intake for 10 to 20 years resulted in a *severe skeletal fluorosis* characterized by *osteosclerosis, calcification of the tendons, and the appearance of multiple exostoses*. This same crippling bone fluorosis can also occur from long-term consumption of naturally fluoridated waters found *in some parts of the world, which contain 14 ppm or more of fluoride*. Other factors that increase the severity of bone fluorosis are high temperatures with a concomitant increase in drinking episodes, an elevated intake of fluoride in food, nutritional diseases, and low-calcium diets. *No* cases of skeletal fluorosis have been

reported in the United States where water fluoridation concentrations were under 3.9 ppm.[181]

Despite all precautions, there is a potential for signs and symptoms of fluoride toxicity in dental office and home use of topical fluoride. The most probable cause is in *children in the 15- to 30-month age bracket* having an excess of dentifrice placed on the toothbrush and then swallowing the fluoride-laden saliva. In most cases, this results in a very mild, often unnoticeable change in the enamel of erupting teeth around age 6. A more serious toxicity can arise in the dental office from the mishandling and ingestion of fluoride salts used for professional purposes. To be prepared for such an unlikely emergency, the professional staff should be prepared for instituting possible emergency procedures.

Home Security of Fluoride Products

The *lack of home storage security* of OTC and prescription fluoride products poses hazards to consumers. As presently packaged, the fluoride content of OTC fluoride products *can* exceed the PTD for children.[182] That the danger at home is real is attested by two *deaths of children after swallowing fluoride tablets*: one in Austria, and the other in Australia.[183] In one year (1986-1987), 13 cases of fluoride poisoning were reported to the North Carolina Poison Center. It was noted by the poison center that *no health-care providers who contacted the center were familiar with the treatment of the GI symptoms induced by fluoride poisoning*.[184] Clearly, parents need to be educated about the hazards of fluoride-containing dental products. Dentifrices, mouthrinses, and fluoride supplements need to be securely stored. Equally, health professionals need to be educated about the emergency treatment protocol following excessive intake of fluoride.

In a larger study, the American Association of Poison Control Centers reported that the number of fluoride-related calls had increased from 3,856 cases in 1984 to 7,794 in 1989. Of these, the number seeking clinical treatment was 366 in 1984 and 668 in 1989. In each of these years, young children were involved in 90% of the calls.[185]

Question 9

Which of the following statements, if any, are correct?

A. The ingestion of 360 mg of fluoride by a 90-kg, 21-year-old adult is not as dangerous as the ingestion of 280 mg of fluoride by a 40-kg, 15-year-old adolescent.

B. Hypocalcemia and hyperkalemia are signs of an impending cardiotoxicity.

C. If a patient has ingested an excessive amount of fluoride, the occurrence of nausea and vomiting is a favorable sign.

D. The frequent topical applications of fluoride to the teeth at age 6 is believed to be the cause of fluorosis.

E. The ingestion of 15 mg/K of fluoride requires that the patient be under appropriate emergency medical care before 30 minutes has elapsed.

Summary

A number of different aspects of topical fluoride therapy have been reviewed in the foregoing material. Without doubt, the use of topical fluoride therapy contributes significantly to the control of dental caries; however, one cannot expect to control dental caries completely through the use of fluorides alone. Furthermore, because no single fluoride treatment procedure provides the maximal degree of caries protection possible with fluoride, the use of multiple fluoride therapy is advocated. In particular, the dentist should identify the needs of each patient and institute a multiple fluoride treatment program designed specifically to fulfill those needs.

Answers and Explanations

1. C—correct.

A—incorrect. Enamel maturation is very rapid the first month, slows down over the next year or so, and then remains relatively stable.

B—incorrect. Fluoride content decreases very rapidly in the first 10 mm and then more slowly, until the dentinoenamel junction is reached.

D—incorrect. As the pH falls, the fluoride becomes more effective in protecting the enamel; at a neutral pH no protection is needed.

E—incorrect. The main reaction product is calcium fluoride.

2. A, B, C, and D—correct.

E—incorrect. There is a lesser concentration on the tooth with MFP and about the same cariostatic action as for other inorganic fluorides.

3. A, D, and E—correct.

B—incorrect. Stannous fluoride in water goes to a hydroxide and then a white oxide.

C—incorrect. Thixotropic gel looks like a gel, acts like a gel, but is not a gel.

4. B—correct.

A—incorrect. A series of applications within a short period appears best.

C—incorrect. There appears to be little, if any, difference in their efficacy.

D—incorrect. The first part is correct; the second part is incorrect; both are effective, but the amount of difference between the SnF_2 and APF is debatable.

E—incorrect. In the critical field studies, the 8 % solution wins easily.

5. D—correct.

A—incorrect. Topical fluoride therapy should be started as soon as possible after the eruption of the first deciduous tooth.

B—incorrect. Fluoride is a lifelong adjunct for dental health.

C—incorrect. Recent studies have shown that it is not necessary to give a prophylaxis prior to a fluoride application.

E—incorrect. The use of these products did not result in significant reductions in caries.

6. A, B, and C—correct.

D—incorrect. Vice versa. Crest started with SnF_2 but now has NaF.

E—incorrect. People use the fluoride dentifrices—up to 90%.

7. A, C, D, and E—correct.

B—incorrect. The effects of fluoride are additive; the more often it is applied, the better.

8. A, B, and D—correct.

C—incorrect. Glass ionomer cements have greater adjacent demineralization inhibition.

9. B, C, and E—correct.

A—incorrect. First the age of the individual is not pertinent; what is critical is the mg/K. In this case, the first individual has had 3 mg/K (360/90), whereas the second individual has ingested 7 mg/K, 280/40—a potentially lethal dose.

D—incorrect. Fluoride ingestion at age 6 or later will not cause fluorosis since pre-eruptive enamel formation is essentially completed.

Self-evaluation Questions

1. There is an (inverse) (direct) relationship between the amount of fluoride (F) in the surface of the enamel and the number of caries. It requires about _____ (time) for the enamel surface to mature following eruption. The greatest amount of F in the enamel is located in the outer _____ (distance) of the enamel.

2. The reaction of elevated concentrations of fluoride with hydroxyapatite (HA) is accompanied by the formation of _____ (on the surface) (in the apatite crystal) and a loss of _____ (one of the key elements of HA). This element is not lost when SnF_2 is one of the reactants, in which case, the compound _____ (name) is

formed. Along with this compound, _____ (another F compound) is formed on the surface.

3. The calcium fluoride (CaF_2) formed on the surface of the tooth with neutral sodium fluoride, APF, or SnF_2, is lost relatively rapidly for _____ (time) and almost completely lost in _____ (time). During this period, the calcium fluoride (is) (is not) protective. Along with the formation—then loss—of CaF_2, there is a slow change in the apatite crystal from _____ (apatite) to _____ (name of crystalline form), which is more permanent. Because studies indicate that the CaF_2 is leached from the tooth, the long-term benefit must be from the _____ (crystalline form). Thus, if the build-up of the crystalline form is slow, (multiple) (single) applications of fluoride probably provide the best long-term prevention.

4. Fluoride accumulates to a greater extent in demineralized areas (true); two fluoride compounds with a low pH that de-mineralize enamel (and thus increase F uptake) are _____ and _____. Two times when the tooth is not optimally mineralized are just after _____ (event) of the tooth and just after bacterial _____ (event) of enamel; in either event, fluoride aids in the mineralization or remineralization process.

5. The three different solutions of fluoride used in office applied topical applications are NaF, _____ %; APF, _____ %; and SnF_2, _____ %. The APF is made acidic by adding two acids, _____ and _____ to a _____ _____ %.

6. The three "C's" indicating impending death from fluoride intoxication are _____, _____, and _____.

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Objectives

At the end of this chapter, it will be possible to

1. Explain how sealants can provide a primary preventive means of reducing the need for operative treatment as 77% of the children 12 to 17 years old in the United States have dental caries in their permanent teeth.[1]

2. Discuss the history of sealant development through the 20th century.

3. List the criteria for selecting teeth for sealant placement and the four essentials in attaining maximum retention of sealants.

4. Describe the several steps preliminary to, during, and after the placement of a sealant—including surface cleanliness, dry fields, details of the application procedure, and remedial measures following the excess application of sealant.

5. Explain the rationale for adding fluorides to sealants.

6. Compare the advantages and disadvantages of light-cured and self-cured sealants.

7. Discuss the advantages of protecting the occlusal surfaces of teeth with sealants.

8. Cite five reasons given for the underuse of sealants by practitioners and analyze the validity of the reasons.

Introduction

Fluorides are highly effective in reducing the number of carious lesions occurring on the *smooth surfaces* of enamel and cementum. Unfortunately, fluorides are *not* equally effective in protecting the occlusal pits and fissures, where the majority of carious lesions occur.[2] Considering the fact that the occlusal surfaces constitute only 12% of the total number of tooth surfaces, it means that the *pits and fissures are approximately eight times as vulnerable as the smooth surfaces.* The placement of sealants is a highly effective means of preventing these.[3]

Historically several agents have been tried to protect deep pits and fissures on occlusal surfaces.

- In 1895, Wilson reported the placement of *dental cement* in pits and fissures to prevent caries.[2] In 1929, Bodecker[4] suggested that deep fissures could be broadened with a large round bur to make the occlusal areas more self-cleansing, a procedure that is called *enameloplasty*.[5] Two major disadvantages, however, accompany enameloplasty. First, it requires a dentist, which immediately limits its use. Second, in modifying a deep fissure by this method, it is often necessary to remove more sound tooth structure than would be required to insert a small restoration.
- In 1923 and again in 1936, Hyatt[6] advocated the early insertion of small restorations in deep pits and fissures before carious lesions had the opportunity to develop. He termed this procedure *prophylactic odontotomy*. Again, this operation is more of a

treatment procedure than a preventive approach, because it requires a dentist for the cutting of tooth structure.

- Several methods have been unsuccessfully used in an attempt either to seal or to make the fissures more resistant to caries. These attempts have included the use of topically applied zinc chloride and potassium ferrocyanide[7] and the use of ammoniacal silver nitrate;[8] they have also included the use of copper amalgam packed into the fissures.[9]
- Fluorides that protect the smooth surfaces of the teeth are less effective in protecting the occlusal surfaces.[10] Following the use of fluorides, there is a large reduction of incidence in smooth-surface caries but a smaller reduction in occlusal pit-and-fissure caries. This results in an *increased proportion* in the ratio of occlusal to interproximal lesions, even though the total number may be less.
- A final course of action to deal with pit-and-fissure caries is one that is often used: *do nothing; wait and watch.* This option avoids the need to cut good tooth structure until a definite carious lesion is identified. It also results in many teeth being lost when individuals do not return for periodic exams. This approach, although frequently used is a violation of the ethical principle of beneficence and patient autonomy.

In the late 1960s and early 1970s, another option became available—the use of pit-and-fissure sealants.[11] With this option, a liquid resin is flowed over the occlusal surface of the tooth where it penetrates the deep fissures to fill areas that cannot be cleaned with the toothbrush (Figure 10-1).[12] The hardened sealant presents a barrier between the tooth and the hostile oral environment. Concurrently, there is a significant reduction of Streptococcus mutans on the treated tooth surface.[13] Pits and fissures serve as reservoirs for mutans streptococci, sealing the niche thereby reduces the oral count.

Figure 10-1 One of the reasons that 50% of the carious lesions occur on the occlusal surface. Note that the toothbrush bristle has a greater diameter than the width of the fissure. (Courtesy of Dr. J. McCune, Johnson & Johnson.)

Criteria for Selecting Teeth for Sealant Placement

Following are the criteria for selecting teeth for sealing. Because no harm can occur from sealing, when in doubt, seal *and monitor*.

- A deep occlusal fissure, fossa, or incisal lingual pit is present.

A sealant is contraindicated if:

- Patient behavior does not permit use of adequate dry-field techniques throughout the procedure.
- An open carious lesion exists.
- Caries exist on other surfaces of the same tooth in which restoring will disrupt an intact sealant.
- A large occlusal restoration is already present.

A sealant is *probably indicated if:*

- The fossa selected for sealant placement is well isolated from another fossa with a

restoration.
- The area selected is confined to a fully erupted fossa, even though the distal fossa is impossible to seal due to inadequate eruption.
- An intact occlusal surface is present where the *contralateral tooth* surface is carious or restored; this is because teeth on opposite sides of the mouth are usually equally prone to caries.
- An *incipient* lesion exists in the pit-and-fissure.
- Sealant material can be flowed over a conservative class I composite or amalgam to improve the marginal integrity, and into the remaining pits and fissures to achieve a *de facto* extension for prevention.

Other Considerations in Tooth Selection

All teeth meeting the previous criteria should be sealed and resealed as needed. Where the cost-benefit is critical and priorities must be established, such as occurs in many public health programs, ages 3 and 4 years are the most important times for sealing the eligible deciduous teeth; ages 6 to 7 years for the first permanent molars;[14] and ages 11 to 13 years for the second permanent molars and premolars.[15] Currently, 77% of the children 12-to-17-years-old in the United States have dental caries in their permanent teeth.[1] Many school days would be saved, and better dental health would be achieved in School Dental Health Clinic programs by combining sealant placement and regular fluoride exposure.[16]

The disease susceptibility of the tooth should be considered when selecting teeth for sealants, not the age of the individual. Sealants appear to be equally retained on occlusal surfaces in primary, as well as permanent teeth.[3] Sealants should be placed on the teeth of adults if there is evidence of existing or impending caries susceptibility, as would occur following excessive intake of sugar or as a result of a drug- or radiation-induced xerostomia. They should also be used in areas where fluoride levels in community water is optimized, as well as in non-fluoridated areas.[17]

The following are two good illustrations of this philosophy. After a 3-year study, Ripa and colleagues[18] concluded that the time the teeth had been in the mouth (some for 7 to 10 years) had no effect on the vulnerability of occlusal surfaces to caries attack. Also, the incidence of occlusal caries in young Navy[19] and Air Force[20] recruits (who are usually in their late teens or early 20s) is relatively high.

Background of Sealants

Buonocore first described the fundamental principles of placing sealants in the late 1960s.[10,21] He describes a method to bond poly-methylmethacrylate (PMMA) to human enamel conditioned with phosphoric acid. Practical use of this concept however, was not realized until the development of bisphenol A-glycidyl methacrylate (Bis-GMA), urethane dimethacrylates (UDMA) and trithylene glycol dimethacrylates (TEGDMA) resins that possess better physical properties than PMMA. The first successful use of resin sealants was reported by Buonocore in the 1960s.[22]

Bisphenol A-Glycidyl Methylacrylate Sealants

Bisphenol A-glycidyl methylacrylate (Bis-GMA) is now the sealant of choice. It is a mixture of Bis-GMA and methyl methacrylate.[23] Products currently accepted by the American Dental Association (ADA) include:[24]

- Baritone L3, Type II Confi-Dental Products Co.
- Alpha-Dent Chemical Cure Pit and Fissure Sealant Dental Technologies, Inc.
- Alpha-Dent Light Cure Pit and Fissure Sealant Dental Technologies, Inc.
- Prisma-Shield Compules Tips VLC Tinted Pit & Fissure Sealant Dentsply L.D. Caulk Division
- Prisma-Shield VLC Filled Pit & Fissure Sealant Dentsply L.D. Caulk Division
- Helioseal F, Type II Ivoclar-Vivadent, Inc.
- Helioseal, Type II Ivoclar-Vivadent, Inc.
- Seal-Rite Low Viscosity, Type II Pulpdent Corp.
- Seal-Rite, Type II Pulpdent Corp.

The ADA National Standard sets aside specific criteria of pit-and-fissure sealants stating; Specification No. 39 established the following requirements:

- That the working time for type I sealants is not less than 45 seconds;
- That the setting time is within 30 seconds of the manufacturer's instruction and does not exceed three minutes;
- That the curing time for type II sealants is not more the 60 seconds;
- That the depth of cure for type II sealant is not less than 0.75 millimeter;
- That the uncured film thickness is not more than 0.1 millimeter;
- That sealants meet the bicompatibility requirements of American Nation a Standard/American Dental Association Document No. 41 for Recommended Standard Practices for Biological Evaluation of Dental Materials.[25]

Sealant products accepted by the American Dental Association carried the statement: "[Product name] has been shown to be acceptable as an agent for sealing off an anatomically deficient region of the tooth to supplement the regular professional care in a program of preventive dentistry."[26]

Nuva-Seal was the first successful commercial sealant to be placed on the market, in 1972. Since then more effective second- and third-generation sealants have become available see Table 10-1. The first sealant clinical trials used cyanoacrylate-based materials. Dimethacrylate-based products replaced these. The primary difference between sealants is their method of polymerization. First-generation sealants were initiated by ultraviolet light, second-generation sealants are autopolymerized, and third-generation sealants use visible light.

Some sealants contain *fillers*, which makes it desirable to classify the commercial products into *filled* and *unfilled* sealants. The *filled* sealants contain microscopic glass beads, quartz particles, and other fillers used in composite resins. The fillers are coated with products such as *silane*, to facilitate their combination with the Bis-GMA resin. The fillers make the sealant more *resistant to abrasion and wear*. Because they are more resistant to abrasion the occlusion should be checked and the sealant height may need to be adjusted after placement. In contrast, unfilled sealants wear quicker but usually do not need occlusal adjustment.

Fluoride-Releasing Sealants

The addition of fluoride to sealants was considered about 20 years ago,[27] and it was probably attempted based on the fact that the incidence and severity of secondary caries *was* reduced around fluoride-releasing materials such as the silicate cements used for anterior restorations.[28,29] Because fluoride uptake increases the enamel's resistance to caries,[30] the use of a fluoridated resin-based sealant may provide an additional anticariogenic effect if the fluoride released from its matrix is incorporated into the adjacent enamel.

Fluoride-releasing sealants have shown antibacterial properties[31-33] as well as a greater artificial caries resistance compared to a nonfluoridated sealant.[34-36] A recent *in vitro* study showed that pit-and-fissure sealants containing fluoride provided a caries-inhibiting effect with a significant reduction in lesion depth in the surface enamel adjacent and a reduction in the frequency of wall lesion.[37] Moreover, the fluoridated sealant laboratory bond strength to enamel,[38] and clinical performance,[39,40] is similar to that of nonfluoridated sealants.[41,42] In a recent study, it was shown that teeth sealed with Teethmate F fluoridated sealant revealed high amounts of enamel fluoride uptake in vitro and in vivo to a depth ranging from 10 to 20 um from the surface.[43] The residual fluoride was also observed within the sealing material. This agrees with another study showing the high amount of fluoride released from Teethmate F-1.[44]

The addition of fluoride to the sealants will greatly increase their value in the preventive and restorative use as mentioned above. Fluoride is added to sealants by two methods. The first is by adding a soluble fluoride to the unpolymerized resin. The fluoride can be expected to leach out over a period of time into the adjacent enamel. Eventually the fluoride content of the sealant should be exhausted, but the content of the enamel greatly increased.

The second method of incorporating fluoride is by the addition of an organic fluoride compound that is chemically bound to the resin to form an ion exchange resin. As such, when fluoride is low in the saliva, fluoride would be released. Vice versa, when the fluoride in the environment is high, it should bind to the resin to form—at least theoretically—a continuous reservoir for fluoride release and recharge.[45] See Table 10-2 on page 292 for a list of current available sealant materials.

Polymerization of the Sealants

The liquid resin is called the *monomer*. When the catalyst acts on the monomer, repeating chemical bonds begin to form, increasing in number and complexity as the hardening process (*polymerization*) proceeds. Finally, the resultant hard product is known as a polymer. Two methods have been employed to catalyze polymerization: (1) light curing by use of a visible blue light (synonyms: photocure, photoactivation, light activation) and (2) self-curing, in which a monomer and a catalyst are mixed together (synonyms: cold cure, autopolymerization, and chemical activation).

The two original Caulk products, Nuva-Seal and Nuva-Cote, were the only sealants in the United States requiring ultraviolet light for activation. Both have been *replaced* by other light-cured sealants that require *visible blue light.* In the manufacture of these

latter products, a catalyst, such as *camphoroquinone,* which is sensitive to visible blue-light frequencies, is placed in the monomer at the time of manufacture. Later, when the monomer is exposed to the visible blue light, polymerization is initiated.

With the autopolymerizing sealants, the catalyst is incorporated with the monomer; in addition, another bottle contains an *initiator*—usually *benzoyl peroxide.* When the monomer and the initiator are mixed, *polymerization* begins.

Light-Cured Versus Self-Cured Sealants

The main advantage of the light-cured sealant is that the operator can initiate polymerization at *any suitable time.* Polymerization time is shorter with the light-cured products than with the self-curing sealants. The light-cured process does require the purchase of a light source, which adds to the expense of the procedure. This light, however, is the same one that is used for polymerization of composite restorations, making it available in all dental offices. When using a light-cured sealant in the office, it is prudent to store the product away from bright office lighting, which can sometimes initiate polymerization.

Conversely, the self-curing resins do not require an expensive light source. They do, however, have the great disadvantage that once mixing has commenced, if some minor problem is experienced in the operating field, the operator must either continue mixing or stop and make a new mix. For the autopolymerizing resin, the time allowed for sealant manipulation and placement *must not be exceeded,* even though the material might still appear liquid. Once the hardening begins, *it occurs very rapidly, and any manipulation of the material during this critical time jeopardizes retention.*

The light-cured sealants have a higher compressive strength and a smoother surface;[46] which is probably caused by air being introduced into the self-cure resins during mixing[47] Despite these differences, both the photocured and the autopolymerizing products appear to be equal in retention.[43,48-50]

The High-Intensity Light Source

The light-emitting device consists of a high-intensity *white light,* a blue filter to produce the *desired blue color,* usually between 400 to 500 nm, and a light-conducting rod. Some other systems consist of a blue light produced by light-emitting diodes (LED) (Figure 10-2). Most have timers for automatically switching off the lights after a predetermined time interval. In use, the end of the rod is held only a few millimeters above the sealant during the first 10 seconds, after which it can be rested on the hardened surface of the partially polymerized sealant. The time required for polymerization is *set by the manufacturer* and is usually around *20 to 30* seconds. The *depth* of cure is influenced by the *intensity of light,* which can differ greatly with different products and length of exposure. Often it is desirable to set the automatic light timer for longer than the manufacturer's instructions.[51] Even after cessation of light exposure, a final, slow polymerization can *continue* over a 24-hour period.[52]

It is not known whether long-term exposure to the intense light can damage the eye. Staring at the lighted operating field is uncomfortable and does produce afterimages. This problem is circumvented by the use of a round, 4-inch dark-yellow disk, which

fits over the light housing. The disk filters out the intense blue light in the 400- to 500-nanometers range as well as being sufficiently dark to subdue other light frequencies.

Figure 10-2 Light emitting diode (LED) curing unit for direct, intraoral exposure.

Question 1

Which of the following statements, if any, are correct?

A. In an area with fluoridated water, a *lower incidence* of caries can be expected, along with a *lower proportion* of occlusal to smooth-surface lesions.

B. Sealants should *never* be flowed over incipient caries.

C. Bis-GMA are the initials used to specify the chemical family of resins containing bisphenol A-glycidyl methyl-acrylate.

D. A monomer can polymerize, but a polymer cannot monomerize.

E. Sealants are contraindicated for adults.

Requisites for Sealant Retention

For sealant retention the surface of the tooth must (1) have a *maximum surface area*, (2) have *deep, irregular pits and fissures*, (3) be *clean*, and (4) be *absolutely dry* at the time of sealant placement and uncontaminated with saliva residue. *These are the four commandments for successful sealant placement, and they cannot be violated.*

Increasing the Surface Area

Sealants do not bond directly to the teeth. Instead, they are retained mainly by *adhesive forces.*[53] To increase the surface area, which in turn increases the adhesive potential, *tooth conditioners* (also called *etchants*), which are composed of a 30 to 50% concentration of phosphoric acid, are placed on the occlusal surface prior to the placement of the sealant.[54] The etchant may be either in *liquid* or *gel* form. The former is easier to apply and easier to remove. Both are equal in abetting retention.[55,56] If any etched areas on the tooth surface are not covered by the sealant or if the sealant is not retained, the normal appearance of the enamel returns to the tooth within 1 hour to a few weeks *due to a remineralization* from constituents in the saliva.[57] The etchant should be carefully applied to avoid contact with the soft tissues. If not confined to the occlusal surface, the acid may produce a mild inflammatory response. It also produces a sharp acid taste that is often objectionable.

Pit-and-Fissure Depth

Deep, irregular pits and fissures offer a much more favorable surface contour for sealant retention compared with broad, shallow fossae (Figure 10-3). The deeper fissures protect the resin sealant from the shear forces occurring as a result of masticatory movements. Of parallel importance is the possibility of caries

development increasing as the *fissure depth and slope* of the inclined planes increases.[58,59] Thus, *as the potential for caries increases, so does the potential for sealant retention.*

Figure 10-3 An electron scanning microscope view of the deep pits and fissures of the occlusal surface of a molar. (Courtesy of Dr. A. J. Gwinnett, State University of New York, Stony Brook.)

Surface Cleanliness

The need and method for cleaning the tooth surface prior to sealant placement are controversial. Usually the acid etching alone is sufficient for surface cleaning. This is attested to by the fact that two of the most cited and most effective sealant longevity studies by Simonsen[60] and Mertz-Fairhurst[61] were accomplished without use of a prior prophylaxis. Recently, however, it was shown that cleaning teeth with the newer prophylaxis pastes with or without fluoride (NuPro, Topex) did not affect the bond strength of sealants,[62] composites,[63] or orthodontic brackets.

Other methods used to clean the tooth surface prior to placing the sealant included, air-polishing, hydrogen peroxide, and enameloplasty.[63-65] The use of an air-polisher has proven to thoroughly clean and removes residual debris from pits and fissures.[65-68] Hydrogen peroxide has the disadvantage that it produces a precipitate on the enamel surface.[68] Enameloplasty, achieved by bur or air abrasion has proven effective. Yet, no significant differences were observed in comparison with either etching or bur preparation of the fissures on the penetration to the base of the sealant. However, the use of enameloplasty, even if equal or slightly superior would have very serious ramifications. The laws of most states require a dentist to use air abrasion and/or to cut a tooth, a requirement that would severely curtail hygienists and assistants participation in office and school preventive dentistry programs.[69]

Whatever the cleaning preferences—either by acid etching or other methods—all heavy stains, deposits, debris, and plaque should be removed from the occlusal surface before applying the sealant.

Preparing the Tooth for Sealant Application

The preliminary steps for the light-activated and the autopolymerized resins are similar up to the time of application of the resin to the teeth. After the selected teeth are isolated, they are thoroughly dried for approximately *10 seconds.* The 10-second drying period can be mentally estimated by counting off the seconds—1,000, 2,000—until 10,000 has been reached. The liquid etchant is then placed on the tooth with a small resin sponge or cotton pledget held with cotton pliers. Traditionally, the etching solution is gently daubed, *not rubbed,* on the surface for *1 minute* for permanent teeth and for 11/2 *minutes* for deciduous teeth.[70,71] Other clinical studies, however, have shown that acid etching the enamel of both primary and permanent teeth for only 20 seconds produced similar sealant[70] and composite[72] retention as those etched for 1 and 11/2 minutes. Currently, *20 to 30 seconds* enamel-etching time is recommended. Alternatively, acid gels are applied with a supplied syringe and left undisturbed. Another 15 seconds of etching is indicated for fluorosed teeth to compensate for the greater acid resistance of the enamel. The etching period should be timed with a *clock.* At the end of the etching period, the aspirator tip is positioned

with the bevel interposed *between the cotton roll and the tooth.* For 10 seconds the water from the syringe is flowed over the occlusal surface and thence into the aspirator tip. Again, this 10-second period can be mentally counted. Care should be exercised to ensure that the aspirator tip is close enough to the tooth to prevent any water from reaching the cotton rolls, yet not so close that it diverts the stream of water directly into the aspirator (see Figure 10-5).

Following the water flush, the tooth surface is dried for *10 seconds.* The air supply needs to be absolutely dry. The dried tooth surface should have a white, dull, frosty appearance. This is because the etching will remove approximately 5 to 10 um of the original surface,[73] although at times interrod penetrations of up to 100 um may occur.[74] The etching *does not always* involve the interrod areas; sometimes the central portion of the rod is etched, and the periphery is unaffected. The pattern on any one tooth is unpredictable.[75] In any event, the surface area is greatly increased by the acid etch.

Figure 10-5 Showing position of aspirator tip between the bicuspid and cotton roll during flushing, A, and between water flow and cotton roll looped around second molar, B. *Complete* dryness of the cotton rolls can be maintained with this technique.

Question 2

Which of the following statements, if any, are correct?

A. Autopolymerizing sealants and light-cured sealants have approximately the same record for longevity.

B. A 40% phosphoric acid etchant should be satisfactory for both etching and cleaning the average tooth surface prior to sealant placement.

C. Fossae with deep inclined planes tend to have more carious fissures; fossae with deep inclined planes tend to retain sealants better.

D. In studies in which a rubber dam was used to maintain a dry field for sealant placement, the retention of sealants was greater than when cotton rolls were used.

E. In placing a sealant, 10 seconds are devoted to each of the drying and etching phases and 1 minute to the flushing of the etchant from the tooth.

Dryness

The teeth *must* be dry at the time of sealant placement because sealants are hydrophobic. The presence of saliva on the tooth is even more detrimental than water because its organic components interpose a barrier between the tooth and the sealant. Whenever the teeth are dried with an air syringe, the air stream should be *checked* to ensure that it is not moisture-laden. Otherwise, sufficient moisture sprayed on the tooth will prevent adhesion of the sealant to the enamel. A check for moisture can be accomplished by directing the air stream onto a cool mouth mirror; any fogging indicates the presence of moisture. Possibly the omission of this simple step accounts for the inter-operator variability in the retention of fissure sealants.

A dry field can be maintained in several ways, including use of a *rubber dam*, employment of *cotton rolls,* and the placement of *bibulous pads* over the opening of the parotid duct. The rubber dam provides an ideal way to maintain dryness for an extended time. Because a rubber dam is usually employed in accomplishing quadrant dentistry, sealant placement for the quadrant should also be accomplished during the operation. Under most operating conditions, however, it is not feasible to apply the dam to the different quadrants of the mouth; instead it is necessary to employ cotton rolls, combined with the use of an effective *high-volume, low-vacuum aspirator.* Under such routine operating conditions, cotton rolls, with and without the use of bibulous pads, can usually be employed as effectively as the dam for the relatively short time needed for the procedure. *The two most successful sealant studies have used cotton rolls for isolation.*[60,61] In one study in which retention was tested using a rubber dam versus cotton rolls, the sealant retention was approximately *equal.*[76] Others have shown excellent sealant retention after 3 years[77] and after 10 to 20 years.[60,78]

In programs with *high patient volume* where cotton rolls are used, it is best to have two individuals involved—the *operator,* whose main task is to prepare the tooth and to apply the sealant, and the *assistant,* whose task is to maintain dryness. An operator working alone, however, can maintain a maximum dry field for the time needed to place the sealants, although it is not recommended, particularly for young children or those that are difficult to manage. For the maxilla, there should be little problem with the placement of *cotton rolls* in the buccal vestibule and, if desirable, the placement of a *bibulous pad* over the parotid duct. For the mandible, a 5-inch segment of a 6-inch cotton roll should be looped around the last molar and then held in place by the patient using the index and third fingers of the opposite hand from the side being worked on (Figure 10-4). With aid from the patient and with appropriate aspiration techniques, the cotton rolls can usually be kept dry throughout the entire procedure. Cotton roll holders may be used, but they can be cumbersome when using the aspirator or when attempting to manipulate or remove a roll. If a cotton roll does become *slightly* moist, many times another short cotton roll can be placed on top of the moist segment and held in place for the duration of the procedure. In the event that it becomes necessary to replace a wet cotton roll, it is essential that *no* saliva contacts the etched tooth surface; if there is *any* doubt, it is necessary to repeat all procedures up to the time the dry field was compromised. This includes a 15-second etch to remove any residual saliva, in lieu of the original 1-minute etch.

Another promising dry-field isolating device that can be used for single operator use, especially when used with cotton rolls, is by using ejector moisture-control systems.[a] In one study comparing the Vac-Ejector versus the cotton roll for maintaining dryness, the two were found to be equally effective.[79]

[a]Whaledent International, New York, NY

Figure 10-4 Four-handed dentistry with no assistant. The patient holds the cotton rolls with the index and third finger, thumb under chin. Patient also holds aspirator with other hand when it is not being used by operator.

Application of the Sealant

With either the light-cured or autopolymerized sealants, the material should first be placed in the fissures where there is the maximum depth. At times penetration of the fissure is negated by the presence of debris, air entrapment, narrow orifices, and excessive viscosity of the sealant.[80] The sealant should not only fill the fissures but should have some *bulk over the fissure.* After the fissures are adequately covered, the material is then brought to a knife edge approximately *halfway* up the inclined plane.

Following polymerization, the sealants should be examined carefully *before* discontinuing the dry field. If any voids are evident, additional sealant can be added *without* the need for any additional etching. The hardened sealant has an oil residue on the surface. This is unreacted monomer that can be either wiped off with a gauze sponge or can be left. If a sealant requires repair at any time after the dry field is discontinued, it is prudent to repeat the same etching and drying procedures as initially used. Because all the commercial sealants—both the light-cured and self-cured—are of the same Bis-GMA chemical family, *they easily bond to one another*.[81]

Occlusal and Interproximal Discrepancies

At times an excess of sealant may be inadvertently flowed into a fossa or into the adjoining interproximal spaces. To remedy the first problem, the occlusion should be checked visually or, if indicated, with articulating paper. Usually *any minor* discrepancies in occlusion are rapidly removed by normal chewing action. If the premature contact of the occlusal contact is unacceptable, a large, *no. 8. round cutting* bur may be used to rapidly create a broad resin fossa.

The integrity of the interproximal spaces can be checked with the use of dental floss. If any sealant is present, the use of scalers may be required to accomplish removal. These corrective actions are rarely needed once proficiency of placement is attained.

Question 3

Which of the following statements, if any, are correct?

A. The etchant *predictably* attacks the center of the enamel prism, leaving the periphery intact.

B. When the data of a study indicate that 65% of the original sealants are retained for 7 years, it is the same as saying that an average of 5% is lost each year.

C. Bis-GMA products by different manufacturers are incompatible with one another.

D. An etched area that is not rapidly sealed will retain its rough, porous surface *indefinitely.*

E. The cleansing and etching of the occlusal surface with phospohoric acid is accomplished by *rubbing* the surface during the etching process.

Evaluating Retention of Sealants

The finished sealant should be checked for retention without using undue force. In the

event that the sealant does not adhere, the placement procedures should be repeated, with only about 15 seconds of etching needed to remove the residual saliva before again flushing, drying, and applying the sealant. If *two* attempts are unsuccessful, the sealant application should be postponed until remineralization occurs.

Resin sealants are retained better on recently erupted teeth than in teeth with a more mature surface; they are retained better on first molars than on second molars. They are better retained on mandibular than on maxillary teeth. This latter finding is possibly caused by the lower teeth being more accessible, direct sight is also possible; also, gravity aids the flow of the sealant into the fissures.[41]

Teeth that have been sealed and then have lost the sealant have had fewer lesions than control teeth.[82] This is possibly due to the presence of tags that are *retained in the enamel* after the bulk of the sealant has been sheared from the tooth surface. When the resin sealant flows over the prepared surface, it penetrates the finger-like depressions created by the etching solution. These projections of resin into the etched areas are called *tags.*[83] (Figure 10-6). The tags are essential for retention. Scanning electron microscopic studies of sealants that have not been retained have demonstrated large areas devoid of tags or incomplete tags, usually caused by saliva contamination. If a sealant is forcefully separated from the tooth by masticatory pressures, many of these tags are *retained* in the etched depressions.

The number of retained sealants decreases at a *curvolinear rate.*[41] Over the first 3 months, the rapid loss of sealants is probably caused by *faulty technique* in placement. The fallout rate then begins to plateau, with the ensuing sealant losses probably being due to abnormal *masticatory stresses.* After a year or so, the sealants become very difficult to see or to discern tactilely, especially if they are abraded to the point that they fill only the fissures. In research studies this lack of visibility often leads to *underestimating* the effectiveness of the sealants that remain but cannot be identified. Because the most rapid falloff of sealants occurs in the early stages, an initial 3-month recall following placement should be routine for determining if sealants have been lost. If so, the teeth should be resealed. Teeth successfully sealed for 6 to 7 years are likely to remain sealed.[83]

In a review of the literature, longest-term study reported that at the follow-up examination of the first molars, 20-years after sealant had been applied, 65% showed *complete* retention and 27% partial retention *without* caries. At a 15-year follow-up of the same sealants the second molars demonstrated the corresponding figures 65% and 30%, respectively. This study showed that pit-and-fissure sealants applied during childhood have a *long-lasting, caries preventive effect.*[60,77] Mertz-Fairhurst[83] cited studies in which 90 to 100% of the original sealants were retained over a 1-year period (Table 10-1). One 10-year study using 3M Concise Sealant had a 57% complete retention and a 21% partial retention of sealant, *all with no caries.* Another study, using Delton, registered 68% retention after 6 years.[108] (Figure 10-7). These are studies in which the sealant was placed and then observed at periodic intervals; there was no resealing when a sealant was lost. *Where resealing is accomplished as needed at recall appointments, a higher and more continuous level of protection is achieved.* More recent studies report 82% of the sealants placed are retained for 5 years.[70]

Figure 10-6 Tags, 30 um. Sealant was flowed over etched surface, allowed to polylmerize, and tooth surface subsequently dissolved away in acid. (Courtesy,

Silverstone LM, Dogon IL. *The Acid Etch Technique.* St. Paul, MN: North Central Publishing Co, 1975.)

Figure 10-7 A: 5-year sealant: Five years after placement of a white pit-and-fissure sealant in the matched pair to the control subject. Sealant and control subjects were matched on age, sex, caries history and other factors. B: 5-year control: This matched pair to the sealed patient. This subject did not receive sealant. The first permanent molar has already been restored with two amalgam restorations in the previous 5-year period. C: 15-year sealant: 15 years after the single application of a white pit-and-fissure sealant. This is the same tooth as seen in Figure 1, 5-year sealant, but 10 years later. As can be seen, the sealant has served its purpose even though there has been some loss in the peripheral fissures. (Courtesy of Dr. Richard J. Simonsen, D.)

Colored Versus Clear Sealants

Both clear and colored sealants are available. They vary from translucent to white, yellow, and pink. Some manufacturers sell both clear and colored sealants in either the light-curing or autopolymerizing form. The selection of a colored versus a clear sealant is a matter of individual preference. The colored products permit a *more precise placement* of the sealant, with the visual assurance that the periphery extends halfway up the inclined planes. *Retention can be more accurately monitored* by both the patient and the operator placing the sealant. On the other hand, a clear sealant may be considered more *esthetically* acceptable.

Some clinicians prefer the clear sealants because they are more discrete than white. Others prefer the white sealants as they are easier to monitor at recall appointments. On the other hand, some clinicians seem to prefer the clear sealants because it is possible to see under the sealant if a carious lesion is active or advancing. However, no clinical study has comprehensively compared these issues. Recently, some pit-and-fissure sealants have been introduced that will change color as they are being light-polymerized. This property has not been fully investigated and seems to be only of relative advantage to the dental personnel applying the sealant.

Placement of Sealants Over Carious Areas

Sealing over a carious lesion is important because of the professionals' concern about the possibility of caries progression under the sealant sites. In teeth that have been examined *in vivo* and later subjected to histologic examination following extraction for orthodontic reasons, it has been found that areas of incipient or overt caries often occur under many fissures, which *cannot* be detected with the explorer.[85] In some studies, sealants have been purposely placed over small, overt lesions.[83,86] When compared with control teeth, many of the sealed carious teeth have been diagnosed as sound 3 and 5 years later.[87] Handelman has indicated that sealants can be considered a viable modality for *arrest* of pit-and-fissure caries.[88] In other studies of sealed lesions, the number of bacteria recovered from the sealed area decreased rapidly.[33,34,86-89] This decrease in bacterial population is probably due to the integrity of the seal of the resin to the etched tooth surface[90] seal that does not permit the movement of fluids or tracer isotopes between the sealant and the tooth.[91]

Sealants have been placed over more extensive lesions in which carious dentin is

involved.[92] Even with these larger lesions, there is a decrease in the bacterial population and arrest of the carious process as a function of time. In another study, clinically detectable lesions into the dentin were covered for 5 years with Nuva-Seal. After that time the bacterial cultures were essentially negative, and an apparent 83% *reversal* from a caries-active to a caries-inactive state was achieved.[86] Jordan and Suzuki[93] sealed small lesions in 300 teeth. During clinical and x-ray observations over a 5-year period, they found *no change in size of the carious lesion*, so long as the sealant remained intact. More recently, Mertz-Fairhurst and colleagues[94] demonstrated that sealed lesions became *inactive* bacteriologically, with the residual carious material suggesting decay cessation. This ability to arrest incipient and early lesions is highlighted by the statement in the 1979 publication of the ADA's Council on Dental Therapeutics: "Studies indicate that there is an apparent reduction in microorganisms in infected dentin covered with sealant. . .. These studies appear to substantiate that there is no hazard in sealing carious lesions." The statements end with the *cautionary* note: "However, additional long-term studies are required before this procedure can be evaluated as an alternative to traditional restorative procedures.[95] When sealing incipient lesions, care should be taken to monitor their retention at subsequent recall/annual dental examinations. In addition, there have been reports of sealants being used to achieve penetration of incipient smooth-surface lesions ("white spots") of facial surfaces."[96]

Question 4

Which of the following statements, if any, are correct?

A. Tags can be easily determined by their rough feel when checking the *surface* of a sealant with an explorer.

B. Teeth that lose a sealant are more susceptible to caries than ones that retain a sealant but less caries-prone than a control tooth that was never sealed.

C. The falloff of sealants is *linear* as a function of time.

D. A study in which the periodic resealing of fissures occurs would be expected to have a *lesser* caries rate than a long-term study in which the same annual falloff is experienced, but where no resealing is accomplished.

E. Following placement of a sealant over a fissure with an undetectable carious lesion, the size of the subsurface lesion gradually *increases.*

Sealants Versus Amalgams

Comparing sealants and amalgams is not an equitable comparison because sealants are used to *prevent* occlusal lesions, and amalgam is used to *treat* occlusal lesions that could have been prevented. Yet, the comparison is necessary. One of the major obstacles to more extensive use of sealants has been the belief that amalgams, and not sealants, should be placed in anatomically defective fissures; this belief stems from *misinformation* that amalgams can be placed in less time, and that once placed, they are a permanent restoration. Several studies have addressed these suppositions. For instance, sealants require approximately 6 to 9 minutes to place initially, amalgams 13

to 15 minutes.[97,98]

Many studies on *amalgam* restorations have indicated a *longevity* from only a few years to an average life span of 10 years.[99-102] Equally perturbing is the fact that in one large study of schoolchildren, 16.2% of all surfaces filled with amalgam had marginal leakage and *needed replacement.*[103] The life span of an amalgam is shorter with younger children than with adults.[104] To emphasize the problem of replacement of older restorations, a recent questionnaire study from 91 dentists in Iceland was conducted to determine the cause for replacement of 8,395 restorations. The reason given for the replacement of composites, amalgams, glass-ionomers, and for resin modified glass ionomers was failed restorations (47.2%), primary caries (45.3%) and non-carious defects (7.5%). For every restoration inserted for an overt lesion, there was a need for one to be reinserted previously![105]

The retention data from the earlier sealant studies were discouraging. In recent years, using later-generation sealants, along with the *greater care in technique* used for their insertion, much longer retention periods have been reported. In five long-term studies from 3 to 7 years, the average sealant loss per year ranged from 1.3 to 7%.[106] If the yearly loss of these studies is extrapolated, the average life of these sealants compares favorably or exceeds that of amalgam.[107] When properly placed, sealants are no longer a temporary expedient for prevention; instead, they are the *only effective predictable* clinical procedure available for preventing occlusal caries.

The most frequent cause for sealant replacement is *loss of material,* which mainly occurs during the first 6 months; the most likely cause for amalgam replacement is *marginal decay,*[108] with 4 to 8 years being the average life span.[103] To replace the sealant, only resealing is necessary. No damage occurs to the tooth. Amalgam replacement usually requires cutting more tooth structure with each replacement. Even if longevity merits were equal, the sealant has the advantage of being painless to apply and aesthetic, as well as emphasizing the *highest objectives* of the dental profession—*prevention and sound teeth.*

Options for Protecting the Occlusal Surfaces

The use of sealants has spawned an entirely different concept of conservation of occlusal tooth structure in the management of deep pits and fissures before, or early in caries involvement. The *preventive dentistry restoration* embodies the concepts of both prophylactic odontotomy insertion of a restoration and *covering the restoration and the connecting fissure system with a resin based sealant.* Pain and apprehension are slight, and aesthetics and tooth conservation are maximized.[108] Several options are now available to protect the occlusal surfaces, with the selection depending *on risk and professional's judgment.*[109] The first level of protection is simply to place a conventional sealant over the occlusal fissure system. This sealing preempts future pit-and-fissure caries, as well as arrests incipient or reverses small overt lesions.

The second option reported by Simonsen in 1978,[110] advocated the use of the *smallest* bur to remove the carious material from the bottom of a pit or fissure and then using an appropriate instrument to tease *either sealant or composite* into the cavity preparation. This he termed a preventive dentistry restoration. Following insertion of the restoration, sealant was placed *over* the polymerized material as well as flowed

over the remaining fissure system. Aside from protecting the fissures from future caries, it also protects the composite or inserted sealant from abrasion.[111]

The third option is use of glass-ionomers material for sealants, which is controversial. Due to their fluoride release and cariostatic effect, glass-ionomers have been used in place of traditional materials, as a pit-and-fissure sealant, however, resin sealants have shown much higher bond strength to enamel than glass-ionomers. Clinical trials[112,113] have shown poor retention over periods as short as 6 to 12 months. Though, in vitro studies have suggested that etching previous to application enhances the bonding of glass-ionomer sealant in fissure enamel.[114-116] One study showed that a conventional silver-reinforced glass-ionomer had superior clinical performance compared to a conventional resin sealant.[117]

Resin-reinforced glass-ionomer cements have been investigated for their effectiveness as pit-and-fissure sealants. The 1-year results revealed that although clinically the glass-ionomer wears at a faster rate than a conventional resin sealant, in the scanning electron microscopic evaluation the material could be seen at the deep recesses of the pits-and-fissures with no carious lesion present.[113] A recent study showed that after 3 years the glass-ionomer sealant was completely lost in almost 90% of the teeth compared to less than 10% of the resin sealed teeth; the relative risk of a tooth sealed with glass-ionomer over that of a tooth sealed with resin was higher. Also, the glass-ionomer sealant had poorer retention and less caries protective effect.[118]

Glass-ionomer does not carry the ADA seal of approval as sealant material. The readers should decide their personal philosophy based on the evidence.

A fourth option reported by Garcia-Godoy in 1986 involves the use of a glass-ionomer cement as the *preventive glass-ionomer restoration* (PGIR).[119] The glass-ionomer cement (conventional or resin-modified) is placed only in the cavity preparation. (Figure 10-8). The occlusal surface is then etched with a gel etchant avoiding, if possible, etching the glass-ionomer. Etching the glass-ionomer may remove some of the glass particles weakening the material. The conventional resin sealant is placed *over the glass-ionomer and the entire occlusal fissure system.* In the event sealant is lost, the fluoride content of the glass-ionomer *helps prevent* future primary and *secondary* caries formation. The same technique has successfully protected the marginal integrity of very small amalgam restoration, as well as providing a protection to the entire fissure system.

Each of these options requires a judgment decision by the clinician. That decision can well be based on the criterion that if an overt lesion cannot be *visualized,* it should be sealed; if it can be visualized, the smallest possible preventive dentistry restoration should be used along with its required sealant "topping." Mertz-Fairhurst and associates[120] have pointed out that the first option could provide the preferred model for conservative treatment of *incipient* and *small overt,* pit-and-fissure caries. It could also serve as an interim treatment for larger lesions. These options would be especially valuable in areas of the world with insufficient professional dental personnel and where preventive dental auxiliaries have been trained to place sealants. In all cases, the preventive dental filling should be considered as an alternative to the traditional class I amalgam with its accompanying extension for prevention that often includes the entire fissure system.

Figure 10-8 Preventive glass ionomer restoration (PGIR). Cavity preparation for reception of glass-ionomer cement. (Courtesy of Dr. Franklin Garcia-Godoy, University of Texas Dental School, San Antonio.)

The Sealant as Part of a Total Preventive Package

The sealant is used to protect the occlusal surfaces. A major effort should be made to incorporate the use of sealants along with other primary preventive dentistry procedures, such as plaque control, fluoride therapy, and sugar discipline. Whenever a sealant is placed, a topical application of fluoride should follow if at all possible. In this manner the whole tooth can be protected. Ripa and colleagues[121] completed a 2-year study for children in second and third grades assessing the effectiveness of a 0.2% fluoride mouthrinse used alone compared with a rinse plus sealants. Twenty-four occlusal lesions developed in the 51 rinse subjects, and *only* 3 in the 84 subjects receiving the rinse plus sealants. The conclusion was that caries could be *almost completely eliminated* by the *combined* use of these two preventive procedures. In many public-health programs, however, it is not possible to institute full-scale prevention programs, either because of apathy or lack of time and money. In such cases, there is some consolation in knowing that at least the *most vulnerable* of all tooth surfaces (the occlusal) is being protected.

Manpower

The *cost* of sealant placement *increases* directly with the level of professional education of the operator. Dentists, hygienists, assistants, and other auxiliaries can be trained to place sealants.[122-124] In view of the cost-effectiveness, dental auxiliaries should be considered as the logical individuals to place sealants. This is important if manpower is to be increased.

Often auxiliaries who have received sealant instruction, either through continuing-education courses or as part of a curriculum, are stymied either because of state laws interdicting their placing sealants or by the nature and philosophy of the practice of the employing dentist.[125] Only fourteen states allow hygienists to practice under less restrictive or unsupervised practice models in which they can initiate treatment based on assessment of patient, treat the patient, and maintain a provider-patient relationship without the participation of the patients' dentist of record. For example, Maine and New Hampshire have a separate supervision for settings outside of the dental office public-health supervision, which is less restrictive than general supervision. New Mexico allows for a collaborative-practice agreement between dentists and hygienists in outside settings. Yet, in states such as Georgia and Illinois, hygienists are required to practice under direct supervision. This means the dentist must be present in the office while the care is being provided.[126]

In a Swedish study, 77 *dental assistants* working in 12 dental clinics sealed 3,218 first and second molars with a 5-year retention rate of between 74 and 94%.[127] Because many dentists consider the placement of sealants to be a relatively simple procedure, few are returning for continuing-education programs to learn the exacting and precise process necessary to ensure maximum sealant retention. Even when the dental professionals desire to participate in such continuing education, a survey found relatively few courses available.[128]

Economics

Bear in mind that not every tooth receiving a sealant would necessarily become carious; hence the cost of preventing a single carious lesion is greater than the cost of a single sealant application. For instance, Leverett and colleagues calculated that five sealants would need to be placed on sound teeth to prevent one lesion over a 5-year period,[129] and Rock and Anderson estimated one tooth for every three sealant applications are prevented from becoming carious.[130] Sealants would be most cost-effective if they could be placed in only those pits and fissures that are destined to become carious. Unfortunately, we do not have a caries predictor test of such exactitude, but, the use of vision plus an economic, portable electronic device that objectively measures conductance (or resistance) would greatly aid in evaluating occlusal risk.[131] Without such a device, it is necessary to rely on professional judgment, based on the severity of the caries activity indicators: number of "sticky" fissures, level of plaque index, number of incipient and overt lesions, and microbiologic test indications.

In an office setting, it is estimated that it costs 1.6 times more to treat a tooth than to seal.[55] The Task Force on Community Preventive Services, an independent, non-federal group formed to evaluated oral-health interventions, was charged with determining interventions that promote and improve oral health. The Task Force examined six public-health programs cost of placing pit-and-fissure sealants revealing a mean cost of $39.10 per person.[132] However, even these numbers are misleading. For instance, what is the value of an intact tooth to its owner? How much does it cost for a dentist and assistant to restore a tooth, compared to the cost of sealing a tooth? Later in life, what is the cost of bridges and dentures that had their genesis when children were at high risk with little access to dental care?

Use of Pit-and-Fissure Sealants

By the mid-1980s most of the answers were available as to the need and *effectiveness* of Bis-GMA sealants to reduce the incidence of occlusal caries, and the *techniques* of placement of pit-and-fissure sealants were known.[133] The *safety* of their placement has been demonstrated by many studies showing that even when placed over incipient and minimally overt caries sites, there was no progression *as long as the sealant remained intact.*[134] Finally, several clinical studies have pointed out that sealants could be *applied by properly trained auxiliaries,* thus providing a more economical source of manpower for private and military practices as well as for large school and public health programs.

Bis-GMA sealant usage has been strongly supported by the ADA "as a safe and effective means for caries control."[25] The United States Public Health Service, in a request for a proposal for a school pit-and-fissure study, stated *"This combination of preventive techniques (combined use of fluoride and sealants) is expected to essentially eliminate caries in teeth erupting after the initiation of the study."*[135] Despite the support from the two largest organizations most interested in the dental health of the nation, the rank-and-file of the dental profession *have not accepted sealants as a routine method for prevention.*

In spite of all the knowledge of the properties and successes of the sealants usage has lagged, with about 10% of the posterior teeth of children demonstrating the presence of sealants.[136] For example, a 1994 examination of 117,000 children in North Carolina between the ages of 6 and 17 found that approximately 12% had sealants,[137] while the percentage for 927,000 in Tennessee was 10%.[138] Other states demonstrate similar sealant usage. One study revealed that 88 children did have sealants while 508 did not have needed sealants.[139] For recruits entering the U.S. Air Force, sealants were found on 13.1% of the teeth while there was a need for 47.5% more. In the latter case, it was noted that a third of these personnel had occlusal caries that might have been prevented by the sealants.[20]

Many barriers exist in meeting the Healthy People 2010 Objective for sealants. In 2001, the State of Alabama was planning how to meet national dental objectives, when 50% of U.S. children are expected to have dental sealants on at least one permanent molar by the age of 14 years.[140] (Currently, 22% of the children between 12 to 14 years have at least one sealant claim.) A final assessment of the 2010 prospects and the current State's demographics concluded that racial and gender disparities, difficulty in accessing care, the nonavailability of Medicaid-participating dentists in a country, and a lower payment/claim ratio may make the national sealant objective difficult to achieve.[140] It should be mentioned that in many surveys, children from lower socioeconomic groups had greater sealant needs than those from more affluent neighborhoods.

On the other hand, other countries have had marked success with increasing the number of teeth sealed. A study involving 68,704 children living in Lanarkshire, Scotland found approximately 10% of the occlusal surfaces were sealed.[141] Five years later, in England the percentage of children *having sealants dramatically increased* between 20 to 50% in several areas.[142]

The placement of sealants is making slow progress. The 1998-99 Ohio State survey of 3rd-grade students in School Based/School Link programs found that in addition to oral-health benefits, "Providing sealant programs in all eligible, high-risk schools could reduce or eliminate racial and economic disparities in the prevalence of dental sealants".[143] Yet, there are problems in examining the number of sealants *versus* the *need* for sealants.

Dentist Involvement

Pit-and-fissure sealants are underused in private practice and public health. There are many complex reasons for such under use, but efforts should be undertaken to increase sealant use.[3] Increasing sealant use is dependent, in part, on dentists' acceptance and understanding of the preventive technique. In a mail survey in Minnesota, 95% of 375 dentists reported the use of sealants, varying from 1 to 25 per week. Possibly, the incongruity of numbers stems from the fact that although the majority of dentists use sealants, the *frequency* of use is *low*.[144]

Reasons for this apathy have ranged from alleged concerns of sealing over carious lesions, lack of technical skill, short longevity of sealants, and the need for more research—all problems that have been adequately addressed in the literature.[133] Probably the most important factor now restricting the placement of sealants is the

lack of an adequate insurance fee schedule.[145] Another is that most dentists are treatment-oriented. This fact is amplified by an explanation by Galarneau and Brodeur that "A dentists lack of comfort with withholding treatment may stop him/her from offering preventive care and cause him to follow a restoration-oriented practice."[146] Another factor is that dentists rarely explain the oral-health advantages of sealants over dental restorations.[147]

In attempting to alter the attitudes of dentist on sealant use, several studies have been conducted to measure *changes in knowledge and attitudes* following continuing-education courses. The follow-up indicated that there had been an increase in *knowledge* but little change in *attitudes* concerning sealant use.[148] In Colorado, pediatric dentists, who are continually involved in treating children, placed more sealants than general dentists—again, probably a manifestation of attitudes.[145]

Regardless of increased rhetoric about prevention, the concepts and actions of prevention are *not* being fully implemented in dental schools.[149] Dental school faculties need to be educated about the effectiveness and methods of applying sealants.[150,151] Possibly the development of a model curriculum for teaching pit-and-fissure sealant usage would help.[152] The dental community must develop a consensus about the value and economic effect of preventive measures.[150]

Other barriers to effective delivery of sealants include (1) state-board restrictions on auxiliary placement of sealants, (2) lack of consumer knowledge of the effectiveness of sealants, and, resultantly, a lack of demand for the product.[122] *The economics and education of the profession and of the public are the prime requisites for expanded sealant acceptance.*[153]

Question 5

Which of the following statements, if any, are correct?

A. The longevity expectation for a properly placed amalgam restoration is approximately twice that of a properly placed sealant.

B. Sealants should be placed only on permanent teeth of children up to age 16.

C. Sealants are found on approximately 54% of U.S. children.

D. Following the graduation of students presently in dental schools, a large increase in the use of sealants can be expected.

E. Caries does not progress under a properly sealed composite or amalgam.

Other Pit-and-Fissure Initiatives

The findings of the following studies must be considered an *important extension* of the present use of pit-and-fissure sealants, which are used to prevent the development of incipient lesions and to arrest minimal overt lesions. If professional judgment dictates, conservative sealed amalgams or composites could be used to maintain *marginal integrity, extend the longevity of the restorative materials, and for achieving*

a de facto extension for prevention without the need to remove sound tooth structure to extend the restoration over the entire fissure system. These two uses of resins for prevention and restorations without major operative considerations should be of great value in developing countries where professional manpower is at a minimum and the demand for dental care is great.

Probably the most important recent research on the use of Bis-GMA sealants and carious lesions were described by Mertz-Fairhurst and coworkers.[87,154] In the 10-year study,[154] patients with paired permanent molars or premolars with *obvious clinical and radiographic class I lesions* were selected. The carious lesions extended halfway into the dentin or to the nearest pulp horn. The randomized placement of restorations for each of the tooth pairs consisted of two of the following: (1) a *classic* amalgam restoration, complete with extension for prevention of all connecting fissures (79 subjects); (2) a *conservative* amalgam restoration involving only the carious site with a sealant "topping," the latter which was extended into the entire pit-and-fissure system (77 subjects); and (3) with each one of the amalgam restorations, a paired composite restoration placed over the carious tissue with a "topping" of sealant that included all the pits and fissures (156 subjects). In the preparation for the composite, *no attempt was made to remove the carious tissue.* A 1-millimeter wide, 40- to 60-degree bevel was made in the sound enamel surrounding the lesion. The area was washed, dried, and a bonding agent was placed on the bevel. Hand instruments were used to place the composite, after which rotary instruments were used to shape the occlusal anatomy. Following this step, the occlusal surface was treated as for the placement of the average sealant—dry, etch, rinse, and dry before placing the resin over the composite and the entire fissure system.

The conclusions of this study after 10 years were: (1) *both* the sealed composites and the sealed amalgam restorations exhibited *superior clinical performance and longevity* compared to the unsealed amalgam restorations; (2) bonded and sealed composite restorations placed over the frank cavitated lesions *arrested the clinical progress of these lesions for the 10 years of the study.*

Summary

The majority of all carious lesions that occur in the mouth occur on the occlusal surfaces. Which teeth will become carious cannot be predicted; however, if the surface is sealed with a pit-and-fissure sealant, no caries will develop as long as the sealant remains in place. Recent studies indicate an approximate 90% retention rate of sealants 1-year after placement. Even when sealants are eventually lost, most studies indicate that the caries incidence for teeth that have lost sealants is less than that of control surfaces that had never been sealed. Research data also indicate that many incipient and small overt lesions are arrested when sealed. Not one report has shown that caries developed in pits or fissures when under an intact sealant. Sealants are easy to apply, but the application of sealants is an extremely sensitive technique. The surfaces that are to receive the sealant must be completely isolated from the saliva during the entire procedure, and etching, flushing, and drying procedures must be timed to ensure adequate preparation of the surface for the sealant. Sealants are comparable to amalgam restorations for longevity and do not require the cutting of tooth structure. Sealants do not cost as much to place as amalgams. Despite their advantages, the use of sealants has not been embraced by all dentists, even though

endorsed by the ADA and the U.S. Public Health Service. Even when small overt pit-and-fissure lesions exist, they can be dealt with conservatively by use of preventive dentistry restorations. What now appears to be required is that the dental schools teach sealants as an effective intervention, that the dental professional use them, that the hygienists and the auxiliary personnel be permitted to apply them, and the public demand them.

Answers and Explanations

1. C and D—correct.

A—incorrect. Because the fluorides protect the smooth surface, there will be a greater proportion of pit-and-fissure lesions.

B—incorrect. By definition, an incipient lesion has not been invaded by bacteria; thus the use of a sealant is an ideal preventive measure.

E—incorrect. Remember, it is the caries susceptibility of the teeth that is important—not the age of the individual.

2. A, B, and C—correct.

D—incorrect. All the major, successful, long-term retention studies have used cotton-roll isolation; in the one study of rubber dam versus cotton rolls, the rolls were equal to, or better than, the dam.

E—incorrect. Ten seconds are used for the drying and flushing procedures, and 20 to 30 seconds for the etching.

3. A and B—correct.

C—incorrect. Bis-GMA plastics are of the same chemical family and will bond to each other regardless of manufacturer.

D—incorrect. Remineralization from saliva constituents occurs rapidly in a period of hours to days.

E—incorrect. Cleansing and etching do occur; however, rubbing tends to obliterate the delicate etching pattern and reduce retention potential.

4. B and D—correct.

A—incorrect. The tags of the sealant cannot be felt with the explorer; they extend into the enamel from the underneath side of the plastic.

C—incorrect. The curvolinear falloff is greatest at 3 months, less at 6 months, after which it gradually plateaus.

E—incorrect. The literature is unanimous that caries does not progress under an intact

sealant.

5. C and E—correct.

A—incorrect. There is little difference between the longevity of a well-placed amalgam compared with a well-placed sealant.

B—incorrect. If a tooth is susceptible to caries, it should be sealed, whatever the patient's age.

D—incorrect. All signs indicate that the teaching of sealant placement is greatly neglected in dental schools.

Self-evaluation Questions

1. Approximately _____ % of all carious lesions occur on the occlusal surfaces; the continual use of fluorides (increases) (decreases) this percentage.

2. Four different methods used prior to the advent of polyurethane, cyanoacrylate, and Bis-GMA sealants, were _____, _____, _____, and _____.

3. One condition that *indicates the use of a sealant is* _____; *four conditions that contraindicate* the use of sealants are _____, _____, _____, and _____; three conditions that *probably indicate* the use of sealants are _____, _____, and _____.

4. Two photoactivated, and two chemically activated sealants that have been accepted, or provisionally accepted, by the ADA are (photoactivated) _____, _____, and (chemically activated) _____ and _____.

5. The liquid resin in a sealant kit is known as the _____; when it is catalyzed the hardening process is known as _____. The catalyst used for the polymerization of chemically activated sealants is _____ and for visible photoactivation, _____.

6. Two advantages to light-cured sealants are _____ and _____; and two advantages of autopolymerized sealants are _____ and _____.

7. _____ forces, not chemical bonding, causes retention of the sealant to the tooth; the four commandments to ensure maximum retention are _____, _____, _____, and _____.

8. Three methods by which a dry field can be established are _____, _____, and _____.

9. The placement of sealants is extremely technique-sensitive; after selection of the tooth for sealant placement, it should be dried for _____ (time); then etched for _____ (time), followed by a water flush of _____ (time), and finally, dried for _____ (time) before placing the sealant.

10. Excessively high sealants that interfere with occlusion can be reduced by use of a number _____ (cutting) (finishing) bur.

11. The falloff of sealants is (linear) (curvilinear); long-term studies where 65% of the sealants are retained after 7 years indicate an average yearly loss of _____ %. After 10 years, _____ _____ % would be retained. This contracts to an average life expectancy of an amalgam of approximately _____ (years).

12. To protect the total tooth, the application of a sealant should be followed by an application of _____.

13. To ensure that sealant placement techniques have been perfected in dental and dental hygiene schools, it should be necessary for _____ (state dental-regulating agency) to require a demonstration of proficiency for all candidates prior to state licensure.

14. The three key components of a light source of polymerizing sealants are _____, _____, and _____ (which results in the blue color).

15. The three basic options for a preventive dentistry restoration are _____, _____, and _____.

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Chapter 10. Pit-and-Fissure Sealants - *Franklin Garcia-Godoy Norman O. Harris Denise Muesch Helm*

Objectives

At the end of this chapter, it will be possible to

1. Explain how sealants can provide a primary preventive means of reducing the need for operative treatment as 77% of the children 12 to 17 years old in the United States have dental caries in their permanent teeth.[1]

2. Discuss the history of sealant development through the 20th century.

3. List the criteria for selecting teeth for sealant placement and the four essentials in attaining maximum retention of sealants.

4. Describe the several steps preliminary to, during, and after the placement of a sealant—including surface cleanliness, dry fields, details of the application procedure, and remedial measures following the excess application of sealant.

5. Explain the rationale for adding fluorides to sealants.

6. Compare the advantages and disadvantages of light-cured and self-cured sealants.

7. Discuss the advantages of protecting the occlusal surfaces of teeth with sealants.

8. Cite five reasons given for the underuse of sealants by practitioners and analyze the validity of the reasons.

Introduction

Fluorides are highly effective in reducing the number of carious lesions occurring on the *smooth surfaces* of enamel and cementum. Unfortunately, fluorides are *not* equally effective in protecting the occlusal pits and fissures, where the majority of carious lesions occur.[2] Considering the fact that the occlusal surfaces constitute only 12% of the total number of tooth surfaces, it means that the *pits and fissures are approximately eight times as vulnerable as the smooth surfaces.* The placement of sealants is a highly effective means of preventing these.[3]

Historically several agents have been tried to protect deep pits and fissures on occlusal surfaces.

- In 1895, Wilson reported the placement of *dental cement* in pits and fissures to prevent caries.[2] In 1929, Bodecker[4] suggested that deep fissures could be broadened with a large round bur to make the occlusal areas more self-cleansing, a procedure that is called *enameloplasty*.[5] Two major disadvantages, however, accompany enameloplasty. First, it requires a dentist, which immediately limits its use. Second, in modifying a deep fissure by this method, it is often necessary to remove more sound tooth structure than would be required to insert a small restoration.
- In 1923 and again in 1936, Hyatt[6] advocated the early insertion of small restorations in deep pits and fissures before carious lesions had the opportunity to develop. He termed this procedure *prophylactic odontotomy*. Again, this operation is more of a

treatment procedure than a preventive approach, because it requires a dentist for the cutting of tooth structure.

- Several methods have been unsuccessfully used in an attempt either to seal or to make the fissures more resistant to caries. These attempts have included the use of topically applied zinc chloride and potassium ferrocyanide[7] and the use of ammoniacal silver nitrate;[8] they have also included the use of copper amalgam packed into the fissures.[9]
- Fluorides that protect the smooth surfaces of the teeth are less effective in protecting the occlusal surfaces.[10] Following the use of fluorides, there is a large reduction of incidence in smooth-surface caries but a smaller reduction in occlusal pit-and-fissure caries. This results in an *increased proportion* in the ratio of occlusal to interproximal lesions, even though the total number may be less.
- A final course of action to deal with pit-and-fissure caries is one that is often used: *do nothing; wait and watch.* This option avoids the need to cut good tooth structure until a definite carious lesion is identified. It also results in many teeth being lost when individuals do not return for periodic exams. This approach, although frequently used is a violation of the ethical principle of beneficence and patient autonomy.

In the late 1960s and early 1970s, another option became available—the use of pit-and-fissure sealants.[11] With this option, a liquid resin is flowed over the occlusal surface of the tooth where it penetrates the deep fissures to fill areas that cannot be cleaned with the toothbrush (Figure 10-1).[12] The hardened sealant presents a barrier between the tooth and the hostile oral environment. Concurrently, there is a significant reduction of Streptococcus mutans on the treated tooth surface.[13] Pits and fissures serve as reservoirs for mutans streptococci, sealing the niche thereby reduces the oral count.

Figure 10-1 One of the reasons that 50% of the carious lesions occur on the occlusal surface. Note that the toothbrush bristle has a greater diameter than the width of the fissure. (Courtesy of Dr. J. McCune, Johnson & Johnson.)

Criteria for Selecting Teeth for Sealant Placement

Following are the criteria for selecting teeth for sealing. Because no harm can occur from sealing, when in doubt, seal *and monitor*.

- A deep occlusal fissure, fossa, or incisal lingual pit is present.

A sealant is contraindicated if:

- Patient behavior does not permit use of adequate dry-field techniques throughout the procedure.
- An open carious lesion exists.
- Caries exist on other surfaces of the same tooth in which restoring will disrupt an intact sealant.
- A large occlusal restoration is already present.

A sealant is *probably indicated if:*

- The fossa selected for sealant placement is well isolated from another fossa with a

restoration.
- The area selected is confined to a fully erupted fossa, even though the distal fossa is impossible to seal due to inadequate eruption.
- An intact occlusal surface is present where the *contralateral tooth* surface is carious or restored; this is because teeth on opposite sides of the mouth are usually equally prone to caries.
- An *incipient* lesion exists in the pit-and-fissure.
- Sealant material can be flowed over a conservative class I composite or amalgam to improve the marginal integrity, and into the remaining pits and fissures to achieve a *de facto* extension for prevention.

Other Considerations in Tooth Selection

All teeth meeting the previous criteria should be sealed and resealed as needed. Where the cost-benefit is critical and priorities must be established, such as occurs in many public health programs, ages 3 and 4 years are the most important times for sealing the eligible deciduous teeth; ages 6 to 7 years for the first permanent molars;[14] and ages 11 to 13 years for the second permanent molars and premolars.[15] Currently, 77% of the children 12-to-17-years-old in the United States have dental caries in their permanent teeth.[1] Many school days would be saved, and better dental health would be achieved in School Dental Health Clinic programs by combining sealant placement and regular fluoride exposure.[16]

The disease susceptibility of the tooth should be considered when selecting teeth for sealants, not the age of the individual. Sealants appear to be equally retained on occlusal surfaces in primary, as well as permanent teeth.[3] Sealants should be placed on the teeth of adults if there is evidence of existing or impending caries susceptibility, as would occur following excessive intake of sugar or as a result of a drug- or radiation-induced xerostomia. They should also be used in areas where fluoride levels in community water is optimized, as well as in non-fluoridated areas.[17]

The following are two good illustrations of this philosophy. After a 3-year study, Ripa and colleagues[18] concluded that the time the teeth had been in the mouth (some for 7 to 10 years) had no effect on the vulnerability of occlusal surfaces to caries attack. Also, the incidence of occlusal caries in young Navy[19] and Air Force[20] recruits (who are usually in their late teens or early 20s) is relatively high.

Background of Sealants

Buonocore first described the fundamental principles of placing sealants in the late 1960s.[10,21] He describes a method to bond poly-methylmethacrylate (PMMA) to human enamel conditioned with phosphoric acid. Practical use of this concept however, was not realized until the development of bisphenol A-glycidyl methacrylate (Bis-GMA), urethane dimethacrylates (UDMA) and trithylene glycol dimethacrylates (TEGDMA) resins that possess better physical properties than PMMA. The first successful use of resin sealants was reported by Buonocore in the 1960s.[22]

Bisphenol A-Glycidyl Methylacrylate Sealants

Bisphenol A-glycidyl methylacrylate (Bis-GMA) is now the sealant of choice. It is a mixture of Bis-GMA and methyl methacrylate.[23] Products currently accepted by the American Dental Association (ADA) include:[24]

- Baritone L3, Type II Confi-Dental Products Co.
- Alpha-Dent Chemical Cure Pit and Fissure Sealant Dental Technologies, Inc.
- Alpha-Dent Light Cure Pit and Fissure Sealant Dental Technologies, Inc.
- Prisma-Shield Compules Tips VLC Tinted Pit & Fissure Sealant Dentsply L.D. Caulk Division
- Prisma-Shield VLC Filled Pit & Fissure Sealant Dentsply L.D. Caulk Division
- Helioseal F, Type II Ivoclar-Vivadent, Inc.
- Helioseal, Type II Ivoclar-Vivadent, Inc.
- Seal-Rite Low Viscosity, Type II Pulpdent Corp.
- Seal-Rite, Type II Pulpdent Corp.

The ADA National Standard sets aside specific criteria of pit-and-fissure sealants stating; Specification No. 39 established the following requirements:

- That the working time for type I sealants is not less than 45 seconds;
- That the setting time is within 30 seconds of the manufacturer's instruction and does not exceed three minutes;
- That the curing time for type II sealants is not more the 60 seconds;
- That the depth of cure for type II sealant is not less than 0.75 millimeter;
- That the uncured film thickness is not more than 0.1 millimeter;
- That sealants meet the bicompatibility requirements of American Nation a Standard/American Dental Association Document No. 41 for Recommended Standard Practices for Biological Evaluation of Dental Materials.[25]

Sealant products accepted by the American Dental Association carried the statement: "[Product name] has been shown to be acceptable as an agent for sealing off an anatomically deficient region of the tooth to supplement the regular professional care in a program of preventive dentistry."[26]

Nuva-Seal was the first successful commercial sealant to be placed on the market, in 1972. Since then more effective second- and third-generation sealants have become available see Table 10-1. The first sealant clinical trials used cyanoacrylate-based materials. Dimethacrylate-based products replaced these. The primary difference between sealants is their method of polymerization. First-generation sealants were initiated by ultraviolet light, second-generation sealants are autopolymerized, and third-generation sealants use visible light.

Some sealants contain *fillers*, which makes it desirable to classify the commercial products into *filled* and *unfilled* sealants. The *filled* sealants contain microscopic glass beads, quartz particles, and other fillers used in composite resins. The fillers are coated with products such as *silane*, to facilitate their combination with the Bis-GMA resin. The fillers make the sealant more *resistant to abrasion and wear*. Because they are more resistant to abrasion the occlusion should be checked and the sealant height may need to be adjusted after placement. In contrast, unfilled sealants wear quicker but usually do not need occlusal adjustment.

Fluoride-Releasing Sealants

The addition of fluoride to sealants was considered about 20 years ago,[27] and it was probably attempted based on the fact that the incidence and severity of secondary caries *was* reduced around fluoride-releasing materials such as the silicate cements used for anterior restorations.[28,29] Because fluoride uptake increases the enamel's resistance to caries,[30] the use of a fluoridated resin-based sealant may provide an additional anticariogenic effect if the fluoride released from its matrix is incorporated into the adjacent enamel.

Fluoride-releasing sealants have shown antibacterial properties[31-33] as well as a greater artificial caries resistance compared to a nonfluoridated sealant.[34-36] A recent *in vitro* study showed that pit-and-fissure sealants containing fluoride provided a caries-inhibiting effect with a significant reduction in lesion depth in the surface enamel adjacent and a reduction in the frequency of wall lesion.[37] Moreover, the fluoridated sealant laboratory bond strength to enamel,[38] and clinical performance,[39,40] is similar to that of nonfluoridated sealants.[41,42] In a recent study, it was shown that teeth sealed with Teethmate F fluoridated sealant revealed high amounts of enamel fluoride uptake in vitro and in vivo to a depth ranging from 10 to 20 um from the surface.[43] The residual fluoride was also observed within the sealing material. This agrees with another study showing the high amount of fluoride released from Teethmate F-1.[44]

The addition of fluoride to the sealants will greatly increase their value in the preventive and restorative use as mentioned above. Fluoride is added to sealants by two methods. The first is by adding a soluble fluoride to the unpolymerized resin. The fluoride can be expected to leach out over a period of time into the adjacent enamel. Eventually the fluoride content of the sealant should be exhausted, but the content of the enamel greatly increased.

The second method of incorporating fluoride is by the addition of an organic fluoride compound that is chemically bound to the resin to form an ion exchange resin. As such, when fluoride is low in the saliva, fluoride would be released. Vice versa, when the fluoride in the environment is high, it should bind to the resin to form—at least theoretically—a continuous reservoir for fluoride release and recharge.[45] See Table 10-2 on page 292 for a list of current available sealant materials.

Polymerization of the Sealants

The liquid resin is called the *monomer*. When the catalyst acts on the monomer, repeating chemical bonds begin to form, increasing in number and complexity as the hardening process (*polymerization*) proceeds. Finally, the resultant hard product is known as a polymer. Two methods have been employed to catalyze polymerization: (1) light curing by use of a visible blue light (synonyms: photocure, photoactivation, light activation) and (2) self-curing, in which a monomer and a catalyst are mixed together (synonyms: cold cure, autopolymerization, and chemical activation).

The two original Caulk products, Nuva-Seal and Nuva-Cote, were the only sealants in the United States requiring ultraviolet light for activation. Both have been *replaced* by other light-cured sealants that require *visible blue light.* In the manufacture of these

latter products, a catalyst, such as *camphoroquinone,* which is sensitive to visible blue-light frequencies, is placed in the monomer at the time of manufacture. Later, when the monomer is exposed to the visible blue light, polymerization is initiated.

With the autopolymerizing sealants, the catalyst is incorporated with the monomer; in addition, another bottle contains an *initiator*—usually *benzoyl peroxide.* When the monomer and the initiator are mixed, *polymerization* begins.

Light-Cured Versus Self-Cured Sealants

The main advantage of the light-cured sealant is that the operator can initiate polymerization at *any suitable time.* Polymerization time is shorter with the light-cured products than with the self-curing sealants. The light-cured process does require the purchase of a light source, which adds to the expense of the procedure. This light, however, is the same one that is used for polymerization of composite restorations, making it available in all dental offices. When using a light-cured sealant in the office, it is prudent to store the product away from bright office lighting, which can sometimes initiate polymerization.

Conversely, the self-curing resins do not require an expensive light source. They do, however, have the great disadvantage that once mixing has commenced, if some minor problem is experienced in the operating field, the operator must either continue mixing or stop and make a new mix. For the autopolymerizing resin, the time allowed for sealant manipulation and placement *must not be exceeded,* even though the material might still appear liquid. Once the hardening begins, *it occurs very rapidly, and any manipulation of the material during this critical time jeopardizes retention.*

The light-cured sealants have a higher compressive strength and a smoother surface;[46] which is probably caused by air being introduced into the self-cure resins during mixing[47] Despite these differences, both the photocured and the autopolymerizing products appear to be equal in retention.[43,48-50]

The High-Intensity Light Source

The light-emitting device consists of a high-intensity *white light,* a blue filter to produce the *desired blue color,* usually between 400 to 500 nm, and a light-conducting rod. Some other systems consist of a blue light produced by light-emitting diodes (LED) (Figure 10-2). Most have timers for automatically switching off the lights after a predetermined time interval. In use, the end of the rod is held only a few millimeters above the sealant during the first 10 seconds, after which it can be rested on the hardened surface of the partially polymerized sealant. The time required for polymerization is *set by the manufacturer* and is usually around *20 to 30* seconds. The *depth* of cure is influenced by the *intensity of light,* which can differ greatly with different products and length of exposure. Often it is desirable to set the automatic light timer for longer than the manufacturer's instructions.[51] Even after cessation of light exposure, a final, slow polymerization can *continue* over a 24-hour period.[52]

It is not known whether long-term exposure to the intense light can damage the eye. Staring at the lighted operating field is uncomfortable and does produce afterimages. This problem is circumvented by the use of a round, 4-inch dark-yellow disk, which

fits over the light housing. The disk filters out the intense blue light in the 400- to 500-nanometers range as well as being sufficiently dark to subdue other light frequencies.

Figure 10-2 Light emitting diode (LED) curing unit for direct, intraoral exposure.

Question 1

Which of the following statements, if any, are correct?

A. In an area with fluoridated water, a *lower incidence* of caries can be expected, along with a *lower proportion* of occlusal to smooth-surface lesions.

B. Sealants should *never* be flowed over incipient caries.

C. Bis-GMA are the initials used to specify the chemical family of resins containing bisphenol A-glycidyl methyl-acrylate.

D. A monomer can polymerize, but a polymer cannot monomerize.

E. Sealants are contraindicated for adults.

Requisites for Sealant Retention

For sealant retention the surface of the tooth must (1) have a *maximum surface area*, (2) have *deep, irregular pits and fissures*, (3) be *clean*, and (4) be *absolutely dry* at the time of sealant placement and uncontaminated with saliva residue. *These are the four commandments for successful sealant placement, and they cannot be violated.*

Increasing the Surface Area

Sealants do not bond directly to the teeth. Instead, they are retained mainly by *adhesive forces.*[53] To increase the surface area, which in turn increases the adhesive potential, *tooth conditioners* (also called *etchants*), which are composed of a 30 to 50% concentration of phosphoric acid, are placed on the occlusal surface prior to the placement of the sealant.[54] The etchant may be either in *liquid* or *gel* form. The former is easier to apply and easier to remove. Both are equal in abetting retention.[55,56] If any etched areas on the tooth surface are not covered by the sealant or if the sealant is not retained, the normal appearance of the enamel returns to the tooth within 1 hour to a few weeks *due to a remineralization* from constituents in the saliva.[57] The etchant should be carefully applied to avoid contact with the soft tissues. If not confined to the occlusal surface, the acid may produce a mild inflammatory response. It also produces a sharp acid taste that is often objectionable.

Pit-and-Fissure Depth

Deep, irregular pits and fissures offer a much more favorable surface contour for sealant retention compared with broad, shallow fossae (Figure 10-3). The deeper fissures protect the resin sealant from the shear forces occurring as a result of masticatory movements. Of parallel importance is the possibility of caries

development increasing as the *fissure depth and slope* of the inclined planes increases.[58,59] Thus, *as the potential for caries increases, so does the potential for sealant retention.*

Figure 10-3 An electron scanning microscope view of the deep pits and fissures of the occlusal surface of a molar. (Courtesy of Dr. A. J. Gwinnett, State University of New York, Stony Brook.)

Surface Cleanliness

The need and method for cleaning the tooth surface prior to sealant placement are controversial. Usually the acid etching alone is sufficient for surface cleaning. This is attested to by the fact that two of the most cited and most effective sealant longevity studies by Simonsen[60] and Mertz-Fairhurst[61] were accomplished without use of a prior prophylaxis. Recently, however, it was shown that cleaning teeth with the newer prophylaxis pastes with or without fluoride (NuPro, Topex) did not affect the bond strength of sealants,[62] composites,[63] or orthodontic brackets.

Other methods used to clean the tooth surface prior to placing the sealant included, air-polishing, hydrogen peroxide, and enameloplasty.[63-65] The use of an air-polisher has proven to thoroughly clean and removes residual debris from pits and fissures.[65-68] Hydrogen peroxide has the disadvantage that it produces a precipitate on the enamel surface.[68] Enameloplasty, achieved by bur or air abrasion has proven effective. Yet, no significant differences were observed in comparison with either etching or bur preparation of the fissures on the penetration to the base of the sealant. However, the use of enameloplasty, even if equal or slightly superior would have very serious ramifications. The laws of most states require a dentist to use air abrasion and/or to cut a tooth, a requirement that would severely curtail hygienists and assistants participation in office and school preventive dentistry programs.[69]

Whatever the cleaning preferences—either by acid etching or other methods—all heavy stains, deposits, debris, and plaque should be removed from the occlusal surface before applying the sealant.

Preparing the Tooth for Sealant Application

The preliminary steps for the light-activated and the autopolymerized resins are similar up to the time of application of the resin to the teeth. After the selected teeth are isolated, they are thoroughly dried for approximately *10 seconds.* The 10-second drying period can be mentally estimated by counting off the seconds—1,000, 2,000—until 10,000 has been reached. The liquid etchant is then placed on the tooth with a small resin sponge or cotton pledget held with cotton pliers. Traditionally, the etching solution is gently daubed, *not rubbed,* on the surface for *1 minute* for permanent teeth and for 11/2 *minutes* for deciduous teeth.[70,71] Other clinical studies, however, have shown that acid etching the enamel of both primary and permanent teeth for only 20 seconds produced similar sealant[70] and composite[72] retention as those etched for 1 and 11/2 minutes. Currently, *20 to 30 seconds* enamel-etching time is recommended. Alternatively, acid gels are applied with a supplied syringe and left undisturbed. Another 15 seconds of etching is indicated for fluorosed teeth to compensate for the greater acid resistance of the enamel. The etching period should be timed with a *clock.* At the end of the etching period, the aspirator tip is positioned

with the bevel interposed *between the cotton roll and the tooth.* For 10 seconds the water from the syringe is flowed over the occlusal surface and thence into the aspirator tip. Again, this 10-second period can be mentally counted. Care should be exercised to ensure that the aspirator tip is close enough to the tooth to prevent any water from reaching the cotton rolls, yet not so close that it diverts the stream of water directly into the aspirator (see Figure 10-5).

Following the water flush, the tooth surface is dried for *10 seconds.* The air supply needs to be absolutely dry. The dried tooth surface should have a white, dull, frosty appearance. This is because the etching will remove approximately 5 to 10 um of the original surface,[73] although at times interrod penetrations of up to 100 um may occur.[74] The etching *does not always* involve the interrod areas; sometimes the central portion of the rod is etched, and the periphery is unaffected. The pattern on any one tooth is unpredictable.[75] In any event, the surface area is greatly increased by the acid etch.

Figure 10-5 Showing position of aspirator tip between the bicuspid and cotton roll during flushing, A, and between water flow and cotton roll looped around second molar, B. *Complete* dryness of the cotton rolls can be maintained with this technique.

Question 2

Which of the following statements, if any, are correct?

A. Autopolymerizing sealants and light-cured sealants have approximately the same record for longevity.

B. A 40% phosphoric acid etchant should be satisfactory for both etching and cleaning the average tooth surface prior to sealant placement.

C. Fossae with deep inclined planes tend to have more carious fissures; fossae with deep inclined planes tend to retain sealants better.

D. In studies in which a rubber dam was used to maintain a dry field for sealant placement, the retention of sealants was greater than when cotton rolls were used.

E. In placing a sealant, 10 seconds are devoted to each of the drying and etching phases and 1 minute to the flushing of the etchant from the tooth.

Dryness

The teeth *must* be dry at the time of sealant placement because sealants are hydrophobic. The presence of saliva on the tooth is even more detrimental than water because its organic components interpose a barrier between the tooth and the sealant. Whenever the teeth are dried with an air syringe, the air stream should be *checked* to ensure that it is not moisture-laden. Otherwise, sufficient moisture sprayed on the tooth will prevent adhesion of the sealant to the enamel. A check for moisture can be accomplished by directing the air stream onto a cool mouth mirror; any fogging indicates the presence of moisture. Possibly the omission of this simple step accounts for the inter-operator variability in the retention of fissure sealants.

A dry field can be maintained in several ways, including use of a *rubber dam*, employment of *cotton rolls,* and the placement of *bibulous pads* over the opening of the parotid duct. The rubber dam provides an ideal way to maintain dryness for an extended time. Because a rubber dam is usually employed in accomplishing quadrant dentistry, sealant placement for the quadrant should also be accomplished during the operation. Under most operating conditions, however, it is not feasible to apply the dam to the different quadrants of the mouth; instead it is necessary to employ cotton rolls, combined with the use of an effective *high-volume, low-vacuum aspirator.* Under such routine operating conditions, cotton rolls, with and without the use of bibulous pads, can usually be employed as effectively as the dam for the relatively short time needed for the procedure. *The two most successful sealant studies have used cotton rolls for isolation.*[60,61] In one study in which retention was tested using a rubber dam versus cotton rolls, the sealant retention was approximately *equal.*[76] Others have shown excellent sealant retention after 3 years[77] and after 10 to 20 years.[60,78]

In programs with *high patient volume* where cotton rolls are used, it is best to have two individuals involved—the *operator,* whose main task is to prepare the tooth and to apply the sealant, and the *assistant,* whose task is to maintain dryness. An operator working alone, however, can maintain a maximum dry field for the time needed to place the sealants, although it is not recommended, particularly for young children or those that are difficult to manage. For the maxilla, there should be little problem with the placement of *cotton rolls* in the buccal vestibule and, if desirable, the placement of a *bibulous pad* over the parotid duct. For the mandible, a 5-inch segment of a 6-inch cotton roll should be looped around the last molar and then held in place by the patient using the index and third fingers of the opposite hand from the side being worked on (Figure 10-4). With aid from the patient and with appropriate aspiration techniques, the cotton rolls can usually be kept dry throughout the entire procedure. Cotton roll holders may be used, but they can be cumbersome when using the aspirator or when attempting to manipulate or remove a roll. If a cotton roll does become *slightly* moist, many times another short cotton roll can be placed on top of the moist segment and held in place for the duration of the procedure. In the event that it becomes necessary to replace a wet cotton roll, it is essential that *no* saliva contacts the etched tooth surface; if there is *any* doubt, it is necessary to repeat all procedures up to the time the dry field was compromised. This includes a 15-second etch to remove any residual saliva, in lieu of the original 1-minute etch.

Another promising dry-field isolating device that can be used for single operator use, especially when used with cotton rolls, is by using ejector moisture-control systems.[a] In one study comparing the Vac-Ejector versus the cotton roll for maintaining dryness, the two were found to be equally effective.[79]

[a]Whaledent International, New York, NY

Figure 10-4 Four-handed dentistry with no assistant. The patient holds the cotton rolls with the index and third finger, thumb under chin. Patient also holds aspirator with other hand when it is not being used by operator.

Application of the Sealant

With either the light-cured or autopolymerized sealants, the material should first be placed in the fissures where there is the maximum depth. At times penetration of the fissure is negated by the presence of debris, air entrapment, narrow orifices, and excessive viscosity of the sealant.[80] The sealant should not only fill the fissures but should have some *bulk over the fissure.* After the fissures are adequately covered, the material is then brought to a knife edge approximately *halfway* up the inclined plane.

Following polymerization, the sealants should be examined carefully *before* discontinuing the dry field. If any voids are evident, additional sealant can be added *without* the need for any additional etching. The hardened sealant has an oil residue on the surface. This is unreacted monomer that can be either wiped off with a gauze sponge or can be left. If a sealant requires repair at any time after the dry field is discontinued, it is prudent to repeat the same etching and drying procedures as initially used. Because all the commercial sealants—both the light-cured and self-cured—are of the same Bis-GMA chemical family, *they easily bond to one another*.[81]

Occlusal and Interproximal Discrepancies

At times an excess of sealant may be inadvertently flowed into a fossa or into the adjoining interproximal spaces. To remedy the first problem, the occlusion should be checked visually or, if indicated, with articulating paper. Usually *any minor* discrepancies in occlusion are rapidly removed by normal chewing action. If the premature contact of the occlusal contact is unacceptable, a large, *no. 8. round cutting* bur may be used to rapidly create a broad resin fossa.

The integrity of the interproximal spaces can be checked with the use of dental floss. If any sealant is present, the use of scalers may be required to accomplish removal. These corrective actions are rarely needed once proficiency of placement is attained.

Question 3

Which of the following statements, if any, are correct?

A. The etchant *predictably* attacks the center of the enamel prism, leaving the periphery intact.

B. When the data of a study indicate that 65% of the original sealants are retained for 7 years, it is the same as saying that an average of 5% is lost each year.

C. Bis-GMA products by different manufacturers are incompatible with one another.

D. An etched area that is not rapidly sealed will retain its rough, porous surface *indefinitely.*

E. The cleansing and etching of the occlusal surface with phospohoric acid is accomplished by *rubbing* the surface during the etching process.

Evaluating Retention of Sealants

The finished sealant should be checked for retention without using undue force. In the

event that the sealant does not adhere, the placement procedures should be repeated, with only about 15 seconds of etching needed to remove the residual saliva before again flushing, drying, and applying the sealant. If *two* attempts are unsuccessful, the sealant application should be postponed until remineralization occurs.

Resin sealants are retained better on recently erupted teeth than in teeth with a more mature surface; they are retained better on first molars than on second molars. They are better retained on mandibular than on maxillary teeth. This latter finding is possibly caused by the lower teeth being more accessible, direct sight is also possible; also, gravity aids the flow of the sealant into the fissures.[41]

Teeth that have been sealed and then have lost the sealant have had fewer lesions than control teeth.[82] This is possibly due to the presence of tags that are *retained in the enamel* after the bulk of the sealant has been sheared from the tooth surface. When the resin sealant flows over the prepared surface, it penetrates the finger-like depressions created by the etching solution. These projections of resin into the etched areas are called *tags.*[83] (Figure 10-6). The tags are essential for retention. Scanning electron microscopic studies of sealants that have not been retained have demonstrated large areas devoid of tags or incomplete tags, usually caused by saliva contamination. If a sealant is forcefully separated from the tooth by masticatory pressures, many of these tags are *retained* in the etched depressions.

The number of retained sealants decreases at a *curvolinear rate.*[41] Over the first 3 months, the rapid loss of sealants is probably caused by *faulty technique* in placement. The fallout rate then begins to plateau, with the ensuing sealant losses probably being due to abnormal *masticatory stresses.* After a year or so, the sealants become very difficult to see or to discern tactilely, especially if they are abraded to the point that they fill only the fissures. In research studies this lack of visibility often leads to *underestimating* the effectiveness of the sealants that remain but cannot be identified. Because the most rapid falloff of sealants occurs in the early stages, an initial 3-month recall following placement should be routine for determining if sealants have been lost. If so, the teeth should be resealed. Teeth successfully sealed for 6 to 7 years are likely to remain sealed.[83]

In a review of the literature, longest-term study reported that at the follow-up examination of the first molars, 20-years after sealant had been applied, 65% showed *complete* retention and 27% partial retention *without* caries. At a 15-year follow-up of the same sealants the second molars demonstrated the corresponding figures 65% and 30%, respectively. This study showed that pit-and-fissure sealants applied during childhood have a *long-lasting, caries preventive effect.*[60,77] Mertz-Fairhurst[83] cited studies in which 90 to 100% of the original sealants were retained over a 1-year period (Table 10-1). One 10-year study using 3M Concise Sealant had a 57% complete retention and a 21% partial retention of sealant, *all with no caries.* Another study, using Delton, registered 68% retention after 6 years.[108] (Figure 10-7). These are studies in which the sealant was placed and then observed at periodic intervals; there was no resealing when a sealant was lost. *Where resealing is accomplished as needed at recall appointments, a higher and more continuous level of protection is achieved.* More recent studies report 82% of the sealants placed are retained for 5 years.[70]

Figure 10-6 Tags, 30 um. Sealant was flowed over etched surface, allowed to polylmerize, and tooth surface subsequently dissolved away in acid. (Courtesy,

Silverstone LM, Dogon IL. *The Acid Etch Technique.* St. Paul, MN: North Central Publishing Co, 1975.)

Figure 10-7 A: 5-year sealant: Five years after placement of a white pit-and-fissure sealant in the matched pair to the control subject. Sealant and control subjects were matched on age, sex, caries history and other factors. B: 5-year control: This matched pair to the sealed patient. This subject did not receive sealant. The first permanent molar has already been restored with two amalgam restorations in the previous 5-year period. C: 15-year sealant: 15 years after the single application of a white pit-and-fissure sealant. This is the same tooth as seen in Figure 1, 5-year sealant, but 10 years later. As can be seen, the sealant has served its purpose even though there has been some loss in the peripheral fissures. (Courtesy of Dr. Richard J. Simonsen, D.)

Colored Versus Clear Sealants

Both clear and colored sealants are available. They vary from translucent to white, yellow, and pink. Some manufacturers sell both clear and colored sealants in either the light-curing or autopolymerizing form. The selection of a colored versus a clear sealant is a matter of individual preference. The colored products permit a *more precise placement* of the sealant, with the visual assurance that the periphery extends halfway up the inclined planes. *Retention can be more accurately monitored* by both the patient and the operator placing the sealant. On the other hand, a clear sealant may be considered more *esthetically* acceptable.

Some clinicians prefer the clear sealants because they are more discrete than white. Others prefer the white sealants as they are easier to monitor at recall appointments. On the other hand, some clinicians seem to prefer the clear sealants because it is possible to see under the sealant if a carious lesion is active or advancing. However, no clinical study has comprehensively compared these issues. Recently, some pit-and-fissure sealants have been introduced that will change color as they are being light-polymerized. This property has not been fully investigated and seems to be only of relative advantage to the dental personnel applying the sealant.

Placement of Sealants Over Carious Areas

Sealing over a carious lesion is important because of the professionals' concern about the possibility of caries progression under the sealant sites. In teeth that have been examined *in vivo* and later subjected to histologic examination following extraction for orthodontic reasons, it has been found that areas of incipient or overt caries often occur under many fissures, which *cannot* be detected with the explorer.[85] In some studies, sealants have been purposely placed over small, overt lesions.[83,86] When compared with control teeth, many of the sealed carious teeth have been diagnosed as sound 3 and 5 years later.[87] Handelman has indicated that sealants can be considered a viable modality for *arrest* of pit-and-fissure caries.[88] In other studies of sealed lesions, the number of bacteria recovered from the sealed area decreased rapidly.[33,34,86-89] This decrease in bacterial population is probably due to the integrity of the seal of the resin to the etched tooth surface[90] seal that does not permit the movement of fluids or tracer isotopes between the sealant and the tooth.[91]

Sealants have been placed over more extensive lesions in which carious dentin is

involved.[92] Even with these larger lesions, there is a decrease in the bacterial population and arrest of the carious process as a function of time. In another study, clinically detectable lesions into the dentin were covered for 5 years with Nuva-Seal. After that time the bacterial cultures were essentially negative, and an apparent 83% *reversal* from a caries-active to a caries-inactive state was achieved.[86] Jordan and Suzuki[93] sealed small lesions in 300 teeth. During clinical and x-ray observations over a 5-year period, they found *no change in size of the carious lesion*, so long as the sealant remained intact. More recently, Mertz-Fairhurst and colleagues[94] demonstrated that sealed lesions became *inactive* bacteriologically, with the residual carious material suggesting decay cessation. This ability to arrest incipient and early lesions is highlighted by the statement in the 1979 publication of the ADA's Council on Dental Therapeutics: "Studies indicate that there is an apparent reduction in microorganisms in infected dentin covered with sealant. . .. These studies appear to substantiate that there is no hazard in sealing carious lesions." The statements end with the *cautionary* note: "However, additional long-term studies are required before this procedure can be evaluated as an alternative to traditional restorative procedures.[95] When sealing incipient lesions, care should be taken to monitor their retention at subsequent recall/annual dental examinations. In addition, there have been reports of sealants being used to achieve penetration of incipient smooth-surface lesions ("white spots") of facial surfaces."[96]

Question 4

Which of the following statements, if any, are correct?

A. Tags can be easily determined by their rough feel when checking the *surface* of a sealant with an explorer.

B. Teeth that lose a sealant are more susceptible to caries than ones that retain a sealant but less caries-prone than a control tooth that was never sealed.

C. The falloff of sealants is *linear* as a function of time.

D. A study in which the periodic resealing of fissures occurs would be expected to have a *lesser* caries rate than a long-term study in which the same annual falloff is experienced, but where no resealing is accomplished.

E. Following placement of a sealant over a fissure with an undetectable carious lesion, the size of the subsurface lesion gradually *increases.*

Sealants Versus Amalgams

Comparing sealants and amalgams is not an equitable comparison because sealants are used to *prevent* occlusal lesions, and amalgam is used to *treat* occlusal lesions that could have been prevented. Yet, the comparison is necessary. One of the major obstacles to more extensive use of sealants has been the belief that amalgams, and not sealants, should be placed in anatomically defective fissures; this belief stems from *misinformation* that amalgams can be placed in less time, and that once placed, they are a permanent restoration. Several studies have addressed these suppositions. For instance, sealants require approximately 6 to 9 minutes to place initially, amalgams 13

to 15 minutes.[97,98]

Many studies on *amalgam* restorations have indicated a *longevity* from only a few years to an average life span of 10 years.[99-102] Equally perturbing is the fact that in one large study of schoolchildren, 16.2% of all surfaces filled with amalgam had marginal leakage and *needed replacement.*[103] The life span of an amalgam is shorter with younger children than with adults.[104] To emphasize the problem of replacement of older restorations, a recent questionnaire study from 91 dentists in Iceland was conducted to determine the cause for replacement of 8,395 restorations. The reason given for the replacement of composites, amalgams, glass-ionomers, and for resin modified glass ionomers was failed restorations (47.2%), primary caries (45.3%) and non-carious defects (7.5%). For every restoration inserted for an overt lesion, there was a need for one to be reinserted previously![105]

The retention data from the earlier sealant studies were discouraging. In recent years, using later-generation sealants, along with the *greater care in technique* used for their insertion, much longer retention periods have been reported. In five long-term studies from 3 to 7 years, the average sealant loss per year ranged from 1.3 to 7%.[106] If the yearly loss of these studies is extrapolated, the average life of these sealants compares favorably or exceeds that of amalgam.[107] When properly placed, sealants are no longer a temporary expedient for prevention; instead, they are the *only effective predictable* clinical procedure available for preventing occlusal caries.

The most frequent cause for sealant replacement is *loss of material,* which mainly occurs during the first 6 months; the most likely cause for amalgam replacement is *marginal decay,*[108] with 4 to 8 years being the average life span.[103] To replace the sealant, only resealing is necessary. No damage occurs to the tooth. Amalgam replacement usually requires cutting more tooth structure with each replacement. Even if longevity merits were equal, the sealant has the advantage of being painless to apply and aesthetic, as well as emphasizing the *highest objectives* of the dental profession—*prevention and sound teeth.*

Options for Protecting the Occlusal Surfaces

The use of sealants has spawned an entirely different concept of conservation of occlusal tooth structure in the management of deep pits and fissures before, or early in caries involvement. The *preventive dentistry restoration* embodies the concepts of both prophylactic odontotomy insertion of a restoration and *covering the restoration and the connecting fissure system with a resin based sealant.* Pain and apprehension are slight, and aesthetics and tooth conservation are maximized.[108] Several options are now available to protect the occlusal surfaces, with the selection depending *on risk and professional's judgment.*[109] The first level of protection is simply to place a conventional sealant over the occlusal fissure system. This sealing preempts future pit-and-fissure caries, as well as arrests incipient or reverses small overt lesions.

The second option reported by Simonsen in 1978,[110] advocated the use of the *smallest* bur to remove the carious material from the bottom of a pit or fissure and then using an appropriate instrument to tease *either sealant or composite* into the cavity preparation. This he termed a preventive dentistry restoration. Following insertion of the restoration, sealant was placed *over* the polymerized material as well as flowed

over the remaining fissure system. Aside from protecting the fissures from future caries, it also protects the composite or inserted sealant from abrasion.[111]

The third option is use of glass-ionomers material for sealants, which is controversial. Due to their fluoride release and cariostatic effect, glass-ionomers have been used in place of traditional materials, as a pit-and-fissure sealant, however, resin sealants have shown much higher bond strength to enamel than glass-ionomers. Clinical trials[112,113] have shown poor retention over periods as short as 6 to 12 months. Though, in vitro studies have suggested that etching previous to application enhances the bonding of glass-ionomer sealant in fissure enamel.[114-116] One study showed that a conventional silver-reinforced glass-ionomer had superior clinical performance compared to a conventional resin sealant.[117]

Resin-reinforced glass-ionomer cements have been investigated for their effectiveness as pit-and-fissure sealants. The 1-year results revealed that although clinically the glass-ionomer wears at a faster rate than a conventional resin sealant, in the scanning electron microscopic evaluation the material could be seen at the deep recesses of the pits-and-fissures with no carious lesion present.[113] A recent study showed that after 3 years the glass-ionomer sealant was completely lost in almost 90% of the teeth compared to less than 10% of the resin sealed teeth; the relative risk of a tooth sealed with glass-ionomer over that of a tooth sealed with resin was higher. Also, the glass-ionomer sealant had poorer retention and less caries protective effect.[118]

Glass-ionomer does not carry the ADA seal of approval as sealant material. The readers should decide their personal philosophy based on the evidence.

A fourth option reported by Garcia-Godoy in 1986 involves the use of a glass-ionomer cement as the *preventive glass-ionomer restoration* (PGIR).[119] The glass-ionomer cement (conventional or resin-modified) is placed only in the cavity preparation. (Figure 10-8). The occlusal surface is then etched with a gel etchant avoiding, if possible, etching the glass-ionomer. Etching the glass-ionomer may remove some of the glass particles weakening the material. The conventional resin sealant is placed *over the glass-ionomer and the entire occlusal fissure system.* In the event sealant is lost, the fluoride content of the glass-ionomer *helps prevent* future primary and *secondary* caries formation. The same technique has successfully protected the marginal integrity of very small amalgam restoration, as well as providing a protection to the entire fissure system.

Each of these options requires a judgment decision by the clinician. That decision can well be based on the criterion that if an overt lesion cannot be *visualized,* it should be sealed; if it can be visualized, the smallest possible preventive dentistry restoration should be used along with its required sealant "topping." Mertz-Fairhurst and associates[120] have pointed out that the first option could provide the preferred model for conservative treatment of *incipient* and *small overt,* pit-and-fissure caries. It could also serve as an interim treatment for larger lesions. These options would be especially valuable in areas of the world with insufficient professional dental personnel and where preventive dental auxiliaries have been trained to place sealants. In all cases, the preventive dental filling should be considered as an alternative to the traditional class I amalgam with its accompanying extension for prevention that often includes the entire fissure system.

Figure 10-8 Preventive glass ionomer restoration (PGIR). Cavity preparation for reception of glass-ionomer cement. (Courtesy of Dr. Franklin Garcia-Godoy, University of Texas Dental School, San Antonio.)

The Sealant as Part of a Total Preventive Package

The sealant is used to protect the occlusal surfaces. A major effort should be made to incorporate the use of sealants along with other primary preventive dentistry procedures, such as plaque control, fluoride therapy, and sugar discipline. Whenever a sealant is placed, a topical application of fluoride should follow if at all possible. In this manner the whole tooth can be protected. Ripa and colleagues[121] completed a 2-year study for children in second and third grades assessing the effectiveness of a 0.2% fluoride mouthrinse used alone compared with a rinse plus sealants. Twenty-four occlusal lesions developed in the 51 rinse subjects, and *only* 3 in the 84 subjects receiving the rinse plus sealants. The conclusion was that caries could be *almost completely eliminated* by the *combined* use of these two preventive procedures. In many public-health programs, however, it is not possible to institute full-scale prevention programs, either because of apathy or lack of time and money. In such cases, there is some consolation in knowing that at least the *most vulnerable* of all tooth surfaces (the occlusal) is being protected.

Manpower

The *cost* of sealant placement *increases* directly with the level of professional education of the operator. Dentists, hygienists, assistants, and other auxiliaries can be trained to place sealants.[122-124] In view of the cost-effectiveness, dental auxiliaries should be considered as the logical individuals to place sealants. This is important if manpower is to be increased.

Often auxiliaries who have received sealant instruction, either through continuing-education courses or as part of a curriculum, are stymied either because of state laws interdicting their placing sealants or by the nature and philosophy of the practice of the employing dentist.[125] Only fourteen states allow hygienists to practice under less restrictive or unsupervised practice models in which they can initiate treatment based on assessment of patient, treat the patient, and maintain a provider-patient relationship without the participation of the patients' dentist of record. For example, Maine and New Hampshire have a separate supervision for settings outside of the dental office public-health supervision, which is less restrictive than general supervision. New Mexico allows for a collaborative-practice agreement between dentists and hygienists in outside settings. Yet, in states such as Georgia and Illinois, hygienists are required to practice under direct supervision. This means the dentist must be present in the office while the care is being provided.[126]

In a Swedish study, 77 *dental assistants* working in 12 dental clinics sealed 3,218 first and second molars with a 5-year retention rate of between 74 and 94%.[127] Because many dentists consider the placement of sealants to be a relatively simple procedure, few are returning for continuing-education programs to learn the exacting and precise process necessary to ensure maximum sealant retention. Even when the dental professionals desire to participate in such continuing education, a survey found relatively few courses available.[128]

Economics

Bear in mind that not every tooth receiving a sealant would necessarily become carious; hence the cost of preventing a single carious lesion is greater than the cost of a single sealant application. For instance, Leverett and colleagues calculated that five sealants would need to be placed on sound teeth to prevent one lesion over a 5-year period,[129] and Rock and Anderson estimated one tooth for every three sealant applications are prevented from becoming carious.[130] Sealants would be most cost-effective if they could be placed in only those pits and fissures that are destined to become carious. Unfortunately, we do not have a caries predictor test of such exactitude, but, the use of vision plus an economic, portable electronic device that objectively measures conductance (or resistance) would greatly aid in evaluating occlusal risk.[131] Without such a device, it is necessary to rely on professional judgment, based on the severity of the caries activity indicators: number of "sticky" fissures, level of plaque index, number of incipient and overt lesions, and microbiologic test indications.

In an office setting, it is estimated that it costs 1.6 times more to treat a tooth than to seal.[55] The Task Force on Community Preventive Services, an independent, non-federal group formed to evaluated oral-health interventions, was charged with determining interventions that promote and improve oral health. The Task Force examined six public-health programs cost of placing pit-and-fissure sealants revealing a mean cost of $39.10 per person.[132] However, even these numbers are misleading. For instance, what is the value of an intact tooth to its owner? How much does it cost for a dentist and assistant to restore a tooth, compared to the cost of sealing a tooth? Later in life, what is the cost of bridges and dentures that had their genesis when children were at high risk with little access to dental care?

Use of Pit-and-Fissure Sealants

By the mid-1980s most of the answers were available as to the need and *effectiveness* of Bis-GMA sealants to reduce the incidence of occlusal caries, and the *techniques* of placement of pit-and-fissure sealants were known.[133] The *safety* of their placement has been demonstrated by many studies showing that even when placed over incipient and minimally overt caries sites, there was no progression *as long as the sealant remained intact.*[134] Finally, several clinical studies have pointed out that sealants could be *applied by properly trained auxiliaries,* thus providing a more economical source of manpower for private and military practices as well as for large school and public health programs.

Bis-GMA sealant usage has been strongly supported by the ADA "as a safe and effective means for caries control."[25] The United States Public Health Service, in a request for a proposal for a school pit-and-fissure study, stated *"This combination of preventive techniques (combined use of fluoride and sealants) is expected to essentially eliminate caries in teeth erupting after the initiation of the study."*[135] Despite the support from the two largest organizations most interested in the dental health of the nation, the rank-and-file of the dental profession *have not accepted sealants as a routine method for prevention.*

In spite of all the knowledge of the properties and successes of the sealants usage has lagged, with about 10% of the posterior teeth of children demonstrating the presence of sealants.[136] For example, a 1994 examination of 117,000 children in North Carolina between the ages of 6 and 17 found that approximately 12% had sealants,[137] while the percentage for 927,000 in Tennessee was 10%.[138] Other states demonstrate similar sealant usage. One study revealed that 88 children did have sealants while 508 did not have needed sealants.[139] For recruits entering the U.S. Air Force, sealants were found on 13.1% of the teeth while there was a need for 47.5% more. In the latter case, it was noted that a third of these personnel had occlusal caries that might have been prevented by the sealants.[20]

Many barriers exist in meeting the Healthy People 2010 Objective for sealants. In 2001, the State of Alabama was planning how to meet national dental objectives, when 50% of U.S. children are expected to have dental sealants on at least one permanent molar by the age of 14 years.[140] (Currently, 22% of the children between 12 to 14 years have at least one sealant claim.) A final assessment of the 2010 prospects and the current State's demographics concluded that racial and gender disparities, difficulty in accessing care, the nonavailability of Medicaid-participating dentists in a country, and a lower payment/claim ratio may make the national sealant objective difficult to achieve.[140] It should be mentioned that in many surveys, children from lower socioeconomic groups had greater sealant needs than those from more affluent neighborhoods.

On the other hand, other countries have had marked success with increasing the number of teeth sealed. A study involving 68,704 children living in Lanarkshire, Scotland found approximately 10% of the occlusal surfaces were sealed.[141] Five years later, in England the percentage of children *having sealants dramatically increased* between 20 to 50% in several areas.[142]

The placement of sealants is making slow progress. The 1998-99 Ohio State survey of 3rd-grade students in School Based/School Link programs found that in addition to oral-health benefits, "Providing sealant programs in all eligible, high-risk schools could reduce or eliminate racial and economic disparities in the prevalence of dental sealants".[143] Yet, there are problems in examining the number of sealants *versus* the *need* for sealants.

Dentist Involvement

Pit-and-fissure sealants are underused in private practice and public health. There are many complex reasons for such under use, but efforts should be undertaken to increase sealant use.[3] Increasing sealant use is dependent, in part, on dentists' acceptance and understanding of the preventive technique. In a mail survey in Minnesota, 95% of 375 dentists reported the use of sealants, varying from 1 to 25 per week. Possibly, the incongruity of numbers stems from the fact that although the majority of dentists use sealants, the *frequency* of use is *low*.[144]

Reasons for this apathy have ranged from alleged concerns of sealing over carious lesions, lack of technical skill, short longevity of sealants, and the need for more research—all problems that have been adequately addressed in the literature.[133] Probably the most important factor now restricting the placement of sealants is the

lack of an adequate insurance fee schedule.[145] Another is that most dentists are treatment-oriented. This fact is amplified by an explanation by Galarneau and Brodeur that "A dentists lack of comfort with withholding treatment may stop him/her from offering preventive care and cause him to follow a restoration-oriented practice."[146] Another factor is that dentists rarely explain the oral-health advantages of sealants over dental restorations.[147]

In attempting to alter the attitudes of dentist on sealant use, several studies have been conducted to measure *changes in knowledge and attitudes* following continuing-education courses. The follow-up indicated that there had been an increase in *knowledge* but little change in *attitudes* concerning sealant use.[148] In Colorado, pediatric dentists, who are continually involved in treating children, placed more sealants than general dentists—again, probably a manifestation of attitudes.[145]

Regardless of increased rhetoric about prevention, the concepts and actions of prevention are *not* being fully implemented in dental schools.[149] Dental school faculties need to be educated about the effectiveness and methods of applying sealants.[150,151] Possibly the development of a model curriculum for teaching pit-and-fissure sealant usage would help.[152] The dental community must develop a consensus about the value and economic effect of preventive measures.[150]

Other barriers to effective delivery of sealants include (1) state-board restrictions on auxiliary placement of sealants, (2) lack of consumer knowledge of the effectiveness of sealants, and, resultantly, a lack of demand for the product.[122] *The economics and education of the profession and of the public are the prime requisites for expanded sealant acceptance.*[153]

Question 5

Which of the following statements, if any, are correct?

A. The longevity expectation for a properly placed amalgam restoration is approximately twice that of a properly placed sealant.

B. Sealants should be placed only on permanent teeth of children up to age 16.

C. Sealants are found on approximately 54% of U.S. children.

D. Following the graduation of students presently in dental schools, a large increase in the use of sealants can be expected.

E. Caries does not progress under a properly sealed composite or amalgam.

Other Pit-and-Fissure Initiatives

The findings of the following studies must be considered an *important extension* of the present use of pit-and-fissure sealants, which are used to prevent the development of incipient lesions and to arrest minimal overt lesions. If professional judgment dictates, conservative sealed amalgams or composites could be used to maintain *marginal integrity, extend the longevity of the restorative materials, and for achieving*

a de facto extension for prevention without the need to remove sound tooth structure to extend the restoration over the entire fissure system. These two uses of resins for prevention and restorations without major operative considerations should be of great value in developing countries where professional manpower is at a minimum and the demand for dental care is great.

Probably the most important recent research on the use of Bis-GMA sealants and carious lesions were described by Mertz-Fairhurst and coworkers.[87,154] In the 10-year study,[154] patients with paired permanent molars or premolars with *obvious clinical and radiographic class I lesions* were selected. The carious lesions extended halfway into the dentin or to the nearest pulp horn. The randomized placement of restorations for each of the tooth pairs consisted of two of the following: (1) a *classic* amalgam restoration, complete with extension for prevention of all connecting fissures (79 subjects); (2) a *conservative* amalgam restoration involving only the carious site with a sealant "topping," the latter which was extended into the entire pit-and-fissure system (77 subjects); and (3) with each one of the amalgam restorations, a paired composite restoration placed over the carious tissue with a "topping" of sealant that included all the pits and fissures (156 subjects). In the preparation for the composite, *no attempt was made to remove the carious tissue.* A 1-millimeter wide, 40- to 60-degree bevel was made in the sound enamel surrounding the lesion. The area was washed, dried, and a bonding agent was placed on the bevel. Hand instruments were used to place the composite, after which rotary instruments were used to shape the occlusal anatomy. Following this step, the occlusal surface was treated as for the placement of the average sealant—dry, etch, rinse, and dry before placing the resin over the composite and the entire fissure system.

The conclusions of this study after 10 years were: (1) *both* the sealed composites and the sealed amalgam restorations exhibited *superior clinical performance and longevity* compared to the unsealed amalgam restorations; (2) bonded and sealed composite restorations placed over the frank cavitated lesions *arrested the clinical progress of these lesions for the 10 years of the study.*

Summary

The majority of all carious lesions that occur in the mouth occur on the occlusal surfaces. Which teeth will become carious cannot be predicted; however, if the surface is sealed with a pit-and-fissure sealant, no caries will develop as long as the sealant remains in place. Recent studies indicate an approximate 90% retention rate of sealants 1-year after placement. Even when sealants are eventually lost, most studies indicate that the caries incidence for teeth that have lost sealants is less than that of control surfaces that had never been sealed. Research data also indicate that many incipient and small overt lesions are arrested when sealed. Not one report has shown that caries developed in pits or fissures when under an intact sealant. Sealants are easy to apply, but the application of sealants is an extremely sensitive technique. The surfaces that are to receive the sealant must be completely isolated from the saliva during the entire procedure, and etching, flushing, and drying procedures must be timed to ensure adequate preparation of the surface for the sealant. Sealants are comparable to amalgam restorations for longevity and do not require the cutting of tooth structure. Sealants do not cost as much to place as amalgams. Despite their advantages, the use of sealants has not been embraced by all dentists, even though

endorsed by the ADA and the U.S. Public Health Service. Even when small overt pit-and-fissure lesions exist, they can be dealt with conservatively by use of preventive dentistry restorations. What now appears to be required is that the dental schools teach sealants as an effective intervention, that the dental professional use them, that the hygienists and the auxiliary personnel be permitted to apply them, and the public demand them.

Answers and Explanations

1. C and D—correct.

A—incorrect. Because the fluorides protect the smooth surface, there will be a greater proportion of pit-and-fissure lesions.

B—incorrect. By definition, an incipient lesion has not been invaded by bacteria; thus the use of a sealant is an ideal preventive measure.

E—incorrect. Remember, it is the caries susceptibility of the teeth that is important—not the age of the individual.

2. A, B, and C—correct.

D—incorrect. All the major, successful, long-term retention studies have used cotton-roll isolation; in the one study of rubber dam versus cotton rolls, the rolls were equal to, or better than, the dam.

E—incorrect. Ten seconds are used for the drying and flushing procedures, and 20 to 30 seconds for the etching.

3. A and B—correct.

C—incorrect. Bis-GMA plastics are of the same chemical family and will bond to each other regardless of manufacturer.

D—incorrect. Remineralization from saliva constituents occurs rapidly in a period of hours to days.

E—incorrect. Cleansing and etching do occur; however, rubbing tends to obliterate the delicate etching pattern and reduce retention potential.

4. B and D—correct.

A—incorrect. The tags of the sealant cannot be felt with the explorer; they extend into the enamel from the underneath side of the plastic.

C—incorrect. The curvolinear falloff is greatest at 3 months, less at 6 months, after which it gradually plateaus.

E—incorrect. The literature is unanimous that caries does not progress under an intact

sealant.

5. C and E—correct.

A—incorrect. There is little difference between the longevity of a well-placed amalgam compared with a well-placed sealant.

B—incorrect. If a tooth is susceptible to caries, it should be sealed, whatever the patient's age.

D—incorrect. All signs indicate that the teaching of sealant placement is greatly neglected in dental schools.

Self-evaluation Questions

1. Approximately _____ % of all carious lesions occur on the occlusal surfaces; the continual use of fluorides (increases) (decreases) this percentage.

2. Four different methods used prior to the advent of polyurethane, cyanoacrylate, and Bis-GMA sealants, were _____, _____, _____, and _____.

3. One condition that *indicates the use of a sealant is* _____; *four conditions that contraindicate* the use of sealants are _____, _____, _____, and _____; three conditions that *probably indicate* the use of sealants are _____, _____, and _____.

4. Two photoactivated, and two chemically activated sealants that have been accepted, or provisionally accepted, by the ADA are (photoactivated) _____, _____, and (chemically activated) _____ and _____.

5. The liquid resin in a sealant kit is known as the _____; when it is catalyzed the hardening process is known as _____. The catalyst used for the polymerization of chemically activated sealants is _____ and for visible photoactivation, _____.

6. Two advantages to light-cured sealants are _____ and _____; and two advantages of autopolymerized sealants are _____ and _____.

7. _____ forces, not chemical bonding, causes retention of the sealant to the tooth; the four commandments to ensure maximum retention are _____, _____, _____, and _____.

8. Three methods by which a dry field can be established are _____, _____, and _____.

9. The placement of sealants is extremely technique-sensitive; after selection of the tooth for sealant placement, it should be dried for _____ (time); then etched for _____ (time), followed by a water flush of _____ (time), and finally, dried for _____ (time) before placing the sealant.

10. Excessively high sealants that interfere with occlusion can be reduced by use of a number _____ (cutting) (finishing) bur.

11. The falloff of sealants is (linear) (curvilinear); long-term studies where 65% of the sealants are retained after 7 years indicate an average yearly loss of _____ %. After 10 years, _____ _____ % would be retained. This contracts to an average life expectancy of an amalgam of approximately _____ (years).

12. To protect the total tooth, the application of a sealant should be followed by an application of _____.

13. To ensure that sealant placement techniques have been perfected in dental and dental hygiene schools, it should be necessary for _____ (state dental-regulating agency) to require a demonstration of proficiency for all candidates prior to state licensure.

14. The three key components of a light source of polymerizing sealants are _____, _____, and _____ (which results in the blue color).

15. The three basic options for a preventive dentistry restoration are _____, _____, and _____.

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Objectives

At the end of this chapter, it will be possible to

1. Explain how sealants can provide a primary preventive means of reducing the need for operative treatment as 77% of the children 12 to 17 years old in the United States have dental caries in their permanent teeth.[1]

2. Discuss the history of sealant development through the 20th century.

3. List the criteria for selecting teeth for sealant placement and the four essentials in attaining maximum retention of sealants.

4. Describe the several steps preliminary to, during, and after the placement of a sealant—including surface cleanliness, dry fields, details of the application procedure, and remedial measures following the excess application of sealant.

5. Explain the rationale for adding fluorides to sealants.

6. Compare the advantages and disadvantages of light-cured and self-cured sealants.

7. Discuss the advantages of protecting the occlusal surfaces of teeth with sealants.

8. Cite five reasons given for the underuse of sealants by practitioners and analyze the validity of the reasons.

Introduction

Fluorides are highly effective in reducing the number of carious lesions occurring on the *smooth surfaces* of enamel and cementum. Unfortunately, fluorides are *not* equally effective in protecting the occlusal pits and fissures, where the majority of carious lesions occur.[2] Considering the fact that the occlusal surfaces constitute only 12% of the total number of tooth surfaces, it means that the *pits and fissures are approximately eight times as vulnerable as the smooth surfaces.* The placement of sealants is a highly effective means of preventing these.[3]

Historically several agents have been tried to protect deep pits and fissures on occlusal surfaces.

- In 1895, Wilson reported the placement of *dental cement* in pits and fissures to prevent caries.[2] In 1929, Bodecker[4] suggested that deep fissures could be broadened with a large round bur to make the occlusal areas more self-cleansing, a procedure that is called *enameloplasty*.[5] Two major disadvantages, however, accompany enameloplasty. First, it requires a dentist, which immediately limits its use. Second, in modifying a deep fissure by this method, it is often necessary to remove more sound tooth structure than would be required to insert a small restoration.
- In 1923 and again in 1936, Hyatt[6] advocated the early insertion of small restorations in deep pits and fissures before carious lesions had the opportunity to develop. He termed this procedure *prophylactic odontotomy*. Again, this operation is more of a treatment procedure than a preventive approach, because it requires a dentist for the

cutting of tooth structure.

- Several methods have been unsuccessfully used in an attempt either to seal or to make the fissures more resistant to caries. These attempts have included the use of topically applied zinc chloride and potassium ferrocyanide[7] and the use of ammoniacal silver nitrate;[8] they have also included the use of copper amalgam packed into the fissures.[9]
- Fluorides that protect the smooth surfaces of the teeth are less effective in protecting the occlusal surfaces.[10] Following the use of fluorides, there is a large reduction of incidence in smooth-surface caries but a smaller reduction in occlusal pit-and-fissure caries. This results in an *increased proportion* in the ratio of occlusal to interproximal lesions, even though the total number may be less.
- A final course of action to deal with pit-and-fissure caries is one that is often used: *do nothing; wait and watch.* This option avoids the need to cut good tooth structure until a definite carious lesion is identified. It also results in many teeth being lost when individuals do not return for periodic exams. This approach, although frequently used is a violation of the ethical principle of beneficence and patient autonomy.

In the late 1960s and early 1970s, another option became available—the use of pit-and-fissure sealants.[11] With this option, a liquid resin is flowed over the occlusal surface of the tooth where it penetrates the deep fissures to fill areas that cannot be cleaned with the toothbrush (Figure 10-1).[12] The hardened sealant presents a barrier between the tooth and the hostile oral environment. Concurrently, there is a significant reduction of Streptococcus mutans on the treated tooth surface.[13] Pits and fissures serve as reservoirs for mutans streptococci, sealing the niche thereby reduces the oral count.

Figure 10-1 One of the reasons that 50% of the carious lesions occur on the occlusal surface. Note that the toothbrush bristle has a greater diameter than the width of the fissure. (Courtesy of Dr. J. McCune, Johnson & Johnson.)

Criteria for Selecting Teeth for Sealant Placement

Following are the criteria for selecting teeth for sealing. Because no harm can occur from sealing, when in doubt, seal *and monitor*.

- A deep occlusal fissure, fossa, or incisal lingual pit is present.

A sealant is contraindicated if:

- Patient behavior does not permit use of adequate dry-field techniques throughout the procedure.
- An open carious lesion exists.
- Caries exist on other surfaces of the same tooth in which restoring will disrupt an intact sealant.
- A large occlusal restoration is already present.

A sealant is *probably indicated if:*

- The fossa selected for sealant placement is well isolated from another fossa with a restoration.

- The area selected is confined to a fully erupted fossa, even though the distal fossa is impossible to seal due to inadequate eruption.
- An intact occlusal surface is present where the *contralateral tooth* surface is carious or restored; this is because teeth on opposite sides of the mouth are usually equally prone to caries.
- An *incipient* lesion exists in the pit-and-fissure.
- Sealant material can be flowed over a conservative class I composite or amalgam to improve the marginal integrity, and into the remaining pits and fissures to achieve a *de facto* extension for prevention.

Other Considerations in Tooth Selection

All teeth meeting the previous criteria should be sealed and resealed as needed. Where the cost-benefit is critical and priorities must be established, such as occurs in many public health programs, ages 3 and 4 years are the most important times for sealing the eligible deciduous teeth; ages 6 to 7 years for the first permanent molars;[14] and ages 11 to 13 years for the second permanent molars and premolars.[15] Currently, 77% of the children 12-to-17-years-old in the United States have dental caries in their permanent teeth.[1] Many school days would be saved, and better dental health would be achieved in School Dental Health Clinic programs by combining sealant placement and regular fluoride exposure.[16]

The disease susceptibility of the tooth should be considered when selecting teeth for sealants, not the age of the individual. Sealants appear to be equally retained on occlusal surfaces in primary, as well as permanent teeth.[3] Sealants should be placed on the teeth of adults if there is evidence of existing or impending caries susceptibility, as would occur following excessive intake of sugar or as a result of a drug- or radiation-induced xerostomia. They should also be used in areas where fluoride levels in community water is optimized, as well as in non-fluoridated areas.[17]

The following are two good illustrations of this philosophy. After a 3-year study, Ripa and colleagues[18] concluded that the time the teeth had been in the mouth (some for 7 to 10 years) had no effect on the vulnerability of occlusal surfaces to caries attack. Also, the incidence of occlusal caries in young Navy[19] and Air Force[20] recruits (who are usually in their late teens or early 20s) is relatively high.

Background of Sealants

Buonocore first described the fundamental principles of placing sealants in the late 1960s.[10,21] He describes a method to bond poly-methylmethacrylate (PMMA) to human enamel conditioned with phosphoric acid. Practical use of this concept however, was not realized until the development of bisphenol A-glycidyl methacrylate (Bis-GMA), urethane dimethacrylates (UDMA) and trithylene glycol dimethacrylates (TEGDMA) resins that possess better physical properties than PMMA. The first successful use of resin sealants was reported by Buonocore in the 1960s.[22]

Bisphenol A-Glycidyl Methylacrylate Sealants

Bisphenol A-glycidyl methylacrylate (Bis-GMA) is now the sealant of choice. It is a

mixture of Bis-GMA and methyl methacrylate.[23] Products currently accepted by the American Dental Association (ADA) include:[24]

- Baritone L3, Type II Confi-Dental Products Co.
- Alpha-Dent Chemical Cure Pit and Fissure Sealant Dental Technologies, Inc.
- Alpha-Dent Light Cure Pit and Fissure Sealant Dental Technologies, Inc.
- Prisma-Shield Compules Tips VLC Tinted Pit & Fissure Sealant Dentsply L.D. Caulk Division
- Prisma-Shield VLC Filled Pit & Fissure Sealant Dentsply L.D. Caulk Division
- Helioseal F, Type II Ivoclar-Vivadent, Inc.
- Helioseal, Type II Ivoclar-Vivadent, Inc.
- Seal-Rite Low Viscosity, Type II Pulpdent Corp.
- Seal-Rite, Type II Pulpdent Corp.

The ADA National Standard sets aside specific criteria of pit-and-fissure sealants stating; Specification No. 39 established the following requirements:

- That the working time for type I sealants is not less than 45 seconds;
- That the setting time is within 30 seconds of the manufacturer's instruction and does not exceed three minutes;
- That the curing time for type II sealants is not more the 60 seconds;
- That the depth of cure for type II sealant is not less than 0.75 millimeter;
- That the uncured film thickness is not more than 0.1 millimeter;
- That sealants meet the bicompatibility requirements of American Nation a Standard/American Dental Association Document No. 41 for Recommended Standard Practices for Biological Evaluation of Dental Materials.[25]

Sealant products accepted by the American Dental Association carried the statement: "[Product name] has been shown to be acceptable as an agent for sealing off an anatomically deficient region of the tooth to supplement the regular professional care in a program of preventive dentistry."[26]

Nuva-Seal was the first successful commercial sealant to be placed on the market, in 1972. Since then more effective second- and third-generation sealants have become available see Table 10-1. The first sealant clinical trials used cyanoacrylate-based materials. Dimethacrylate-based products replaced these. The primary difference between sealants is their method of polymerization. First-generation sealants were initiated by ultraviolet light, second-generation sealants are autopolymerized, and third-generation sealants use visible light.

Some sealants contain *fillers,* which makes it desirable to classify the commercial products into *filled* and *unfilled* sealants. The *filled* sealants contain microscopic glass beads, quartz particles, and other fillers used in composite resins. The fillers are coated with products such as *silane,* to facilitate their combination with the Bis-GMA resin. The fillers make the sealant more *resistant to abrasion and wear.* Because they are more resistant to abrasion the occlusion should be checked and the sealant height may need to be adjusted after placement. In contrast, unfilled sealants wear quicker but usually do not need occlusal adjustment.

Fluoride-Releasing Sealants

The addition of fluoride to sealants was considered about 20 years ago,[27] and it was probably attempted based on the fact that the incidence and severity of secondary caries *was* reduced around fluoride-releasing materials such as the silicate cements used for anterior restorations.[28,29] Because fluoride uptake increases the enamel's resistance to caries,[30] the use of a fluoridated resin-based sealant may provide an additional anticariogenic effect if the fluoride released from its matrix is incorporated into the adjacent enamel.

Fluoride-releasing sealants have shown antibacterial properties[31-33] as well as a greater artificial caries resistance compared to a nonfluoridated sealant.[34-36] A recent *in vitro* study showed that pit-and-fissure sealants containing fluoride provided a caries-inhibiting effect with a significant reduction in lesion depth in the surface enamel adjacent and a reduction in the frequency of wall lesion.[37] Moreover, the fluoridated sealant laboratory bond strength to enamel,[38] and clinical performance,[39,40] is similar to that of nonfluoridated sealants.[41,42] In a recent study, it was shown that teeth sealed with Teethmate F fluoridated sealant revealed high amounts of enamel fluoride uptake in vitro and in vivo to a depth ranging from 10 to 20 um from the surface.[43] The residual fluoride was also observed within the sealing material. This agrees with another study showing the high amount of fluoride released from Teethmate F-1.[44]

The addition of fluoride to the sealants will greatly increase their value in the preventive and restorative use as mentioned above. Fluoride is added to sealants by two methods. The first is by adding a soluble fluoride to the unpolymerized resin. The fluoride can be expected to leach out over a period of time into the adjacent enamel. Eventually the fluoride content of the sealant should be exhausted, but the content of the enamel greatly increased.

The second method of incorporating fluoride is by the addition of an organic fluoride compound that is chemically bound to the resin to form an ion exchange resin. As such, when fluoride is low in the saliva, fluoride would be released. Vice versa, when the fluoride in the environment is high, it should bind to the resin to form—at least theoretically—a continuous reservoir for fluoride release and recharge.[45] See Table 10-2 on page 292 for a list of current available sealant materials.

Polymerization of the Sealants

The liquid resin is called the *monomer*. When the catalyst acts on the monomer, repeating chemical bonds begin to form, increasing in number and complexity as the hardening process (*polymerization*) proceeds. Finally, the resultant hard product is known as a polymer. Two methods have been employed to catalyze polymerization: (1) light curing by use of a visible blue light (synonyms: photocure, photoactivation, light activation) and (2) self-curing, in which a monomer and a catalyst are mixed together (synonyms: cold cure, autopolymerization, and chemical activation).

The two original Caulk products, Nuva-Seal and Nuva-Cote, were the only sealants in the United States requiring ultraviolet light for activation. Both have been *replaced* by other light-cured sealants that require *visible blue light.* In the manufacture of these latter products, a catalyst, such as *camphoroquinone,* which is sensitive to visible

blue-light frequencies, is placed in the monomer at the time of manufacture. Later, when the monomer is exposed to the visible blue light, polymerization is initiated.

With the autopolymerizing sealants, the catalyst is incorporated with the monomer; in addition, another bottle contains an *initiator*—usually *benzoyl peroxide.* When the monomer and the initiator are mixed, *polymerization* begins.

Light-Cured Versus Self-Cured Sealants

The main advantage of the light-cured sealant is that the operator can initiate polymerization at *any suitable time.* Polymerization time is shorter with the light-cured products than with the self-curing sealants. The light-cured process does require the purchase of a light source, which adds to the expense of the procedure. This light, however, is the same one that is used for polymerization of composite restorations, making it available in all dental offices. When using a light-cured sealant in the office, it is prudent to store the product away from bright office lighting, which can sometimes initiate polymerization.

Conversely, the self-curing resins do not require an expensive light source. They do, however, have the great disadvantage that once mixing has commenced, if some minor problem is experienced in the operating field, the operator must either continue mixing or stop and make a new mix. For the autopolymerizing resin, the time allowed for sealant manipulation and placement *must not be exceeded,* even though the material might still appear liquid. Once the hardening begins, *it occurs very rapidly, and any manipulation of the material during this critical time jeopardizes retention.*

The light-cured sealants have a higher compressive strength and a smoother surface;[46] which is probably caused by air being introduced into the self-cure resins during mixing[47] Despite these differences, both the photocured and the autopolymerizing products appear to be equal in retention.[43,48-50]

The High-Intensity Light Source

The light-emitting device consists of a high-intensity *white light,* a blue filter to produce the *desired blue color,* usually between 400 to 500 nm, and a light-conducting rod. Some other systems consist of a blue light produced by light-emitting diodes (LED) (Figure 10-2). Most have timers for automatically switching off the lights after a predetermined time interval. In use, the end of the rod is held only a few millimeters above the sealant during the first 10 seconds, after which it can be rested on the hardened surface of the partially polymerized sealant. The time required for polymerization is *set by the manufacturer* and is usually around *20 to 30* seconds. The *depth* of cure is influenced by the *intensity of light,* which can differ greatly with different products and length of exposure. Often it is desirable to set the automatic light timer for longer than the manufacturer's instructions.[51] Even after cessation of light exposure, a final, slow polymerization can *continue* over a 24-hour period.[52]

It is not known whether long-term exposure to the intense light can damage the eye. Staring at the lighted operating field is uncomfortable and does produce afterimages. This problem is circumvented by the use of a round, 4-inch dark-yellow disk, which fits over the light housing. The disk filters out the intense blue light in the 400- to

500-nanometers range as well as being sufficiently dark to subdue other light frequencies.

Figure 10-2 Light emitting diode (LED) curing unit for direct, intraoral exposure.

Question 1

Which of the following statements, if any, are correct?

A. In an area with fluoridated water, a *lower incidence* of caries can be expected, along with a *lower proportion* of occlusal to smooth-surface lesions.

B. Sealants should *never* be flowed over incipient caries.

C. Bis-GMA are the initials used to specify the chemical family of resins containing bisphenol A-glycidyl methyl-acrylate.

D. A monomer can polymerize, but a polymer cannot monomerize.

E. Sealants are contraindicated for adults.

Requisites for Sealant Retention

For sealant retention the surface of the tooth must (1) have a *maximum surface area,* (2) have *deep, irregular pits and fissures,* (3) be *clean,* and (4) be *absolutely dry* at the time of sealant placement and uncontaminated with saliva residue. *These are the four commandments for successful sealant placement, and they cannot be violated.*

Increasing the Surface Area

Sealants do not bond directly to the teeth. Instead, they are retained mainly by *adhesive forces.*[53] To increase the surface area, which in turn increases the adhesive potential, *tooth conditioners* (also called *etchants*), which are composed of a 30 to 50% concentration of phosphoric acid, are placed on the occlusal surface prior to the placement of the sealant.[54] The etchant may be either in *liquid* or *gel* form. The former is easier to apply and easier to remove. Both are equal in abetting retention.[55,56] If any etched areas on the tooth surface are not covered by the sealant or if the sealant is not retained, the normal appearance of the enamel returns to the tooth within 1 hour to a few weeks *due to a remineralization* from constituents in the saliva.[57] The etchant should be carefully applied to avoid contact with the soft tissues. If not confined to the occlusal surface, the acid may produce a mild inflammatory response. It also produces a sharp acid taste that is often objectionable.

Pit-and-Fissure Depth

Deep, irregular pits and fissures offer a much more favorable surface contour for sealant retention compared with broad, shallow fossae (Figure 10-3). The deeper fissures protect the resin sealant from the shear forces occurring as a result of masticatory movements. Of parallel importance is the possibility of caries development increasing as the *fissure depth and slope* of the inclined planes

increases.[58,59] Thus, *as the potential for caries increases, so does the potential for sealant retention.*

Figure 10-3 An electron scanning microscope view of the deep pits and fissures of the occlusal surface of a molar. (Courtesy of Dr. A. J. Gwinnett, State University of New York, Stony Brook.)

Surface Cleanliness

The need and method for cleaning the tooth surface prior to sealant placement are controversial. Usually the acid etching alone is sufficient for surface cleaning. This is attested to by the fact that two of the most cited and most effective sealant longevity studies by Simonsen[60] and Mertz-Fairhurst[61] were accomplished without use of a prior prophylaxis. Recently, however, it was shown that cleaning teeth with the newer prophylaxis pastes with or without fluoride (NuPro, Topex) did not affect the bond strength of sealants,[62] composites,[63] or orthodontic brackets.

Other methods used to clean the tooth surface prior to placing the sealant included, air-polishing, hydrogen peroxide, and enameloplasty.[63-65] The use of an air-polisher has proven to thoroughly clean and removes residual debris from pits and fissures.[65-68] Hydrogen peroxide has the disadvantage that it produces a precipitate on the enamel surface.[68] Enameloplasty, achieved by bur or air abrasion has proven effective. Yet, no significant differences were observed in comparison with either etching or bur preparation of the fissures on the penetration to the base of the sealant. However, the use of enameloplasty, even if equal or slightly superior would have very serious ramifications. The laws of most states require a dentist to use air abrasion and/or to cut a tooth, a requirement that would severely curtail hygienists and assistants participation in office and school preventive dentistry programs.[69]

Whatever the cleaning preferences—either by acid etching or other methods—all heavy stains, deposits, debris, and plaque should be removed from the occlusal surface before applying the sealant.

Preparing the Tooth for Sealant Application

The preliminary steps for the light-activated and the autopolymerized resins are similar up to the time of application of the resin to the teeth. After the selected teeth are isolated, they are thoroughly dried for approximately *10 seconds.* The 10-second drying period can be mentally estimated by counting off the seconds—1,000, 2,000—until 10,000 has been reached. The liquid etchant is then placed on the tooth with a small resin sponge or cotton pledget held with cotton pliers. Traditionally, the etching solution is gently daubed, *not rubbed,* on the surface for *1 minute* for permanent teeth and for 11/2 *minutes* for deciduous teeth.[70,71] Other clinical studies, however, have shown that acid etching the enamel of both primary and permanent teeth for only 20 seconds produced similar sealant[70] and composite[72] retention as those etched for 1 and 11/2 minutes. Currently, *20 to 30 seconds* enamel-etching time is recommended. Alternatively, acid gels are applied with a supplied syringe and left undisturbed. Another 15 seconds of etching is indicated for fluorosed teeth to compensate for the greater acid resistance of the enamel. The etching period should be timed with a *clock.* At the end of the etching period, the aspirator tip is positioned with the bevel interposed *between the cotton roll and the tooth.* For 10 seconds the

water from the syringe is flowed over the occlusal surface and thence into the aspirator tip. Again, this 10-second period can be mentally counted. Care should be exercised to ensure that the aspirator tip is close enough to the tooth to prevent any water from reaching the cotton rolls, yet not so close that it diverts the stream of water directly into the aspirator (see Figure 10-5).

Following the water flush, the tooth surface is dried for *10 seconds*. The air supply needs to be absolutely dry. The dried tooth surface should have a white, dull, frosty appearance. This is because the etching will remove approximately 5 to 10 um of the original surface,[73] although at times interrod penetrations of up to 100 um may occur.[74] The etching *does not always* involve the interrod areas; sometimes the central portion of the rod is etched, and the periphery is unaffected. The pattern on any one tooth is unpredictable.[75] In any event, the surface area is greatly increased by the acid etch.

Figure 10-5 Showing position of aspirator tip between the bicuspid and cotton roll during flushing, A, and between water flow and cotton roll looped around second molar, B. *Complete* dryness of the cotton rolls can be maintained with this technique.

Question 2

Which of the following statements, if any, are correct?

A. Autopolymerizing sealants and light-cured sealants have approximately the same record for longevity.

B. A 40% phosphoric acid etchant should be satisfactory for both etching and cleaning the average tooth surface prior to sealant placement.

C. Fossae with deep inclined planes tend to have more carious fissures; fossae with deep inclined planes tend to retain sealants better.

D. In studies in which a rubber dam was used to maintain a dry field for sealant placement, the retention of sealants was greater than when cotton rolls were used.

E. In placing a sealant, 10 seconds are devoted to each of the drying and etching phases and 1 minute to the flushing of the etchant from the tooth.

Dryness

The teeth *must* be dry at the time of sealant placement because sealants are hydrophobic. The presence of saliva on the tooth is even more detrimental than water because its organic components interpose a barrier between the tooth and the sealant. Whenever the teeth are dried with an air syringe, the air stream should be *checked* to ensure that it is not moisture-laden. Otherwise, sufficient moisture sprayed on the tooth will prevent adhesion of the sealant to the enamel. A check for moisture can be accomplished by directing the air stream onto a cool mouth mirror; any fogging indicates the presence of moisture. Possibly the omission of this simple step accounts for the inter-operator variability in the retention of fissure sealants.

A dry field can be maintained in several ways, including use of a *rubber dam,* employment of *cotton rolls,* and the placement of *bibulous pads* over the opening of the parotid duct. The rubber dam provides an ideal way to maintain dryness for an extended time. Because a rubber dam is usually employed in accomplishing quadrant dentistry, sealant placement for the quadrant should also be accomplished during the operation. Under most operating conditions, however, it is not feasible to apply the dam to the different quadrants of the mouth; instead it is necessary to employ cotton rolls, combined with the use of an effective *high-volume, low-vacuum aspirator.* Under such routine operating conditions, cotton rolls, with and without the use of bibulous pads, can usually be employed as effectively as the dam for the relatively short time needed for the procedure. *The two most successful sealant studies have used cotton rolls for isolation.*[60,61] In one study in which retention was tested using a rubber dam versus cotton rolls, the sealant retention was approximately *equal.*[76] Others have shown excellent sealant retention after 3 years[77] and after 10 to 20 years.[60,78]

In programs with *high patient volume* where cotton rolls are used, it is best to have two individuals involved—the *operator,* whose main task is to prepare the tooth and to apply the sealant, and the *assistant,* whose task is to maintain dryness. An operator working alone, however, can maintain a maximum dry field for the time needed to place the sealants, although it is not recommended, particularly for young children or those that are difficult to manage. For the maxilla, there should be little problem with the placement of *cotton rolls* in the buccal vestibule and, if desirable, the placement of a *bibulous pad* over the parotid duct. For the mandible, a 5-inch segment of a 6-inch cotton roll should be looped around the last molar and then held in place by the patient using the index and third fingers of the opposite hand from the side being worked on (Figure 10-4). With aid from the patient and with appropriate aspiration techniques, the cotton rolls can usually be kept dry throughout the entire procedure. Cotton roll holders may be used, but they can be cumbersome when using the aspirator or when attempting to manipulate or remove a roll. If a cotton roll does become *slightly* moist, many times another short cotton roll can be placed on top of the moist segment and held in place for the duration of the procedure. In the event that it becomes necessary to replace a wet cotton roll, it is essential that *no* saliva contacts the etched tooth surface; if there is *any* doubt, it is necessary to repeat all procedures up to the time the dry field was compromised. This includes a 15-second etch to remove any residual saliva, in lieu of the original 1-minute etch.

Another promising dry-field isolating device that can be used for single operator use, especially when used with cotton rolls, is by using ejector moisture-control systems.[a] In one study comparing the Vac-Ejector versus the cotton roll for maintaining dryness, the two were found to be equally effective.[79]

[a]Whaledent International, New York, NY

Figure 10-4 Four-handed dentistry with no assistant. The patient holds the cotton rolls with the index and third finger, thumb under chin. Patient also holds aspirator with other hand when it is not being used by operator.

Application of the Sealant

With either the light-cured or autopolymerized sealants, the material should first be

placed in the fissures where there is the maximum depth. At times penetration of the fissure is negated by the presence of debris, air entrapment, narrow orifices, and excessive viscosity of the sealant.[80] The sealant should not only fill the fissures but should have some *bulk over the fissure.* After the fissures are adequately covered, the material is then brought to a knife edge approximately *halfway* up the inclined plane.

Following polymerization, the sealants should be examined carefully *before* discontinuing the dry field. If any voids are evident, additional sealant can be added *without* the need for any additional etching. The hardened sealant has an oil residue on the surface. This is unreacted monomer that can be either wiped off with a gauze sponge or can be left. If a sealant requires repair at any time after the dry field is discontinued, it is prudent to repeat the same etching and drying procedures as initially used. Because all the commercial sealants—both the light-cured and self-cured—are of the same Bis-GMA chemical family, *they easily bond to one another.*[81]

Occlusal and Interproximal Discrepancies

At times an excess of sealant may be inadvertently flowed into a fossa or into the adjoining interproximal spaces. To remedy the first problem, the occlusion should be checked visually or, if indicated, with articulating paper. Usually *any minor* discrepancies in occlusion are rapidly removed by normal chewing action. If the premature contact of the occlusal contact is unacceptable, a large, *no. 8. round cutting* bur may be used to rapidly create a broad resin fossa.

The integrity of the interproximal spaces can be checked with the use of dental floss. If any sealant is present, the use of scalers may be required to accomplish removal. These corrective actions are rarely needed once proficiency of placement is attained.

Question 3

Which of the following statements, if any, are correct?

A. The etchant *predictably* attacks the center of the enamel prism, leaving the periphery intact.

B. When the data of a study indicate that 65% of the original sealants are retained for 7 years, it is the same as saying that an average of 5% is lost each year.

C. Bis-GMA products by different manufacturers are incompatible with one another.

D. An etched area that is not rapidly sealed will retain its rough, porous surface *indefinitely.*

E. The cleansing and etching of the occlusal surface with phospohoric acid is accomplished by *rubbing* the surface during the etching process.

Evaluating Retention of Sealants

The finished sealant should be checked for retention without using undue force. In the event that the sealant does not adhere, the placement procedures should be repeated,

with only about 15 seconds of etching needed to remove the residual saliva before again flushing, drying, and applying the sealant. If *two* attempts are unsuccessful, the sealant application should be postponed until remineralization occurs.

Resin sealants are retained better on recently erupted teeth than in teeth with a more mature surface; they are retained better on first molars than on second molars. They are better retained on mandibular than on maxillary teeth. This latter finding is possibly caused by the lower teeth being more accessible, direct sight is also possible; also, gravity aids the flow of the sealant into the fissures.[41]

Teeth that have been sealed and then have lost the sealant have had fewer lesions than control teeth.[82] This is possibly due to the presence of tags that are *retained in the enamel* after the bulk of the sealant has been sheared from the tooth surface. When the resin sealant flows over the prepared surface, it penetrates the finger-like depressions created by the etching solution. These projections of resin into the etched areas are called *tags.*[83] (Figure 10-6). The tags are essential for retention. Scanning electron microscopic studies of sealants that have not been retained have demonstrated large areas devoid of tags or incomplete tags, usually caused by saliva contamination. If a sealant is forcefully separated from the tooth by masticatory pressures, many of these tags are *retained* in the etched depressions.

The number of retained sealants decreases at a *curvolinear rate.*[41] Over the first 3 months, the rapid loss of sealants is probably caused by *faulty technique* in placement. The fallout rate then begins to plateau, with the ensuing sealant losses probably being due to abnormal *masticatory stresses.* After a year or so, the sealants become very difficult to see or to discern tactilely, especially if they are abraded to the point that they fill only the fissures. In research studies this lack of visibility often leads to *underestimating* the effectiveness of the sealants that remain but cannot be identified. Because the most rapid falloff of sealants occurs in the early stages, an initial 3-month recall following placement should be routine for determining if sealants have been lost. If so, the teeth should be resealed. Teeth successfully sealed for 6 to 7 years are likely to remain sealed.[83]

In a review of the literature, longest-term study reported that at the follow-up examination of the first molars, 20-years after sealant had been applied, 65% showed *complete* retention and 27% partial retention *without* caries. At a 15-year follow-up of the same sealants the second molars demonstrated the corresponding figures 65% and 30%, respectively. This study showed that pit-and-fissure sealants applied during childhood have a *long-lasting, caries preventive effect.*[60,77] Mertz-Fairhurst[83] cited studies in which 90 to 100% of the original sealants were retained over a 1-year period (Table 10-1). One 10-year study using 3M Concise Sealant had a 57% complete retention and a 21% partial retention of sealant, *all with no caries.* Another study, using Delton, registered 68% retention after 6 years.[108] (Figure 10-7). These are studies in which the sealant was placed and then observed at periodic intervals; there was no resealing when a sealant was lost. *Where resealing is accomplished as needed at recall appointments, a higher and more continuous level of protection is achieved.* More recent studies report 82% of the sealants placed are retained for 5 years.[70]

Figure 10-6 Tags, 30 um. Sealant was flowed over etched surface, allowed to polylmerize, and tooth surface subsequently dissolved away in acid. (Courtesy, Silverstone LM, Dogon IL. *The Acid Etch Technique.* St. Paul, MN: North

Central Publishing Co, 1975.)

Figure 10-7 A: 5-year sealant: Five years after placement of a white pit-and-fissure sealant in the matched pair to the control subject. Sealant and control subjects were matched on age, sex, caries history and other factors. B: 5-year control: This matched pair to the sealed patient. This subject did not receive sealant. The first permanent molar has already been restored with two amalgam restorations in the previous 5-year period. C: 15-year sealant: 15 years after the single application of a white pit-and-fissure sealant. This is the same tooth as seen in Figure 1, 5-year sealant, but 10 years later. As can be seen, the sealant has served its purpose even though there has been some loss in the peripheral fissures. (Courtesy of Dr. Richard J. Simonsen, D.)

Colored Versus Clear Sealants

Both clear and colored sealants are available. They vary from translucent to white, yellow, and pink. Some manufacturers sell both clear and colored sealants in either the light-curing or autopolymerizing form. The selection of a colored versus a clear sealant is a matter of individual preference. The colored products permit a *more precise placement* of the sealant, with the visual assurance that the periphery extends halfway up the inclined planes. *Retention can be more accurately monitored* by both the patient and the operator placing the sealant. On the other hand, a clear sealant may be considered more *esthetically* acceptable.

Some clinicians prefer the clear sealants because they are more discrete than white. Others prefer the white sealants as they are easier to monitor at recall appointments. On the other hand, some clinicians seem to prefer the clear sealants because it is possible to see under the sealant if a carious lesion is active or advancing. However, no clinical study has comprehensively compared these issues. Recently, some pit-and-fissure sealants have been introduced that will change color as they are being light-polymerized. This property has not been fully investigated and seems to be only of relative advantage to the dental personnel applying the sealant.

Placement of Sealants Over Carious Areas

Sealing over a carious lesion is important because of the professionals' concern about the possibility of caries progression under the sealant sites. In teeth that have been examined *in vivo* and later subjected to histologic examination following extraction for orthodontic reasons, it has been found that areas of incipient or overt caries often occur under many fissures, which *cannot* be detected with the explorer.[85] In some studies, sealants have been purposely placed over small, overt lesions.[83,86] When compared with control teeth, many of the sealed carious teeth have been diagnosed as sound 3 and 5 years later.[87] Handelman has indicated that sealants can be considered a viable modality for *arrest* of pit-and-fissure caries.[88] In other studies of sealed lesions, the number of bacteria recovered from the sealed area decreased rapidly.[33,34,86-89] This decrease in bacterial population is probably due to the integrity of the seal of the resin to the etched tooth surface[90] seal that does not permit the movement of fluids or tracer isotopes between the sealant and the tooth.[91]

Sealants have been placed over more extensive lesions in which carious dentin is involved.[92] Even with these larger lesions, there is a decrease in the bacterial

population and arrest of the carious process as a function of time. In another study, clinically detectable lesions into the dentin were covered for 5 years with Nuva-Seal. After that time the bacterial cultures were essentially negative, and an apparent 83% *reversal* from a caries-active to a caries-inactive state was achieved.[86] Jordan and Suzuki[93] sealed small lesions in 300 teeth. During clinical and x-ray observations over a 5-year period, they found *no change in size of the carious lesion,* so long as the sealant remained intact. More recently, Mertz-Fairhurst and colleagues[94] demonstrated that sealed lesions became *inactive* bacteriologically, with the residual carious material suggesting decay cessation. This ability to arrest incipient and early lesions is highlighted by the statement in the 1979 publication of the ADA's Council on Dental Therapeutics: "Studies indicate that there is an apparent reduction in microorganisms in infected dentin covered with sealant. . .. These studies appear to substantiate that there is no hazard in sealing carious lesions." The statements end with the *cautionary* note: "However, additional long-term studies are required before this procedure can be evaluated as an alternative to traditional restorative procedures.[95] When sealing incipient lesions, care should be taken to monitor their retention at subsequent recall/annual dental examinations. In addition, there have been reports of sealants being used to achieve penetration of incipient smooth-surface lesions ("white spots") of facial surfaces."[96]

Question 4

Which of the following statements, if any, are correct?

A. Tags can be easily determined by their rough feel when checking the *surface* of a sealant with an explorer.

B. Teeth that lose a sealant are more susceptible to caries than ones that retain a sealant but less caries-prone than a control tooth that was never sealed.

C. The falloff of sealants is *linear* as a function of time.

D. A study in which the periodic resealing of fissures occurs would be expected to have a *lesser* caries rate than a long-term study in which the same annual falloff is experienced, but where no resealing is accomplished.

E. Following placement of a sealant over a fissure with an undetectable carious lesion, the size of the subsurface lesion gradually *increases.*

Sealants Versus Amalgams

Comparing sealants and amalgams is not an equitable comparison because sealants are used to *prevent* occlusal lesions, and amalgam is used to *treat* occlusal lesions that could have been prevented. Yet, the comparison is necessary. One of the major obstacles to more extensive use of sealants has been the belief that amalgams, and not sealants, should be placed in anatomically defective fissures; this belief stems from *misinformation* that amalgams can be placed in less time, and that once placed, they are a permanent restoration. Several studies have addressed these suppositions. For instance, sealants require approximately 6 to 9 minutes to place initially, amalgams 13 to 15 minutes.[97,98]

Many studies on *amalgam* restorations have indicated a *longevity* from only a few years to an average life span of 10 years.[99-102] Equally perturbing is the fact that in one large study of schoolchildren, 16.2% of all surfaces filled with amalgam had marginal leakage and *needed replacement.*[103] The life span of an amalgam is shorter with younger children than with adults.[104] To emphasize the problem of replacement of older restorations, a recent questionnaire study from 91 dentists in Iceland was conducted to determine the cause for replacement of 8,395 restorations. The reason given for the replacement of composites, amalgams, glass-ionomers, and for resin modified glass ionomers was failed restorations (47.2%), primary caries (45.3%) and non-carious defects (7.5%). For every restoration inserted for an overt lesion, there was a need for one to be reinserted previously![105]

The retention data from the earlier sealant studies were discouraging. In recent years, using later-generation sealants, along with the *greater care in technique* used for their insertion, much longer retention periods have been reported. In five long-term studies from 3 to 7 years, the average sealant loss per year ranged from 1.3 to 7%.[106] If the yearly loss of these studies is extrapolated, the average life of these sealants compares favorably or exceeds that of amalgam.[107] When properly placed, sealants are no longer a temporary expedient for prevention; instead, they are the *only effective predictable* clinical procedure available for preventing occlusal caries.

The most frequent cause for sealant replacement is *loss of material,* which mainly occurs during the first 6 months; the most likely cause for amalgam replacement is *marginal decay,*[108] with 4 to 8 years being the average life span.[103] To replace the sealant, only resealing is necessary. No damage occurs to the tooth. Amalgam replacement usually requires cutting more tooth structure with each replacement. Even if longevity merits were equal, the sealant has the advantage of being painless to apply and aesthetic, as well as emphasizing the *highest objectives* of the dental profession—*prevention and sound teeth.*

Options for Protecting the Occlusal Surfaces

The use of sealants has spawned an entirely different concept of conservation of occlusal tooth structure in the management of deep pits and fissures before, or early in caries involvement. The *preventive dentistry restoration* embodies the concepts of both prophylactic odontotomy insertion of a restoration and *covering the restoration and the connecting fissure system with a resin based sealant.* Pain and apprehension are slight, and aesthetics and tooth conservation are maximized.[108] Several options are now available to protect the occlusal surfaces, with the selection depending *on risk and professional's judgment.*[109] The first level of protection is simply to place a conventional sealant over the occlusal fissure system. This sealing preempts future pit-and-fissure caries, as well as arrests incipient or reverses small overt lesions.

The second option reported by Simonsen in 1978,[110] advocated the use of the *smallest* bur to remove the carious material from the bottom of a pit or fissure and then using an appropriate instrument to tease *either sealant or composite* into the cavity preparation. This he termed a preventive dentistry restoration. Following insertion of the restoration, sealant was placed *over* the polymerized material as well as flowed *over the remaining fissure system.* Aside from protecting the fissures from future

caries, it also protects the composite or inserted sealant from abrasion.[111]

The third option is use of glass-ionomers material for sealants, which is controversial. Due to their fluoride release and cariostatic effect, glass-ionomers have been used in place of traditional materials, as a pit-and-fissure sealant, however, resin sealants have shown much higher bond strength to enamel than glass-ionomers. Clinical trials[112,113] have shown poor retention over periods as short as 6 to 12 months. Though, in vitro studies have suggested that etching previous to application enhances the bonding of glass-ionomer sealant in fissure enamel.[114-116] One study showed that a conventional silver-reinforced glass-ionomer had superior clinical performance compared to a conventional resin sealant.[117]

Resin-reinforced glass-ionomer cements have been investigated for their effectiveness as pit-and-fissure sealants. The 1-year results revealed that although clinically the glass-ionomer wears at a faster rate than a conventional resin sealant, in the scanning electron microscopic evaluation the material could be seen at the deep recesses of the pits-and-fissures with no carious lesion present.[113] A recent study showed that after 3 years the glass-ionomer sealant was completely lost in almost 90% of the teeth compared to less than 10% of the resin sealed teeth; the relative risk of a tooth sealed with glass-ionomer over that of a tooth sealed with resin was higher. Also, the glass-ionomer sealant had poorer retention and less caries protective effect.[118]

Glass-ionomer does not carry the ADA seal of approval as sealant material. The readers should decide their personal philosophy based on the evidence.

A fourth option reported by Garcia-Godoy in 1986 involves the use of a glass-ionomer cement as the *preventive glass-ionomer restoration* (PGIR).[119] The glass-ionomer cement (conventional or resin-modified) is placed only in the cavity preparation. (Figure 10-8). The occlusal surface is then etched with a gel etchant avoiding, if possible, etching the glass-ionomer. Etching the glass-ionomer may remove some of the glass particles weakening the material. The conventional resin sealant is placed *over the glass-ionomer and the entire occlusal fissure system.* In the event sealant is lost, the fluoride content of the glass-ionomer *helps prevent* future primary and *secondary* caries formation. The same technique has successfully protected the marginal integrity of very small amalgam restoration, as well as providing a protection to the entire fissure system.

Each of these options requires a judgment decision by the clinician. That decision can well be based on the criterion that if an overt lesion cannot be *visualized,* it should be sealed; if it can be visualized, the smallest possible preventive dentistry restoration should be used along with its required sealant "topping." Mertz-Fairhurst and associates[120] have pointed out that the first option could provide the preferred model for conservative treatment of *incipient* and *small overt,* pit-and-fissure caries. It could also serve as an interim treatment for larger lesions. These options would be especially valuable in areas of the world with insufficient professional dental personnel and where preventive dental auxiliaries have been trained to place sealants. In all cases, the preventive dental filling should be considered as an alternative to the traditional class I amalgam with its accompanying extension for prevention that often includes the entire fissure system.

Figure 10-8 Preventive glass ionomer restoration (PGIR). Cavity preparation for reception of glass-ionomer cement. (Courtesy of Dr. Franklin Garcia-Godoy, University of Texas Dental School, San Antonio.)

The Sealant as Part of a Total Preventive Package

The sealant is used to protect the occlusal surfaces. A major effort should be made to incorporate the use of sealants along with other primary preventive dentistry procedures, such as plaque control, fluoride therapy, and sugar discipline. Whenever a sealant is placed, a topical application of fluoride should follow if at all possible. In this manner the whole tooth can be protected. Ripa and colleagues[121] completed a 2-year study for children in second and third grades assessing the effectiveness of a 0.2% fluoride mouthrinse used alone compared with a rinse plus sealants. Twenty-four occlusal lesions developed in the 51 rinse subjects, and *only* 3 in the 84 subjects receiving the rinse plus sealants. The conclusion was that caries could be *almost completely eliminated* by the *combined* use of these two preventive procedures. In many public-health programs, however, it is not possible to institute full-scale prevention programs, either because of apathy or lack of time and money. In such cases, there is some consolation in knowing that at least the *most vulnerable* of all tooth surfaces (the occlusal) is being protected.

Manpower

The *cost* of sealant placement *increases* directly with the level of professional education of the operator. Dentists, hygienists, assistants, and other auxiliaries can be trained to place sealants.[122-124] In view of the cost-effectiveness, dental auxiliaries should be considered as the logical individuals to place sealants. This is important if manpower is to be increased.

Often auxiliaries who have received sealant instruction, either through continuing-education courses or as part of a curriculum, are stymied either because of state laws interdicting their placing sealants or by the nature and philosophy of the practice of the employing dentist.[125] Only fourteen states allow hygienists to practice under less restrictive or unsupervised practice models in which they can initiate treatment based on assessment of patient, treat the patient, and maintain a provider-patient relationship without the participation of the patients' dentist of record. For example, Maine and New Hampshire have a separate supervision for settings outside of the dental office public-health supervision, which is less restrictive than general supervision. New Mexico allows for a collaborative-practice agreement between dentists and hygienists in outside settings. Yet, in states such as Georgia and Illinois, hygienists are required to practice under direct supervision. This means the dentist must be present in the office while the care is being provided.[126]

In a Swedish study, 77 *dental assistants* working in 12 dental clinics sealed 3,218 first and second molars with a 5-year retention rate of between 74 and 94%.[127] Because many dentists consider the placement of sealants to be a relatively simple procedure, few are returning for continuing-education programs to learn the exacting and precise process necessary to ensure maximum sealant retention. Even when the dental professionals desire to participate in such continuing education, a survey found relatively few courses available.[128]

Economics

Bear in mind that not every tooth receiving a sealant would necessarily become carious; hence the cost of preventing a single carious lesion is greater than the cost of a single sealant application. For instance, Leverett and colleagues calculated that five sealants would need to be placed on sound teeth to prevent one lesion over a 5-year period,[129] and Rock and Anderson estimated one tooth for every three sealant applications are prevented from becoming carious.[130] Sealants would be most cost-effective if they could be placed in only those pits and fissures that are destined to become carious. Unfortunately, we do not have a caries predictor test of such exactitude, but, the use of vision plus an economic, portable electronic device that objectively measures conductance (or resistance) would greatly aid in evaluating occlusal risk.[131] Without such a device, it is necessary to rely on professional judgment, based on the severity of the caries activity indicators: number of "sticky" fissures, level of plaque index, number of incipient and overt lesions, and microbiologic test indications.

In an office setting, it is estimated that it costs 1.6 times more to treat a tooth than to seal.[55] The Task Force on Community Preventive Services, an independent, non-federal group formed to evaluated oral-health interventions, was charged with determining interventions that promote and improve oral health. The Task Force examined six public-health programs cost of placing pit-and-fissure sealants revealing a mean cost of $39.10 per person.[132] However, even these numbers are misleading. For instance, what is the value of an intact tooth to its owner? How much does it cost for a dentist and assistant to restore a tooth, compared to the cost of sealing a tooth? Later in life, what is the cost of bridges and dentures that had their genesis when children were at high risk with little access to dental care?

Use of Pit-and-Fissure Sealants

By the mid-1980s most of the answers were available as to the need and *effectiveness* of Bis-GMA sealants to reduce the incidence of occlusal caries, and the *techniques* of placement of pit-and-fissure sealants were known.[133] The *safety* of their placement has been demonstrated by many studies showing that even when placed over incipient and minimally overt caries sites, there was no progression *as long as the sealant remained intact.*[134] Finally, several clinical studies have pointed out that sealants could be *applied by properly trained auxiliaries,* thus providing a more economical source of manpower for private and military practices as well as for large school and public health programs.

Bis-GMA sealant usage has been strongly supported by the ADA "as a safe and effective means for caries control."[25] The United States Public Health Service, in a request for a proposal for a school pit-and-fissure study, stated *"This combination of preventive techniques (combined use of fluoride and sealants) is expected to essentially eliminate caries in teeth erupting after the initiation of the study."*[135] Despite the support from the two largest organizations most interested in the dental health of the nation, the rank-and-file of the dental profession *have not accepted sealants as a routine method for prevention.*

In spite of all the knowledge of the properties and successes of the sealants usage has lagged, with about 10% of the posterior teeth of children demonstrating the presence of sealants.[136] For example, a 1994 examination of 117,000 children in North Carolina between the ages of 6 and 17 found that approximately 12% had sealants,[137] while the percentage for 927,000 in Tennessee was 10%.[138] Other states demonstrate similar sealant usage. One study revealed that 88 children did have sealants while 508 did not have needed sealants.[139] For recruits entering the U.S. Air Force, sealants were found on 13.1% of the teeth while there was a need for 47.5% more. In the latter case, it was noted that a third of these personnel had occlusal caries that might have been prevented by the sealants.[20]

Many barriers exist in meeting the Healthy People 2010 Objective for sealants. In 2001, the State of Alabama was planning how to meet national dental objectives, when 50% of U.S. children are expected to have dental sealants on at least one permanent molar by the age of 14 years.[140] (Currently, 22% of the children between 12 to 14 years have at least one sealant claim.) A final assessment of the 2010 prospects and the current State's demographics concluded that racial and gender disparities, difficulty in accessing care, the nonavailability of Medicaid-participating dentists in a country, and a lower payment/claim ratio may make the national sealant objective difficult to achieve.[140] It should be mentioned that in many surveys, children from lower socioeconomic groups had greater sealant needs than those from more affluent neighborhoods.

On the other hand, other countries have had marked success with increasing the number of teeth sealed. A study involving 68,704 children living in Lanarkshire, Scotland found approximately 10% of the occlusal surfaces were sealed.[141] Five years later, in England the percentage of children *having sealants dramatically increased* between 20 to 50% in several areas.[142]

The placement of sealants is making slow progress. The 1998-99 Ohio State survey of 3rd-grade students in School Based/School Link programs found that in addition to oral-health benefits, "Providing sealant programs in all eligible, high-risk schools could reduce or eliminate racial and economic disparities in the prevalence of dental sealants".[143] Yet, there are problems in examining the number of sealants *versus* the *need* for sealants.

Dentist Involvement

Pit-and-fissure sealants are underused in private practice and public health. There are many complex reasons for such under use, but efforts should be undertaken to increase sealant use.[3] Increasing sealant use is dependent, in part, on dentists' acceptance and understanding of the preventive technique. In a mail survey in Minnesota, 95% of 375 dentists reported the use of sealants, varying from 1 to 25 per week. Possibly, the incongruity of numbers stems from the fact that although the majority of dentists use sealants, the *frequency* of use is *low*.[144]

Reasons for this apathy have ranged from alleged concerns of sealing over carious lesions, lack of technical skill, short longevity of sealants, and the need for more research—all problems that have been adequately addressed in the literature.[133] Probably the most important factor now restricting the placement of sealants is the

lack of an adequate insurance fee schedule.[145] Another is that most dentists are treatment-oriented. This fact is amplified by an explanation by Galarneau and Brodeur that "A dentists lack of comfort with withholding treatment may stop him/her from offering preventive care and cause him to follow a restoration-oriented practice."[146] Another factor is that dentists rarely explain the oral-health advantages of sealants over dental restorations.[147]

In attempting to alter the attitudes of dentist on sealant use, several studies have been conducted to measure *changes in knowledge and attitudes* following continuing-education courses. The follow-up indicated that there had been an increase in *knowledge* but little change in *attitudes* concerning sealant use.[148] In Colorado, pediatric dentists, who are continually involved in treating children, placed more sealants than general dentists—again, probably a manifestation of attitudes.[145]

Regardless of increased rhetoric about prevention, the concepts and actions of prevention are *not* being fully implemented in dental schools.[149] Dental school faculties need to be educated about the effectiveness and methods of applying sealants.[150,151] Possibly the development of a model curriculum for teaching pit-and-fissure sealant usage would help.[152] The dental community must develop a consensus about the value and economic effect of preventive measures.[150]

Other barriers to effective delivery of sealants include (1) state-board restrictions on auxiliary placement of sealants, (2) lack of consumer knowledge of the effectiveness of sealants, and, resultantly, a lack of demand for the product.[122] *The economics and education of the profession and of the public are the prime requisites for expanded sealant acceptance.*[153]

Question 5

Which of the following statements, if any, are correct?

A. The longevity expectation for a properly placed amalgam restoration is approximately twice that of a properly placed sealant.

B. Sealants should be placed only on permanent teeth of children up to age 16.

C. Sealants are found on approximately 54% of U.S. children.

D. Following the graduation of students presently in dental schools, a large increase in the use of sealants can be expected.

E. Caries does not progress under a properly sealed composite or amalgam.

Other Pit-and-Fissure Initiatives

The findings of the following studies must be considered an *important extension* of the present use of pit-and-fissure sealants, which are used to prevent the development of incipient lesions and to arrest minimal overt lesions. If professional judgment dictates, conservative sealed amalgams or composites could be used to maintain *marginal integrity, extend the longevity of the restorative materials, and for achieving*

a de facto extension for prevention without the need to remove sound tooth structure to extend the restoration over the entire fissure system. These two uses of resins for prevention and restorations without major operative considerations should be of great value in developing countries where professional manpower is at a minimum and the demand for dental care is great.

Probably the most important recent research on the use of Bis-GMA sealants and carious lesions were described by Mertz-Fairhurst and coworkers.[87,154] In the 10-year study,[154] patients with paired permanent molars or premolars with *obvious clinical and radiographic class I lesions* were selected. The carious lesions extended halfway into the dentin or to the nearest pulp horn. The randomized placement of restorations for each of the tooth pairs consisted of two of the following: (1) a *classic* amalgam restoration, complete with extension for prevention of all connecting fissures (79 subjects); (2) a *conservative* amalgam restoration involving only the carious site with a sealant "topping," the latter which was extended into the entire pit-and-fissure system (77 subjects); and (3) with each one of the amalgam restorations, a paired composite restoration placed over the carious tissue with a "topping" of sealant that included all the pits and fissures (156 subjects). In the preparation for the composite, *no attempt was made to remove the carious tissue.* A 1-millimeter wide, 40- to 60-degree bevel was made in the sound enamel surrounding the lesion. The area was washed, dried, and a bonding agent was placed on the bevel. Hand instruments were used to place the composite, after which rotary instruments were used to shape the occlusal anatomy. Following this step, the occlusal surface was treated as for the placement of the average sealant—dry, etch, rinse, and dry before placing the resin over the composite and the entire fissure system.

The conclusions of this study after 10 years were: (1) *both* the sealed composites and the sealed amalgam restorations exhibited *superior clinical performance and longevity* compared to the unsealed amalgam restorations; (2) bonded and sealed composite restorations placed over the frank cavitated lesions *arrested the clinical progress of these lesions for the 10 years of the study.*

Summary

The majority of all carious lesions that occur in the mouth occur on the occlusal surfaces. Which teeth will become carious cannot be predicted; however, if the surface is sealed with a pit-and-fissure sealant, no caries will develop as long as the sealant remains in place. Recent studies indicate an approximate 90% retention rate of sealants 1-year after placement. Even when sealants are eventually lost, most studies indicate that the caries incidence for teeth that have lost sealants is less than that of control surfaces that had never been sealed. Research data also indicate that many incipient and small overt lesions are arrested when sealed. Not one report has shown that caries developed in pits or fissures when under an intact sealant. Sealants are easy to apply, but the application of sealants is an extremely sensitive technique. The surfaces that are to receive the sealant must be completely isolated from the saliva during the entire procedure, and etching, flushing, and drying procedures must be timed to ensure adequate preparation of the surface for the sealant. Sealants are comparable to amalgam restorations for longevity and do not require the cutting of tooth structure. Sealants do not cost as much to place as amalgams. Despite their advantages, the use of sealants has not been embraced by all dentists, even though

endorsed by the ADA and the U.S. Public Health Service. Even when small overt pit-and-fissure lesions exist, they can be dealt with conservatively by use of preventive dentistry restorations. What now appears to be required is that the dental schools teach sealants as an effective intervention, that the dental professional use them, that the hygienists and the auxiliary personnel be permitted to apply them, and the public demand them.

Answers and Explanations

1. C and D—correct.

A—incorrect. Because the fluorides protect the smooth surface, there will be a greater proportion of pit-and-fissure lesions.

B—incorrect. By definition, an incipient lesion has not been invaded by bacteria; thus the use of a sealant is an ideal preventive measure.

E—incorrect. Remember, it is the caries susceptibility of the teeth that is important—not the age of the individual.

2. A, B, and C—correct.

D—incorrect. All the major, successful, long-term retention studies have used cotton-roll isolation; in the one study of rubber dam versus cotton rolls, the rolls were equal to, or better than, the dam.

E—incorrect. Ten seconds are used for the drying and flushing procedures, and 20 to 30 seconds for the etching.

3. A and B—correct.

C—incorrect. Bis-GMA plastics are of the same chemical family and will bond to each other regardless of manufacturer.

D—incorrect. Remineralization from saliva constituents occurs rapidly in a period of hours to days.

E—incorrect. Cleansing and etching do occur; however, rubbing tends to obliterate the delicate etching pattern and reduce retention potential.

4. B and D—correct.

A—incorrect. The tags of the sealant cannot be felt with the explorer; they extend into the enamel from the underneath side of the plastic.

C—incorrect. The curvolinear falloff is greatest at 3 months, less at 6 months, after which it gradually plateaus.

E—incorrect. The literature is unanimous that caries does not progress under an intact

sealant.

5. C and E—correct.

A—incorrect. There is little difference between the longevity of a well-placed amalgam compared with a well-placed sealant.

B—incorrect. If a tooth is susceptible to caries, it should be sealed, whatever the patient's age.

D—incorrect. All signs indicate that the teaching of sealant placement is greatly neglected in dental schools.

Self-evaluation Questions

1. Approximately _____ % of all carious lesions occur on the occlusal surfaces; the continual use of fluorides (increases) (decreases) this percentage.

2. Four different methods used prior to the advent of polyurethane, cyanoacrylate, and Bis-GMA sealants, were _____, _____, _____, and _____.

3. One condition that *indicates the use of a sealant is* _____; *four conditions that contraindicate* the use of sealants are _____, _____, _____, and _____; three conditions that *probably indicate* the use of sealants are _____, _____, and _____.

4. Two photoactivated, and two chemically activated sealants that have been accepted, or provisionally accepted, by the ADA are (photoactivated) _____, _____, and (chemically activated) _____ and _____.

5. The liquid resin in a sealant kit is known as the _____; when it is catalyzed the hardening process is known as _____. The catalyst used for the polymerization of chemically activated sealants is _____ and for visible photoactivation, _____.

6. Two advantages to light-cured sealants are _____ and _____; and two advantages of autopolymerized sealants are _____ and _____.

7. _____ forces, not chemical bonding, causes retention of the sealant to the tooth; the four commandments to ensure maximum retention are _____, _____, _____, and _____.

8. Three methods by which a dry field can be established are _____, _____, and _____.

9. The placement of sealants is extremely technique-sensitive; after selection of the tooth for sealant placement, it should be dried for _____ (time); then etched for _____ (time), followed by a water flush of _____ (time), and finally, dried for _____ (time) before placing the sealant.

10. Excessively high sealants that interfere with occlusion can be reduced by use of a number _____ (cutting) (finishing) bur.

11. The falloff of sealants is (linear) (curvilinear); long-term studies where 65% of the sealants are retained after 7 years indicate an average yearly loss of _____ %. After 10 years, _____ _____ % would be retained. This contracts to an average life expectancy of an amalgam of approximately _____ (years).

12. To protect the total tooth, the application of a sealant should be followed by an application of _____.

13. To ensure that sealant placement techniques have been perfected in dental and dental hygiene schools, it should be necessary for _____ (state dental-regulating agency) to require a demonstration of proficiency for all candidates prior to state licensure.

14. The three key components of a light source of polymerizing sealants are _____, _____, and _____ (which results in the blue color).

15. The three basic options for a preventive dentistry restoration are _____, _____, and _____.

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Chapter 11. Oral Biologic Defenses in Tooth Demineralization and Remineralization - *Norman O. Harris John Hicks*

Objectives

1. Describe at least five body defense systems that are operational in and-around the oral cavity.

2. List the names of the major salivary glands in rank order of both their daily output of unstimulated (resting) saliva and the amount of stimulated output.

3. List three means of stimulating saliva output and three methods of inhibiting saliva output.

4. Define and compare the terms sialorrhea, xerostomia, and ptyalism.

5. Describe the appearance and the implications of the contour of the Stephan Curve.

6. Describe how the fluid viscosity of the plaque affects diffusion within the plaque.

7. Describe the ultramicroscopic morphology of enamel rods, enamel crystals and the unit cell of hydroxyapatite (HAP).

8. Explain why an extracted tooth immersed in a liquid acid solution (*in vitro*) will not yield an incipient lesion, whereas, if it is immersed in a buffered gel of similar pH, the incipient lesion develops.

9. Explain why a newly erupted tooth is at high risk to develop a carious lesion.

10. Recount the key events that cause and occur in demineralization, and how the reverse events of remineralization can often repair the damage.

Introduction

[Chapter 11 is a continuation of Chapter 3. Whereas Chapter 3 emphasized the basics of the caries process, chapter 11 concentrates on the saliva and the ultramicroscopic structure of the tooth, as they affect de- and remineralization.]

The mouth is the gateway for food and drink destined for the gastrointestinal (GI) tract. To ensure the safety of the body from the oft-unknown quality of foods being brought into the mouth, two powerful evolutionary monitoring sensory systems exist to help determine safety and quality *before* ingesting the gustatory fare—*vision and smell*. Both of these senses allow the host to reject food deemed to be undesirable. Once within the oral cavity, there is the protective umbrella of the body's immune system—*the cellular and the secretory immune systems.* The former is cell-mediated and consists of the phagocytic and lymphoid elements involved in preventing infection. The secretory system mainly protects mucous membranes with secretions of antibodies, such as sIgA (secretory immunoglobulin A).[1] Two other defense mechanisms are *taste*[2] and *tactile sense.* As an example, tactile sense allows for proprioception[a] via nerves in the oral tissues to evaluate morsel size, texture and shape of the entering food; to segregate foods that need to be chewed from food that needs to be incised; and to determine when a bolus of food is of the correct size and consistency from chewing to be safely swallowed.[3]

The defense functions of the *saliva* are part of the total body's ability to maintain *homeostasis*, i.e., the ability to resist routine daily challenges by chemical and bacterial agents, and to repair limited amounts of tissue damage typical of the wear and tear of daily life.[4] It is only when the bacterial challenge exceeds the body's defense capabilities and/or there is a lack of a person's commitment to self-care, that dental caries ensues.

The *saliva* helps modulate and augment the previously described major body defense systems in protecting oral tissues. However, in the demineraliztion and remineraliztion process of tooth structure (caries and repair), the saliva cannot be isolated from an interrelated three compartment model consisting of *saliva, plaque and teeth.*[5]

[a]Proprioception = The reception of sensory nerve stimuli that locate the location of position of parts of the body. Example: While eating, a diner with every bite, provides the brain with information as to where the opposing teeth are in time to prevent a traumatic occlusion.

The Saliva Compartment

The saliva is derived mainly from the major salivary glands—the parotid, submandibular, and sublingual glands. Of these, the parotid elaborates a serous (watery, mucous-poor) fluid containing eletrolytes, but is relatively low in organic substances. The parotid gland secretes the majority of the *sodium bicarbonate* that is essential in neutralizing acids produced by cariogenic bacteria in the dental plaque,[6,7] and the majority of the enzyme *amylase* that initiates intraoral digestion of carbohydrates. The submandibular gland secretes a mixed serous and mucuos fluid, while the sublingual gland has a greater proportion of mucous output than the other major glands. The minor glands—palatal, lingual, buccal, and labial salivary glands empty onto the mucus membrane in many locations—on the palate, under the tongue, and on the inner side of the cheeks and lips. These minor glands are mainly mucous secreting glands that lubricate these surfaces and allows for improved mastication and passage of food substance into the esophagus.[3] The minor salivary glands also contribute fluoride that bathes the teeth and enhances caries resistance.[8,9,10]

Pure saliva produced by the oral glands is sterile, until it is discharged into the mouth. When the fluids from all major and minor glands mix with each other, this secretion becomes known as *whole saliva.* Whole saliva is further altered by the presence of particles of food, tissue fluid, lysed bacteria, and sloughed epithelial cells. It becomes even more complex with the inclusions of *living cells and their metabolic products*, for example, bacteria and leucocytes, the latter derived from the gingival crevices and tonsils.

Functions of saliva

The physical and chemical protective functions of saliva can be divided into five convenient categories—(1) lubrication, (1) flushing/rinsing, (2) chemical, (3) antimicrobial (includes antibacterial, antifungal and antiviral), and (4) maintenance of supersaturation of calcium and phosphate level batheing the enamel, helping to stymie demineralization and/or to aid remineralization of tooth structure.[11,12] To reinforce the

concept expressed in (4), Peretz aptly opined that saliva can be considered similar to enamel but in a liquid phase.[13]

The salivary defensive system functions continuously, but its secretion becomes greatest and most active during foodstuff ingestion. It has the lowest flow rate during the sleep period of the daily 24-hour cycle.

Lubrication and Flushing

A very thin microscopic layer of mucus protects the oral hard and soft tissues from the often harsh and abrasive foods, as they are being chewed and swallowed. It also protects the soft tissues from dessication and the teeth from abrasion. The moistening of food by saliva *facilitates chewing and swallowing.* Speech is enhanced by the reduced friction between the dry tongue and soft tissues. Coversely, a lack of saliva (*xerostomia*) results in a greatly increased risk of caries with its accompaniment of an extremely *annoying dry-mouth sensation.* Chewing, swallowing and speaking can all be difficult and uncomfortable with *dry-mouth syndrome* and often requires frequent ameliorating sips of water.

Flow Rate

Providing a constant fluid flow is probably the most important defense function of the salivary glands, because it is the fluid that transports the buffering agents, the antimicrobials, and the mineral content of saliva to help control the equilibrium between the demineralization and remineralization of tooth structure. Also, the fluid output of the glands is essential for *diluting* acids, *flushing* food particles embedded around the teeth, *clearing* refined carbohydrates (acid-producing sugar substrates) and physically *removing any displaced* bacteria[12] Oral fluids in contact with food particles results in solubilizing food substances that interact with the taste buds to provide an accurate assessment of taste.[2]

The composition of saliva varies, depending on whether it is *stimulated* or *unstimulated (resting).* During the day, submandibular glands secrete the greatest proportion of the unstimulated saliva, although the flow rate of resting saliva for *all* three glands is very low, being about one tenth that during stimulated flow. Approximately 2/3 of the *resting* saliva is derived from the submandibular glands, one-quarter is from the parotids, and about 1/20 is from the sublingual glands. The minor salivary glands secrete almost 1/10 of the total amount of saliva. The unstimulated flow rate of the salivary glands is subject to a circadian rhythm, with the highest flow in mid-afternoon and the lowest around 4:00 A.M.

Upon *moderate stimulation,* the submandibular and parotid glands secrete approximately equal amounts of saliva, whereas at full stimulation the parotid has the greatest output. When salivary flow is stimulated by chewing gum or paraffin, 1 to 2 mL of whole saliva per minute can be expected. The minimum level of stimulated salivary flow necessary to maintain hard- and soft-tissue health is unknown, but when it is *below 1 mL/minute,* there is cause for concern regarding a possible dry mouth and caries formation. Once the flow rate is below *0.7 mL/minute,* a diagnosis of xerostomia may be rendered. In the course of a single day, up to 1 liter (1 quart) of saliva is secreted into the oral cavity.

The total amount of saliva secreted varies considerably between and within individuals, depending on the environmental factors. Seasonal variations occur, with flow being lower in warm weather and higher in cold. The act of smoking increases flow rates. Flow is greater while standing than when sitting and greater when recumbent, with these postural changes paralleling changes in systemic blood pressure.

Saliva flow may be *stimulated* (1) physiologically, (2) pharmacologically (over the counter drugs, herbals and prescription medications) and by (3) many different disease states[14,15] Examples of physiologic stimulation are the simple acts of *chewing* food and gum, *gustatory stimuli* caused by *tasting* an enjoyable food, while *psychologic* stimulation for food can be evoked by anticipating the first bite of a delicious food via the sense of sight and/or smell. Saliva can also be stimulated by the use of *drugs,* such as pilocarpine. Under certain conditions, saliva flow can be abnormally high—a condition termed *sialorrhea,*(or *ptyalism*) which is often manifested by drooling. Under some conditions drug therapy can be used,[16, 10] but *sialorrhea* or *ptyalism* may be so severe as to require surgical removal (excision)of the responsible gland or *ligation* of the gland duct.[17]

Saliva flow can also be *suppressed* physiologically, pharmacologically[16] and/or by disease. The dry mouth sensation (xerostomia) that accompanies *fear* is an example of a physiological response; pharmacologically it may follow the intake, among others, of antidepressant and antihypertensive drugs;[18,19] it occurs when there are *sialoliths* (stones) within the gland ducts resulting in obstruction of saliva flow,[19,20] or following radiation exposure of the glands during cancer therapy.

The concentration of the various saliva components secreted by the glands is closely related to the flow rate. Stimulation of the rate of flow by stimulation increases the concentration of *some* constituents and decreases it for others. Stimulation of the parotid glands causes an increase in calcium, sodium, chloride, bicarbonate and pH. The same saliva demonstrates a concomitant decrease in phosphate and potassium.

In addition to the secretion of different proportions of electrolytes, *organic* molecules are secreted that can be categorized into five major groups: amylase, mucins, phosphoproteins, glycoproteins, and immunoglobulins. Two of the families of small salivary proteins—histadine and statherin—deserve specific mention because they help control the status of calcium and phosphate in the saliva. These proteins prevent fall-out of the calcium and phosphate that maintain supersaturation in relation to HAP. They prevent a rapid drop in saliva pH and aid in its quicker recovery. In addition, they both are antifungal and help prevent mucosal infections.

Question 1

Which of the following statements, if any, is correct?

A. sIgA (secretory immunoglobulin A) is a guardian of moist epithelial surfaces (mucous membranes).

B. The major salivary glands are the parotid, palatal, and the submandibular.

C. The saliva output of the major salivary glands increases in defense effectiveness at the time of chewing.

D. In the order of maximum flow rate, the parotid is first, the sublingual second, and the minor salivary glands third.

E. All the major salivary glands can be both stimulated or retarded in flow rate by physiological stimulus, drugs, or disease.

Protective Functions of Saliva

The protective functions of saliva are from its *physical, chemical,* and *antimicrobial* properties.[10] Saliva is not equally distributed around the oral cavity because of differences in anatomical and orthodontic features. It also has a tendency to stay on the side it was secreted.[21] These differences mean there is an increased risk for caries formation owing to retention of refined carbohydrates at difficult-to-reach sites in the mouth.[22] Of parallel importance, a viscid saliva is not as effective in clearing food particles and snacks, as is normal saliva.

Antibacterial Functions

The most easily understood major antibacterial function is performed by one of the glycoproteins—the mucins—that trap or *aggregate* bacteria that are eventually swallowed. The same mucins provide a thin film over the mucous membrane and teeth to serve as lubricants.

Four important antimicrobial proteins found in saliva are: lysozyme, lactoferrin, salivary peroxidase and secretory immunoblobulin A (sIgA). *In vitro,* lactoferrin strongly inhibited adherence of mutans streptococci to saliva coated hydroxyapatite (HAP) blocks.[23] Lactoferrin combines with iron and copper to *deprive* bacteria of these essential nutrients. Salivary peroxidase reacts with saliva to form the antimicrobial compound *hypothiocyanate,* which in turn inhibits the capability of the bacteria to *fully use glucose.* Lactoperoxidase strongly adsorbs to hydroxyapatite as a component of the acquired pellicle, and can *influence* the qualitative and quantitative characteristics of the microbial population of dental plaque. The role of the body's cellular and immunologic defense systems in moderating the course of the plaque-induced disease needs clarification. The main access that phagocytic cells and their antibacterial products, have to the oral cavity is through the gingival crevice and the tonsils. It is difficult to conceive of the cellular immune system operating in the bacterial plaque, yet about 500 leukocytes per second are estimated as emigrating from the tissues through the gingival crevice into the oral cavity. The majority of these soon disintegrate in the saliva, a phenomenon that may be related to the fact that more intact polymorphonuclear leukocytes occur in caries-free than in caries-susceptible individuals. On a research basis, there is reason to believe that a linkage exists between normal humoral and cellular defenses, and both caries and periodontal disease. How the cells and immunoglobulins exercise this potential is unclear. The development of a successful vaccine against caries and possibly, against periodontal disease will ultimately depend on such a clarification.

The Plaque Compartment

The plaque compartment begins with the acquired (salivary) pellicle, which is an acellular protein layer of saliva components that is adsorbed onto the surface of the enamel (Chapter 2). Upon this pellicle, the bacteria colonize. The pellicle plus the bacteria and the gel they create, constitutes a *biofilm (*dental placque*).* For several hours after a prophylaxis (that removes biofilm) there is a steady change in the quantity and composition of the pellicle as new proteins are added from the saliva. Glycoproteins appear to mediate the attachment sites of the subsequent colonizing plaque bacteria. Even though mucins are a minor component of the pellicle, they can be very protective *against acid diffusion.*

To understand the effect that plaque has on teeth, it is neccesary to focus on the action of acid in demineralizing teeth. To reduce the potential of demineralization, it is necessary to (1) reduce the *number* of bacteria producing the acid, (2) reduce the *amount of acid* produced by the existing bacteria, and/or (3) negate the effects of the acid produced by plaque.

Physical Character of Plaque

A major consideration in the defense of the tooth is the physical character of plaque itself. In order for the fluid and chemical components of saliva and plaque to function, they must be able to diffuse freely (intermix) with the constituents of the plaque. This diffusion requires time, which is contingent on two important factors. (1) If the fluid content in the plaque is relatively high, incoming and exiting ions diffuse rapidly. (2) If the colloid and glucan content of the plaque is high, the diffusion is slow, thus *retaining any acid against the tooth surface longer*.

Probably the most unpredictable factor relating to the plaque diffusion is the character of the microbial population. Variations in bacterial species from one plaque to another or in different parts of the same plaque result in different diffusion patterns. In other words the bacteria and their metabolites can act as either a *barrier,* or as a *gateway* to the passage of selected anions, cations and proteins. For example, bacteria use phosphate in their metabolism—a metabolic need that is *accentuated* during periods of acidogenesis. *Thus, the bacterial need for phosphate from the plaque metabolic pool occurs at the same time that the same phosphate is required to maintain supersaturation at the plaque tooth interface.*

Not all bacteria are bad. Veillonella, when present, metabolizes lactic acid generated by mutans streptococci, lactobacilli, actinomycetes, and other acidogenic organism. Presumably this action *decreases* the amount of acid available to demineralize tooth structure. Several studies indicate that the presence of Veillonella, indeed, decreases caries risk. Thus the varieties, metabolic characteristics and interrelationships of the plaque bacteria at any one time, are important in determining whether caries will occur.

Question 2

Which of the following statements, if any, are correct?

A. All parts of the mouth are equally assessable to the flushing effect of saliva.

B. The following are anti-microbial agents found in saliva: lysozme, lactoferrin, and salivary peroxidase.

C. If a cross section of a plaque coated crown of a tooth is studied, the following structures would be seen starting with the tooth surface: the enamel surface, acquired (salivary pellicle), bacterial plaque, and finally, saliva.

D. The bacteria of the plaque cannot use the phosphate diffusing out of the pores for their own metbolism.

E. Plaque acidogenesis could probably be reduced to inoculous levels by a major commitment to sugar discipline as a part of self-care.

Reducing Acid Production

Toothbrushing, flossing and irrigation ("brush, floss and flush") are ideal for personal self-care. However, there are *natural* oral defense mechanisms that exist in the body that are *not dependent on the frailities of human motivation, memories* or techniques.

1. Great numbers of bacteria in the saliva are eliminated by *flushing,* aggregation and *swallowing.*

2. The bacterial populations in the saliva and plaque are continually exposed to the *antimicrobial elements of saliva.*

Reducing the amount of acid produced by the bacteria is mainly a function of limiting the intake of refined carbohydrates (i.e., *sugar discipline*). This subject is discussed in detail in later chapters dealing with sugars, nutrition and clinical preventive dentistry. *The ingestion of refined sugars makes dental caries a self-inflicted disease.*

Reducing the Acid Damage

The plaque pH can drop to as low as 4.0 on the Stephan Curve after a glucose mouth rinse. Damage control from acid in the plaque, is achieved by dilution, chemical buffering, and by increasing the protective ions (mainly, calcium, phosphate and fluoride) in the environs of the teeth.[24,10] The water content of the saliva and plaque aid greatly in diluting the acid and in transporting acid into the main flow of saliva where it is further diluted and swallowed. This dilutional effect is supplemented by the buffering capacity of the plaque which can be 10 times higher than for the fluoride in the saliva. This higher adsorption capacity for fluoride in the plaque also occurs to differing extents in increasing bicarbonates, phosphates and ammonia concentrations derived from the saliva. These neutralizing actions serve as a brake in the rapidity and extent to which the pH can drop during periods of acidogenesis.

Each individul has a different potential for modifying the drop and recovery of the pH represented by his/her individual Stephan Curve. As an example, if a group of individuals is given a glucose mouth rinse, each person demonstrates a different, but reproducible pH pattern. Once the pH has started to fall, the availability of statherin

and other salivary buffers help to shorten the time that the pH is at its lowest and most damaging level.

The Tooth Compartment

Coronal caries involves the enamel cap[b] and the underlying dentin. Enamel is more mineralized than bone or dentin. It is estimated that enamel is composed of approximately 96% mineral by weight with an average volume of 87%. The enamel contains millions of enamel rods that run from the dentinoenamel junction (DEJ) to the tooth surface. The rods are approximately 4 to 7 micrometers, and by 6 to 8 micrometers in cross section for primary and permanent teeth, respectively. In cross sections they resemble keyholes, more than rods. Around each rod there is an enveloping protein matrix. During formation of the crown, this organic matrix forms the template that is involved in determining crystal and rod size and orientation (Chapter 3).

The inorganic phase of enamel is based on the mineral, hydroxyapatite (HAP), made up mainly of calcium (Ca), phosphate (PO_4) and hydroxyl (OH) ions. It also contains trace amounts of other elements that happen to be in the bloodstream during enamel formation, in fact more than 40 elements have been identified in analysis of enamel. Each rod is made up of millions of *crystals* each which are shaped much like a carpenters hexagonal lead pencil—one that is slightly flattened on two opposite sides between the submicroscopic *crystals* there are also *submicroscopic amounts of matrix.* These enveloping protein wraps of both the enamel rods and crystals are the main channels for diffusion for demineralizing acids and remineralizing electrolytes as explained in Chapter 3.

[In order to better understand the tooth histology at increasing magnifications, this is to invite you to join the following art and photographic tour featuring the "Anatomy of a Tooth." The starting point is Figure 11-1. You will need this information throughout your career.

Illustration 11-1a is a cross section of enamel, showing how each of the tails are cradled between the heads of the adjoining rods. The drawing 11-1b provides a concept of a single enamel rod.[c] With these two background schematics, the head and tail positioning becomes even more understandable when viewed on an electron micrograph, (Figure 11-2) that shows the rod as a crude keyhole structure. Figure 11-1 c is of a single crystal portrayed as a carpenter-shaped pencil configuration. Each crystal is composed of Ca, PO_4 and (OH) (and other extraneous contaminants). Each of the crystals making up the enamel rod is considered as a unit cell.(11-1d) A unit cell is the smallest subdivision of a crystalline substance that is entirely representative of the structure of the crystal. This means that all rods of any dimensions can be constructed (or remineralized) by adding additional unit cells, much as a building can be increased in size by adding additional bricks. It is important to recognize that unit cells, unlike the bricks, have no physical meaning as such; they are just a convenient means of conceptualizing the atomic structure and relationship of crystals at the simplest level.

If one unit cell could be detached along the c-axis, it would resemble a windchimes on a string, with each successive triangular grouping being comprised of calcium,

phosphate and hydroxl ions equidistant from adjacent groupings (Figure 11-1 e) When looking at the arrangement from the top of the column, the center position is occupied by hydroxyl ions, surrounded by a trianglular configuration with a calcium ion at each point of the triangle. Immediately peripheral to each calcium ion is a phosphate grouping (11-1f). Each successive triangular grouping grouping along the c-axis is rotated 180-degrees from the ones above and below, as illustrated by the solid and dotted lines in Figure 11-1 f). Each of the atoms can be replaced by other atoms. For instance, a hydroxyl group can be substituted by fluoride; a calcium ion by strontium, and a phosphate by a carbonate.

Next, let us take a more detailed look at how a *crystal* dissolves starting with Figures 11-1 c, and then illustrations in Figure 11-3 parts 1, 2, and 3 that show the sequence of dissolution of a crystal, which starts with a central *etch pit.* The etch pits on the basal faces are beautifully illustrated at electron microscope level, as are the images of hallowed out crystals shown in Figure 11-4, parts 1 and 4, respectively.

[b]If an intact tooth is stripped of all dentin and cementum, the remaining portion of the tooth is the "enamel cap".

[c]Enamel rods can be correctly called enamel prisms.

Demineralization

There were a few early interesting experiments by Silverstone who first focused worldwide attention to the overall subject of de- and remineralization. Several decades ago, researchers could not understand the reason why a typical cavity did not form when a tooth was directly placed in acid Instead the outer layers of the tooth would continue to dissolve, but there were no white spots. There were no incipient (subsurface) lesions. However, when Silverstone used an acidified pH *gel* (instead of an acid solution) in which to immerse the tooth, an incipient lesion *did* form with the expected four zones of enamel caries.[25-27] The surface zone had sufficient calcium and phosphate exiting from the body of the lesion to the surface zone to create a supersaturation of calcium and phosphate ions to cause a HAP precipitation *between the gel and the tooth surface.* The next study by Silverstone was to grind off the entire mature surface of the crown and again immerse the tooth in the buffered gel. The entire surface area of the tooth was recreated, showing that the outward diffusing minerals had attained sufficient saturation to precipitate and form the exterior of the enamel. This was interesting, but he carried the study one or two steps further towards practical application.

When a tooth with a carefully *preserved pellicle* was subjected to the same gel immersion treatment, there was the same build-up of mature enamel and closing of the *pores between the tooth surface and the pellicle.* He reasoned that the pellicle acted as a template to maintain the contour of the remineralized area. This demonstrated for the first time that *the pellicle served as a protective layer.*

When using saliva as the remineralization solution, the ability to remineralize tooth sections in vitro varies with the saliva from *different individuals*, but occurs consistently with the saliva of each individual, indictating that some people have a greater capacity for remineralization *(host resistance*) than others.

Fluoride has a major influence on both demineralization and remineralization.[28] Fortunately, only small concentrations of fluoride are needed to inhibit demineralization or to enhance remineralization. As little as 0.1 ppm fluoride can reduce the amount of enamel dissolution *in vitro.* The presence of fluoride at the remineralizing site can accelerate rehardening by a factor of up to fivefold. In the mouth, the fluoride can come from four sources (1) transitory contact with fluoridated drinking water; (2) the continual low fluoride ouput of the salivary glands; (3) the bound fluoride occurring in the plaque which is released when the pH drops to around 5.5; or, (4) from the fluoride contained in the mature enamel layer following demineralization.

Figure 11-1 Enamel: From the electron microscope to the molecule. a. An electron microscope model of the keyhole morphology of enamel. Note that the crystals (dotted lines) within any single prism are coaxial with the prism in the head region. From Meckel, A. H., Griebstein, W. J. and Neal. R. J. *International Symposium on the Composition, Properties and Fundamental Structure of Tooth Enamel.* April 1964. Courtesy: Ed. Stack, M. V., and Fearnhead, R. W., Bristol, England: John Write and Sons, Ltd, 1965. b. Individual enamel rod, showing different crystal orientations in head and tail. c. Illustration of a crystal with labels a, b, and c axes. d. Theoretic presentation of unit cells that make up the crystallites. e. Vertical arrangement of hydroxyapatite along C axis of the unit cell. f. Showing how every other molecular configuration is rotated 180° as illustrated by the solid and then the dotted lines. (Courtesty of N. O. Harris, University of Texas Dental School, San Antonio.)

Figure 11-2 Same electron micrograph as 3-2a. Same caption. (A repeat) Electron micrograph of rod cut perpendicular to long axis, showing head (H) and tail (T) relationship.

Figure 11-3 Dissolution of the crystal schematic: Each enamel prism is made up of parallel crystals of hydroxyapatite that have a slightly flattened hexagonal appearance. 1. The initial etching of the crystal begins at the ends with, 2. the formation of etchpits. 3. These etchpits deepen along the c-axis to eventually produce a hollow core. (From Arends J. Jangerbloed WL. Ultrastructure studies of synthetic apatite crystals. *J Dent Res,* 1979 [Special Issue B]; 58:837-843.)

Figure 11-4 Dissolution of the crystal, photographic. 1. Artificially grown apatite crystal with etchpit on basal face, original magnifiction × 500. 2. A hexagonal etchpit in fluorapatite, original magnifiction × 2500. 3. TEM picture of sound enamel crystallites, original magnification × 100,000; and 4. TEM picture of etched enamel crystallites that are partially hollowed out, original magnification × 100,000. (Courtesy of Dr. W.L. Jongebloed, I. Molenaar and L. Arends. University of Groningen, *The Netherlands and Joel News,* Japan. 1976; 13e(2):14.)

Question 3

Which of the following statements, if any, is correct?

A. The enamel is a solid piece of hydroxyapatite.

B. The crystal of a rod is the first component of the enamel cap to dissolve; it is also

the first to be reconstituted in remineralization.

C. A protein matrix envelops each crystal as well as each rod.

D. The central configuration of the unit cell is made up of calcium and phosphate, the OH is at the corners of the triangle.

E. It requires more acid of the same pH to dissolve a crystal than to dissolve the rod.

Remineralization

Remineralization is the *repair* of enamel rod structure following acidogenic episodes. When teeth erupt, they are anatomically complete, but crystallographically incomplete and immature. Following eruption, the missing ions are supplied from the saliva, a process termed *post-eruptive maturation*, Throughout life, minerals from the saliva are used to repair acid-damaged tooth structure. This repair process can range from an almost immediate replacement of daily ion losses from the enamel surface, to a slow repair (under proper conditions) of more extensive subsurface (white spot) lesions. Without specific knowledge of the caries process, a lay person is likely to envision the development of a caries lesion as a continuous process, accompanied by an ever-increasing loss of tooth mineral until the stage is reached when a clinically discernible cavity is present. Fortunately, this conception is incorrect. The process of demineralization is *not irreversible or inevitably progressive.* If damage has not progressed beyond a still yet to be defined point, lost mineral *can be replaced.*

Considerable clinical evidence exists for remineralization. Head, a physician and a dentist, pointed out in 1912 that teeth underwent cycles of softening and hardening.[29] By 1933, Boedecker[30] advocated the use of Andreasen's method of remineralizing "soft" teeth and "white spots." Andreasen's remineralizing powder consisted of tartaric acid, gelatin, calcium phosphate, calcium carbonate, magnesium carbonate, sodium bicarbonate and sodium chloride. Boedecker commented as follows: "The purpose that this powder is to fulfill, is to go into solution in the saliva and in this state, permeate and recalcify the porous area in the enamel . . . and after the remineralizing powder has been used for 6 weeks, decay around fillings will come to a standstill."

Muhler, in several clinical studies of the anticaries effectiveness of stannous fluoride, often found that the experimental subjects had more sound teeth later in the study than at the initial examination.[31] Invariably, the number of these reversals was greater in the stannous fluoride treatment groups than in the controls. Von der Fehr and colleagues were able to induce white spots with sucrose mouth rinses and reversed the process with fluoride rinses.[32] Backer-Dirks,[33] in a long-term study, noted that over 50 percent of the interproximal lesions seen at the initial examination did not progress, indicating an arrestment phenomenon due to remineralization. Additional support for remineralization is derived from the frequent observations of teeth that are acid-etched prior to placement of pit-and-fissure sealants. For those etched areas not covered with the resin, the chalky white appearance disappears over a period of a few days and the enamel regains its initial translucent, glossy appearance.

Except under unusual circumstances, such as occur following the destruction of the

salivary glands during cancer radiotherapy or diseases of the glands, deviations from remineralizing conditions in the mouth are transient. For example, the local pH may be lowered to where enamel demineralization occurs during the ingestion of acid foods or from the production of acid by the plaque bacteria following the ingestion of refined carbohydrates. If the insults are *brief* and *widely* separated in time, remineralizing conditions can be restored in the intervening periods and the slight damage repaired. On the other hand, frequent or protracted periods of acidogenesis, with insufficient time intervals for remineralization, ultimately lead to the development of overt caries.

Crystal Size in Demineralization and Remineralization

Siverstone, when he published the article, "The significance of remineralization in caries prevention." opened up a new area of conservative dentistry—an era that ten Cate calls "noninvasive restorative care." In his review of remineralization. Silverstone pointed out that crystal sizes differ predictably in each of the zones of the incipient lesion and in remineralized caries areas.[34] In the incipient carious lesions, the crystals in the two zones of *de*mineralization—the body of the lesion and the *translucent zone*—were smaller than in sound enamel. (Figure 11-5). The crystals in the two zones of *re*mineralization—the dark and the surface zones—were equal to, or greater in size than those found in normal enamel. Predictably, when a remineralizing solution with fluoride is used to remineralize the subsurface lesion, the crystal sizes are greater than for normal sound enamel.

Figure 11-5 Illustration of the relative crystal diameters in sound enamel (bottom) and in the four histological zones of the enamel lesions (right). (Courtesy of Silverstone LM. The significance of remineraliztion in caries prevention. *J Can Dent Assn.* 1984; 50: 157-184.)

Question 4

Which of the following statements, if any, are correct?

A. The concept of remineralization dates from the last quarter of the 20th century.

B. The crystals of the body of the lesion are larger than those of the dark zone.

C. An incipient lesion with a low pH and a low saliva calcium and phophate concentration is more likely to remineralize than one with a high saliva pH and is supersaturated with calcium and phosphate.

D. The anti-caries benefits of saliva during the Stephan Curve both slows demineralization and accelerates remineralization.

E. A remineralized rod in the presence of fluoride is a more acid resistant rod than one originally made up of hydroxyapatite.

Summary

It has been emphasized that oral disease, in fact, all disease occurs when the challenge

posed by pathogens exceed the body's capability for defense and repair. In the case of dental caries, the defense and self-repair mechanisms of the body operate continuously in the saliva, in the plaque, and in the enamel cap. Aside from the host's usual humoral and cellular defense functions to destroy pathogens, the oral cavity is protected by the senses of smell and vision, taste and tactile sensation, the body's immunological defenses, and the saliva. Demineralization is dependent on two major factors—pH of the plaque, and saturation of the tooth minerals. If the saturation is high and the pH high, demineralization will not occur. If both the pH and the saturation are low, the risk of caries is high. The output of resting saliva is moderate to low throughout the day; it is only through the period of the Stephan Curve that the maximum stimulated saliva protection occurs.

Bacterial acidogenesis in the dental plaque causes the plaque pH to fall and recover is a manner predicted by the Stephan Curve. If the maximum drop in pH is below the 5.5 to 5.0 range, demineralization occurs with the extent dependent on calcium and phosphate saturation level as well as the duration and frequency of the acid attacks. The increased secretions of haptins and statherin slow the drop in pH. An increased amount of salivary buffering minimizes the affect of the acidogenic end-products of the plaque bacteria. The increased flow of saliva, with its high fluid content, enhances the removal of cariogenic residues. As the pH drops, supersaturation of calcium and phosphate ions decline along the plaque-tooth interface. Ions, such as magnesium and carbonate that are adsorbed onto the tooth, dissolve preferentially and add to the buffering capacity of the local environment. When undersaturation occurs, somewhere between pH 5.5 and 5.0, calcium fluoride, HAP and FHA begin to dissolve in successive order. These dissolving crystals add to the saturation along the dissolving plaque-tooth interface, thus slowing and eventually arresting tooth demineralization. At that time, remineralization takes over. Ions necessary for mineral repair are again available from the inorganic components of the plaque that participate in the remineralization process and are ready for combating the next acidogenic cycle.

Answers and Explanations

1. A, C, and E—correct.

B—incorrect. The *palatal* glands are minor salivary glands. The correct answer should have included the submandibular gland, not the palatal gland.

D—incorrect. The order should be: parotid, submaxillary, and sublingual glands.

2. B, C, and E—correct.

A—incorrect. There can be teeth that overlap, the palate can be malformed and/or an abnormally large tongue can block saliva flow to some parts of the oral cavity. This problem of difficult-to-reach areas is best solved by a counseling session with a dental hygienist.

D—Bacteria need phosphate for energy; there is no way to tell the PO_4 from one source compared to another.

3. B and C—correct.

A—incorrect. The enamel cap is porous with over 10% of the spacing being between the rods and crystals—also in areas such as the hypomineralization of the DEJ, stria of Retzius, spindles and tufts.

D—incorrect. The central core of the HAP crystal is made up of mainly hydroxyl ions, but can include exchanged elements.

E—It requires much less acid to dissolve an individual crystal than a rod. (Just remember the rod is made up of crystals, not vice versa.)

4. D and E—correct.

A—incorrect. Remineralization is mentioned in *Dental Cosmos* (an early dental journal) prior to the turn of the 20th Century and became of interest to researchers in the mid 20th century. If is rarely used in private or public health practice in the United States. (Now routinely used in New Zealand and Scandinavia public health school programs).

B—incorrect. The crystals in the two zones of recrystalization—the surface and the dark zones are the larger.

C—incorrect. The higher the pH and saturation of the saliva the greater the chance for remineralization.

Self-evaluation Questions

1. The ability of the brain to continually monitor the location and action of a body part is known as _____.

2. The parotid gland produces the most amylase (enzyme to break down carbohydrates) and _____. (neutralizing agent).

3. The ability of the body to balance the factors causing disease, and the events promoting body health is known as maintaining _____.

4. The quantity and quality associated with Stephan's Curve in (resting)(stimulated) saliva is usually seen at the time of (eating)(fasting between meals). Circle correct responses.

5. Sialorrhea is best treated with (a saliva stimulant) (an antisialogogue).[d] Circle correct response.

6. A desalivated animal (glands removed) or a person with excised glands would have a problem with _____.

7. A pellicle acts to slow the transit of acid from the plaque to the subsurface _____.

8. The glycoprotein of the saliva that serves to lubricate the oral tissues (to reduce friction and abrasion) and to aggregate bacteria for swallowing is _____.

9. It was _____ (name of individual) who gave the major impetus to the modern basic concepts of de- and remineralization.

[d] *Antisialogogue = an antidote to sialorrhea.*

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Chapter 12. Caries Risk Assessment and Caries Activity Testing - *Svante Twetman Franklin Garcia-Godoy*

Objectives

At the end of this chapter, it will be possible to

1. State the purpose of caries activity tests.

2. Indicate the limitations and advantages of caries activity tests.

3. Identify the two bacteria most often measured in caries activity tests to determine the magnitude of the bacterial challenge to the teeth.

4. Understand terms used in caries-prediction tests.

5. Explain the general approach of caries-risk assessment.

6. List the background data of importance for caries activity.

7. Perform a clinical examination for evaluation of caries activity.

8. Name caries activity tests used in the dental office.

9. Cite new methods available to assess caries risk.

Introduction

Dental caries can be defined as a carbohydrate-modified transmissible local infection with saliva as a critical regulator.[1] The diagnosis is most often based on a clinical examination. Terms like primary and secondary caries, initial and cavitated lesions, white spot lesions, arrested caries, and root caries are often used in an effort to describe the activity and severity of the disease. Although these observations certainly are important, the modern dentistry diagnosis should be extended with identification and evaluation of factors *related* to, or the *causative agents of, the disease.* The multifactorial etiology of dental caries today is relatively well known and the disease is therefore not only a treatable, but in most aspects also a *preventable, infection.* Evaluation of etiologic factors can be made before clinical signs occur as well as in cases with already existing lesions or fillings. Subsequently, measures can be taken to reduce risk factors considered to create problems in the future. This chapter deals with different methods to determine caries risk and caries activity, focusing on the prevention of the disease. The presentation is focused on the description of clinical tests and methods that can be incorporated in the daily work and concentrated on the individual patient; therefore, the community approach is less discussed. For a more thorough discussion of the background of caries, the reader should refer to appropriate textbooks and overview papers cited in the references at the end of this chapter. To be able to discuss clinical implications, however, it might be helpful to take a closer look at the events leading to demineralization.

Caries—A Transmissible Local Infection

It is generally recognized that certain strains of *mutans streptococci*[a] and *lactobacilli* are highly cariogenic (reviewed by van Houte[1]). The former group plays an active role in the early stages of lesion formation while the latter is linked to the *progression of the cavity.* Evidence suggesting a microbial three-step event in caries development exists (Figure 12-1).[2]

The first step is the *primary infection* with mutans streptococci; the second is a local accumulation of mutans streptococci and other aciduric microorganisms to *pathogenic levels* in the dental plaque—a microbial shift that is an ecologic consequence of local acid conditions; the third step is the *demineralization and cavitation* of the enamel. Consequently, three levels of prevention—linked to the steps—could be defined, each with its own profile and characteristic.

- *Primary prevention* (step 1)—to prevent the intrafamily transmission of mutans streptococci and delay the establishment in infants, toddlers, and young children.
- *Secondary prevention* (step 2)—to prevent, arrest, or reverse the microbial shift before any clinical manifestations of the disease occur.
- *Tertiary prevention* (step 3)—focuses on limiting (stopping) the progression of the caries process by initiating remineralization therapy of the existing lesions.

[a]The term mutans streptococci includes several species of streptococci which historically were often collectively referred to as Streptococcus mutans.

First Step—Transmission and Establishment of Mutans Streptococci

Before the eruption of the first primary teeth, *no* mutans streptococci can be harbored permanently in the oral cavity because the bacteria need a *nonshedding surface* (i.e., a tooth) to colonize. From several studies it has been suggested that the transmission of mutans streptococci in most cases occurs vertical within the family. *The main source is the mother of the child*[3] but recent studies using DNA fingerprinting suggest that the child may also be infected by fathers and other caretakers from outside the family.[4] The most common routes of infection are close contacts and everyday nursing items such as pacifiers, baby bottles, and spoons (Figure 12-2). The colonization and establishment of mutans streptococci is highly facilitated by a frequent and sucrose-rich *diet of the parent as well as the child.* The higher the counts in mothers, the more frequent and the earlier risk for colonization in their children.[5-7] There are also a variety of other factors, such as salivary immunoglobulins and agglutinins, presence of competing bacteria, tooth anatomy, and pH, that might influence the colonization. Furthermore, the earlier the establishment and the more mutans streptococci present in children, the more caries is likely to develop in the primary and permanent dentition.[8,9]

The prevalence of mutans streptococci infection increases with age and the number of erupted teeth. Among toddlers, approximately 5 to 10% of a population harbor the bacterium and a rapid increase in prevalence up to around 50 to 60% in the late preschool ages has been observed.[b,10,11] In fact, it has been suggested that children are most susceptible for mutans streptococci colonization between *19 and 31 months of age, a so-called "window of infectivity."*[12,13] This is mainly explained by a combination of frequent and close maternal contacts, cessation of lactation with its protective antibodies, and an immature immune response of the child as well as an individual susceptibility. Approximately 80% of all adolescents and adults are positive for mutans streptococci, so the oral cavity can be regarded as a natural habitat for the bacteria.

[b]These data are representative for Sweden and the percentages may be different in other countries.

Second Step—Microbial Shift

Once a mutans streptococci- and lactobacilli-containing microflora is established in the oral cavity, there is a *risk* for future caries development. It is however a general misunderstanding that the disease is an inevitable result of the colonization. Instead, it is more common to harbor mutans streptococci without subsequent decay. A crucial process develops *if*, and *when*, the caries associated microorganisms turn pathogenic and this in turn is regulated and modified by local environment. Early in life, and especially in connection to nocturnal and nightly nursing bottle meals, the microbial shift is often *concomitant* with colonization while at later ages, the shift might occur at any time due to a disturbed homeostasis of the oral ecology.

Once established, the "normal" content of mutans streptococci and lactobacilli constitute less than 1% of the total microbial community in saliva and dental plaque. *During long-term acidic conditions, however, aciduric bacterial strains will be favored.* Lactobacilli are the most acid-tolerating of the species in the dental plaque and are able to maintain their metabolic activity down to pH 3.0.[14] Mutans streptococci are also highly aciduric and can grow at pH 5.0 and continue acid production to under pH 4.5.[15] Common reasons for the prolonged acid conditions are increased frequency of sugar intake, reduced oral clearance due to low saliva secretion or impaired buffer capacity, and plaque accumulation due to insufficient oral hygiene or interference by fixed orthodontic appliances. Consequently, the proportion of the extremely acid-tolerating mutans streptococci and lactobacilli will increase on the expense of nonmutans streptococci and other bacteria. The higher the proportion of acidogenic and aciduric microorganisms in plaque, the more acid is produced and a negative trend has been started. For example, the proportion of mutans streptococci in plaque associated with nursing bottle caries can be up to 30 percent of the total viable counts.[16] It is important, however, to point out that the microbial shift is a *local* event and it does not happen at the same time in the whole oral cavity. The shift is most likely to occur in fissures and interdental areas that are well-known predilection sites for caries. They are also the sites of greatest plaque accumulation. Furthermore, it should be stressed that microbial shifts *can be reversed* by temporary means (i.e., drugs, antibacterial agents) and permanent changes (i.e., diet alteration, sugar restriction) in the local environment. By using selective antibacterial measures, the cariogenic flora can be suppressed and a noncariogenic flora can be reestablished (reviewed by Emilson[17]). However, if the causative factors of the local shift remain unchanged, a recolonization back to pathogenic level is sooner or later likely to occur.[18] It should also be mentioned that the range of bacteria potentially involved in enamel demineralization has widened in recent years. There is evidence suggesting that Actinomyces species and "low pH," non-mutans streptococci under selected environmental conditions can contribute to caries in the absence of mutans streptococci.[1,19] In rare occasions, these organisms resembling *S. anginosus, S. mitis, S. gordonii,* and *S. oralis*, are thought to be responsible for the establishment of an initial low-pH environment.

Third Step—Demineralization of Enamel

At food intake, the accumulated plaque is fed with carbohydrates, which rapidly will be converted to organic acids, mainly *lactic acid*, through the metabolism of acidogenic bacteria.[20] This will further trigger the acidic milieu and a result in a local pH drop of the plaque fluids. During this drop, protons are diffusing into the enamel with calcium and phosphates leaving the tooth and a demineralization of the hard tissue occurs. From the time of the local microbial shift on a clinically sound enamel surface, an "incubation" period of approximately 6 to 9 *weeks* can be expected before the first visual signs of enamel demineralization ("white spots") on a smooth surface can be observed. In everyday practice, this is not an uncommon side effect associated with fixed orthodontic appliances.[21] Interdentally, however, it takes up to 9 *months* or more before the enamel lesion is visible as radiolucency on a bitewing radiograph.[22] This is a serious concern in modern dentistry that puts high demands on the diagnostic process. There is an evident risk that patients, who in fact have undergone a bacterial shift, still are clinically uneventful when examined and not given the appropriate preventive care.

In normal cases, after carbohydrate (sugar) intake the acid production diminishes when the bacterial substrate is consumed or washed away by saliva dilution. The pH will return to normal and a period of "repair" (remineralization) will again occur. This process is facilitated if fluorides are present locally. *The balance between net loss and net gain of mineral is crucial whether progression or regression of a lesion will occur.* It is important to stress that this is not a continuous process in either direction and that caries-active and caries-inactive periods will follow each other through the lifespan. A "white spot" enamel lesion *without cavitation* can be completely repaired through remineralization while a lesion that has reached the dentin, irrespective of enamel surface breakdown, has usually passed the "point of no return." Although it is unlikely that such a lesion can be totally repaired by aggressive remineralization therapy, a conservative attitude toward operative treatment is recommended favoring "watchful-waiting" and close monitoring during the preventive treatment period. Several clinical studies have demonstrated an extreme slow progression rate, if any, of enamel and dentine lesions subjected to effective and continuous prevention.[23,24] For example, the progression rate in enamel was found to be much slower than through the outer half of the dentin that, in turn, exhibited a median survival time of 3.1 years in a cohort of children that was followed prospectively from 11 to 22 years of age.[25]

Figure 12-1 Enamel demineralization - caries - can be described as a three-step event.

Figure 12-2 Common routes of mutans streptococci transmission: an infected dummy and spoon.

Caries Risk and Caries Activity

It is well known that certain individuals develop much more caries than others. The object of *caries prediction*[c] *and caries risk assessment*[d] is *to improve oral health* in children, adolescents, and adults and to utilize resources in a cost-effective way. Risk is defined as the *probability that a harmful (or unwanted) event will occur.* From the material presented above, it might be useful to separate caries risk assessment from attempts to determine the *caries activity.* By definition, a caries risk assessment is a procedure to *predict future caries development* before the clinical onset of the disease, thus limited to steps 1 and 2; while a caries activity test preferably should estimate the actual state of disease activity (progression/regression), as found in step 3 (Table 12-1).

The caries-risk assessment is performed in order to introduce causal measures before irreversible lesions have become established, while the caries activity test is carried out in order to decide and monitor correct and efficient treatment of a patient. When applied on populations, the caries-risk procedure is termed "caries prediction." As recently expressed by Hausen,[26] "clinicians assess risk, researchers predict." For the clinician it might be useful in this aspect to distinguish between *risk factors and risk indicators* (risk markers). A risk factor plays an essential role in the etiology and occurrence of the disease, while a risk indicator is a factor or circumstance that is indirectly associated with the disease.[27] Risk factors are the life-style and biochemical determinants to which the tooth is directly exposed and which contribute to the development or progression of the lesion (plaque, saliva, diet, etc.). Examples on risk indicators are socioeconomic factors (socially deprived, low education level, poor economy, self-esteem), factors related to general health (diseases, handicap), and

epidemiologic factors (living in a high-caries area or country, high past-caries experience). Caries-risk assessment as well as caries-activity evaluation are based on defined and selected risk factors and risk indicators that are evaluated and put together to an individual profile.

[c]Prediction is a clinical decision about the outcome of a disease process based on available information and professional experience.

[d]Risk assessment is a professional judgement of an individual's future risk of disease based on the best information available.

Terms Used in Prediction

To study the validity[e] of a caries diagnostic test and whether or not it is useful for caries prediction, the relation to caries incidence[f] must be established. It must be immediately pointed out that a close association in cross-sectional studies between en etiologic factor or its measure in a test on one hand and caries on the other does not necessarily mean that it also is a powerful predictor of the disease. The predictive ability of caries tests or risk indicators must therefore be evaluated in longitudinal studies. The results are generally expressed in terms of *sensitivity*, *specificity*, and *prognostic values*. The proportion of diseased subjects whose test (or risk factor) is positive is termed sensitivity. Similarly, the proportion of non-diseased subjects whose test (or risk factor) is negative is called specificity. The predictive values are perhaps of higher interest for the clinician, and a higher predictive value indicates a more valid test. The positive predictive value (PV1) denotes the probability of an individual to develop the disease, while the negative predictive value (PV2) gives the probability to stay healthy. All the above measures should be looked at as pairs. For instance, knowing the sensitivity of a caries test has a limited meaning if one does not know the respective specificity of the test. More recently, *odds-ratio values*, referring to the chance of an event versus the chance of a nonevent, have been introduced in prediction models. The odds-ratio provides information on the chance that the disease will occur given a specific condition. It must, however, be understood that all values above are highly dependent on a number of factors that must be defined and considered to be able to understand the predictive power:[28]

- The level of caries prevalence and incidence in the study population or study group
- The methods used for data collection and especially the criteria for caries scoring
- The validity of the test method
- The number of tests and/or the combinations of tests applied
- The patient's access to preventive and restorative care
- Age of the participants

For instance, the predictive value of a mutans streptococci saliva sample is dependent on the disease level. It is fairly easy to understand that it is harder to predict the eruption of a rare disease compared with a common one. When a test is applied to a population with a high prevalence of disease compared to a population with a lower prevalence, the positive predictive value will increase and the negative predictive value will decrease. The definition of caries activity (disease) is of course very important. Were only new lesions or also progression of previous lesions considered? Is a certain number of new cavities within the dentin or a single early enamel lesion

regarded as disease? The selected cutoff points for positive tests and disease and the motivation why they were chosen must be considered. Furthermore, *restorative treatment and preventive efforts conducted in predictive investigations may obscure the predictive power of the tested model.* Environmental factors, such as the natural content of fluoride in the drinking water, can interfere with the predictive process.[11] The predictive power may also be influenced by age. It is generally believed that caries risk assessments are more accurate in preschoolers compared with older age groups and adults. For instance, Swedish investigators demonstrated in a prospective study that mutans streptococci colonization, immigrant background, consumption of candy and mothers' level of education were significant predictors for caries before 3.5 years of age.[29] When all these variables were present at 1 year of age, the probability for caries development was 87%. Thus, one can state that predictive values are valid *only* for the population studied and for the *given time* when the investigation was conducted. This does not mean that such studies lack merit—on the contrary—considerable experience has been gathered within the field and forms the base for many national oral health care programs.

The number of false responses following a test should naturally be as few as possible. For caries, however, the occurrence of *false-positive* answers are less crucial as long as they only result in an intensified prevention program and not in unneeded restorations. *False-negative* results, on the other hand, can seriously jeopardize dental health due to neglected treatment.

[e]Validity means sufficiently supported by actual fact, sound, good or effective means.

[f]Caries incidence is the number of lesions that occur over a given period of time. Thus, two examinations are required to determine incidence; one before and one at the end of a selected period.

General Approach of Caries Risk Assessment

As discussed earlier, a risk-assessment strategy can be applied on three different levels, each with somewhat different aims: (1) *for populations,* (2) *for groups,* and (3) *for the individual.* For preventive strategies today, it is important to distinguish populations with contrasting prevalence of the disease. On a national level, it seems important to evaluate the actual "challenge" to the teeth and data can be obtained through caries tests in epidemiologic investigations. Such findings evaluated in combination with, for example, sugar-consumption figures, are of highest relevance for oral-health planners. Based on such data, the community can implement preventive programs or dispense health care resources in a cost-effective way. The same approach can be applied to groups for which the current prognostic value can be established. For the individual, caries activity tests can map etiologic factors for caries and serve as a measure of compliance to a given treatment. However, before describing the methods used in the risk assessment procedure, it is valuable to discuss historically why the risk approach has gained so much attention during the recent decades.[30]

In communities with a high prevalence of caries, the need for a risk approach is limited. This was the situation in most industrialized Western countries a few decades ago. A whole population strategy with general preventive measures given to

everyone, such as water fluoridation and fluoride supplements, were highly cost-effective since the vast majority of the population benefited from these programs. Hence, caries declined rapidly and the polarized distribution of today became evident.[31] A large and increasing proportion of the population became caries-free, while others still exhibited a high caries activity. In order to limit the costs for dental health care in a period of economic recession and limited resources, the efforts were then directed to the relative minority of individuals that developed oral diseases.

Consequently, a risk approach was instituted and caries-risk assessment became a part of dental practice. After caries-risk assessment, the persons with the highest need for treatment were provided intensive targeted actions with individualized recalls. Low-risk individuals did not receive further attention with recall periods extended up to 24 months. Epidemiologic and analytic surveys, however, soon identified certain subgroups of individuals with a high prevalence and incidence within the skewed caries distribution.[32] Common examples are ethnic and cultural groups, immigrants (especially refugees), and inhabitants of low-socioeconomic areas. Furthermore, medically compromised individuals could also constitute such a group. In these groups and subgroups, screening procedures of children at various ages in school have been proven rational and cost-effective. However, in comparison with whole population strategies, the general effects of the individualized approach are less evaluated and documented. Therefore, *a balanced mix of a risk selection strategy and collective measures seem to be the "state of the art" in many communities today*. It should be emphasized that in an extreme low-caries population where practically no persons develop caries, a risk assessment would be of little use. It has also been claimed that the risk selection procedure, after proper education, can be *delegated to auxiliary personnel*, which may reduce the cost for the patient and the society.[33]

Question 1

Which of the following statements, if any, are correct?

A. Caries can be defined as a carbohydrate-modified transmissible local infection.

B. A caries *risk factor* incidence test requires only *one* dental examination.

C. Sensitivity of a caries activity test denotes the probability for caries development.

D. A risk-assessment strategy is most important in communities with high-caries prevalence.

E. For preventive care, a false-positive caries-activity test would probably be more advantageous to the patient than a false-negative test.

Community and Group Approach of Caries Risk Assessment

Since the prevalence of caries in many countries shows a skewed distribution, the interest of finding methods for prediction of individuals or groups of patients at risk within the community has become widespread. *The methods used for this identification are predominantly based on past dental caries, oral hygiene, dietary variables, microbial and salivary factors, and social variables.*[34,35] It has been

proposed that both the sensitivity and specificity must be at least 80% to be useful in caries-predictive models or when added, exceed 150%.[36] In practice, this means that every fifth individual with a true high risk would remain undetected and not receive intensified prevention. Correspondingly, every fifth individual with a true low risk would receive treatment with no or little effect. Furthermore, it is thought that a risk model should select not more than 20 to 30% of a population believed to be at risk to be manageable. With such precautions, the sensitivity of the bacterial tests and past caries experience, single or combined, are commonly reported within the range of 60 to 80%. On the other hand, the prediction of patients at low risk seems more reliable than those at high risk with a specificity around 80 to 90%. This means that it appears to be more relevant to select patients with low risk for future caries development than predicting those at risk.

Due to the complex and multifactorial etiology of dental caries, it is generally thought that a multivariate approach rather than the use of single variables improves the predictive ability. Biochemical variables have been combined with sociodemographic and dental behavior data.[37,38] However, results from recent studies using such multivariate methods have been unexpectedly poor.[35,39] The accuracy proved to be much lower than anticipated considering the power of the individual predictors. In fact, the highly complicated relationship has raised the question whether prediction of caries with reasonable, simple, and inexpensive methods will ever be a reality. While the prediction of caries risk using currently available methods is useful in certain communities with a skewed caries distribution and in groups of individuals with medium and high caries levels, its value in a low caries community can be questioned. The clinical value of caries *prediction models,* therefore, may differ from population to population and from children to adults, as recently reviewed by Powell.[40]

The number and the degree of sophistication of predictive methods are of course limiting factors in the population because of practicalities and costs. Therefore, few and strong variables have to be advocated on the population level while a higher degree of combinations can be utilized in smaller groups and for the individual. An example of a very simple community protocol for risk selection is given in Table 12-2. The concept can—and should—of course be altered depending on local conditions and the age of the target group. The past caries experience is thought to be the most powerful single indicator of future caries development in children.[39] It can however be argued that it does not precede the disease but rather is a result of an already existing—or treated—disease and represents an accumulation of long-term disease experience. Thus, considerable interest has been focused on anticipated risk factors and risk indicators, i.e., simple chairside methods for saliva evaluation, directly dependent on the state of oral microbiology at a given time, which will be discussed below.

Ages of Interest for Caries Risk Assessment

Data on the time when caries-risk assessment is most cost-effective in childhood are sparse. *It is well known, however, that all newly erupted teeth are more or less deficient in mineral content and thus more susceptible for caries than after some years of posteruptive maturation.*[41] Moreover, the eruption of teeth constitutes a caries risk *per se,* since new surfaces become available for the disease. Therefore, in this aspect it might be possible to define certain *risk ages* of utmost importance of risk

assessment procedures.

An early dental examination and diagnosis must be regarded as extremely important for efficient preventive intervention. Recent studies have unveiled that approximately 75% of all cavities found in 2-year-old children were located in the upper incisors and that 90% of those exhibiting caries in the upper front also developed cavities in the primary molars.[42,43] Furthermore, the primary molars are caries-prone between 4 and 6 years of age,[44] and the eruption of the first permanent molars constitute a well-known occlusal risk. Finally, the early teenage period (12 to 16 years) offers a high number of newly erupted surfaces susceptible for decay. Based on recent epidemiologic data on caries incidence, the "key ages" as suggested in Table 12-3 might be considered for caries-risk assessment.

Individual Approach of Caries Activity

For an appropriate assessment of caries activity, facts from the case history, clinical and radiographic examinations, dietary history, and supplementary laboratory tests must be taken into account.[45] An individual approach has been suggested and described by Bratthall and Tynelius-Bratthall.[46] According to their method, biochemical and demographic parameters should be combined with a *clinical judgment* ("gut feeling") of the dental professional to elicit proper results. First, determine which particular factors are involved. Next, find out *why* these factors are present. Finally, try to change the situation by targeted actions against identified factors. Some examples are given in Table 12-4 to illustrate this concept.

When reviewing the findings of an individual, the trading of multiple pros and cons becomes very complex to evaluate. For example, think about two caries-free children with contrasting levels of mutans streptococci in their saliva. If each has the same diet, it is very likely that the child with the high counts will develop more caries than the one with low counts. However, if the child with low counts eats candy frequently and the one with high counts has a very restricted sugar intake, which one now has the greatest chance of developing caries? The question becomes harder to answer even though most professionals probably would have an opinion. Imagine in the next step that the first child with low bacterial counts and frequent sugar intake is supplemented with fluoride and the other is opposing the use of fluoride; who will, under such circumstances, develop the most caries? Is the buffer capacity contrasting? In this way, by adding several aggravating and counteracting risk factors and risk indicators, there are thousands of possible combinations to consider. To be able to handle this, it is important to realize that a risk value, such as high mutans streptococci levels, indicates a certain "pressure" on the teeth. With a proper diet and optimal fluoride administration, the risk can be controlled. When several risk values are disclosed, a very strong pressure is evident and more counteracting factors are needed to balance the situation. Suggested cut-off values for commonly used risk factors in pediatric dentistry are listed in Table 12-5. It can be recommended to use interactive software tool that are developed as an aid for the clinician in the caries predictive process. Such an example is the educational "Cariogram."[g,47] Graphically, the PC-program maps the interaction between caries related factors, a process known as cariography (Figure 12-3). Background data and clinical findings, with their varying impact on caries, are entered into a computer and the factors are weighed against each other forming a "risk profile" of the patient. The program can be adjusted for local conditions such as

socioeconomic status and fluoride content in the piped water. The *chance*, expressed as percentage, for a person *to avoid new decay* is thereafter presented graphically on the screen. The program also provides an individualized suggestion on suitable preventive activities when needed. The Cariogram concept has recently been evaluated in a prospective study in which the assessed risk at baseline in a group of 10 to 11-year-old schoolchildren was compared with the actual caries increment after 2 years.[48] Although the general caries incidence was low in the study population, the Cariogram was the most powerful explanatory variable. For example, children in the highest risk group (0 to 20% chance to avoid caries) had 50 times higher risk (odds ratio) than the children in the lowest risk group (81 to 100% chance to avoid caries).

We will now focus and comment on the clinical implication of caries-risk assessment of the individual, covering background data (case history), clinical examination, and caries activity tests.

[g]

Figure 12-3 The Cariogram—an educational interactive PC-program for caries risk evaluation.

Background Data of Importance for Caries Activity

The background factors that *directly or indirectly* can be of importance for the disease usually belong to one or more of the following groups:

- General diseases
- Medication
- Social/family situation
- Dietary habits/feeding
- Oral-hygiene routines, fluoride support

When interviewing the patient for the case history, questions should be asked to clarify these points. There are few general diseases that directly affect the teeth, although there are several that *indirectly* influence the carious process. In fact, and especially regarding children, being ill with medication in combination with anxious and sometimes overprotective parents constitutes a greater caries risk than the disease itself. Several drugs contain a high content of fermentable carbohydrates and have a low pH. Furthermore, the depressing influence on saliva secretion exerted by various medicines is a well-documented risk.[49] A troubled family or social situation might be reflected by factors such as stress (decreased saliva secretion), lack of interest in hygiene (poor plaque control), and low income (cariogenic diet). The topic of diet and caries is vast and needs its own textbook to be adequately covered, but it can be stated that the diet clearly affects the teeth in a direct (in the form of erosion) and indirect (through tooth formation, saliva secretion, and bacterial activity) way. In developing a risk profile, the diet should always be considered. An interview or a "3-day record" of all food and snack intakes are common methods used to obtain information about the diet of a patient.

Clinical Examination for Evaluation of Caries Activity

One aim of the clinical examination is to get a quantitative estimation of the caries problem up to the present and another aim is to reveal if the disease is ongoing or if observed lesions or fillings reflect a past disease activity. A few important points for estimating caries activity are discussed below.

For the individual patient, it is important to collect data in a standardized and systematic way. As stated earlier, the past caries prevalence is always important, but one is not necessarily at risk in spite of 30 previous fillings. It is very useful to try to look "behind" the recorded decayed, missing, or filled primary or permanent teeth (dmft/DMFT) values as indicated in Table 12-6. First, one should consider if there are more or fewer fillings or extractions than considered normal for a particular age group. The patients should then be asked questions such as "*Why* and *when* were the teeth extracted (caries, periodontal disease, orthodontics), or were the restorations placed long ago or recently?" Check the number, extension, and appearance of the lesions, cavities, and fillings. The texture and localization of lesions might provide important hints for caries activity (Figure 12-4). For example, presence of early enamel lesions ("white spots") on newly erupted teeth indicates an active demineralization process. The evaluation will provide the examiner with information on the extent of the problem and if caries seems to be a past or present problem. In the next step, local aggravating factors such as crowded arches, deep fissures, imperfect fillings and exposed root surfaces are evaluated. The morphology of the enamel must always be checked. Although the present evidence supporting an inherited susceptibility to dental caries is limited, altered enamel development, such as increased porosity and decreased mineral content are directly linked to an increased caries risk.[50] Finally, the estimation of the oral hygiene standard with a disclosing solution can be recommended. It should be emphasized that visible plaque on the labial surfaces of maxillary incisors of a young child is a serious sign of caries risk.[51]

Figure 12-4 Caries in a disturbed homeostasis. At the clinical examination, inspect carefully the appearance of the lesion. An active lesion, as in the figure, is often soft light in color, or surrounded by enamel with early whitish demineralization. An arrested lesion is usually darker with a harder surface.

Caries-Activity Tests for the Dental Office

It is well known that dental caries has a multifactorial and complex etiology, and unfortunately, there is no single test available that can fully explain or predict the disease. The term "caries-activity test" may furthermore be misleading since, at best, information can only be obtained on selected factors of importance for the process. Ideally, laboratory tests should be simple, inexpensive, rapid, and accurately reflect the three overlapping circles presented by Keyes[52] in 1962: (1) the bacterial challenge, (2) the sugar content of the diet, and (3) tooth and host resistance (susceptibility) with remineralization potential. In the light of these requirements and circles, the following set-up of tests might be suggested:

- *Bacterial challenge*—determination of mutans streptococci as an indicator of relative risk.
- *Diet*—determination of lactobacilli as an indicator of sugar content in diet.
- *Remineralization potential*—salivary flow rate and buffer capacity as an indicator of potential biologic repair.

• *Host suspectibility*—caries experience as an indicator of past activity.

After sampling, the clinician can choose between sending the tests to a fully equipped microbiologic laboratory or to use commercial test kits that can be processed within the dental office. In both cases, the results will be available after a few days. The simplified testing methods now available can be characterized as semiquantitative, although they are generally considered to significantly correspond with conventional agar plate techniques.[53,54] The most common sources for sampling are saliva and dental plaque. *The saliva test gives an overall estimation whether or not the patient is colonized and reflects the number of colonized surfaces and the prevalence in plaque.*[55] It does not however indicate where the bacteria are harbored, which is important to understand since the cariogenic microorganisms colonize the dentition in a milieu-regulated, localized way.[56] The plaque sample can therefore be used for a detailed "mapping" of the patient's dentition with special references to selected sites. This approach enables an assessment of not only *at-risk individuals* but also *at-risk teeth* and even *risk surfaces.* Salivary tests are generally more practical than tests based on plaque since the collection is less demanding. In addition to the diagnostic value of saliva and plaque tests, the didactic properties as an individualized patient-motivating tool in caries prevention are today widely acknowledged (Figure 12-5).[57-59]

When Should Tests Be Used?

This issue has been lively debated through the last decades. It is, of course, not realistic or even justified by cost-effective means to test all patients living in a low-caries society each time they are recalled or with certain regularity. A simple answer is that a test should be performed each time there is a need for extra information on factors of importance for caries—quantitative as well as qualitative.[46] Thus, selected tests could be justified in several clinical situations to:

• Clarify the reasons behind an ongoing disease and formulate and motivate the preventive strategy to the patient
• Determine the effect of a causal treatment at follow-up visits
• Predict caries development (i.e., make a prognosis) at check-up visits

For the selected subjects, repeated tests are preferred compared to single in order to establish the normal values of the patient and be able to monitor any deviation from the "norm," indicating an altered oral environment. For example, if the unstimulated secretion rate suddenly decreases, this is a sign of an altered ecologic environment with increased caries risk and should be followed by a closer check-up of the medical and psychosocial conditions and drug intake.

Mutans Streptococci Counts

As already mentioned, mutans streptococci are strongly associated with the initiation of dental caries.[1,60,61] They have several cariogenic properties that are enhanced in the presence of sucrose. The most important are the ability to:

• Colonize and grow on nonshedding surfaces
• Produce acids, acidogenic

- Withstand low pH, aciduric
- Form and store extra- and intracellular polysaccharides
- Tolerate high-sucrose concentrations

The human dentition is the natural habitat for mutans streptococci (Figure 12-6). They have a localized way of growing which means that in one individual's mouth, some teeth may harbor bacteria while others do not. The levels of mutans streptococci in saliva and on the tooth surfaces (plaque) reflect the number of colonized sites in the mouth.[62] The *higher the mutans streptococci count on the teeth, the more caries.*[63] A number of different strains can be found in humans and the prevalence seems to vary by age and population. The most common inhabitants in humans worldwide are *Streptococcus mutans* and *Streptococcus sobrinus.*[64] About 10 to 30% of a population have low levels of the bacterium, while 10 to 50% are highly colonized.

The quantitative evaluation of mutans streptococci in saliva and plaque is performed at the laboratory on agar plates with a selective media, the mitis-salivarius-bacitracin agar.[65] A serial dilution of the sample is performed and aliquots are placed on the agar surface with a pipette. After 4 days of anaerobic incubation, the number of colony forming units (CFU)[h] are counted. The bacteria have a significant morphologic appearance and it is possible to distinguish between various strains. Variations of this technique have been suggested to adopt and facilitate use in the dental office, i.e., direct impressions on a wooden spatula on an elevated agar.[66] However, the short storing duration of the plates in combination with demanding incubation requirements make the plate counts best suited for the research laboratory.

[h]Colony-forming units (CFUs) denote the number of visual bacterial colonies that are formed following incubation, not the actual number of bacterial cells. Each colony can consist of many single bacteria.

Strip Mutans Method for Mutans Streptococci Counts

Several simple chairside methods have been developed in recent years for the estimation of mutans streptococci levels in saliva.[67,68] These simplified methods, however, are not only used in the modern dental office but also in dental research as described in numerous papers. The most common method today is the *Strip mutans* technique (Dentocult-SM)[i] developed by Jensen and Bratthall.[69] This method utilizes the ability of mutans streptococci to grow on a hard surface in a selective mitis salivarius broth containing 20% sucrose. The kit includes a specially prepared rounded plastic strip for the sampling. The surface of the strip is slightly roughened on one side to promote bacterial adherence. Fifteen minutes prior to the sampling, a 5-mg bacitracin tablet is added to the broth. As the bacitracin can be added just before use, the shelf-life of the test is prolonged compared to agar plates. After 2 minutes of paraffin-chewing, the plastic strip is rotated a couple of times on the dorsum of the tongue and then withdrawn *through lightly closed lips*, hereby coated with a defined amount of saliva film. The strip is then immediately attached to a cap that is screwed to a glass vial, and incubated at 37°C for 48 hours.

The mutans streptococci colonies will appear on the strip as small blue dots but the color can vary from dark blue to pale blue. The density of the colonies is evaluated against a chart provided by the manufacturer (Figure 12-7) and scored 0 to 3, where

the scores 2 and 3 correspond to approximately 1×10^5 CFU and $>1 \times 10^6$ CFU/mL saliva, respectively. When dry, the strips can also be evaluated and divided into groups with the aid of a special device in a stereo microscope with 6-25 × magnification.[70] The strip mutans technique has been proven reliable to use and significantly related to conventional techniques. Sometimes, gas-forming or other nonmutans bacteria can grow in the broth (grayish), but not on the strip. Furthermore, large mutans streptococci colonies may be found on the bottom of the tube. They normally fall from the smooth side of the strip, but usually this will not affect the scoring. A useful advantage with this method is that the strips can be stored for years in a plastic foil for future comparisons.[71]

A further modification of the Strip mutans technique has recently been developed for *site-specific* microbial plaque diagnosis.[72] The sampling of selected sites is carried out either with a wooden toothpick[73] or a small saline-wetted brush[74] and transferred straight across the plastic strip on an elevated pad, thus enabling four sites to be sampled on each strip. After the cultivation procedure as described above, the CFUs are counted and scored on the predetermined area in a microscope or by aid of a chart (Figure 12-8). This method is especially useful for monitoring the outcome of a site-specific antibacterial treatment.[75]

[i]Available from Orion Diagnostica, Helsinki, Finland.

Lactobacilli Counts

Lactobacilli constitute an acidogenic and aciduric group of microorganisms associated with dental caries.[76] The bacteria need retentive sites for the colonization of tooth surfaces, such as fissures, fillings, gaps, overhangs, etc. Lactobacilli are often found in the deep parts of the caries lesion. Thus, they are considered as secondary invaders and responsible for the *progression of already established lesions.*[14]

Lactobacilli levels are highly influenced by the intake of dietary carbohydrates, thus reflecting the amount of bacterial substrate and indicating an acid environment within the oral cavity. The prevalence of lactobacilli is lower compared with mutans streptococci. Approximately 50% of a population exhibits low values while 10 to 20% have high counts.[77] It could however be noted that reports from Scandinavia suggest a decreasing prevalence of lactobacilli in children[78] in spite of unchanged or even increased sugar consumption, and this may partly be explained by diminishing numbers of retentive sites (cavities and restorations) in the young population. The proportion of lactobacilli in the plaque is normally low (<1%), and the presence in saliva and plaque are determined using a selective medium (Rogosa SL-agar[79]) with conventional culturing methods at the laboratory.

Dip Slide Method for Lactobacilli Counts

The number of salivary lactobacilli can be estimated with the aid of the Dentocult-LB[h] method,[80] consisting of a plastic device covered with selective agar. Paraffin-stimulated saliva is collected in a cup or a tube. The saliva is poured over both sides of the slide, and the excess allowed dropping off. The slide is then inserted in a plastic container and incubated at 37°C for 4 days. After incubation, the lactobacilli appear as small whitish dots and the number on the agar surface is estimated by comparison

with a chart supplied by the manufacturer (see Figure 12-7). As an alternative, the slide can be incubated in room temperature for 7 days. However, this may lead to an increased recovery of yeasts, which may jeopardize the evaluation of the slide.[81] The results of the test can be shown directly to the patient but the slides cannot be stored for longer periods, unless they are stored in a computer with the use of a video camera.

[h]Colony-forming units (CFUs) denote the number of visual bacterial colonies that are formed following incubation, not the actual number of bacterial cells. Each colony can consist of many single bacteria.

General Comments on Bacterial Values

Most selective media underestimate the real number of targeted bacterial strains. This does not impair the value of the sample as long as the levels are within those of clinical relevance. It is highly advisable not to regard the test results as exact bacterial numbers but rather as *ranges* of bacterial counts. Furthermore, it must be emphasized that counts obtained with different methods cannot be directly compared with each other. There is no apparent association between mutans streptococci and lactobacilli counts although a tendency to high levels of *both* species often can be seen in caries-active patients.[82] This fact emphasizes the assumption that the tests measure two separate stages of the caries process in the oral milieu and cannot be substituted by each other.

It is recommended not to have a "fixed" position to threshold values regarded as a caries risk. The risk level for one factor depends on the influence of other factors. One million mutans streptococci per ml of saliva may, under certain conditions, lead to cavity formation, but with a proper diet and fluoride administration, the risk will be considerably lower. For example, a certain level of bacteria or saliva secretion rate does not mean the same for an individual living in a fluoridated area compared with an individual from a low fluoridated or nonfluoridated area.[83,84]

A common question is to what extent must the sampling for the use of the tests be standardized. It is well known that a number of factors such as antibiotics, diet, smoking, toothbrushing, saliva secretion, and retentive sites can affect the number of bacteria in the oral cavity.[28] A normal variation over time of both lactobacilli, mutans streptococci, buffer capacity, and saliva flow rate should always be expected.[85] Studies have shown that *pronounced* natural variations are rare in the short-term perspective.[55,71] The highest bacterial counts in saliva are usually found in the morning before toothbrushing. During daytime, the levels seem to be fairly stable if no particular measures are taken. In epidemiologic surveys, a strict and well-defined collection procedure is of course crucial while such precautions are somewhat limiting for the daily routine work. For the individual patient, the results must be evaluated in the light of practicalities—they represent the *challenge at the time they were taken.* However, check if the patient is taking or recently had (within 1 month) an antibiotic medication and, if possible, try to avoid sampling just after toothbrushing or eating.

Figure 12-5 Saliva tests are useful as didactic and motivating tools in caries prevention.

Figure 12-6 Close-up of mutans streptococci, cultivated on MSB-agar (A) and as appearing in a scanning electron microscope (B).

Figure 12-7 A chart for evaluating chairside saliva tests as indicated. Four classes are used for bacterial enumeration while three levels are used for buffering capacity.

Figure 12-8 A specially designed strip for site-specific enumeration of mutans streptococci in plaque.

Question 2

Which of the following statements, if any, are correct?

A. A valid test can be both accurate and reliable.

B. Presence of mutans streptococci in the oral cavity always implies a caries risk.

C. A useful caries predictor should have a strong and stable association to caries prevalence.

D. A better correlation usually exists between low-bacterial counts and low-caries risk than between high counts and high risk.

E. Lactobacilli, being highly aciduric, are linked to the initiation of the enamel lesion.

Saliva Flow Rate

An appropriate flow of saliva is essential for the maintenance of oral health. It is evident that the oral bacteria are subjected to several important salivary functions, which affect their colonization, survival, and metabolism. The most important mechanisms by which saliva can affect caries are:

- Mechanical cleansing of debris and plaque bacteria
- Antibacterial activity against the oral microflora, i.e., lysis and aggregation
- Buffering and neutralization of plaque acids
- Enhancement of remineralization

The salivary flow from both major and minor glands is controlled by parasympathetic (water, electrolytes) and sympathetic (proteins) stimuli. The water fraction is most important for the clearance process while the antimicrobial activity resides mainly in the protein fraction.

Salivary flow rate is considered as a "key" parameter in caries-risk assessment.[86] Although there is no linear association between salivary flow rate and caries activity, it is important to evaluate whether the secretion is normal or impaired. Absence of saliva, xerostomia, or hyposalivation can result in an extremely increased caries risk. A decreased flow rate is a common side effect to a large number of medicines and radiation therapy.[87] For the individual, a regular and longitudinal followup of the flow rate is of higher clinical value than a single measurement to be able to identify reduction and alterations over time.[85]

Measurement of Saliva Flow Rate

When measuring the flow rate, one can either sample unstimulated or stimulated whole saliva. In addition, saliva from separated secretions, parotid or submandibular/sublingual, can be collected. Stimulated whole saliva samples are *most often* used for routine work. The stimulation can be done by paraffin chewing or by adding droplets of a sour liquid (3% citric acid) on the back of the tongue. The amount of saliva obtained is *divided by the collection time* and the secretion is expressed as ml/minute or mL/5min.

For adult patients, a normal stimulated secretion rate is around *1.0 to 1.5 mL/minute*. Values below 0.7 ml/minute should be considered as low and indicate a caries risk.[88] Women generally have somewhat lower stimulated and unstimulated secretion rates than men. In children, the levels highly depend on age and cooperation, but the corresponding levels in preschoolers for stimulated and unstimulated secretions are around 0.5 and 0.3 mL/minute, respectively.

For collection of unstimulated (resting) saliva, the patient is seated in an upright relaxed position with the head bent forward. The subject lets the saliva passively drip into a graduated tube for 5 to 15 minutes. An unstimulated secretion of less than 0.1 mL/minute is considered as a risk value. In cases of hyposalivation, the saliva is often viscous and "foamy" and the secreted volume is difficult to determine. A gravitation method is therefore advocated. The test tube is weighed before and after sampling and 1 gram corresponds to approximately one milliliter of saliva.

Question 3

Which of the following statements, if any, are correct?

A. An average collection of 7.5 mL of stimulated whole saliva over 5 minutes is considered abnormally low.

B. A lactobacilli test reflects the carbohydrate intake and retentive sites in the oral cavity.

C. A saliva sample provides accurate information on where in the mouth cariogenic bacteria are harbored.

D. Caries-risk assessment for the individual gathers data from case history, clinical examination, and laboratory tests.

E. A person with a mutans streptococci score of 3 (high counts) cannot stay caries-free and demineralization will inevitably occur.

Buffering Capacity of Saliva

The buffering capacity of saliva is important for the maintenance of normal pH levels in saliva and plaque. A low secretion might indicate a low buffering effect and a weak inverse relationship to caries has been noted by several investigators.[89,90] Both the

saliva secretion rate and buffer capacity differ however at different parts of the mouth. The composition and acidogenicity of plaque may be affected differently when situated close to a salivary duct or hidden deep down in a fissure. Nevertheless, unfavorable values of buffer capacity and salivary flow rate should be considered as risk factors for the individual. The tests commonly used are based on the titration technique with the final pH determined by a dye color change.

Dentobuff Method for Measurement of Buffer Capacity

A simple chairside method to measure the buffer capacity of saliva, the Dentobuff strip[j], has been developed by Ericsson and Bratthall.[91] A small amount of acid is impregnated on a pH indicator strip. One droplet of stimulated saliva is placed on the testing pad of the strip in a flat position to dissolve the acid. After exactly 5 minutes, the color of the strip is compared with a provided chart, indicating the final pH. The method reflects mainly the bicarbonate buffer system and identifies saliva with low (yellow), intermediate (green), and normal (blue) buffer capacity (see Figure 12-7). It is important that the test is read after exactly 5 minutes as color will change with time and thus give misleading results. The yellow color indicates a final pH of 4 or less, meaning that the saliva was unable to raise the pH. This result should be considered as a risk value.

[j]Orion Diagnostica, Espoo, Finland

Collecting Bacterial and Saliva Samples

To obtain information from *all* the chairside methods the following procedure is recommended.

- Prepare the chairside kits and inform the patient.
- Start the sampling with the patient in an upright position. Ask the patient to chew paraffin and swallow the saliva after 1 minute. Then start a timer for the secretion rate and instruct the patient to spit frequently into a graded test tube.
- Stop spitting after 5 minutes and take the Strip mutants test on the tongue.
- Measure the amount of saliva and calculate secretion rate.
- Take a droplet of the saliva with a pipette on the Dentobuff strip. Set timer for 5 minutes.
- Pour the remaining saliva on both sides of the Dentocult LB agar and let excess drip off.
- Incubate mutans streptococci and lactobacilli tests.
- Evaluate buffer strip after 5 minutes, mutans streptococci after 2 days, and lactobacilli after 4 days.

What Is the Next Step?

The effectiveness of caries risk and caries activity tests has been evaluated in various populations over the past decades with more or less encouraging findings.[40,92,93] The risk or activity approach *per se* is not a controversial issue but rather by which means this assessment should be done. Even though the multifactorial models are proven as useful in one country or society, it might be less useful in others.[94] Another important fact is that risk assessment programs must be evaluated continuously since the value

can vary over time. A striking example can be taken from lactobacilli tests when used as a didactic tool to reduce sugar-consumption in schoolchildren. Two decades ago, this program reduced caries increment with 50%[57] while it was of only limited value when recently reevaluated within the same community.[95]

For the individual, the identification of factors responsible for caries risk and caries activity should form the basis for targeted action against the etiological factors involved. Knowledge of risk factors gives the patient an opportunity to reflect over his or her situation and an option to take a personal responsibility for the future oral health. It may be argued that there is a weak scientific support for the fact that gained knowledge is an efficient tool to change a nonhealthy dental behavior.[96] This may be true for a non-specific general message and therefore it seems even more important to individualize the information, as disclosed by the tests. Both the therapist and the patient can be made aware on the main problem and focus on one strategy rather then the whole concept. In that aspect, the tests are also a matter of quality care and a guidance simply to do the right thing at the right costs. As previously stated, the relative importance of one risk factor may differ from one patient or group of patients to another. For example, it has been shown that the main risk factor for white-spot lesions during treatment with fixed orthodontic appliances was poor oral hygiene,[97] and therefore, it may not be meaningful or cost-effective to focus on diet. Similarly, as the level of metabolic control seem to be a stronger predictor for caries than mutans streptococci in children with Type 1 diabetes,[98] the focus should be on diet rather than antibacterial measures. In many cases, however, more than one risk factor or risk indicator are strongly involved. A common question is then whether or not it is meaningful to change or improve only one of them. Yes, it is of absolute importance since the balance in the oral environment between demineralization and remineralization is equivocal and in many cases, also a minor improvement may help the patient over the threshold level and to be on track. Moreover, after a successful management of one etiological factor, the self-esteem and motivation may grow to proceed with the next factor.

The other way around is probably of even greater importance. There is consensus in literature on the high specificity of caries risk and activity assessments to select individuals at low risk for future caries. This is a very positive message to communicate and the patient may have an option to extend the recall intervals. Thereby, resources can be redirected and money saved for the patient and for the society. At the end of the day, it is a matter of philosophy and quality—the teeth are, with very few exceptions, healthy when they erupt and it's a challenge for the dental profession to guide and assist their patients to keep them that way in a cost-efficient way.

Other Suggested Caries Activity Tests

In order to predict caries risk or determine the disease activity, a variety of other methods have been suggested. A few of these are briefly described and commented on below.

Snyder Test

In this test, suggested by Snyder,[99] sampled saliva is inoculated into a glucose agar

and acid formation is determined by a color indicator. The procedure reflects the total number and the acidogenicity of the salivary bacteria and can be used as an alternative to the lactobacilli test.[53]

Viscosity of Saliva

The viscosity of saliva is an important factor for the subjective perception of dry mouth and hyposalivation. Today, however, there are no methods of clinical significance in use to estimate the viscosity and furthermore, its relation to caries incidence is not clear. Measurement of oral mucosal friction by the aid of a rheologic device has been developed and may, in the light of the widespread use of xerogenic drugs, grow in importance for elderly patients.[100]

Dip-Slide Measurement of Salivary Yeast

In general, the presence of an oral yeast infection can be considered as a reflection of the host response and indicative of a medically compromised patient. A high number of salivary yeasts are often found in patients with hyposalivation. Moreover, fungi are aciduric and their presence might be a reflection of an acidic environment and caries activity.[101] A dip-slide system for measuring oral yeast (*Candida albicans*) infection, ORICULT-N,[k] (Figure 12-7) has been developed and is commercially available.[102]

[k]Orion Diagnostica, Espoo, Finland

Plaque-forming Rate

General plaque has been suggested as a caries predictor.[103] The speed of plaque development can be estimated by the plaque-forming-rate index (PFRI).[104] Twenty-four hours after professional tooth cleaning, plaque reaccumulation rate is assessed on a scale from 1 to 5 on 6 measuring points per tooth. No oral hygiene measures are carried out during the 24-hour period. Although used in several clinical studies with a positive relationship to caries incidence, the method has not gained a widespread clinical acceptance.

Plaque pH Measurement—Acid Formation by Dental Plaque

Plaque pH can be directly measured intraorally by using either glass or antimony electrodes.[105] Caries-active subjects exhibit lower resting pH and final pH following sucrose rinses compared to caries-free persons. Telemetric monitoring, however, seems more useful in evaluating pH changes after intake of various foods than in determining caries activity.[106] Consequently, the technique is more often used in research laboratories at universities rather than in the everyday dental office.

Future Methods

A serious concern with the culturing methods of today is the time span from sampling until the results are available for the professionals and their patients. Furthermore, sampling must be planned to fit weekends and other activities. It is not likely that the current available tests can be significantly improved, especially if they are to be suitable for chairside use or aimed for field conditions. New tests, measuring for

example bacterial adhesion and bacteria-binding saliva ligands as genetically determining factors for caries, might be developed. Existing immunologic methods like enzyme-linked immunosorbent assay (ELISA) kits will probably be transferred from the specialized laboratory to the dental clinic in coming years. A call for faster and more accurate techniques will certainly stimulate the development of new and improved products. Moreover, improved knowledge of lifestyle-factors such as oral hygiene and sugar consumption pattern obtained through qualitative studies can add precision to the caries risk evaluation process.

Question 4

Which of the following statements, if any, are correct?

A. Salivary mutans streptococci levels are influenced by antibiotic medication.

B. A high-buffer capacity is often found in patients with a low-secretion rate.

C. Microbiologic caries activity tests can be used as didactic and motivating tools in caries prevention.

D. Past caries experience is found to be the most valuable single predictor in many caries risk studies.

E. The predictive ability of a test depends on the prevalence of the disease in the population.

Summary

Caries is a transmissible local infection and aciduric microorganisms, like mutans streptococci and lactobacilli, are the prime pathogens. This chapter has reviewed the ecologic events leading to caries development: (1) early establishment of mutans streptococci, (2) microbial shift and, (3) enamel demineralization. This process can be prevented, arrested, or reversed with the knowledge of factors such as the microbial challenge, intake of refined carbohydrates, and the body's capacity of self-repair.

Caries-risk assessment strategies can be applied for populations, larger or smaller groups, or individuals. There is no single test that can accurately reflect the complex etiology of caries. Although tests of mutans streptococci and lactobacilli show strong correlation with caries in cross-sectional and longitudinal surveys, they are generally of limited value for risk-screening purposes in communities with a low prevalence of caries. In groups of individuals with higher caries incidence such as medically compromised patients, inhabitants of low socioeconomic areas, and low fluoride areas, the predictive power and the value of the microbial tests are increased. Negative or very low counts of mutans streptococci and lactobacilli are highly predictive for subjects at low risk of getting caries. The past caries prevalence is the most powerful single predictor on a population basis.

For the individual patient, a risk assessment is performed by compiling data of importance for caries development from the case history, clinical examination, and chairside tests. Microbiologic tests should be regarded as monitors of the oral ecology

and repeated samplings may indicate deviations from the normality of the individual. Any increase in the challenge factors or decrease in defense and repair factors at any time should be considered as a warning sign. This knowledge should form the basis for an individualized and targeted preventive oral health care program.

Chairside tests covering bacterial challenge, diet, and remineralization potential of saliva are described. The simplified methods can be characterized as semiquantitative although they significantly correspond to conventional laboratory methods. Furthermore, the chairside methods have been proven useful as a didactic tool in patient education and motivation.

Many diagnostic criteria of caries activity that are used today represent historic events. The chairside microbiologic tests improve quality and add a possibility of early risk assessment and diagnosis. We hope that this chapter has given the reader inspiration to incorporate caries activity tests in their daily work, for the benefit of their patients.

Answers and Explanations

1. A and E—correct.

B—incorrect. Two examinations are necessary to determine the number of carious lesions occurring over a given amount of time.

C—incorrect. Sensitivity is the percentage of subjects with a positive test who develop the disease.

D—incorrect. Caries-risk assessment is recommended in a low caries population with a skewed distribution.

2. A, B, C, and D—correct.

E—incorrect. Lactobacilli are more commonly associated with cavitation and progression of existing lesions.

3. B and D—correct.

A—incorrect. The stimulated secretion rate of whole saliva is 1.5 mL/minute, which is normal.

C—incorrect. Plaque samples disclose where the bacteria are harbored.

E—incorrect. Score 3 (corresponding to 10^6 CFUs) indicates caries risk but not necessarily demineralization.

4. A, C, D, and E—correct.

B—incorrect. A low buffering capacity is often found in patients with a low stimulated secretion rate.

Self-evaluation Questions

1. If an epidemiologic clinical caries survey is being conducted, the number of decayed teeth (d; D) present at that time constitutes a caries _____ study; if the same patients are reexamined 1 year later, the number of new decayed teeth constitutes a caries _____ study.

2. The two most common mutans streptococci strains in humans are *Streptococcus* _____ and *Streptococcus* _____.

3. An incipient enamel lesion can be seen with the unaided eye. (True, False)

4. Usually, mutans streptococci are established during childhood, between ages _____ and _____, but may increase in numbers during the following years.

5. Studies have shown a family pattern concerning mutans streptococci, meaning that bacteria often are transferred from _____ to children, but other sources may also be found.

6. Chairside bacterial test results in saliva should be regarded as _____ rather than exact bacterial numbers.

7. A risk factor plays an essential role in the _____ of the disease while a closely associated variable that is not causative is called a _____.

8. With the decreasing prevalence of caries seen over the last decades in the industrialized world, one could expect a greater number of (false-negatives) (false-positives) to be diagnosed.

9. A microbial shift in dental plaque can occur when _____ microorganism(s) are favored.

10. The positive predictive value (PV+) is probably of highest interest for the clinician since the _____ for an individual with a positive test to develop the disease is denoted.

Useful Dental Websites for Caries Activity Information

- World Health Organization (WHO): Oral Health Country Profile Project http//www.whocollab.odont.mah.se/index.html
- International Health Care Foundation (IHCF): Caries-risk assessment; saliva interactive site, WWW-based management of dental prevention; Cariogram; other dental web sites of interest http://www.ihcf.li
- Malmo University, Faculty of Odontology: continuously updated list of references on caries risk assessment, mutans streptococci and lactobacilli. http://www.db.mah.se/car/data/riskbasic.html
- NIH Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life: complete version of papers on caries diagnosis and caries risk. http://nidcr.nih.gov/news/consensus.asp

• Orion Diagnostics, Turku, Finland: manufacturer of kits for saliva diagnosis http://www.oriondiagnostica.fi

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Chapter 13. Periodontal Disease Prevention: Facts, Risk Assessment, and Evaluation - *Norman O. Harris Donald E. Willmann*

Objectives

At the end of this chapter, it will be possible to:

1. Cite the one main sign that delineates gingivitis from periodontitis.

2. Explain the rationale for the latest classification of the periodontal diseases.

3. Explain the purpose of O'Leary's Index, Silness and Loe's Plaque Index, and Loe and Silness's Gingival Index.

4. Describe how manual periodontal probes are used, and contrast them to constant-force electronic probes.

5. Explain how pocket depth and attachment loss are measured and how gingival recession measurements are related to both.

6. Clarify the differences between the Community Periodontal Index of Treatment Needs (CPITN) and the Periodontal Screening and Recording System (PSR).

7. Discuss the value of the gingival crevicular fluid and how the flow is quantitated.

8. Explain why smoking constitutes a high-risk habit that jeopardizes the prevention, treatment and maintenance of the periodontal diseases.

Introduction

In 1875, Riggs' disease,[a] (later known as pyorrhea[b] alveolaris[1,2] and still later as periodontal disease) was easy to diagnose. If pus could be expelled from the gingival crevice by exerting finger pressure over the root, from the apex towards the crown, the correct diagnosis was pyorrhea alveolaris.[3] This diagnosis could be confirmed by placing a drop or two of guaiacum on the exudate producing a deep blue color.[4] At the time it was estimated that 95% of all people over 25 were "more or less affected."[5] Of interest is the fact that systemic conditions were suspect as possibly associated with, or as causal agents of pyorrhea alveolaris—such conditions as gastric dyspepsia, phthisis,[c] adenoids, nasal catarrh, constipation, general congestion due to intemperance, malnutrition, and cold feet or other extremities that indicate poor circulation.[3]

Throughout the first half of the 20th century, pyorrhea alvolaris and receding gums remained the popular terms for the disease by both the profession and lay persons. The cause of pyorrhea alveolaris at the time was attributed to the presence of calculus.[6] Both the long-time terminology and the well-established calculus etiology was to again change. Periodontal disease was to supercede the designation of pyorrhea alveolaris while the accepted etiology of calculus[6] was dropped in favor of a nonspecific plaque hypothesis. According to the nonspecific hypothesis, periodontal disease was caused by a mixed overgrowth of known and unknown organisms in the dental plaque.[7,8] It was still assumed that once a patient was "infected with periodontal disease," the process became more severe with time; in other words, periodontal disease was considered a pathologic penalty for aging. The public still continued to recognize periodontal disease as an inflammatory disease characterized by *periododontal pockets* accompanied with a *silent bone loss.*

By the mid-20th century, the "non" of the nonspecific hypothesis was dropped in favor of a new, "specific bacterial hypothesis" that now postulated that gingivitis and periodontitis were caused by specific as well as still-unknown bacterial species indigent to the plaque.[8] At the same time, a consensus began to emerge that the one-time single periodontal disease was instead, a series of different, but related diseases categorized as (1) gingivitis and (2) adult, prepubertal, juvenile, rapidly progressive, and refractory periodontitis. With this change, calculus rebounded into a secondary etiological role, where its porous surface was, and still is, believed to serve both as a *habitat* for plaque bacteria and their end products and as an *irritant* to the marginal gingival tissues.[9-11] The presence of subgingival calculus contributes to the progression and chronicity of periodontal disease.[9,12]

The relationship between the different periodontal diseases was not, and is not yet well understood. This was underscored by the past classification system that was based on a narrative description that related to the patient's age at the time of onset, rapidity of disease progression, response to therapy, and severity of the disease—and not to definite causal agents like for caries, where mutans streptococci and lactobacilli are the prime cariogenic pathogens.[13]

There are still major voids of knowledge about the specific periodontal pathogens, or those that might be indicted in the future as synergistic or causal to the periodontal disease process. Plaque samples from individuals with periodontitis can demonstrate approximately 350 microbial species in the dental plaque and about 150 species in the supragingival plaque, tongue,and other oral structures.[14] Yet, singularly or in combination, a very strong case can now be made for implicating among others, *Actinobacillus actinomycetemcomitans* (Aa), *Porphyromonas gingivalis* (Pg), *Bacteroides forsythus, Prevotella intermedia* (Pi), *Eikenella corrodens, Fusobacterium nucleatum, Campybacterrectus* and *Treponema* (spirochetes).[15-18]

Routine laboratory, and more sophisticated DNA probe analyses (described later) can be accomplished to identify suspect bacteria. Such positive identifications aid the clinician in selecting drugs to suppress the organisms found in the different periodontal diseases.

In 1998, the profession experienced another nomenclature change. This *new classification system* eliminated the groupings based on age of onset, rapidity of onset, etc., and replaced it with a classification *that attempts to identify the local and systemic causes of gingivitis and periodontitis.* For instance, bacteria in the plaque cause periodontal disease, but the action of any of the same bacteria may be *modified* by systemic factors such as the endocrines, blood dyscrasias, ingested medication, etc. The full classification with sub groupings and examples is contained in Appendix 13-1.[19] This new classification system will be incorporated into future Dental and Dental Hygienist National Board Examinations.

[a]Riggs' disease: Named after a Boston Dentist. Dr. Riggs extracted the first tooth ever to be extracted under general anesthesia. His patient was Dr. Horace Wells, the dentist given credit for the discovery of nitrous oxide as a anesthetic.

[b]Pyorrhea = pus.

[c]Phthisis = asthma.

Facts about Gingivitis and Periodontitis

Although bacteria are the causative factor of the periodontal diseases, there are powerful influencing factors that can modify the course of the diseases such as (1) *smoking,* (2) *genetic differences,* (3) baseline severity of disease, (4) Presence of *P. gingivalis, P. intermedia,* and *B. forsythus,* and *Actinobacillus actinomycetemcomitans,* and (5) *individual compliance* with established standards for oral self-care.[20,21] Of interest to married couples is the fact that spouses and children of an adult periodontitis patient might be at a relatively high risk of developing a

periodontal breakdown.[22] Another strong risk indicator is the observed relationship of several *systemic* diseases to gingivitis and periodontitis. Among these are diabetes mellitus,[23] Down's syndrome,[24] and more rarely diagnosed conditions such as Haim-Munk syndrome and Papillon-Le Fevre syndromes.[25] Also noticed has been a greater frequency of cardiovascular accidents and nonhemorrhagic strokes among individuals with periodontitis.[26,27]

Both gingivitis and periodontitis affect the tissues of the periodontium. By *definition,* a plaque-induced gingivitis is an inflammation of the marginal gingiva *without any loss of the epithelial attachment.* Once there is a loss of the epithelial attachment, again by *definition,* periodontitis begins. The term periodontitis can be defined as (1) an inflammation of the marginal gingival *with* (2) a loss of the epithelial attachment, *plus* (3) irreversible damage to any of the other three remaining components of the periodontium, i.e., the cementum, alveolar bone, and the periodontal ligament that connects the latter two structures.

Question 1

Which of the following statements, if any, are correct?

A. One of the highly suspect bacteria associated with periodontitis has been abbreviated Aa; the correct full spelling of the bacteria is *Actinobacillus actinomycetemcomitans.*

B. The last "official" change of periodontal disease classification in 1998 should make it easier to determine the etiology (or cofactors) to the periodontal diseases.

C. Three powerful nonbacterial factors that influence the course of periodontal disease are genetic differences, smoking and adequate daily self-care.

D. Three systemic diseases that are associated with periodontitis are cardiac disease, diabetes mellitus and most viral diseases.

E. It is possible to have a 4-millimeter deep pocket with a slow apical migration of the epithelial attachment, and yet have a gingivitis.

With good oral hygiene practices, a plaque-induced gingivitis of bacterial origin can be *cured,* i.e., the free margin of the gingiva can be returned to its original histology. On the other hand, because of the irreversible changes that occur to the components of the periodontium in periodontitis, it is usually not possible for the affected tissues to return to normal. Once periodontal treatment is completed, any further preventive and/or treatment therapy is considered as *maintenance* (and not a cure) and is intended to sustain the status quo of the tooth as much as possible throughout a lifetime. The recognition of the early signs and symptoms of gingivitis and/or periodontitis and beginning immediate treatment is crucial to the arrest and control of disease progression. This gatekeeper function can best be served by the *general dentist.*[28]

There is abundant evidence that the microbial population of the *supra*gingival plaque is associated with gingivitis, and the *sub*gingival plaque with periodontitis.

In the earliest stages of gingivitis, there is an infiltration of body defense cells beneath the crevicular epithelium. If the gingivitis is not arrested at an early time, the color of the marginal gingiva changes from a pale *pink-to-red*; the contour of the marginal gingival becomes *edematous,* and there is *bleeding* on probing or during toothbrushing ("pink toothbrush"). Any gingival bleeding at *any age* and at any time is not normal and should be viewed with concern by both the clinician and the patient. Yet, because gingival bleeding is considered such a commonly occurring entity, dentists and patients alike often fail to recognize early inflammatory gingival changes, *even though this is the time that complete recovery (cure) is possible.* Patients are often not informed of the presence of periodontal disease until the opportunity for cure or early arrest is past. In one study, only 48% of the patients with diagnosed advanced periodontitis had been informed of their condition by their dentist. Only 12% of those with gingivitis and 20% of the patients with early periodontitis had been made aware of their conditions.[29,30] These data support the fact that one of the complaints about periodontal disease diagnoses, is that they often occur too late to be really helpful.[28] This finding has both *ethical and legal* overtones.[31,32]

Marginal gingivitis is extremely common among *all* age groups, and is *not* necessarily related to aging per se. Many Senior citizens enjoy excellent periodontal health into old age.[33] On the other hand, many advanced cases of periodontitis seen in aging are the result of lifelong neglect of self-care.

Unfortunately, many periodontal cases can be traced back to youth. For example, in Reykjavik, Iceland, gingival bleeding was found in 16% of 230 6-year old children.[34] In a military population of 1,334 soldiers, 40.3% were found to have gingivitis, while 35.7% of the subjects had pocket depths of 3 to 5 millimeters (considered as possible early periodontitis).[35] Bhat reported 34% of 14- to 17-year-old youths had supragingival, and 23% had subgingival calculus.[36] It is of concern that so many children and young persons are not under professional care.[37] Children or adolescents with gingivitis, subgingival calculus, or early signs of alveolar bone loss should be considered as high periodontitis-risk individuals and should be entered into a monitored preventive program as early as possible. These repeated findings of gingivitis that occur at relatively early ages, are a harbinger for the periodontal disease that becomes the leading cause of tooth loss after the third decade.[38]

Noninvasive Treatment Guidelines for Gingivitis

Gingivitis of plaque origin is a *preventable and curable* periodontal disease. The *objective of professional and home self-care is to eliminate or severely reduce the etiologic organisms in the dental plaque and to prevent or reverse gingival inflammation.* This effort can be abetted by a thorough prophylaxis, supplemented at home by use of the *toothbrush, dental floss* and an *irrigation device.* Generally, an electric toothbrush is more effective than a manual brush (see Chapters 5, 6, and 7). This "brush, floss, and flush" routine can be enhanced by the daily use of a fluoride toothpaste, over-the-counter products with *essential oils,* such as Listerine, or dentist prescribed *chlorhexidine* mouthrinses.

The daily self-care routine should be habituated from early childhood to prevent challenge organisms from significantly populating, or repopulating the plaque. In the

event that these empirical measures fail, a differential diagnosis should be considered to determine if one of the plaque modified etiologies for gingivitis listed in Appendix 13-1 is the primary cause.[19] If so, a medical referral may be in order.

Noninvasive Primary Preventive Care for Periodontitis

Once a patient develops *periodontitis*, therapy usually includes additional measures to those recommended for gingivitis. As the probing depth increases, it becomes more difficult to eliminate the bacteria of the subgingival plaque. In addition to routine calculus removal at the time of the prophylaxis, *scaling* and *root planing* needs to be accomplished.[39] Many clinicians advocate irrigating the deeper pockets. The depth of penetration of irrigation solutions into the pocket depends on the tip design of the irrigator, the fluid pressure, and the calculus present that might divert the irrigant stream.[40] Chlorhexidine,[41] stannous fluoride,[42,43] and Listerine[44] are but a few of the solutions that have been used. Research to find more effective antimicrobial agents is a continuing quest.[45] The *dental hygienist is probably the key person to deliver the subgingival irrigation therapy.*[46] *as well as to instruct the patient on how to accomplish the task at home.*[47] (See Figure 13-1.)

The mouthrinses used in a self-care programs do not penetrate deeply enough into the periodontal pockets. However, when irrigation is accomplished in the office, a greater penetration of the pocket can be attained by placing the therapeutic irrigating solution in the fluid container of the ultrasonic scaler.[48] To complete the treatment, often a slow-delivery medication is placed in the pocket, or antibio- tic therapy can be initiated to eliminate microbes that have invaded the sulcular tissues.[49] Once a maximum treatment success has been achieved, an every-3-month monitoring is *mandatory.*

Invasive Procedures Required to Access the Subgingival Pocket

As the pocket continues to deepen it becomes more difficult to apply noninvasive preventive procedures. To solve this problem, the periodontist can sometimes perform *flap surgery,* a surgical procedure that removes a circumferential portion part of the marginal gingiva and exposes the root. Following the operation, the previous inaccessible subgingival pocket area becomes more accessible to apply routine dental hygiene procedures. It should be emphasized that this surgery is *not* a cure—it only provides a reprieve to help arrest a disease that has been out of control.

Advanced Periodontal Surgery

On the basis of studies, it is estimated that approximately 5 to 20% of adults have severe periodontal disease while the majority have mild-to-moderate periodontitis.[50] In the advanced stage of periodontal disease there is a dramatic loss of the epithelial attachment with a concurrent loss of supporting alveolar bone that can severely compromise the support of a tooth. A discussion of advanced surgical techniques is beyond the scope of this book; however, there are surgical procedures that can often be used to repair damage caused by periodontitis.

In an effort to compensate for losses of bone and tissue, *guided regenerative techniques*[d] have been introduced in the past several years with a divided emphasis on

bone as well as *soft-tissue* regeneration. This is accomplished by use of bone grafts and bone stimulants, as well as plastic surgery procedures to reshape soft tissues. At times when the bony support is minimal, prosthetic devices interlinking several teeth are constructed to act as a splint to prevent any one or more teeth being subjected to excessive lateral tooth movement upon mastication.

Following surgical interventions to manage moderate and severe stages of periodontitis, the preventive actions that must be taken *still require meticulous mechanical ("brush, floss, and flush") and chemical plaque control (antimicrobial mouthrinses).* Chlorhexidine is probably the antimicrobial mouth rinse of choice to protect the integrity of the restored tissues and to help suppress the transmission of periodontopathogens from other soft and hard tissue locations in the mouth.

[d]Periodontal regeneration means healing after periodontal surgery—a healing that results in a partial or complete restoration of the tooth supporting tissues, namely cementum, alveolar bone and periodontal ligament. Ann Periodontology, 1997; 2:215-22.

What Is Peri-implantitis?

To improve esthetics and function following the extraction of a tooth, the void can usually be filled by either a bridge or an implant. For a bridge, it is necessary to prepare two intact teeth as anchor teeth. This can involve a considerable loss of tooth structure. On the other hand, an *implant* can be "implanted" between the two adjacent teeth to function much as a normal tooth.

An implant consists of noncorrosive metallic "root" that is inserted into a cylindrical preparation in the alveolar bone. (Figure 13-2). After bone healing, a prepared crown is cemented to the prepared portion of the implant that remains above the mucosa. This allows the implant to serve the *same function* as other natural teeth. Unfortunately, the implant is also exposed to the same bacterial flora as are all the other normal teeth—teeth that develop gingivitis and periodontitis. The same primary-preventive procedures are necessary for survival of an implant as for the other teeth of the mouth; neglect of self-care results in the same gingival infection and sequela as for periodontitis. The same destructive bacteria are involved as in periodontitis. The failure and removal of an implant parallels the terminal extraction of a natural tooth from periodontal disease(s). In other words, the same problems and the nearly same solutions apply to an implant with *peri-implantitis* as for a natural tooth with periodontitis.

Figure 13-1 A. An irrigator is an outstanding device to use a *plentiful* amount of water to flush debris still remaining after brushing and flossing ("brush and flush"). B. An irrigator with the use of an accessory nozzles can utilize a limited amount of antimicrobial solution to carefully irrigate periodontal pockets. (Courtesy of Hydro Floss, Inc., Birmingham, AL, 35244.)

Figure 13-2 Two bicuspid crowns will be placed on the implants once the bone wound has healed. At that time they will serve the same functions as natural teeth. (Dr. Donald Willmann, University of Texas Dental School at San Antonio, TX.)

Question 2

Which of the following statements, if any, are correct?

A. A case of gingivitis can be cured (cured = gingival tissues returned to orignal histology).

B. A case of periodontitis can be cured. (cured = gingival tissues returned to original histology).

C. The mechanical part of self-care consists of use of a toothbrush, floss, and an irrigator ("brush, floss, and flush").

D. Mouthrinses are effective in the irrigation of deep pockets to treat periodontitis.

E. An implant between two teeth requires abuttment teeth for support.

Epidemiology and Risk Assessment

Periodontal Disease Indicators

Two objectives have been established for this chapter: (1) To provide some *basic facts* about gingivitis and periodontitis, highlighting the role of preventive dentistry—which has been done, and (2) to now explain how some *evidence*-based tests[e] and indices can be used to assess risk, severity, and prevalence of the periodontal diseases. Some of these indicators were developed to *screen populations* to determine the *prevalence* and *severity* of periodontal conditions, while others were developed to evaluate the periodontal *health of individuals in a private practice.* Others serve both purposes, but *no single index is appropriate for all types of studies.*

Those tests and indices used to evaluate the various stages of gingivitis and periodontitis usually include one or more of the following: (1) pocket depth; (2) amount and location of dental plaque; (3) extent of gingival inflammation; (4) calculus deposits; (5) bacterial identification; (6) evidence of epithelial attachment loss; and (7) smoking habits.

The bleeding index is a most positive indicator of *existing gingivitis*, while a smoking history is probably the most reliable *predictor* of periodontal disease.

[e]Evidence-developed indicator = a sign or test that has a scientific backgroud linking the indicator to the disease.

Measuring Dental Plaque

O'Leary's Plaque Record (Index)

The relationship between plaque and gingivitis was first established by Loe et al. in 1965.[51] Seven years later, O'Leary developed one of the first useful and widely used indices to identify the location and extent of plaque. O'Leary's index is useful for monitoring patients' plaque control performance, is easy to accomplish, is economical,

and is reproducible.[52] Only a mouth mirror and explorer are necessary. The completed chart indicates the locations where plaque accumulates and where improved brushing and flossing techniques are required.

The steps for manually recording and interpreting the O'Leary Index are as follows.

1. The smooth surfaces of the teeth in the mouth are divided at the anatomic line angles into four sections—mesial, buccal, distal, and lingual (Figure 13-3).

2. All missing teeth are crossed out, and the total number of remaining teeth are determined. For plaque control purposes, the pontic(s) of a fixed bridge and implants should be scored in a manner similar to that of natural teeth.

3. The patient is first asked to rinse vigorously with water to dislodge any loose food debris.

4. The plaque is then disclosed by applying a *disclosing solution* to all teeth, making sure that the dentogingival junction is covered with the agent. As an alternative, a disclosing *tablet* can be chewed and the colored saliva swished around the mouth.

5. The mouth is again rinsed vigorously with water. The operator then uses the explorer or tip of a periodontal probe to confirm the presence of disclosed accumulations of plaque at the dentogingival junction. If the plaque on a tooth surface is in contact with the gingival margin or papillae, the entire tooth surface space is filled in with a red pencil to increase visibility and to enhance the form's impact on the patient. Areas having stained pellicle alone should *not* be scored as having plaque.

The total number of scored tooth surfaces is then counted; the sum is then divided by the number of available teeth (including pontics and implants), and multiplied by 100 in order to establish the plaque score as a percentage. This baseline plaque score should be compared with future recall scores to objectively *monitor* a patient's progress.

O'Leary and colleagues[52] have stated that a suitable goal in teaching personal self-care is to reduce the plaque index to *10% or less.* It is suggested that no periodontal surgery or fixed prostheses should be started until this goal has been reached. If surgical or prosthetic intervention is not contemplated, an initial reduction to 15% is probably more realistic for most individuals.

Several record forms are available that are modifications of O'Leary's original presentation (Appendix 23-1). With the introduction of the chairside computer recording of dental examinations, these computer generated plaque index records will eventually become part of the "paperless" dental office.

The Plaque Index of Silness and Loe

The plaque index of Silness and Loe provides a modication of O'Leary's index. It too is visual and required only a mouth mirror. A deficiciency of the O'Leary index is that it requires that the surfaces of every tooth be examined for plaque but that there was no gradations between a great amount of plaque and no plaque.

The index of Silness and Loe also requires that the four surfaces of designated teeth be visually examined and a score recorded, viz., the maxillary right first molar, maxillary right lateral incisor, and the left first bicuspid; the the mandible, the mandibular left first molar, the left lateral incisor and the right first bicuspid—a total of six teeth. For each of the surfaces of these teeth a score of 0 to 3 is given that matches the severity of the listings in Table 13-1. In this way, the average amount of plaque for each tooth can be determine by dividing by 4; the scores for all six teeth be divided by 6 to get the average for the mouth. The highest scores can be expected to occur on the interproximal surfaces.

The index has the advantage of providing more data on the self-care habits of the patient, as well as taking less time to evaluate than to record entire dentitions—a fact that is important in large-scale epidemiology studies. The data from the Silness and Loe index can also be used to compare with other indexes, for instance to evaluate the amount of plaque against the gingival bleeding index of Loe and Silness (described later) throughout the months of pregnancy.[53]

Oral-Hygiene Index and Simplified Oral-Hygiene Index

One of the most popular indicators for determining oral hygiene status in epidemiology studies is the Oral Hygiene Index (OHI). It was developed in 1960 by Greene and Vermillion[54] and modified 4 years later as the OHI-S.[55] The simplified (S) version provides much the same information, as did the earlier version, but can be accomplished much more rapidly. It is very useful for large-scale epidemiology surveys but is not generally believed to be sensitive enough to accurately evaluate the oral hygiene status of an individual patient. The OHI has two components: the oral debris score and the calculus score. The term oral debris includes "plaque, materia alba, and food remnants." With the OHI-S, soft and hard deposits are evaluated only on the facial or lingual surfaces of six selected teeth. They are the *buccal* surfaces of the upper *first molars of both sides,* the *labial* surfaces of the *upper right and lower left central* incisors, and the *lingual surfaces of both lower first molars.* The criteria for the OHI-S scores are shown in Table 13-2. The total OHI-S score can be divided by the number of surfaces examined to calculate the average oral hygiene score.

Gingival Bleeding Index of Loe and Silness

One of the most commonly used indexes to determine the prevalence and severity of gingival inflammation is the Gingival Index of Loe and Silness.[56] With the Loe and Silness index it is possible by its coding from 0 to 3 to record bleeding tendencies, color and contour changes of the gingival, alterations in the consistency of tissue and the presence of ulcerations (Table 13-3).

Like the Silness and Loe plaque index, only six teeth are selected. Data can be computed for individual teeth, or for all the six. The evaluation, coding, and recording are quite rapid and useful in larger-scale epidemiology studies.

As a part of a full-scale periodontal examination of a patient, it is desirable to determine gingival bleeding by probing of the marginal gingiva. This procedure *must* be carefully controlled to avoid *false positives and iatrogenic damage to the*

periodontium.[58] *In the test for gingival bleeding, the probe should be run along the soft tissue wall at the orifice of the periodontal sulcus or periodontal pocket.* Probing at the bottom of the pocket is a *poor indicator.*[59] The basic objective of a bleeding index is *not* to determine the sulcus[f] depth, *not* to evaluate the extent of loss of the epithelial attachment, *not* to determine bone loss, but *only* to evaluate whether there is, or is *not,* gingival bleeding. When all teeth are included, the data can be used as an epidemiology instrument or for a patient's clinical record.

[f]Sulcus and crevice are often used interchangeably. For example, gingival sulcus and gingival crevice.

Figure 13-3 The chart used for O'Leary's Plaque Index.

Question 3

Which of the following statements, if any, are correct?

A. Two dental plaque indices are those of O'Leary and Silness and Loe.

B. A plaque score of 10% for the O'Leary plaque index is considered marginally satisfactory.

C. Gingival bleeding when there is no loss of epithelial attachment is a positive sign of gingivitis.

D. The Loe-Silness index is oriented towards monitoring gingival health.

E. Bleeding during toothbrushing ("pink toothbrush") can only be diagnosed by a dentist as a gingivitis.

Periodontal Probes

As illustrated in Figure 13-4, there are several variations of periodontal probes. Each has circumferential markings on the probing tip to aid in determining sulcular depth; others also have color-coding to further facilitate accurate measurements. The probe is used for four main purposes: (1) the measurement of *pocket depth,* (2) the measurement of epithelial *attachment loss,* (3) *induction* of gingival and/or papillary bleeding, and (4) the detection of *subgingival calculus* as part of the periodontal examination. The probe may be of metal, or of a hard polymer.[60] The probing tip is approximately 0.5 millimeter in diameter. Its tactile *reproducibility* and *accuracy* depends much upon the experience of the operator.

There is always a need for caution in probing, especially in the presence of inflammation. Probing inflamed gingival tissue sites with its fragile capillaries risks inducing a *bacteremia.* For individuals at risk of infective *endocarditis,* both a clinical and radiographic assessment is indicated prior to a decision to probe. Prophylactic antibiotic coverage may be indicated.[62] (See Figure 13-5.)

A new era of periodontal probing was ushered in by the coupling of the computer and the constant force electronic probes (Figure 13-6).[g] One example of the electronic

probe is the Florida probe,[e] which has been routinely used since 1955 in the University of Florida's Disease Research Center.[63,64] In one well-controlled study, the Florida probe was shown to be extremely accurate and reproducible. The minimum probing error was found to be around *0.2 millimeter*.[65,66] In contrast, the resolution of the standard manual probe is *1 millimeter*. When using a constant-force probe, as soon as the *resistance* at the bottom of the sulcus reaches a preset level such as 15 to 20 grams, the depth of the sulcus is *automatically* entered into the computer record form (Appendix 13-2). (For comparison, a force of 25 grams is just below the threshold of pain when a probe is inserted under the fingernail; (see Figure 13-7).

Another electronic probe is the Toronto probe that uses air pressure to extend and retract the measuring tip; this action helps control the probing force. An Alabama probe automatically detects the cementoenamel junction and measures the clinical attachment levels to the bottom of the pocket within a 0.2 millimeters tolerance level.[67]

Because of the accuracy of the electronic probes, the time needed to identify periodontal disease activity (attachment loss) between recall intervals can be shortened. Printouts can be made that permit comparisons between different examinations. As can be noted in Appendix 13-2, the same computer software can be used to record the main periodontal disease indicators—pocket depth, gingival recession, plaque, bleeding, and tooth mobility. This is accomplished partially by using red, yellow, and green or other computer color designations for the various entries.

A unique convenience is that as pocket and bleeding sites are seen on the monitor and entered in the record, the information can be called out in a computer-generated male or female voice—not that of the dentist. Additional color-coded copies of the completed patient's record can be printed out for patient records and patient information, as well as for insurance filing.

[e]Evidence-developed indicator = a sign or test that has a scientific backgroud linking the indicator to the disease.

[g]Available from Computerized Probe, Inc., Florida Probe Computerized Systems, Oklahoma City, OK.

Figure 13-4 Different types of calibrated periodontal probes useful in assessing the depth and configuration of periodontal pockets.

Figure 13-5 Diagram of WHO periodontal probe. It has a ball-tip end to avoid false assessment by over-measurement and for easier detection of subgingival calculus. The color-coded part from 3.5 to 5.5 mm greatly facilitates rapid assessment of periodontal pocket depth. (From WHO Technical Report Series 621, 1978.)

Figure 13-6 The Florida Probe. Note the slim barrel and the ease with which it fits within the hand. The tips are removable and sterilizable. (Courtesy Florida Probe Corporation, Gainesville, FL.)

Figure 13-7 Practical test for establishing 20 to 25 g periodontal probing pressure. The periodontal probe is placed underneath the fingernail where the sensitivity approximates that of the bottom of a periodontal pocket. The correct

amount of force should not cause pain to the patient on probing. (Dr. Arden Christen, Indiana University School of Dentistry, Indianapolis, IN.)

Periodontal Probing

As previously mentioned, two of the main purposes of periodontal probing are to determine *pocket depth,* and to measure the amount of *attachment loss.* Both have one requirement in common, namely a careful step-by-step circumferential probing around each tooth.[68] To determine pocket depth; the probe is inserted into the mesial proximal sulcus. It is aligned as vertically as possible, but with a slight angle away from the midpoint of the tooth bucco-lingually because of the contact point. Without being withdrawn, the probe is then "walked" along the facial surface of the crevice until the distal proximal contact area is reached. The probe is then withdrawn and reinserted from the lingual surface and "walked" back to the proximal surface. As the probing proceeds, a record is made of the distance from the deepest site of the pocket to the crest of the free gingival margin on each of the four surfaces. A more detailed second probing might be indicated where the initial sulcular depth has been found to be of concern.[69] Other patterns of probing are acceptable. The main objective is to include all surfaces and all problem areas. Probing depth can be influenced by various factors, such as the type of probe, angulation of the probe to the tooth, pressure used in probing, and inflammation of the free gingival margin—all create possibilities for error in measuring pocket depth or attachment loss. In fact, the probing depth *seldom* corresponds to the exact microscopic (histologic) depth of a normal sulcus or pocket depth. However, the clinical pocket depth does reflect the relative level of the actual pocket depth. It does provide the clinician with a useful *reproducible* estimate of the location of the most coronal insertion of the fibers of the periodontal ligament between the alveolar bone and the cementum. (See Figure 13-8.)

The measurement of *epithelial attachment loss* involves the same format of probing as for determining *pocket depth.* The main *difference* is the *reference point from which* the measurement is recorded. For pocket depth, it is from the depth of the pocket *to the crest* of the free marginal gingiva. For calculating attachment loss, the measurement is made from the depth of the pocket on each surface *to a fixed site,* such as the cementoenamel junction or occlusal plane.[70] Two measurements *separated in time* but at the same site are necessary to estimate the amount of apical migration (if any) of the epithelial attachment. Sites that show a 2-millimeter loss of attachment between two sequential recall examinations should be considered as active.

The dividing line between gingivitis and periodontitis in a practice is objective and reproducible. It is based on the consistent finding that a normal gingival sulcus depth is approximately 3 millimeters in depth. If a probing depth of three millimeters is encountered with no bleeding and no loss of epithelial attachment, the periodontium is considered in good health. A probing depth of 4 to 6 millimeters is considered in the gray area between periodontal health and disease.[71] If bleeding is encountered, the problem may be *either* gingivitis or periodontitis, depending on the examiner's evaluation of epithelial attachment loss. When a patient is in this gray zone, frequent monitoring and scrupulous oral hygiene is required. On the other hand, a loss of greater than 6 millimeters usually constitutes an advanced periodontitis.[71] A patient with a pocket depth measurements of 6 to 9 millimeters can usually expect surgical

treatment, careful monitoring and a lifetime necessity of maximum self-care. In extreme cases, pocket depths of up to 12 millimeters have been recorded before a tooth has been exfoliated or extracted. As the probing depth increases beyond 3 millimeters, *professional judgment* becomes an increasingly major factor in determining whether preventive measures alone, or whether a combination of noninvasive preventive and invasive treatment strategies are necessary.

Gingival Recession

At this point, the term *gingival recession* should be introduced. As the attachment loss continues, the free gingival margin may recede apically along with the epithelial attachment as well as the underlying alveolar bone. In such a case, the pocket depth may be near normal (as measured from the crest of the free gingival margin), while the attachment loss increases (as measured from the cementoenamel junction).

With a receding free marginal gingiva, there is a loss of supporting alveolar bone. There is also a loss of the vertical height of the *attached* gingiva. For every millimeter of attachment loss, there is a corresponding loss of attached gingiva. If for instance, it is assumed that there were 12 millimeters of attached gingiva[h] at the time of periodontal normalcy, but after 20 years, the attachment loss is 6 millimeters, then there is only approximately 6 millimeters of attached gingiva (original 12 mm minus 6 mm epithelial attachment loss = 6 mm present attached gingiva).

[h]The attached gingiva is attached to the side of the alveolus, and extends from the base of the marginal gingival to the mucobuccal fold.

Figure 13-8 Examples of probing. A. Shallow sulcus on lingual side; B. deeper sulcus (pocket) on bucal surface. (Dr. Arden Christen, Indiana University School of Dentistry, Indianapolis, IN.)

Question 4

Which of the following statements, if any, are correct?

A. If one is probing an infected area (meaning, periodontal pocket) and perforates a few capillaries, it can set off a bacteremia which can be dangerous to a cardiovascular patient.

B. The rapidity of apical migration of the epithelial attachment can only be determined by measurements made at two appropriately separated times.

C. The computer linked periodontal probe is reproducible in replicate pocket depth measurements within a range of 1 millimeter; whereas, the manual probe has an equal or better record of reproducibility for pocket depth measurements.

D. The probing for pocket depth is the same as for probing for epithelial attachment loss; the only difference is the reference point from which measurements are made.

E. There is an inverse relationship between the depth of a periodontal pocket and the vertical height of the attached gingival, i.e., the deeper the pocket, the lessening of the height of the attached gingiva.

Community Periodontal Index of Treatment Needs (CPITN)

The previous plaque, oral debris and bleeding indices were epidemiological instruments that could be *visually* accomplished for events occurring *above* the gingival margin. The next two widely used epidemiology indices, the Community Periodontal Index of Treatment Needs (CPITN) and the Periodontal Recording System (PSR) require probing to evaluate the periodontal health (or otherwise) that occurs below the gingival margin.

The Federation Dentaire Internationale (FDI) in collaboration with the Oral Health Unit of the World Health Organization (WHO) developed the Community Periodontal Index of Treatment Needs (CPITN)[72] to attain more uniform worldwide epidemiology data. In this screening index, the periodontal treatment needs are recorded for six segments (sextants). The segments are the anterior and two posterior sets of maxillary and mandibular teeth. The system excludes the third molars, except where the third molars are functioning in the place of the second molars. A sextant must have at least two functional teeth. The *highest* (worst) of the coded conditions in Table 13-4 is recorded for *each sextant.*

A special color-coded black banded probe from 3.5 to 5.5 mm and circular rings at 8.5 and 11.5 facilitates uniformity of scores in the world-wide accomplishment of the CPITN.[72] If the black band cannot be seen above the marginal gingival following insertion, a code 4 is recorded. Other possibilities with lesser periodontal involvement are code 3 if the marginal gingival falls within the range of the black band, code 2 if there is supra- or subgingival calculus, code 1 if gingival bleeding on gentle pressure, and code 0 if there is no sign of disease. There is no rule specifying the number of separate probing to be made.[72]

Periodontal Screening and Recording System (PSR)

This system of screening was introduced by the American Dental Association (ADA)[i] and the American Academy of Periodontology in 1992. It was developed to encourage dentists to screen individual patients 18 years of age and older for undetected periodontal disease. Only 5 minutes is needed to accomplish the screening (probing). The same probe and the same scoring of 0 to 4 basis is used as with the CPITN. The highest recording of "4" indicates a probing depth of over 5.5 millimeters for at least *one tooth* in the sextant. A weight force of no more than 20 to 25 g is considered sufficient to detect pathology without causing pain (see Figure 13-5). The *major difference* between the CPITN and the PSR is that with the latter, a *guideline for treatment* is suggested to match the level of treatment needs with the level of disease severity (Appendix 13-3 and Appendix 13-4).

[i]Additional information can be secured from http://www.ada.org/prof/prac/issues/pubs/psr/.

Identification of Periodontopathogens by DNA Analysis

The DNA analysis, (sometimes called "probe analysis") is an accurate diagnostic method for identifying bacteria.[74-76] It does not depend on the presence of living

bacteria, and thus requires no special packaging before being sent to the laboratory. The test is based on the fact that the two molecular strands of DNA of a bacterium are always complementary (as in a human) and can be separated. One single control strand of the DNA from an unknown bacterial species *taken from the patient's* subgingival plaque is matched with a complementary strand of the DNA bacteria *from a known laboratory culture.* The laboratory strand is marked with a radioisotope so that it can be quanitated if it combines with the patient's DNA complementary strand, for instance from *Actinobacillus actinomycetemcomitans* (Aa), *Porphyromonas gingivalis* (Pg), or *Prevotella intermedia* (Pi). As the number of complementary strand couplings increase, the amount of radioactivity also increases. It is the total level of radioactivity detected that forms the basis of the final report.

The American Dental Association has granted its *Seal of Acceptance* to two DNA assay systems, the DMD and the Pathotek Pathogen Detection Systems. The DMD test measures *individual levels of radioactivity* for Aa or Pg or Pi, with the results reported as negative, low, moderate, or high radioactivity for *each* of the organisms. A color-illustrated report graphically illustrates periodontal risk levels. This report can be used for purposes of treatment, patient education and motivation. Pathotek detects all *three pathogens* and reports on their combined total level.

The DNA assay tests are simple to apply. The site at the orifice of the gingival crevice to be sampled is cleaned of supragingival plaque and a paper point is gently inserted into the sulcus and removed after 10 seconds. It is then placed in a vial that is mailed to the laboratory for analysis. Test results are generally available within 10 days after mailing or within a few days if a telephone report is desired.

The Immune Factor

Since the majority of treatment for periodontitis centers around suppressing or eliminating the bacterial challenge of the plaque organisms, it is natural to begin to look at the body's humoral and cellular immune system for help.[77,78] Many vaccines have been developed for *other diseases*;[79] a *model animal system* has been developed for serious periodontal disease vaccine testing;[80] one promising vaccine study is now underway in England for *caries* (see Chapter 23);[81] and daily, the *function of new genes* is being announced There is no doubt that the terminology of recombinant technology, combination vaccines, genetically tailored vaccines, and preformed antibodies are but a few new terms for the dental lexicon of the future.[79]

The recent completion of the *Human Genome Project* with its decoding of desoxynucleic acid, has set the stage for a tremendous surge of knowledge about chromosomes, genes, and antibodies, and their relation to disease and abnormalities. Efforts are currently directed towards identifying markers helpful in preventing, diagnosing, predicting, and curing diseases. Drug companies are studying antibodies to which drugs can be coupled to develop perfectly targeted "magic bullets." Others are working on high priority vaccines to control diseases such as diabetes mellitus where the successful development of such a vaccine would immediately have the effect on reducing the great number of associated periodontal cases.

A few examples of individual accomplishments can be cited. Kornman has pointed out that the *pro-inflammatory cytokine,* interleukin-1 can be used as a predictor for

future severe periodontitis.[82,83] *Cytokines* are produced by various cell types such as *macrophages, neutrophils,* and *fibrocytes* which would be expected to be the defense and repair elements in the area(s) of inflammation.[84] Another study found that a platelet-activating factor (PAF) progressively increased as the severity of periodontitis increased.[85] There is research being accomplished targeted at adhesins, with the hope that *anti-adhesin* antibodies will negate the ability of a bacterium to adhere to a tooth surface or to soft tissue.[86,87] A dramatic clinical outcome relates to a 6-year old girl diagnosed with chronic, probably congenital neutropenia, replete with recurrent oral ulcerations and significant periodontal breakdown resembling prepubertal periodontitis. Granulocytic colony stimulating factor (G-CSF) was administered, resulting in an increase in granulocyte count. In two weeks, there was a resolution of the neutropenic-induced ulceration.[88]

Unfortunately, there is yet no uniform integrating theory for the myriad of genes involved in the majority of diseases[89]—much as the Periodic Table that made possible the ability to predict the properties of elements that were not yet discovered.

Question 5

Which of the following statements, if any, are correct?

A. The CPITN is a periodontal index in which pocket measurements of all teeth in the (six) sextants are recorded.

B. The PSR suggests the level of treatment of care needed to match the severity of the periodontal findings.

C. If one strand of a bacterium's DNA from a patient's mouth matches a complementary strand of a bacterium from a known laboratory bacterium, it provides a positive identity of the patient's organism.

D. Inflammatory cytokines are released by body defense cells such as the macrophage, neutrophil and fibrocyte.

E. The worldwide effort to successfully decode the desoxynucleic acid molecule is known as the Human Genetic Project.

Crevicular Fluid Assessment

Gingival Crevicular Fluid Measurement and Analysis

The physiologic flow of gingival crevicular fluid serves (1) to flush out metabolic catabolites in the sulcus,[90] and (2) this fluid also contains protective elements of the host's humoral and cellular defense system that *continually* bathes the four smooth surfaces of the teeth. As gingivitis increases in severity, so does the flow of gingival crevicular fluid—usually proportional to severity of the gingivitis,[91] but *not* to the severity of peridontitis. Thus, the measurement of the flow rate of crevicular fluid has been proposed as a means of monitoring the degree of gingival inflammation.[92,93] Others have proposed that the level of some of the chemical constituents of saliva could also be used as markers of gingival disease status.[94]

The presence of gingival crevicular fluid (GCF) as a gingivitis indicator would have numerous advantages. The knowledge that periodontal destruction progresses through periodic but *unpredictable cycles* of acute episodes followed by periods of quiescence has stimulated investigation of the GCF components. The results from GCF analysis may provide information on the host's responses to inflammation and tissue destruction during these episodes. The flow of GCF is site-specific, sometimes affecting only one individual tooth, or even one specific site on that one specific tooth. Gingival crevicular fluid is a conveniently sampled transudate that contains components derived from both the host tissues and the subgingival plaque.[92]

It is relatively simple to measure the rate of crevicular flow. However, the fluid is now mainly used as a research tool, with the hope of discovering some marker that signals either active or inactive status. The procedure for collecting the GCF is as follows.

1. After the gingiva has been isolated with cotton rolls, the tissue is dried with a gentle stream of air for 5 seconds.

2. A paper strip is inserted into the sulcus for 5 seconds, removed, and discarded.

3. A second strip is either placed at the entrance to the gingival sulcus(extrasulcular method) or inserted into the sulcus until a frictional bind is encountered (intrasulcular technique). (The intrasulcular method can itself irritate the crevicular epithelium and trigger the flow of crevicular fluid.)

4. The strips are allowed to remain in place for 5 seconds.

5. The amount of crevicular fluid can be quantitated by placing the strips in a gingival fluid meter measuring device called the Periotron. The extent of wetness of the paper strip produced by the gingival fluid affects the flow of a current through a moisture-sensitive sensor. The amount of current flow is displayed as a digital readout.[91,95]

It is claimed that this method provides an "early warning system" for the detection of gingivitis.

Smoking as a Major Risk Factor for Periodontal Disease

In the United States there are an estimated 50 million smokers, and another unknown number of individuals exposed to passive smoke.[96] It is well recognized that smoking is the cause of many systemic pathologies, of which *lung cancer, cardiac disease,* and *stroke* are best known and most feared.[97] Unfortunately, lethal tobacco products—cigarette and use of smokeless ("spit") tobacco—have gone unnoticed as causal agents for disabling and disfiguring oral and pharyngeal cancer.[98] There have also been many articles published over the past five decades pointing out that tobacco is a causal agent for *periodontal disease,* an oral disease that affects millions. The enormity of this relationship is found in a statement that *smoking accounts for one-half of all periodontal cases and three-fourths of oral cancer cases* in the United States.[99]

The role of smokeless ("spit")[j] tobacco on oral morbidity and mortality has received little media attention.[100,101] For instance, in providing a balanced presentation on the oral hazards of smoking, chewing tobacco must also be associated with an increased risk of root caries as well as periodontal disease.[102,103] Men who used chewing tobacco were four times more likely than those who had never used tobacco, to have one or more decayed or filled root surfaces.

It is now beginning to emerge that probably one of the most important questions that can be asked on the patient's dental and medical history form is, *"Do you smoke or live with someone who smokes, and/or do you use smokeless tobacco?"*

Smoking is a high-risk habit and constitutes a major periodontal problem.[100,104-107] Mirbod has provided a litany of tobacco-associated lesions found in the mouth—squamous-cell carcinoma, gingivitis and periodontitis, burns and keratosis patches, black hairy tongue, palate erosions, leukoplakia and epithelial dysplasia, and tooth staining.[108] All can be visually detected early when aggressive treatment produces the best results.

The use of tobacco products has an adverse effect of the onset, prevention, prognosis, treatment and maintenance phases of periodontitis. Part of this negative outlook is because smoking also causes adverse changes in the body's immune response system that present major barriers to successful periodontal treatment.[107,109] Smoking is associated with alveolar bone loss, with epithelial attachment loss, gingival recession, and with periodontal pocket development.[104] In an extensive review of the literature Haber discusses many of his own studies and observations to explain how a patient's smoking history differs from a nonsmoker and its effect on periodontal disease.[110] For example:

- Tobacco users are 2.5 to 6 times more likely to develop periodontal disease than nonsmokers.[109]
- A patient's smoking history is a useful clinical indicator of *future* periodontal disease activity.[110]
- There are more current smokers who seek professional periodontal care than nonsmokers.[111,112]
- There appears to be a relationship between the *number of cigarettes smoked* and the risk of developing periodontal diseases.[109,113]
- As the number of cigarettes smoked increases, so does the severity of the periodontal disease.[110,105]
- More smokers than nonsmokers are classified as having severe adult periodontitis and severe early-onset generalized periodontitis.[114]
- Almost *100%* of 30- to 40-year-old heavy smokers have periodontitis;[113] the response of these patients to therapy is *not* as favorable as for nonsmokers.[115,98]
- Approximately 86 to 90% of refractory periodontitis (not responsive to treatment) patients are current smokers.[113]
- There is a strong association between smoking and alveolar *bone and tooth loss.*[116-118,106]
- Smoking is associated with adverse changes in the body's immune system.[104]
- The *gingival fibroblastic repair function* is altered, resulting in a thickened fibrotic gingiva.[110,119]

- Following treatment, the improvements in probing depths and epithelial attachments are still more favorable for the nonsmoker.[120,115]
- There is more pocketing of the anterior segments of teeth for smokers than nonsmokers.
- It is very difficult to persuade a patient to quit smoking as part of the treatment plan.

A seminal article, by Barbour and associates, focuses on the degraded immune response of smokers with periodontitis when compared to nonsmokers. They confirm Haber's observation that there is a *neutrophilic deficiency*, but in addition point out many other functions of the immune system that are compromised—including phagocytosis, chemotaxis, immune suppression, immune surveillance, and alterations of immunoglobulin function. Each of the areas is well documented and discussed. One of the considerations of the study was to determine the effects of smoking on periodontal disease, especially how it was related to host defense mechanisms.[109] An intriguing question would be, "In view of the altered immune response, is periodonitis a predictor of susceptibility to other smoking diseases that have a later onset?"

[j]Spit tobacco: A designation of a tobacco associated with this habit.

Smoking Cessation and Recovery

Smoking cessation is an essential component for the successful treatment of periodontal disease—there is little rationale for treating periodontitis without eliminating one of the major causes of the disease.[113] Thus, there is also the question of whether periodontal surgical treatment is indicated without a commitment by the patient to quit smoking.[113] As with other smoking diseases, cessation is only the first step of a long healing process where the smoker often does not approach the lower risk of the nonsmoker for 10 to 20 years.[97,98] Krall and coworkers estimated that the risk of tooth loss 12 years after smoking cessation was reduced by 20%.[118] However, the fact that the rate of tooth loss of ex-smokers falls between the data for current smokers and those who never smoked indicates that recovery is taking place.[118] The periodontal status and bone loss of ex-smokers also appears to be intermediate between current smokers and those who never smoked.[110,116,117]

Gingival improvement is more rapid following smoking cessation although a bit quixotic. Usually a smoker's gingiva has a glazed fibrotic appearance with rolled edges after years of smoking. *Bleeding is minimal* on brushing. It is believed that this is due to the local effect of the tobacco smoke possibly suppressing the inflammatory reaction. This *local* and *direct* effect of components in the cigarette smoke are believed to also account for a greater amount of pocketing that occurs in the anterior teeth. However, about 10 to 12 weeks after quitting smoking, there is an increase in bleeding, possibly caused by a recovery of the inflammatory response. About a year after cessation, the fibrotic, thickened anatomy of the gingiva begins to assume a more normal appearance and the periodontitis appears to stabilize. For the majority of patients, attachment loss ceases or dramatically slows.[110,119]

The present methods for prevention and treatment of gingivitis and periodontal disease emphasize the need for meticulous day-to-day oral hygiene procedures. Even so these preventive actions are not intended to, nor are they adequate to continuously replace a compromised portion of the immune system brought on by smoking. Where

smoking occurs, the chemical and manual methods of plaque control still help to reduce the bacterial challenge. However, with round-the-clock impaired humoral and cellular body defenses, and with an impaired gingival fibroblastic repair capability, the entire defense and recovery process is jeopardized.[120,115]

There is a need to provide smoking cessation counseling programs as an integral part of the periodontal treatment plan. The American Cancer Society, American Heart Association, and American Lung Association[k] sponsor many "No Smoking" classes throughout the nation to facilitate smoking cessation. For *youthful* smokers and potential smokers, the emphasis should be on *smoking avoidance* and *refusal* techniques. This task can probably be best accomplished in the public-school system (see Chapter 19).

The outcome of such counseling will probably be more successful if linked with a discussion of the great amount of preventable *morbidity* and *mortality* caused by active and passive smoking and the use of smokeless tobacco. *To teach dental students* to accept this counseling responsibility, the University of Indiana Dental School has established an antismoking curriculum program for student teaching and patient education.[121] In addition there is an Indiana University Nicotine Dependence Program at the University Cancer Center that is operated in conjunction with the medical school and several area hospitals.[122]

[k]To identify the nearest location of no-smoking classes in the United States, call 1-800-LUNG-USA.

Question 6

Which of the following statements, if any, are correct?

A. An increasing flow of Gingival Crevicular Fluid (GFC) is indicative of an increasing severity of gingivitis, but not of peridontitis.

B. If present tobacco smoking was to cease, approximately one-half of the present need for periodontal care would also cease.

C. Smoking of cigarettes and use of smokeless tobacco can act directly on the oral tissues, as exemplified by the presence of oral and pharyngeal cancer.

D. The smoking history of an individual can aid in predicting the possibility of future onset, as well as the eventual long-range outcome of the treatment of periodontal disease.

E. Smoking can be the cause of the earlier development of periodontitis, an increase in its severity, and reduction in the probability of a successful treatment outcome.

Summary

Two of the most important messages of the entire chapter are: (1) at the first sign of gingival bleeding, regardless of age, a dentist should be seen immediately for diagnosis, treatment, education and monitoring; and, (2) for all patients who smoke, to

encourage and help facilitate their participation in an anti-smoking program.

Many indices are used to determine the prevalence and severity of gingivitis and/or periodontitis among a given population, or to determine the severity of gingivitis and/or periodontitis among individual patients. The most commonly used markers are a plaque index, gingival bleeding, loss of epithelial attachment and pocket depth. With computer software, data collection can be easily extended to include recession, suppuration, furcation involvement, tooth mobility and others. The most important detail that delineates gingivitis from periodontitis is the integrity of the epithelial attachment. As long as the pocket depth measurements approximate 3 millimeters with no bleeding and no recent loss of epithelial attachment, the periodontium can be considered in excellent health. As the pocket probing depths become greater, noninvasive preventive procedures become more difficult to apply while invasive treatment becomes more frequent and complex. Manual probes are used to determine sulcus depth; however, the constant-force electronic probes appear to be more accurate, reproducible and easier to use in recording data. Laboratory tests are often used to determine the microorganisms of the subgingival plaque. Some progress has been made, but the cause-and-effect of these bacteria is not as well understood as are the cariogenic mutans streptococci and lactobacillus. Immune studies are permitting the researchers to better understand the dynamic interaction between the pathogenic organisms and the body defenses. Possibly one of the most important harbingers of gingivitis and periodontitis is cigarette smoking and/or use of smokeless tobacco products. Many studies have found that tooth loss from periodontal disease is associated with tobacco use. Investigators have reported that current smokers have a greater prevalence of severe periodontal problems, as well as accompanying breakdowns of various components of the immune system than do individuals who have never smoked. With smoking, the challenge organisms are rarely confronted by a fully effective immune defense system.

Answers and Explanations

1. A, B, and C—correct.

D—incorrect. The first two, cardiac disease and diabetes are correct; most viral diseases is incorrect.

E—incorrect. If there is a slow apical migration of the epithelial attachment past the CEJ, it is by definition a periodontitis. Once that diagnosis is correctly made, it cannot revert to a gingivitis.

2. A and C—correct.

B—incorrect. Once periodontitis is correctly diagnosed, there is no way to restore the tissues to their previous histolology; however, future progress can be controlled—but not cured.

D—incorrect. Mouthrinses do not penetrate the subgingival plaque to the extent necessary to kill or to remove the periodontopathogens.

E—incorrect. An implant is anchored in alveolar bone. It is a free-standing structure

that needs no support.

3. A and C—correct.

B—incorrect. An O'Leary Index score of 10% is considered excellent—not marginal.

D—incorrect. The Loe-Silness Index is used to record gingival bleeding—not plaque.

E—incorrect. Bleeding can occur as a result of either gingivitis or a periodontitis. It requires a differential diagnosis to determine which.

4. A, B, D, and E—correct.

C—incorrect. It should be vice versa. The computer linked periodontal probe is reproducible to within 0.2 millimeter; the manual probe to within 1 millimeter.

5. B, C, and D—correct.

A—incorrect. Only one tooth—the tooth with the worst (highest) 0 to 4 score in each sextant is recorded.

E—incorrect. The title to the program was the Human *Genome* Project.

6. A, B, C, D, and E—correct.

Self-Evaluation Questions

1. Two popular indices for determining the location of plaque on smooth surfaces of the teeth are the _____ Index and the _____ Index.

2. Greene and Vermillion developed the _____ Index and then truncated it for convenience in epidemiology studies.

3. The _____ is an instrument to measure the flow of gingival curricular fluid.

4. Name three devices used in self-care, of which one is a toothbrush; the other two are _____ and an _____.

5. The equivalent of periodontitis around an implant is called _____.

6. The disease of the periodontium that proceeds periodontitis is _____.

7. The device used to determine pocket depth and epithelial attachment loss is called a _____.

8. For every millimeter loss of epithelial attachment, there is a corresponding loss of the same distance of the _____ gingiva.

9. A personal habit that accounts for approximately one-half of all the periodontal

diseases in the United States is _____.

10. The single delineating factor (by definition) that separates gingivitis from periodontitis is _____.

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Chapter 14. Sugar and Other Sweeteners - *Peter E. Cleaton-Jones Connie Mobley*

Objectives

At the end of this chapter it will be possible to

1. Name the three sugars that are composed of molecules of glucose, fructose, or galactose, all of which can produce caries.

2. Define sugars, sweeteners, and sugar replacers.

3. Describe the potential impact of an excessive intake of added sugars on the quality of the human diet.

4. List three polyols that are sweeteners and cite their advantages and disadvantages in influencing caries incidence.

5. Defend the Food and Drug Administration (FDA) for either removing or attempting to remove saccharin and cyclamate from the marketplace.

6. Name a sweetener that has recently received FDA approval, and list three more that are candidates for approval.

Introduction

To most people the term sugar refers to the common household foodstuff table sugar (sucrose). Yet sucrose is only one of many naturally occurring sugars used in the human diet. Technically the term sugar applies to two classifications of carbohydrates. Free-form monosaccharides (simple sugars) include the more common glucose, fructose, and galactose. Disaccharides (two simple sugar molecules linked together) include the most common sucrose, lactose, and maltose. Naturally occurring sugars are available in fruits, vegetables, grains, and dairy foods.

Sweeteners are added sugars that are used as ingredients to both satisfy our taste and in some cases provide added energy. Grouping sweeteners as "nutritive" or "non-nutritive" acknowledges a difference in the amount of energy provided by the sweetener. Nutritive sweeteners may be referred to as caloric and include sugars and sugar alcohols. Non-nutritive sweeteners offer no energy and can sweeten with little volume. Both the sugar alcohols and non-nutritive sweeteners can replace the sugars and are sometimes referred to as sugar substitutes, sugar replacers or alternative sweeteners.[1] Table 14-1 lists sweeteners available in the food supply and their unique characteristics.

Sensation of Taste

It is difficult to determine whether taste is genetically linked, acquired in utero, neonatal, or influenced by visual, auditory, or taste stimuli during infancy, early childhood, or even adulthood.[2]

Taste buds are present and functioning before birth, a fact demonstrated by injecting sweetening agents into the amniotic fluid during the fourth month of pregnancy.[3] The sweetened amniotic fluid results in an increased rate of swallowing by the fetus. At birth, infants show a taste preference for sucrose, and their taste cells are more responsive to sucrose than to other sugars. Whether it is simply a pleasurable taste or a true metabolic need is not known.

Taste sensation is initiated by the arrival of a stimulus at the taste buds. Taste recognition occurs when the receptor sites of the cells of the taste buds carry, by cranial nerves, a qualitative and quantitative message to the brain. The messages are processed, and the stimulus is recognized as either sweet, sour, salty, or bitter, or some combination of these four.

The Historic Importance of Sweeteners

The first recorded evidence of sweeteners dates to 2600 B.C. Drawings in Egyptian tombs illustrate beekeeping practices for honey production. The honey was reserved for the rich and powerful.

Cultivating sugar cane began in southeast Asia, India, and China around 100 B.C. The earliest known written reference to sugar cane occurs in a scroll dating to 375 A.D. The Arabs developed the first process for refining sugar cane into sucrose. The cultivation of sugar cane was practiced in southern Europe in the 13th century, and eventually knowledge of it spread to the New World. The cultivation of the root crop called sugar beets started more than 200 years ago.[4]

North American Indians had devised a method of bleeding the sap of the sugar maple tree long before the Pilgrims arrived in Massachusetts. The sugar in the sap of the mature sugar maple is almost exclusively sucrose.

Sweeteners made from corn starch began to appear around 1910. The lesser sweetness of the sugars derived from corn starch, mainly glucose (also referred to as dextrose), was responsible for characterizing them as substitute sweeteners. This identity imposed restrictions on their use. In the 1960s and 1970s, new chemical processes were developed which resulted in the ability to convert the glucose contained in corn starch to fructose. This conversion led to the production of a variety of high-fructose corn syrups (HFCS). Because fructose is twice as sweet as glucose, its use has increased rapidly.

The amount of HFCS used as a sweetener surpassed that of sucrose in 1985. Aspartame, which is approximately 180 times sweeter than sucrose, is the most frequently used noncaloric sweetener. From its discovery in 1965, it is now being used by more than 100 million people worldwide.[5]

Sucrose

Sucrose is the most commonly used tabletop sweetener. Absolute usage in a country is not known but the disappearance of sucrose from the market is the commonly used estimate but it must be understood that this estimate includes wastage which is not ingested by people. In the United States estimated usage has decreased from the high of 102 lb (46 kg) per person in 1971 to 65.8 lb (30 kg) per person in 2000[7] (Figure 14-1). For the United Kingdom the 1993 usage was 44 lb (20 kg) per person.[8] In the past, this usage was considered consumption data; however, this is a misnomer. Usage is actually the quantity of sweeteners delivered to commercial establishments to be used in various ways. It does not account for any loss caused by waste, nor does it include any additional natural sugars consumed. It has been reported that up to 31% of total sugars consumed by adolescents are hidden sugars in foods, such as in milk and fruit.[9]

Individual variation in consumption also occurs. Males generally consume more sucrose than females, and teenagers are by far the greatest consumers. Peak consumptions occur among 15- to 18-year-old males.[14] A decreased consumption rates occurs among members of large families.

Figure 14-1 Sweetener use in the United States 1900-2000 in lb per capita.[6,7,11-13]

Uses of Sucrose

Sucrose has several attributes that make it desirable for the food industry. It is ideal in the following roles:

- Sweetening agent: The character of the sweet taste can be varied according to pH and temperature used to make a product, as well as by its interaction with other ingredients in the formulation. The level of sweetness is important to the acceptance of certain foods.
- Flavor blender and modifier: In some foods, such as mayonnaise, sucrose is a flavor blender; in other foods, such as pickles, it reduces the acidic bite and sour taste.
- Texture and bodying agent: Sucrose gives a texture that is highly acceptable to consumers. It provides body and a distinctive "mouth feel" to food products.
- Dispersing/lubricating agent: In dry packaged mixes, sucrose is used as an agent to keep other ingredients from packing too closely. This, in turn, permits a better blending of the ingredients during food preparation.
- Caramelization/color agent: Caramelization during baking produces a brown color, which increases acceptance. It provides a desirable, characteristic flavor and aroma to the food product.
- Bulking agent: When a noncaloric sweetener that may be 200 times sweeter than sugar replaces sucrose, other ingredients must be added to replace the lost sucrose "bulk" to maintain the food's normal appearance and consistency.

Earlier in this century, home canning and baking resulted in a higher per capita consumption of sucrose than for industrially processed foods. Modern-day affluence, the desire to be liberated from the kitchen, and a higher percentage of working women are factors that have helped reverse the trend. Seventy-five percent of the sucrose manufactured between 1910 and 1930 was delivered to households. In 1950, industrial uses of sucrose surpassed that used at home.[3] The food-processing industry has greatly changed the eating habits of the average American by increasing the output of processed foods. No longer are there only three meals during the day; instead, individual food intake patterns have been extended to include a continuous morning-to-night intake of snacks and beverages, many of them containing sucrose. In a less-developed country, South Africa, home use is still 70% of total use and in the 2000/2001 season was 64 lb (29 kg) per person.[15]

Sucrose has several disadvantages that restrict its industrial use.

- The high concentration (osmolarity) used in canning often causes shrinkage and wrinkling of canned fruits. Both characteristics detract from the visual appeal of the product.
- It absorbs moisture (hygroscopic) and accordingly makes it difficult to freeze-dry food containing high concentrations of sucrose.
- It chars at high temperatures; thus, it cannot be used to sweeten items that must be fried—bacon, for instance.
- It supports bacterial growth; hence, its use in prepared food increases the potential

for bacterial contamination and spoilage.

Evaluation of the Health Aspects of Sucrose

Prior to 1958, few regulatory constraints existed on the introduction of new products into foods. If problems developed, the United States Food and Drug Administration (FDA) had to prevail on the conscience of the manufacturer to withdraw the product or to prove in court that the product was not safe. Both options were rather daunting because considerable financial interest was usually involved. In 1958, the U.S. Congress passed the Foods Additive Amendment that required preliminary marketing clearance. The act required the following information on additives: (1) chemical composition, (2) method of manufacture, (3) analytic method used for the detection of the additive, (4) proof that the additive accomplished its intended effect and that it did not occur in excess of the amount required to achieve that effect, and (5) proof that it was safe.[16] The burden of proof in substantiating any of these factors resided in the petitioner applying for the clearance, and not with the FDA.

The Foods Additive Amendment decreed that all components added to processed foodstuffs prior to 1958 were classified as food ingredients, whereas those added thereafter were called food additives. With this act, Congress authorized a list of food ingredients which it called generally regarded as safe (GRAS). Sucrose was listed as a food ingredient and placed on the GRAS list. At that time, items on the list were considered as relatively immune from future regulatory action. With the passage of time, however, all the items listed on the original GRAS list of food ingredients have come under review.[17]

In 1986, the FDA formed a Sugars Task Force that critically reviewed all of the recent scientific literature addressing potentially adverse health effects associated with sugars consumption. They investigated the cause-and-effect relationship between the use of sugar and diabetes, cardiovascular disease, hypertension, heart disease, and obesity. The task force determined that no conclusive body of research links any of the above to sugar consumed in moderation.[14] Nutrition and Your Health: Dietary Guidelines for Americans states, "Choose beverages and foods to moderate your intake of sugar."[18]

In the United Kingdom, the Committee on Medical Aspects of Food Policy (COMA) Report noted that current consumption of sugars, particularly sucrose, played no direct role in the development of cardiovascular disease, essential hypertension, or diabetes mellitus; however, the report stated that sugars are the most important dietary factor in the cause of dental caries.[19] Others share the view, based on epidemiological evidence, that sugars are one of the essential multifactorial agents in the prevalence and progression of caries.[20,21]

It has been suggested that trends in consumption of added sugar raise concern that it may also be associated with increasing rates of obesity and inadequate intakes of essential nutrients, especially calcium. Investigators used data from the U.S. Department of Agriculture's 1994-96 Continuing Survey of Food Intakes by 15,011 individuals between 2 years and older, to identify those who consumed more than 26 teaspoons of added sugars daily. These individuals tended to be both younger and male and to frequently over consume total energy.[22]

Using these same data, Guthrie and Morton identified regular soft drinks, followed by table sugar/sweeteners and sweetened grains like cookies and cakes as the primary source of added sugar in the U.S. diet. Percent total energy from added sugars, ranged from 12% for those 65 years and older to 20% for 12- to 17-year-olds, with a mean intake for the entire population of 16%.[23] Displacement of milk in the diet by regular soft drinks in children and adolescents has been demonstrated by several researchers.[24,25]

Major sources of added sugars in the U.S. diet are: table sugar, honey, syrup, candy, jam or jelly, gelatin desserts, soft drinks, fruitades, lemonades and other fruit punches, sweetened grains like cookies and cakes, dairy desserts such as ice cream, sweetened milks and yogurts. These do not include diet or sugar-free varieties with sugar replacers or substitutes.

Role in Caries Formation

Sugar in plaque is a contributory factor in dental caries.[26] Two animal studies and three human clinical studies have contributed to the understanding of the importance of sugar in the development of caries.

In 1955, the first animal study[27] was conducted with rodents in a gnotobiotic (germ-free) environment. One group of rats was fed a caries-producing diet containing large amounts of sugar. The second group was fed the same diet, but at the same time specific microorganisms were introduced to the otherwise germ-free environment. Those rats receiving the cariogenic diet alone did not develop caries; those with the cariogenic diet plus the bacteria did develop lesions (Table 14-2). Observations at that time and since have conclusively demonstrated that certain microorganisms and strains of organisms are more caries-productive than others.

In a second rodent study,[28] one group of rats was fed a caries-producing diet by means of a stomach tube, with no food coming in contact with the teeth. No caries resulted. When the same diet was fed orally and allowed to come in contact with the teeth, caries did occur (Table 14-3).

These two studies conclusively demonstrate that (1) bacteria are essential for caries development, regardless of diet, and (2) the action of the sugar in carious development is local, not systemic.

Several human studies have reported and further clarified the animal studies. Two of the most often cited occurred at Hopewood House[29] in Australia and at Vipeholm in Sweden.[30]

Hopewood House was an orphanage in Australia that accommodated up to 82 children. From its beginning, sugar and other refined carbohydrates were excluded from the children's diet. Carbohydrates were served in the form of whole meal bread, soybeans, wheat germ, oats, rice, potatoes, and some molasses. Dairy products, fruits, raw vegetables, and nuts were prominently featured in the typical menu. As illustrated in Figure 14-2, dental surveys of these children from the ages of 5 to 11 years revealed a greatly reduced caries incidence compared with the state-school population

in that age group. The children's oral hygiene was poor, with about 75% suffering from gingivitis. When the children became old enough to earn wages in the outside economy, they deviated from the original diet. A steep increase of decayed, missing, and filled teeth (DMFT) after the age of 11 years indicates that the teeth did not acquire any permanent resistance to caries (see Figure 14-2).

The Vipeholm study was conducted at a mental institution in that city located in southern Sweden. Adult patients on a nutritionally adequate diet were observed for several years and found to develop caries at a slow rate. Subsequently, the patients were divided into seven groups to compare the cariogenicity accompanying various changes in frequency and consistency of carbohydrate intake. Sucrose was included in the diet as toffee, chocolate, caramel, in bread, or in liquid form. Caries increased significantly when foods containing sucrose were ingested between meals. In addition to the frequency of eating, the consistency of the sugar-containing food was very important. Sticky or adhesive forms of food that maintained high sugar levels in the mouth for a longer time were much more cariogenic than forms that were rapidly cleared.

The Vipeholm study also demonstrated that it was possible to increase the average consumption of sugar from about 30 to 330 g per day with little increase in caries, provided the additional sugar was consumed at mealtime in solution form.[30] Two points to remember about the Vipeholm study are that abnormal quantities and presentations of food were used and that, by modern standards, the study would not receive ethical clearance.

Finally, some people suffer from a condition known as hereditary fructose intolerance (HFI). After the intake of fructose, these persons become nauseated, vomit, and sweat excessively; malaise, tremor, coma, and convulsions may develop. As a result, these individuals learn to carefully avoid foods with fructose or sucrose where fructose is one of the metabolic products. Those HFI individuals who have survived this disorder by successfully avoiding fructose or sucrose from any source are either caries-free or have very few caries.[31] The low prevalence of caries in HFI patients indicates that starchy foods alone do not produce decay, whereas sugary foods do.

What is the threshold level of sugar content above which a food is highly cariogenic? While many animal and human studies have examined the drop and recovery of plaque pH following consumption of specific foods, a truly safe level has not been established although sugar consumption of between 10 and 15 kg per person per year has been suggested.[32]

Two similar epidemiologic studies of the caries prevalence in 12-year-olds and the per capita sugar use have been done. The first, conducted in 47 countries,[33] revealed a statistically significant relationship between the availability of sugar and the number of DMFT. When daily per capita supply of sugar was less than 50 g, the DMFT index was less than 3.0 (Table 14-4). More recently, a study in 90 countries showed a statistically significant relationship between the logarithm of DMFT and sugar consumption at a slope of 0.021 per kg per person per year.[34] This significant association disappeared when only the data from the 29 industrialized countries were analyzed. This indicates that factors other than sugar consumption (i.e., oral hygiene, professional care, fluoride use), must be taken into account in explaining variation in

caries prevalence.

The erroneous impression that oral hygiene and optimum fluoride exposure will protect teeth from bad dietary practices supports the oversimplified view that just removing "sugar" from the diet is an adequate approach to preventing caries progression. The caries promoting activity of carbohydrates and sweeteners vary based on frequency of intake as well as combined intake with other foods that may vary in protein or fat content. Processed high-starch snacks—whether gelatinized, baked, or fried—produce as much acid in dental plaque as sucrose alone but at a slower rate.[35,36] Foods containing both cooked starch and sucrose, like fried potatoes and bread, have been shown to enhance caries potential.[37] When sucrose is added to cooked starch foods the caries promoting potential is increased because the starch brings the sucrose into closer contact with the tooth surface.[38] Thus, added sugars can be part of a total diet when following guidelines that suggest that few foods or beverages containing sugars or starches be eaten between meals.[18]

Streptococcus mutans is generally regarded as the microorganism having the greatest cariogenic potential in humans. Sucrose enhances the colonization and growth of *S. mutans* in dental plaque more than other monosaccharides or disaccharides. These bacteria (1) ferment sucrose rapidly, producing acids; (2) convert sucrose to extracellular polysaccharides that facilitate the adherence of the bacteria to teeth and may function as a reserve of fermentable carbohydrate necessary for the production of acids; and (3) reduce plaque permeability that in turn decreases the rate at which saliva can neutralize or dilute acids formed in the depths of the plaque.[39]

Figure 14-2 Plot of the mean number of DMFT versus chronologic age in state schools in Australia and in Hopewood House. (Reprinted by permission from Marthaler, *Caries Res.* 1, 1967.[21])

Question 1

Which of the following statements, if any, are correct?

A. Some sweeteners with a per-gram calorie content equal to sucrose can result in a lesser calorie intake because of their intense sweetness.

B. The *Foods Additive Amendment of 1958* established the basis for the GRAS list.

C. A *food additive* is less subject to FDA study than a *food ingredient.*

D. Approximately 50% more sugar is required in the diet of gnotobiotic (germ-free) rats to induce caries than in the diet of control rats.

E. Individuals with hereditary fructose intolerance (HFI) usually have more caries than sugar-tolerant individuals.

Corn Sweetener Use

The large increase in the cost of sucrose in 1974 prompted a search for a less expensive alternative. The availability of high-fructose corn syrup (HFCS) with 42% fructose provided one alternative. By late 1977, a process to produce 55% fructose

syrups was developed. The use of HFCS per capita in the United States has jumped from 0.7 lb (0.3 kg) in 1970 to 61.6 lb (28 kg) in 2000.[7] The wholesale list price of HFCS is approximately two-thirds that of sucrose. The caloric sweetener HFCS seems to be reaching maximum use on a per capita basis, and its production is not expected to continue increasing as rapidly in the future as it did in the 1970s and early 1980s. High-fructose corn syrup use in soft drinks accounted for over 70% of its use in 1992.[40] HFCS usage outside the United States is low.

A study by Scheinin[41] determined the relative cariogenicity of fructose and sucrose. Fructose was used exclusively by one group who developed 3.8 new carious lesions, whereas the sucrose group developed 7.2 new lesions. The large decrease in caries incidence in the United States may partially be explained by the increased use of HFCS sweeteners with a concurrent decrease in sucrose.

The consumption of glucose and dextrose corn syrup has remained fairly constant in the United States over the past 60 years at 3.5 lb (1.6 kg). The three leading uses are the brewing industry, confectionary, and cereal products.[12]

Effects of Other Sugars

Fructose, maltose, and lactose are also caloric sugars found in nature. A considerable amount of the first two sugars is contained in fruits and vegetables. Lactose in varying concentrations is present in all mammalian milk. The sweetness of these other sugars ranges from 0.2 to 1.8 times the sweetness of sucrose (Table 14-5).

The subjective evaluation of the sweetness of a substance is usually judged by taste panels. Several methods are used: (1) having the members of the panel write down in their own words a subjective perception of the sweetness, time of onset, aftertaste, or other descriptive terms; and (2) comparing the test sweetener against a reference sweetener, most likely sucrose. These two evaluations indicate quality but not intensity of the test material. For intensity, threshold detection and recognition levels are noted. For threshold detection testing, extreme dilutions of the sweetener are used. The threshold level is the lowest concentration at which sweetness can be discerned. Recognition tests are based on the lowest concentration at which a panel can recognize the specific sweetener being tested. Testing is accomplished with the sample solutions at 37°C because temperature does modify taste perception. The threshold level is much lower than the recognition level.

The Polyols as Sweeteners

The most commonly known polyols include sorbitol, mannitol, and xylitol. These polyols are not sugars in the strictest sense. Each molecule resembles a sugar, with the exception that an alcohol grouping is attached to each carbon atom of the polyol. Often they are referred to as "sugar alcohols."

The polyols have 40 to 75% of the caloric content of sucrose. Xylitol has the same sweetness as sucrose. Polyols have similar physical characteristics to sucrose, and their substitution does not change the customary size and weight of a product. Browning or caramelization, however, does not occur with food products that have been sweetened with polyols.

Sorbitol

Sorbitol, first isolated in 1872, is mainly used in chewing gum, toothpaste, frozen desserts, and some candy. The dental interest in sorbitol results from its use in so-called sugar-free gum, which has been claimed to be noncariogenic. This claim of noncariogenicity has not been substantiated by clinical trials, but intraoral studies have indicated that the plaque pH seldom drops below 5.7 after chewing sorbitol-sweetened gum.[42] The need for further studies is emphasized by the fact that S. mutans is known to metabolize sorbitol.

Mannitol

Mannitol, which occurs naturally in seaweed, is also derived from the sugar mannose. This sweetener is metabolized very slowly by oral microorganisms and has virtually no cariogenic potential.[43] Mannitol is used in toothpastes, mouth rinses, and as a dusting agent for chewing gum.

The Polyols as Sweeteners

Xylitol

The polyol that has received the greatest amount of attention by the dental profession is xylitol. Xylitol is derived from birch trees, corn cobs, and oats, as well as from bananas, strawberries and certain mushrooms. As with other polyols, the appearance and texture of xylitol is similar to sucrose. Its cost is about 10 times that of sucrose. Even with a significant expansion in xylitol production, the cost cannot be reduced by much more than half.

Clinical, salivary chemistry and microbiologic evidence suggest that xylitol is the best nutritive sucrose substitute with respect to caries prevention. It has been shown to be nonacidogenic and therefore noncariogenic.[a,44] The main use of xylitol appears to be where it is used in partial substitution for other sugars. This takes advantage of its microbial action, with the food item still being competitive in price.

All of these sugar alcohols have been recognized as having a low potential of producing dental caries. Therefore, in the United States, Congress has authorized products containing less than 0.5 g of sugar and a sugar alcohol to be labeled as "reducing" or "not promoting" tooth decay.[45] This labeling went into effect in January 1998.

Regarding dental-caries prevention, the main use today of the polyols, notably sorbitol and xylitol, is in chewing gums.[46] It is believed, however, that the caries preventive effect of substituted chewing gums is the chewing process itself rather than the sugar-substitutes such as the polyols.[47]

[a]Noncariogenic = Does not cause caries.

Question 2

Which of the following statements, if any, are correct?

A. Hopewood House in Australia established the fact that *restricting refined carbohydrate intake* reduces caries incidence, whereas the Vipeholm study in Sweden demonstrated that *frequency of intake and consistency* of sugar products are important in evaluating cariogenicity of foods.

B. A higher concentration of a sweetener is required for recognition than for detection.

C. It is possible for a noncariogenic substance to be anticariogenic,[b] but not all noncariogenic agents are anticariogenic.

D. All products with polyol sweeteners are considered sugar-free.

E. Xylitol is the best nutritive sucrose substitute with respect to caries prevention.

[b]Anticariogenic = Reverses the caries process prior to cavitation by enhancing remineralization.

Intense Sweeteners

The need for intense sweeteners is acute. For primary preventive dentistry practices, a noncarious product that could be used in oral medications, mouthrinses, dentifrices, and all forms of "candy" or between-meal snacks is highly desirable. The American Dental Association (ADA) is encouraging the use of intense, or artificial, sweeteners.

Very small amounts of intense sweeteners can be used to achieve acceptable levels of sweetness. Even though the cost of these sweeteners may be 100 times greater than an equal amount of sucrose, they are 90 percent more economical than sucrose because their equivalent sweetness can be 1,000 times that of sucrose.

In 1977, the U.S. Senate Select Committee on Nutrition and Human Needs proposed as a dietary goal for the United States that no more than 10% of one's total daily calories be from refined sugars and other caloric sweeteners. In 1978, the average daily diet provided 18%[48] of total calories through sugar and other caloric sweeteners. In 1986, however, that percentage was estimated to be 11%[14] (Figure 14-3). Reaching closer to this goal was made possible by a reduction in sugar intake and a possible increase in the use of acceptable intense sweeteners although evidence for the latter is lacking.

The most popular intense sweeteners in the United States are saccharin, aspartame, acesulfame-K, and sucralose.

Saccharin

Saccharin is considered approximately 300 times sweeter than sucrose. In 1988, approximately 6 lb (3 kg) of saccharin per person (sugar-sweetness equivalent weight) was delivered for use as a sweetener in the United States, a drop from 10 lb (5 kg) in 1984. Because of its intense sweetness, the use of saccharin is only about 4% as costly

as an equivalent sweetness derived from sucrose.[49] Saccharin is compatible with most food and drug ingredients. Its major deterrent, a metallic aftertaste, can be recognized by most users.

On April 15, 1977, on the basis of alleged carcinogenicity, the revocation of previous approvals for saccharin was proposed by the FDA with the recommendation that saccharin be classified as a drug, meaning it could only be sold by prescription. This decision set off a consumer furor across the nation, resulting in bills being passed in Congress to postpone the ban on saccharin for 18 months. Congress has reacted with a series of 2-year moratoriums that prohibit the FDA from banning use of saccharin in diet sodas and food while permitting more time for further research. In 1987, 1992, and 1996, 5-year moratoriums were passed by Congress. In 1992, the FDA formally withdrew its 1977 proposal to ban the use of saccharin. The agency did not address the safety of saccharin, but stated it would repropose the ban later should such action be warranted.[50]

Aspartame

Aspartame, better known by one of its tradenames, NutraSweet, is void of an unpleasant aftertaste. It is a dipeptide of two naturally occurring amino acids, phenylalanine and aspartic acid, but it is not found in nature. It was a serendipitous discovery by James Schlatter, a chemist with G. D. Searle & Company, who produced aspartame in 1965 while working on a new antiulcer drug.[5] Aspartame has 4 Cal/g, which is characteristic of proteins; however, because it is 180 times sweeter than sucrose, the caloric intake is insignificant.

Aspartame was originally approved for use in July 1974 by the FDA as a nutritive sweetener. During the review period following the initial approval, objections were filed. In December 1975, the FDA retracted its aspartame approval pending a more detailed inspection of the manufacturer's research and public hearings. In July 1981, aspartame was reapproved for use as an artificial sweetener. In 8 years its per capita use increased to 14 lb (6 kg) (sugar-sweetness equivalent weight). More people have voluntarily consumed considerable quantities of aspartame within a few years of its introduction than any other new chemical entity in history.[51] Originally, aspartame was about 30 times more expensive than saccharin; however, in 1992, the NutraSweet patent expired and the cost of the sweetener decreased substantially. In Canada the expiration of the patent precipitated a 50% drop in cost for the product. It is approved as a free-flowing sugar substitute for table use, and for use by manufacturers in over 100 products, such as cold cereals, drink mixes, instant coffee, instant tea, soft drinks, gelatins, puddings, pie fillings, toppings, dairy products, multivitamin food supplements, and other products where the "standards of identity do not preclude such use."[52] The commissioner of the FDA concluded his statement on aspartame before the Committee on Labor and Human Resources of the United States Senate by stating, "we do not have any medical or scientific evidence that undermines our confidence in the safety of aspartame."[53] Aspartame is a flavor enhancer, especially for sweetening acid flavors. It is also a flavor extender, lengthening the period of flavor for chewing gum for five to seven times as long as gums sweetened with sugar. Aspartame appears to be noncariogenic. The ADA has issued a statement supporting the approval of aspartame as a sweetener.[54] People with phenylketonuria (PKU) should avoid the intake of aspartame because of its phenylalanine content. Products sweetened with

aspartame must be labeled with the statement "Phenylketonurics: Contains Phenylalanine."

Acesulfame K

Acesulfame K is a non-caloric sweetener 200 times sweeter than sucrose, with a pleasant taste. Its sweetness is quickly perceptible and diminishes gradually without any unpleasant aftertaste. It is a derivative of acetoacetic acid. It is marketed under the tradename Sunette by the Hoechst Celanese Corporation and is classified as a noncariogenic sweetener.

This sweetener was discovered in 1967; however, it was not approved for use by the FDA in the United States until the summer of 1988. Acesulfame has been tested in more than 90 studies and was in widespread use in 60 countries before it was approved for use in the United States. In approving the sweetener, the FDA stated that the studies it had reviewed did "not show any toxic effects that could be attributed to the sweetener."[55] It is approved for use in such items as toothpastes, mouthwashes, pharmaceuticals, dry beverage mixes, instant coffee and tea, chewing gum, gelatins, puddings, and as a tabletop sweetener. It has a synergistic action with other low-calorie sweeteners, as do most of the intense sweeteners. This means the combination of ingredients is sweeter than the sum of the individual ingredients in sweetness. It is excreted quickly and totally, unmetabolized by both animals and humans.

Sucralose

Sucralose is a noncaloric sweetener 600 times sweeter than sucrose that is derived from sucrose. It also exhibits synergistic effects. It is not broken down nor absorbed in the human body and therefore provides no calories. Sucralose does not promote tooth decay. More than 100 studies, including human research, support the safety of sucralose. In 1991, Canada was the first country to approve its use in foods. It was approved for use in the United States in 1998.[56]

Cyclamate

Cyclamate has a pleasant, sweet taste and a relative sweetness approximately 30 times greater than sucrose. It was originally included on the GRAS list. In 1960, the FDA requirements for studies were expanded to include testing for teratology and carcinogenicity. In early October 1969, there were indications of some cases of rodent bladder cancer. The FDA ruled in 1970 that cyclamate would no longer be allowed even if it were classified as a drug.

Promising New Noncaloric Sweeteners

Many sweeteners have been submitted to the FDA for approval in the United States. Two of the more promising ones are Alitame and Sweetener 2000.

Alitame is 2000 times sweeter than sucrose. It is composed of two amino acids, L-aspartic acid and D-alanine. It is metabolized in the body; however, because of its intense sweetness, the caloric contribution to the diet is insignificant. It has a synergistic effect with other sweeteners. Alitame has a clean taste and is stable both at

high temperatures and broad pH ranges.

Sweetener 2000 is 10,000 times sweeter than sucrose. Originally discovered and patented by researchers at Claude Bernard University in Lyon, France, Sweetener 2000 is exclusively licensed by the NutraSweet Company. It tastes similar to sugar and promises excellent stability in all possible applications. It could literally change the way the world thinks about sweeteners.[57]

Other sweeteners are being used in other parts of the world (Table 14-6).[58,59] Over 150 plants have been identified as possessing a sweet taste.[60]

Figure 14-3 Sweeteners are making significant inroads into the sucrose market.

Current Legislation Regarding Sweetener Use

The specific use of a sweetener must be stated before it can be approved for commercial use. Will it be used as a flavoring, or will it be used as an anticaries agent? Such differences in intended use can greatly affect the cost of getting the product on the market. If it is to be used as a sweetener, then only safety, teratology, mutagenicity, and carcinogenicity are subjects of investigation. If anticariogenicity is claimed, such as is possible in the use of xylitol, a great amount of additional money must be spent in animal and human caries incidence studies before such claims can be advertised. Estimates on the time and expense of marketing an entirely new sweetener range up to 10 years and as high as $20 million, respectively. If the sweetener is classified as a new drug, it may require a dosage statement and package insert carrying warnings of complications, contraindications, and incompatibility with other drugs.[61]

On the other hand, public safety is paramount. Many of the original food additives were chosen from organic and inorganic compounds that were intended for fabric and paper coloration, with safety being secondary to product appeal.

Question 3

Which of the following statements, if any, are correct?

A. The two amino acids in aspartame are phenylalanine and aspartic acid.

B. Sweetener 2000 has equivalent sweetness to saccharin but no reported aftertaste.

C. Sunette, the sweetener, is known as acesulfame K.

D. Sweetness is related to cariogenicity.

Summary

There is little doubt that the consumption of sugar is associated with the caries process, but sugar alone is not the sole determinant of whether food is cariogenic.[62] Sweetness is such a cultural characteristic, however, that behavior modification to exclude it from the diet is considered an impossibility. Also, the nonsweetening

benefits of sucrose in industry would probably guarantee its continued use. In many industrial applications in the preparation and processing of food, other caloric and noncaloric sweeteners are preferable to sucrose. New sweeteners have been introduced recently that are less cariogenic and many hundred or thousand times sweeter than sucrose. Many of them are nonacidogenic and noncaloric. From a dental standpoint these new sweeteners offer the potential for a considerable decrease in caries incidence. At the present time no one sweetener dominates another from the clinical perspective of caries prevention.[43]

Answers and Explanations

1. A and B—correct

C—incorrect. A food additive is considered suspect, whereas the food ingredient has a long-term record of use and apparent safety.

D—incorrect. Without bacteria, no amount of sugar is going to produce caries in the gnotobiotic rats.

E—incorrect. People with HFI cannot consume sucrose without adverse systemic problems and hence experience few, if any, caries.

2. A, C, D, and E—correct

B—incorrect. It requires more sweetener to identify the product than to identify the sweet taste.

3. A and C—correct

B—incorrect. Sweetener 2000 is 10,000 times sweeter than sucrose, whereas saccharin is 300 times the sweetness of sugar.

D—incorrect. Cariogenicity is related to sugar, not sweetness.

Self-evaluation Questions

1. Two synthetic caloric sweeteners are _____ and _____; two synthetic noncaloric sweeteners are _____ and _____.

2. Peanut brittle made with saccharin would be a very unusual product, mainly because the sweetener lacks the _____ (characteristic) that sucrose imparts to a product. Three other attributes of sucrose that are desirable from a commercial viewpoint are _____, _____, and _____.

3. Four properties of sucrose that make it undesirable for the preparation of some consumer products are _____, _____, _____, and _____.

4. The acronym GRAS refers to _____.

5. The Vipeholm study demonstrated that two key factors relating to cariogenicity of foods were (1) frequency of intake and (2) _____; the lesson learned at Hopewood House was _____.

6. The lowest concentration at which a substance is identified to be sweet is known as the _____ concentration; the tasting of higher concentrations to identify specific sugars is known as _____ testing.

7. The sugar alcohols are more correctly referred to as _____ (name). Three of these compounds are _____, _____, and _____.

8. The noncariogenicity of xylitol is because it is _____.

9. A sweetener that is much sweeter than sucrose is referred to as an _____ sweetener.

10. Three of the most popular sweeteners are _____, _____, and _____.

11. One sweetener that is on the market today because of congressional action is _____.

12. A new product to be accepted must include data relating to carcinogenicity, _____, and _____.

13. Aspartic acid and phenylalanine are the two molecules that make up _____ (name of sweetener).

14. The chemical name for Sunette is _____.

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Chapter 15. Nutrition, Diet, and Oral Conditions - *Carole A. Palmer Linda D. Boyd*

Objectives

At the end of this chapter it will be possible to

1. Explain the underlying rationale for the Reference Daily Intakes, Food Guide Pyramid, and food labels.

2. Discuss the potential oral effects of severe malnutrition during organogenesis.

3. Discuss why foods with equal amounts of sugar are not necessarily equally cariogenic.

4. Describe how dietary patterns and food composition affects cariogenic potential.

5. Discuss the effects of food on buffering capacity.

6. Discuss the role of nutrition in periodontal disease.

7. Explain why elderly patients are at higher nutritional risk than other age groups.

8. Discuss the relevant nutritional considerations for patients who have diabetes, immunocompromising conditions, or head and neck surgery.

Introduction

Oral health, diet, and nutritional status are closely linked (Figure 15-1). Nutrition is an essential for the growth, development, and maintenance of oral structures and tissues. During periods of rapid cellular growth, nutrient deficiencies can have an *irreversible* effect on the developing oral tissues. Prior to tooth eruption, nutritional status can influence tooth enamel maturation and chemical composition as well as tooth morphology and size.[1] Early malnutrition increases a child's susceptibility to dental caries in the deciduous teeth.[2] Throughout life, nutritional deficiencies or toxicities can affect *host resistance*, healing, oral function, and oral-tissue integrity. For example, immune response to local irritants and healing of periodontal tissues may be impaired when nutritional status is compromised. Because the oral epithelium has more rapid cell turnover than most other tissues in the body, clinical signs of malnutrition are often manifest first in the oral cavity.

After tooth eruption, the effects of diet on the dentition are topical rather than systemic. Dietary factors and eating patterns can initiate exacerbate or minimize dental decay. Fermentable carbohydrates are *essential* for the implantation, colonization, and metabolism of bacteria in dental plaque. Factors such as eating frequency and retentiveness of carbohydrates influence the progression of carious lesions, while foods containing calcium and phosphorus, such as cheese, enhance remineralization. Frequent intake of *acidic foods* or beverages can cause enamel erosion. Conversely, impaired dental function may lead to poor nutritional health. Older adults with loose or missing teeth, or ill-fitting dentures often reduce their intake of foods that require chewing, such as fresh fruits, vegetables, meats, and breads.[3] When the variety of foods in a diet is reduced, there is greater risk of nutrient inadequacies. The patient who undergoes oral or periodontal surgery may require dietary guidance to prevent deleterious changes in the diet. Patients with diabetes mellitus, oral cancer, or depressed immune function may suffer from oral conditions that compromise nutritional status. The dental clinician needs to *understand* how diet and nutrition can affect oral health, and how oral conditions can affect food choices and ultimately nutritional status. This chapter provides an overview of the relationships between diet, nutrition and dental practice, and offers appropriate suggestions for patient guidance.

Figure 15-1 Relationships between Nutrition and Health.

Importance of Diet Assessment and Counseling in Dentistry

The modern dental practitioner is not only concerned with educating patients for the prevention of caries and periodontal disease, but also plays an important role in screening patients for other health risks. Just as a medical history and blood pressure evaluation are used to screen for underlying medical conditions, a dietary assessment and screening can help pinpoint potential nutritional problems that may affect or be affected by dental care. Because of the large number of patients seen regularly in dental practice, the *dental team* is in an excellent position to recognize areas of *nutritional risk.* The role of the dental team should be to *screen patients* for nutritional risk, provide *dietary guidance* related to oral health, and *refer patients* to nutrition professionals for treatment of other nutrition-related systemic conditions.[3]

Question 1

What are appropriate nutrition interventions for dental clinicians?

A. Assess patients' nutritional status using laboratory and other biochemical assessment tools.

B. Screen patients for nutritional risk.

C. Recognize dietary problems in denture patients.

D. Provide diet guidance related to oral health.

The Basis for a Healthy Diet

Dietary Reference Intakes

Daily food intake must be sufficient to meet metabolic requirements for energy and provide the essential nutrients that the body cannot synthesize in sufficient quantities to meet physiologic needs. Since the 1940s, the *Food and Nutrition Board* (FNB) of the National Academy of Sciences has published the *Recommended Dietary Allowances* (RDA), which were recommendations for daily nutrient intake that would support growth and maintenance of body tissues, and prevent deficiency diseases. (4-Food and Nutrition Board). Beginning in 1997, the Food and Nutrition Board began to make major changes to the format and purpose of the nutrition recommendations. *The Dietary Reference Intakes (DRI) expands and replaces the RDA*[5] by addressing the prevention of *chronic degenerative diseases* and the risk of *excess intake of nutrients.*[5]

The DRI are quantitative estimates of nutrient values to be used for planning and assessing diets for healthy people.[5] These reference values vary by *gender and life stage group.* DRI consist not only of RDA but also three other types of reference values shown in Table 15-1.

Evaluation of the true nutritional status of an individual requires a *combination* of clinical, biochemical, and anthropometric data.[5] So if an individual reports an intake of a nutrient below the RDA, more information would be necessary to determine if an actual deficiency exists. Conversely, nutrient intakes that meet the RDA over time have a low probability of being inadequate.

Dietary Guidelines for Americans

The *Dietary Guidelines for Americans* were first published in 1980, and are revised every *5* years.[6] The guidelines are designed to complement the DRIs by making recommendations for food choices to promote health. The 2000 Dietary Guidelines for Americans contain 10 recommendations, grouped into *three areas called the ABC* of good health. They are shown in Table 15-2.[6] These *newest* guidelines place more emphasis on *physical activity* and *healthy weight* compared to previous editions. The focus on preventing obesity is caused by the increased risk it presents for many chronic and degenerative diseases, such as heart disease, stroke, diabetes, arthritis, high blood pressure, and some kinds of cancer. The recommendations emphasize balance, moderation, and variety in food choices, and promote increased use of whole grains, fruits and vegetables, and decreased use of saturated fat, cholesterol, and salt. In addition, for the first time, the guidelines address food safety in an effort to combat food-borne illness, an important public health concern.[6]

The 2000 Dietary Guidelines for Americans define a healthy weight according to the *Body Mass Index (BMI).* The BMI is a medical standard for defining obesity that not only is highly correlated with independent measures of body fat, but is also used to determine if a person is at increased health risk due to excess weight[7] (Table 15-3). A healthy BMI of 19 to 25 is associated with the lowest statistical health risk [8-Meisler, 1996]. Persons with BMI above 25 are considered obese, and the recommendation is to lose 1 to 2 BMI units (10 to 15 pounds) to reduce their risk for chronic disease.[7]

Food Guide Pyramid

To help people select nutrient-rich foods and to follow the Dietary Guidelines, the *Food Guide Pyramid* was developed by the U.S. Department of Agriculture.[9] The Food Guide Pyramid displays foods in five categories based on their nutrient composition (Figure 15-2). Whole grains, such as rice, pasta, cereals, and breads, found at the broad base of the Pyramid should form the foundation of a healthful diet. They are good sources of carbohydrate (including fiber) and minerals. Fruits and vegetables form the next level of the Pyramid. The meat group contains good sources of protein, vitamins, and minerals. Meat alternates, legumes, eggs, nuts, and tofu, are included in the meat group. The dairy group is comprised primarily of good calcium sources. The small triangle at the top of the Pyramid is for the fats, oils, and sweets that provide primarily added calories and, thus, should be eaten in small amounts. No single food group is more important than another; each group provides some, but not all, of the essential nutrients.

Standardized serving sizes and the recommended number of servings for various age groups are specified. However, the caloric content of foods varies widely within a food group. The desirable number of servings from each food group depends not only upon age and sex, but also the calorie goal. For example, if 1,600 calories were the daily energy goal, an individual would choose the minimum number of servings of low-fat food choices from each group. If additional calories are needed, increased servings should come from the grain, fruit, and vegetable groups, rather than the top of the pyramid.

Food Labels

The Nutrition Facts panel found on most processed food packages helps the consumer select foods that meet the Dietary Guidelines (Figure 15-3). The *National Labeling and Education Act of 1990* requires that comprehensive nutrition information *must* appear on the labels of most processed foods and processed meats and poultry products. In *addition,* nutrition information at point of purchase is *voluntary* for fresh fruits, vegetables, and raw fish. In accord with the mandatory food labeling regulations published by the Food and Drug Administration in 1994,[10] the nutrition panel on processed foods must include the following:

- *A standardized portion size* (designed to make nutritional comparisons of similar products easier, and reflects the serving sizes that people actually eat).
- The number of *servings per container.*
- The amounts of *total calories* and *calories from fat* per serving.
- The *number of grams* per serving of total fat, saturated fat, cholesterol, sodium, total carbohydrates, dietary fiber, sugars, and protein.

In addition, the nutritional contribution of *one* serving of the product must be stated as a *percentage of the Daily Values.* The Daily Values are based on the RDA for protein, vitamins, and minerals and on standards designed especially for food labels for nutrients not covered in the RDA such as fat, cholesterol, total carbohydrates, dietary fiber, and sodium. The calculations to determine the percents of Daily Values are based on a *2,000-calorie diet.* Depending on a person's age, gender, and activity level, a person may need more or less than 100% of a Daily Value. The Daily Value also helps consumers see how a food fits into an overall daily diet.

Other information, such as the amounts of polyunsaturated or monounsaturated fats or other vitamins and minerals, is optional. In addition, descriptors such as "free," "low," "high," "light," "lean," or "reduced," may be used on the label as long as a standard portion *meets defined criteria.* For example, to be labeled "low-calorie" a serving must have no more than 40 calories. To be labeled "low-fat," no more than 3 grams of fat per serving is allowed.

Health claims for the potential benefit of a nutrient or food in relation to a disease or health condition will be allowed on labels if they are supported by scientific evidence and are approved by the Food and Drug Administration (FDA). The 12 health claims currently allowed to be placed on food labels are shown in Table 15-4.[11]

Figure 15-2 The Food Guide Pyramid: A Guide to Daily Food Choices is an outline of what to eat each day. Not a rigid prescription but a general guide that lets each person choose a healthful diet, the Pyramid calls for eating a variety of foods to get the needed nutrients while consuming the right amount of calories to maintain a healthy weight. (Courtesy U.S. Department of Agriculture, Human Nutrition Information Service.)

Figure 15-3 Food Label.

Question 2

The Daily Reference Intakes (DRI) are set at:

A. the minimum amount of a nutrient needed to prevent deficiency.

B. the maximum amount that will not cause toxicity.

C. the average estimated requirement for healthy people.

D. the average requirement plus a margin of safety.

Nutrition in the Development and Integrity of Oral Tissues and Structures

Nutrition plays an important role in the initial growth and development or oral tissues and in their continuous integrity through the lifespan. Optimal nutrition during periods of hard and soft tissue development allow these tissues to reach their optimal potential for growth and resistance to disease. Malnutrition (either over or under-nutrition) during critical periods of *organogenesis* can have *irreversible* effects on developing tissues. Examples of this effect can be seen in the *tetracycline staining* of teeth, in dental *fluorosis,* and in the fever-induced *enamel hypoplasia* seen in the primary teeth.[12] In the dentition, malnutrition is less well documented in humans than in animals, but it appears that during the "critical periods," malnutrition can result in dentition with increased caries susceptibility.[13] Malnutrition *after* initial organ and tissue development is *usually reversible,* but can still compromise tissue regeneration and healing and increase susceptibility to oral diseases. Nutrients for which deficiencies or excesses have been directly associated with oral conditions are protein; energy; vitamins C, A, D; iodine; and fluoride.

Protein/Calorie Malnutrition

Protein is the most abundant organic compound in the body and is required for the synthesis of virtually all body tissues and structures. Proteins account for the structure of DNA, the tensile strength of collagen, and the viscosity of saliva. Thus, aberrations in protein nutriture can have far reaching oral and systemic effects.

The normal turnover of epithelial tissue in the oral cavity requires a continual supply of nutrients. For example, *every 3 to 6 days, the basal epithelium of the gingiva undergoes renewal.*[14] Thus, any severe deficiency of protein/calorie intake will result in a decrease in mitotic activity in the crevicular epithelium, as well as elsewhere throughout the body.[15] In a comparison of periodontal involvement in patients with severe malnutrition (kwashiorkor) with that of healthy controls in South India,[16] fewer caries and more periodontal disease was found among the undernourished group. Since the oral hygiene indices of both groups were similar, it was assumed that the difference was due to nutritional factors. (It should be noted that any malnutrition of the severity of kwashiorkor represents a multi-nutrient deficiency). Impaired protein synthesis has been found if protein malnutrition occurs during the developmental stage in animals.[17] In animal models, short-term fasting (4 days) resulted in a 40% reduction in collagen production.[18] In the same study, a 10% decrease in collagen synthesis was noted with a reduced dietary intake meeting 20% of requirements.[18] These findings suggest that even short-term states of undernutrition may impact collagen synthesis.

In *chronically malnourished* children, several studies have shown delays in tooth eruption patterns, and increased tooth enamel solubility, leading to increased caries susceptibility.[19-25]

The *linear hypoplasia* reported in the enamel of primary teeth of children in underprivileged populations is thought to contribute to their high prevalence of dental caries. This type of hypoplasia appears to be related to the severity of the malnutrition.[26]

With the exception of the *cleansing* and *diluting* effects of saliva, oral defense mechanisms depend on an adequate supply of proteins. The *glycoproteins* that result in aggregation of bacteria arise from the salivary glands. *Lysozyme, salivary peroxidase,* and *lactoferrin* are also *glycoproteins. Secretory IgA* (sIgA) arises mainly from the labial and buccal glands and is an immunoglobulin. The cell types involved in cellular immunity (polymorphonuclear lymphocytes and macrophages and the enzymes used in phagocytosis) also require protein for their production.[27]

Probably one of the *most deleterious* effects of protein/calorie deficiency is the depletion of the *cellular and immunocellular defenses* of both the oral and the connective sides of the barrier epithelial cells lining the gingival crevice. In general, the severity of the impaired immunologic response parallels the severity of the protein or calorie deficiency.[28]

Minerals

Calcium, in association with vitamin D and phosphorus is essential for proper

development and maintenance of mineralized tissues (teeth and alveolar bone). A deficiency of these nutrients during critical phases of tooth development in children results in hypo-mineralization of developing teeth, and possible delayed eruption patterns.[29] Enamel hypoplasia may be seen in prematurely born very low birth-weight (VLBW) infants due to the higher needs for calcium and phosphorus in these infants.[30] In addition, VLBW infants have immature kidneys and may not metabolize adequate levels of vitamin D.[30]

Iron is of interest since iron deficiency is the *most common deficiency* in the United States. Iron deficiency anemia is manifest in the oral cavity by pallor of oral tissues, especially the tongue. The tongue may appear shiny, with blunted filiform papillae. The effects of iron deficiency on mineralized tissues are less clear. In rats, even a marginal deficiency of iron in the rat diet predisposes the rats to caries. Conversely, supplementing a caries-promoting diet with iron produced a major reduction in caries with the greatest effect shown in the neonatal period.[31] In addition, iron serves as a cofactor with ascorbic acid in collagen synthesis, as is copper.[32]

Zinc regulates *function in inflammation* by inhibiting the release of lysosomal enzymes and histamines. A zinc deficiency can inhibit collagen formation and reduce cell-mediated immunity.[33] The effect of zinc in modifying periodontal defense mechanisms has been shown in rabbits,[35] but has yet to be clearly delineated in humans.[36, 37]

Vitamins

Vitamin A is essential for the development and continued integrity of all body organs and tissues, including the *epithelial mucosa* of the oral cavity. In vitamin-A deficiency, cell differentiation is impaired: *Mucus-secreting cells* are replaced with keratin-producing cells. The result is defective tissue formation, and impaired healing. Vitamin-A deficiency also results in impairment of *both specific and nonspecific immunoprotective mechanisms.* Deficiency can affect tissue response to bacterial infection, mucosal immunity, parasitic and viral infection, natural killer-cell activity, and phagocytosis.[38] Vitamin-A toxicity can show similar effects, with impaired healing response being the most direct affect on the oral cavity.[39] Effects include proliferation of oral epithelium, reduction of the keratin layer, thickening of the basal membrane, and increase in the granular layer. A patient who took 200,000 IU of vitamin A daily for over 6 months presented with painful gingival lesions, along with nausea, vomiting, xerostomia, and headaches. Clinical examination revealed gingival erosions, ulcerations, bleeding, swelling, loss of keratinization, color changes, and desquamation of the lips.[39] All pathologic manifestations disappeared within 2 months of the elimination of the vitamin A supplements when oral hygiene habits were unchanged.

Vitamin C (ascorbic acid) is essential to oral health. Synthesis of *hydroxyproline*, an essential component of *collagen*, requires ascorbic acid. Defects in collagen synthesis are responsible for the many manifestations of vitamin-C deficiency (*scurvy*). In the oral cavity these include spontaneous bleeding, infusions of blood into interdental papillae, loosening and exfoliation of teeth, detachment of oral epithelial tissue, and impaired wound healing.

The effects of vitamin-C deficiency are best studied in animal models, where all factors can be controlled. Acute scurvy can be produced by placing monkeys on a vitamin-C deficient diet for 12 weeks. The hydroxyproline content of the gingiva started to decline in the first four weeks and occurred at a faster rate than in skin.[40] By the end of the 8th week, the synthesis of hydroxyproline was totally impaired.[40] The results are extensive gingival pocket formation and tooth mobility due to degradation of the collagen making up periodontal ligament fibers.[25]

Although frank scurvy is rare, even marginal deficiencies may result in alterations in collagen synthesis. Thus deficient or marginal ascorbic acid intakes may be a conditioning factor in the development of gingivitis and one of the early manifestations of vitamin-C deficiency.[41] The most recent epidemiologic data from NHANES III (National Health and Nutrition Examination Survey) suggests that the odds of having periodontal disease are 1.2 times greater in those with low dietary vitamin-C intakes.[42] In the same study, *smokers and former smokers* with low vitamin-C intake are at 1.6 times greater risk of having periodontal disease.[42] Research findings suggest that people with marginal vitamin-C deficiency supplemented with ascorbic acid have a statistically significant increase in hydroxyproline in periodontal tissues.[43]

Ascorbic acid is essential to immune related functions, such as resistance to oral infection, via its role in leukocyte formation and subsequent *phagocytosis.*

Conversely, chronic vitamin C excess may precipitate a scurvy-like condition (rebound scurvy) upon cessation of the vitamin. Because the impact of deficient levels of vitamin C is first observed in gingival tissues, dentists and dental hygienists in clinical practice may be the first to diagnose the phenomenon.[44] The B-complex vitamins primarily function as *co-enzymes in energy metabolism.* B-complex vitamins are found widely in foods, and usually together. With the exception of B_{12} in the elderly and folic acid in pregnant women, deficiencies of single B vitamins are uncommon. Oral signs and symptoms of B-complex vitamin deficiencies include cracks in the corners of the mouth (cheilosis), inflammation, burning, redness, pain and swelling of the tongue.[45]

Question 3

Which is *true* about vitamins and oral health?

A. Vitamin-C-deficient wounds heal as well as non-vitamin-C-deficient wounds

B. Vitamin A-toxicity does not have oral effects

C. The oral manifestations of vitamin-C deficiency are related to defects in collagen formation

D. Effects of deficiency and toxicity are best studied in humans

Diet and Nutrition in Oral Conditions: Background and Counseling Strategies

Who Needs Diet Guidance Caries Prevention

Dietary *education and guidance* are important for the prevention and control of dental caries. Patients should be carefully assessed to determine the level of prevention and nutrition guidance needed following these Institute of Medicine prevention guidelines:[46]

Selective Prevention: This strategy targets subset of the total population that are deemed to be at risk for caries for a variety of reasons. Examples include:

Adolescents at risk of caries because of high intake of soft drinks and snack foods.

Caries-prevention counseling for patients with xerostomia or cariogenic diet patterns.

Proactive diet suggestions for new denture wearers or those having jaw fixation.

Diet advice prior to radiation or chemotherapy.

Using current diet patterns as a basis for discussion, patients should be taught the role of diet in caries, what are cariogenic and noncariogenic eating patterns, and how to adapt current diet to lower cariogenic risk.

Indicated Prevention: This strategy targets individuals showing early danger signs of caries, such as extensive cervical demineralization. These individuals need the immediate aforementioned interventions as well as more detailed guidance on how to reduce cariogenicity of their current diet. This will involve determining the factors influencing current habits, and working with the patient to develop appropriate and acceptable strategies for improvement. Patients need to be followed up on a regular basis to promote long-term change.

Question 4

The diet assessment process in dentistry is designed to:

A. diagnose nutrient deficiencies

B. help screen patients for oral-health risk factors

C. serve as a teaching tool

D. determine patients' daily caloric intake

E. provide a therapeutic diet prescription for patients

F. be part of total preventive assessment

Dental Caries: Role of Carbohydrates in Caries Development

Dental caries is a common plaque-dependent bacterial infection that is strongly affected by diet. Development of clinical caries is contingent upon the interaction of three local factors in the mouth: *a susceptible tooth, cariogenic bacteria, and*

fermentable carbohydrate (Figure 15-4). Absence of one of these factors dramatically reduces caries risk. Mutans streptococci are the predominant oral bacteria that initiate the caries process. Newly erupted teeth with a thin enamel layer are very caries susceptible. Tooth morphology, especially the presence of deep pits and fissures, influences the likelihood that mutans streptococci will attach to and colonize the tooth's surface. Plaque bacteria ferment starches and sugars, producing organic acids. These acids demineralize dental enamel.[47]

Other dietary factors *counteract* the damaging effects of carbohydrates. The presence of protective minerals and ions such as *fluoride, calcium, and phosphorus* in *plaque and saliva,* promote remineralization of incipient lesions. In addition to transporting minerals, saliva contains *buffering agents, bicarbonate and phosphates,* that neutralize organic acids. Thus, the amount and composition of saliva affect the caries process. Other host factors that influence caries risk include: genetic predisposition, immune status, malnutrition during tooth formation, education level, and income status.

In the most recent national health and examination survey (NHANES III, Phase I) 94% of adults showed evidence of coronal caries and 22.5% of adults had root caries.[48] In the same survey, 25% of the children and teens aged 5 to 17 had 80% of the dental caries detected in the permanent teeth.[49] For these caries-prone children and adults, *nutrition counseling* about the damaging effects of fermentable carbohydrates on teeth is essential.

Through epidemiological and clinical studies discussed in Chapter 14, the causal relationship between sugar consumption and dental caries has been established. Animal studies suggest that an increase in the concentration of sucrose in the diet reduces dental plaque formation and increases the incidence of dental caries.[50,51] People with very low sugar intakes have low-caries scores. People in nations that have high sugar intakes have high rates of caries.[52] It is unclear if this is primarily the topical effect of sugar consumption or systemic effects on dentin formation. However, the amount of sugar consumed is not the sole dietary variable associated with caries development. Sucrose plays a more dominant role than other sugars in the development of smooth surface caries. One of sucrose's metabolic by-products, an extracellular polysaccharide called *glucan,* enables the mutans streptococci to adhere to the smooth enamel surfaces.[53] However, the amount of sucrose necessary for the implantation of mutans streptococci is very low.

Although sugar intake is high among most persons in industrialized countries, it is more difficult to demonstrate a *correlation* between caries prevalence and the amount of sugar consumed than in developing countries where sugar intake is lower. Three recent clinical trials of English, United States, and Canadian schoolchildren examined the relationship between sugar intake and dental caries. In England, 405 children with a mean age of 11.6 years were followed for 2 years. Total sugar intake (118 grams per day or 21% of total calorie intake) had the highest significant correlation with caries rates.[54] Intake of sugary foods before bedtime was highly correlated with caries incidence. In the United States, 499 children aged 11 to 15 years, living in nonfluoridated rural Michigan communities, were followed for 3 years. The average increase in decayed, missing, and filled surfaces (DMFS) over the 3 years was 3.1 in girls and 2.7 for boys. The daily average sugar intake was 142 grams, this represented

26.5% of their total energy intake. Children who obtained a higher percent of their total calories from sugars had more proximal surface caries. The average number of eating occasions and the number of sugary between-meal snacks consumed were not related to caries increment.[55]

Fifty percent of 232 11-year-old children in a Canadian study had *inadequate diets.* Children with superior diets tended to develop fewer caries; however, the association was not statistically significant.[56] Differences in eating patterns and intake of caries-promoting foods among the children in these studies may have been too small to result in significant differences in caries experience. Other factors contributing to the caries decline in western countries are: fluoride intake from water, the use of fluoridated dentifrices, improved plaque control, the use of dental sealants, and more frequent visits to the dentist.[57]

The use of *sugar alcohols and alternative sweeteners* in foods also has had a role in *reducing* caries. Perhaps one of the most promising sugar substitutes to be studied is *xylitol,* a sugar alcohol that has been demonstrated to be non-cariogenic as well as promoting remineralization.[58] Xylitol's ability to inhibit metabolic acid production by mutans streptococci results in minimal depression of plaque pH. Maintenance of the *plaque pH close to the saliva pH* also fosters remineralization of teeth.[59] In addition, the substitution of xylitol for fermentable sugars in the diet results in a less cariogenic bacterial flora. The importance of other non-fermentable sweeteners in caries control is detailed in Chapter 14.

Simple sugars are not the only carbohydrate that influences the development of a carious lesion. Highly refined *cooked starch-sugar combinations* such as doughnuts, cookies, potato chips, and some ready-to-eat breakfast cereals *produce a prolonged acidogenic response* when retained in interproximal spaces.[60] When starches are cooked, they are partially degraded. This allows the salivary alpha-amylase to convert starch particles retained on the tongue, oral mucosa, and teeth to *maltose.* Making maltose available to plaque bacteria extends the length of time the plaque pH will remain low and permit enamel demineralization to occur. Thus, retentive high starch foods may be more acidogenic than high-sugar-low-starch foods that are rapidly eliminated from the mouth.[61]

Effects of Eating Patterns and Physical Form of Foods

Other dietary factors that may hinder or enhance caries development include: the frequency of eating, the *physical form* of the carbohydrate (liquid vs. solid), *retentiveness* of a food on the tooth surface, the *sequence* in which foods are consumed (e.g., cheese eaten before a sweet food limits the pH drop), and the presence of minerals in a food.

Frequent between-meal snacking on sugar or processed starch-containing foods increases plaque formation and extends the length of time that bacterial acid production can occur. When total daily sugar intake was held constant, increasing the frequency of sugar intake for groups of rats resulted in increased number of *Streptococci mutans* in plaque and the amount of caries experienced.[62] The positive relationship between frequency of sugar intake and caries in humans was first demonstrated in the *Vipeholm* study.[63] Subjects who consumed candies *between meals*

developed *more* caries than those who were fed equal amounts of sugars *with* meals. Frequent snacking between meals keeps the plaque pH low and extends the time for enamel and dentin demineralization to occur.

Bacterial fermentation can continue as long as carbohydrate adheres to the enamel and exposed dentinal tooth surfaces. Even though starchy foods vary in their cariogenic potential, the highly refined starchy foods, such as soft bread and potato chips, that are retained on tooth surfaces for prolonged periods of time, result in a lowered pH which may last *up to 60 minutes.*[64,61] High-sucrose confectionery foods deliver high levels of sugar to the oral bacteria immediately after the foods are consumed, whereas high-starch foods deliver progressively increasing concentrations of sugars over a considerably longer period of time.

The *sequence* in which foods are eaten affects how much the plaque pH falls. Sugared coffee consumed at the *end* of a meal will cause the plaque pH to remain *low for a longer time* than when an unsweetened food is eaten *following* intake of sugared coffee.[65] If peanuts are eaten before or after sugar-containing foods, the plaque pH is less depressed.[66]

Some components of foods *are protective* against dental caries. Protein, fat, phosphorus, and calcium inhibit caries in rats.[67] Aged natural cheeses have been shown to be cariostatic.[68] When cheese is eaten following a sucrose rinse, the plaque pH remains higher than when no cheese follows a sucrose rinse. In addition, enamel demineralization, measured using the intraoral cariogenicity test, is reduced. The protective effect of cheeses is attributed to *their texture* that *stimulates salivary flow,* and their protein, calcium, and phosphate content that *neutralizes plaque acids.* Fluoride found in drinking water, foods, and dentifrices increases a tooth's resistance to decay and enhances remineralization of carious lesions.

Lipids seem to accelerate oral clearance of food particles. Some fatty acids, linoleic and oleic, in low concentration, inhibit growth of mutans streptococcus. Lectins, proteins found in plants, appear to interfere with microbial colonization and may affect salivary function.[69]

Figure 15-4 Factors Required for Caries Development.

Question 5

In the diet of a patient with rampant dental caries, which is *most relevant* to the problem?

A. total amount of sucrose consumed

B. total amount of sticky sweets consumed

C. nutrient quality of the meals and snacks

D. number of meals and snacks

E. what is eaten for desert in the evening

Measuring the Cariogenic Potential of Foods

Since it is unethical to conduct human experiments to measure the true cariogenic potential of foods, other indirect tests have been developed. These tests enable researchers to classify foods into at least three categories: protective, low, and high cariogenic potential. Currently the cariogenic potential or the ability to induce caries in humans may be assessed indirectly by measuring the ability of a test food to cause: caries formation in animals, acid production in dental plaque, or demineralization of enamel.[1]

Animal studies have been conducted using a *programmed feeding machine*. In one study, 20 common snack foods were presented to rats at specified intervals during the day.[70] After sulcal and smooth surface caries were scored in the animals, cariogenic potential indices (CPI's) were computed for each food (the sucrose group had a CPI value of one) (Table 15-5). A food with a CPI of 0.4 had low cariogenic potential. Those snack foods with high cariogenic potential had 1% or more hydrolyzable starch in combination with sucrose or other sugars.

Acid production in the mouth during bacterial fermentation of a food is predictive of the contribution of that food to the caries process. Measurement of plaque acidogenicity can be measured by determining the pH of a plaque sample taken from the mouth or *in situ*.[1] Foods that cause the plaque pH to fall below the critical demineralization level (*pH 5.5 to 5.0*) are considered acidogenic. Measurement of oral plaque pH requires placement of a wire-*telemetric appliance* containing a pH microelectrode in the space where a tooth is missing in the mouth. As the test food is chewed, the pH under undisturbed plaque at the site of the indwelling electrode is continually transmitted to an external receiver. The rate of the fall and rise of the pH at an interproximal site can be recorded continuously using plaque telemetry. Foods found to have low acidogenic potential using this method include: aged cheeses, some vegetables, meats, fish, and nuts.[71]

To assess the ability of a food to demineralize dental enamel, an *intraoral cariogenicity test* has been developed. Bovine or human dental enamel slabs are imbedded in a prosthesis and placed in the mouth where a tooth is missing. After ingesting a test food, changes in surface microhardness or enamel porosity are determined.[72] Since each test measures a different aspect of cariogenicity, foods will be ranked differently. It is recommended that two testing methods be used to determine food acido/cariogenicity potential.[73,74] Table 15-6 shows the acidogenic potential of foods. Table 15-7 provides diet suggestions for caries prevention.

Question 6

Tooth erosion can be caused by

A. acid from vomiting

B. sugar-containing carbonated beverages

C. gastro-esophageal reflux

D. sugar-free carbonated beverages

E. all of the above

Early Childhood Caries

One of the most severe forms of caries occurs in infants. Inappropriate feeding practices may result in progressive dental caries on the buccal and lingual surfaces of newly erupted primary maxillary anterior teeth of infants and toddlers. The overall prevalence of early childhood caries (also called *baby bottle tooth* decay or *nursing caries*) is estimated to be 5%.[75] However, a much higher prevalence has been seen among Alaskan and Oklahoma Native American children (53%) and Navajo (72%) and Cherokee (55%) Head Start children attending Head Start programs.[76, 77]

Primary risk factors for early childhood caries include putting a child to sleep at naptime or bedtime with a bottle containing a liquid *other than plain water*, allowing an infant to breast-feed at will during the night, and extended use of the nursing bottle or sippy cup beyond 1 year of age. Results of the 1991 National Health Interview Survey show that 16.7% or 3.5 million children between 6 months and 5 years of age are put to sleep with a liquid in the bottle other than plain water.[78] Inappropriate feeding practices were reported more often by parents with *less than a high school education,* low incomes, Hispanic backgrounds, and those parents whose children had not been to a dentist in the past year.

Children who develop maxillary anterior caries are at increased risk of developing posterior caries in the future.[79] To prevent early childhood caries, dentists, pediatricians, and other health care professionals should ask parents about their infant feeding practices. Those parents who report inappropriate feeding practices should receive counseling. Programs serving low-income families, such as the Special Supplemental Food Program for Women, Infant, and Children (WIC), can play a major role in providing education to parents at higher risk for using inappropriate feeding practices.

Nutrition and Periodontal Disease

Like caries, periodontal disease is an infectious disease, multifactorial in etiology, and occurs when virulence of the bacterial challenge is *greater than the host defense and repair capability.* The course of periodontal disease involves periods of progression and remission. Unlike the direct causative relationship between carbohydrates and caries, nutritional factors seem to play a much more subtle role in periodontal status. Nutritional factors *can alter host susceptibility* to periodontal disease and/or modulate its progress.[80] The nutritional factors related to *preventing infection* and *enhancing wound healing* in general applies to the prevention and management of periodontal disease as well.[81] *If* both the challenge to and the defense and repair capabilities of the periodontal tissues are in balance, nutrition could be the deciding factor in whether health or disease results. Even when the periodontium is healthy, there is continual need for nutrients to maintain the tissues. Once inflammation is established, the need for nutrients *increases.* There is a close relationship between malnutrition and infection, with infection aggravating malnutrition and malnutrition abetting infection.

Defense in the gingival crevice and connective tissue all require an adequate intake of all nutrients to ensure adequate production and function of defense and supporting cells.[82-86] With the increased needs of cellular immunity and the additional demands by the tissue cells attempting to maintain and repair damaged areas, a greater supply of all nutrients is needed. This has led to evidence showing that nutrient requirements may be higher at local sites of increased stress than in the rest of the body. Such localized challenges may result in *end-organ nutrient deficiencies.*[87, 88]

Diet Guidelines

Whenever routine scaling, prophylaxis, and oral plaque control procedures fail to reverse gingivitis and before any treatment for periodontitis is attempted, a thorough diet evaluation and patient counseling session is indicated. The patient should be informed about the importance of systemic nutrition in the defense and repair of oral tissues. Recommendations should be made to help ensure optimal nutrition to help prevent and manage periodontal disease. These include:

- Eat a *nutritionally adequate diet* following the food pyramid guidelines.
- Increase the use of saliva-stimulating fibrous foods.
- Multivitamin/mineral supplements should be in doses *no higher* than one to two times Recommended Dietary Allowance levels.
- *Avoid fad diets* which could be deficient in nutrients.
- Avoid *single* vitamin supplements.
- *Avoid potentially detrimental megadoses* of vitamins and minerals (10× RDA or higher).

Question 7

Periodontal disease is caused by dietary deficiencies. Calcium deficiency is thought to be a contributing factor in alveolar bone loss in humans.

A. both statements are true.

B. both statements are false.

C. the first statement is true: the second is false.

D. the first statement is false the second is true.

Eating Disorders

Eating disorders, especially *bulimia*, are often first diagnosed in the dental office. Patients, usually young females, present with severe erosion of the lingual tooth surfaces. The oral tissues are often red, sore, and painful. The esophagus may be inflamed, and parotid salivary glands are often swollen. Bulimia is characterized by recurrent episodes of *binge eating* (consumption of large amounts of foods at a time) followed by self-induced regurgitation (purging). The average intake of food during a binge is 3,400 calories over an hour, with some individuals ingesting as much as 50,000 calories in 24 hours.[89] Patients may also use laxatives and/or diuretics to induce malabsorption and fluid loss. The acid from stomach regurgitation irritates the

esophagus and the oropharyngeal soft tissues. The regurgitated acid in combination with xerostomia, results in rapid and extensive destruction of tooth enamel.[90]

Patients often first deny having an eating disorder. However, when confronted with the oral evidence, they often admit to the disorder. The dentist should refer the patient to an eating-disorder management program and elicit patient agreement to undergo treatment. The diagnosis of this disorder by the dentist and the realization of the dental destruction caused by the disorder, often convince patients to agree to treatment. A *multidisciplinary* approach to treatment is needed, including physicians, psychiatrists, psychologists, nutritionists, and social workers. The patient must be cautioned that for dental rehabilitation to be successful, the underlying problem (the eating disorder and its causes) must be resolved.

Question 8

Oral problems that may be seen in patients with eating disorders include:

A. swollen salivary glands

B. orange-stained teeth

C. decreased salivary flow

D. decreased oral pH

E. severe enamel demineralization

The Aging Patient

The aging patient is often faced with a variety of challenges that can undermine both oral health and nutritional status.[91] As a result, the elderly are considered particularly susceptible to malnutrition.[92] Compared to younger individuals, elders have a significantly decreased ability to respond to physiologic challenges. Sensory function decreases leading to *impaired taste and smell.*[93] Changes in the gastrointestinal system can affect the ability to digest, absorb and utilize food properly. Functional problems, such as arthritis or vision difficulties can affect the ability to prepare and eat food. Psychosocial problems such as loneliness, depression, lack of money, and poor access to food can all undermine good eating habits.

Problems in the oral cavity, such as xerostomia and loose teeth, have been considered major contributors to the poor eating habits of the elderly and may be a major contributor to malnutrition.[92,105,94,95] Several studies have shown that dentate status can affect eating ability[96] and subsequent diet quality.[97, 98, 99] Individuals with one or two complete dentures had a 20% decline in diet quality compared to those with at least partial dentition in one or both arches.[100] Another study showed that compared to those with 25 or more teeth, edentulous individuals consumed less fiber and carotene, fewer vegetables, and more cholesterol, saturated fat and calories.[101] Dentures can affect *taste and swallowing ability*, especially if they are maxillary dentures. The denture covers those taste buds found on the upper palate. And when the upper palate is covered, it becomes difficult to detect the location of food in the mouth. For this

reason, dentures are considered to be the *major cause of choking* in adults.[102]

Dry mouth (xerostomia) is common in the older population, in part because of xerostomic medications commonly taken. Xerostomia makes eating more difficult and increases the cariogenic potential of the diet.[103,104] It has also been associated with burning mouth syndrome and inadequate diet.[105]

Conversely, nutrition is an important factor in oral status.[106] In a sample population of 843 elderly people, there was a significant association between low ascorbic acid levels and the prevalence of oral mucosal lesions.[107] Low calcium intake throughout life has been shown to *contribute to osteoporosis.* In turn, osteoporosis in alveolar bone is thought to be an important contributing factor to the resorption of alveolar bone that ultimately results in tooth loss.[108] The alveolar process is composed primarily of trabecular bone, which is more labile to calcium imbalances than is cortical bone. Thus, the alveolar bone provides a potential labile source of calcium available to meet *other* tissue needs. Since the alveolar process is thought to undergo resorption *prior* to other bones; it is projected that changes detected in the alveolar process may eventually be used for early detection of osteoporosis.[109] Mandibular bone mass was correlated with total body calcium and bone mass of the radius and vertebrae in dentate and edentulous postmenopausal women with osteoporosis,[110] with the highest correlation between total body and mandibular bone mass. Thus, the mandible reflects the mineral status of the entire skeleton. Calcium intake in postmenopausal osteoporotic women was also correlated with mandibular density; supporting the hypothesis that low calcium intake may contribute to reduced bone density.[111,112] In a study of 329 healthy post-menopausal women, an inverse relationship was shown between bone mineral density and number of existing teeth, with those women who received dentures after the age of forty having the lowest bone mineral density.[113]

Older patients should be carefully screened for nutritional risk factors, and should be educated about the importance of good nutrition to general and oral health.[114, 115] If major nutritional problems are suspected the patient should be referred to a nutritionist.[116] When new dentures are provided, patients should be counseled on how to adapt their usual diet to a softer consistency for the first few days after denture insertion.

Question 9

Which is true about aging?

A. Dry mouth always occurs with aging.

B. Dentures improve taste perception.

C. Taste and smell acutely tend to decrease.

D. Calcium intake is not of concern with this group.

The Diabetic Patient

The *diabetic* dental patient is at *greater risk* for developing oral infections and periodontal disease than the nondiabetic patient.[117,118] The dental team needs to be aware of current approaches to diabetes management and carefully monitor the patient's health status prior to initiating dental treatment.

The nutrition care plan generally requires that patients have meals and snacks of specific nutrient composition *at regularly scheduled intervals,* coordinated with medications (insulin or oral agents) and exercise. Dietary management has changed from the high fat, low carbohydrate diets of past decades to the more liberal use of complex carbohydrates and the reductions in fat recommended today.[119,107] A well-balanced diabetic diet should be low in cariogenicity, since the use of cariogenic fermentable carbohydrates should be infrequent. Frequent use of hard candies or other foods taken to counteract hypoglycemia are an indication that the diabetes is not well controlled. Patients with uncontrolled diabetes should be referred to their physician for further management. *In the dental office, quickly assimilated carbohydrate sources such as juices, milk, and crackers, should be kept readily available in the event that a diabetic patient develops symptoms of hypoglycemia.*

Patients with Immunocompromising Conditions (Cancer, AIDS)

Immunocompromised patients, such as those with cancer or AIDS, often have *increased* requirements for nutrients while having major physiologic and psychosocial impediments to eating. Cancer often sets up a syndrome of weight loss and wasting in which both metabolism and nutrient losses *increase.* The cancer often causes severe anorexia, taste changes, and early satiety. The pain and discomfort of oral infections such as the herpes simplex and oral candidiasis found in AIDS and chemotherapy patients, can also impair the desire and ability to eat.[120] *Over half of all head and neck cancer patients are nutritionally compromised at initial diagnosis.*[121] Radiation therapy increases eating difficulty by causing painful oral *mucositis, dysphagia,* and severe *xerostomia.*[122]

When providing dental treatment to patients suffering from cancer or AIDS, team members need to understand the nutrition principles underlying the care, so that dental services provided can be coordinated effectively with total care. The nutrition care plan initially focuses on providing *high caloric intake in frequent small meals.* Liquid supplements may be used if optimal nutriture cannot be achieved via food alone. In more serious cases, patients may need enteral (tube) feedings or more advanced nutritional support. A high calorie diet will likely be high in sugars and total calories.[123] In these cases, the dental team should *not* caution patients to reduce the frequency of eating, since this will contradict nutritional management goals. Rather, thorough cleaning after each eating period, and use of fluoride mouth rinses and topical fluoride trays before bed should be stressed. This approach is standard protocol for immunocompromised patients as part of an aggressive preventive dental program.[124] Cancer patients should be cautioned, however, about the potential oral sequelae of an increased frequency of eating. Patients should also be cautioned to avoid the use of slowly dissolving hard candy often used to assuage the xerostomia. The most important *monitoring* tool for these patients is *weight status.* The patient should be queried at each visit about how their weight is being maintained. Involuntary weight loss of 10 pounds or more is a warning for the need for more intensive care.

Oral Surgery and Intermaxillary Fixation

The patient who has had oral surgery, whether therapeutic or as a result of trauma, needs special nutritional consideration (125-Kendall, 1982). An adequate diet *before surgery is needed to support adequate post-surgical response.* If food consumption will be impaired for a short period of time, the risk of nutritional deficiency is low. The risk of deficiency increases with length of eating impairment. The surgery itself can result in an anorexia, inability to chew, and increased metabolic requirements.[126] After surgery, a patient may need a *liquid diet for 1 or 2 days,* but should progress as soon as possible to a soft diet of high nutritional quality, until a normal diet can be resumed. In some cases, nutritionally complete liquid supplements may be appropriate and should be prescribed in consultation with the patient's dietitian and physician. Often patients prefer purees of normal foods over commercial liquid supplements.[127] Multivitamin/ mineral supplements may be appropriate as well.

Question 10

Which of the following is/are true?

A. A well-controlled diabetic diet should be low in caries risk.

B. Patients with cancer often have increased nutrient needs.

C. Patients with immune-compromising conditions should be told to reduce the frequency of eating to reduce caries risk.

D. The oral surgery patient may require a liquid diet for 1 to 2 days after surgery but should return to a normal diet as soon as it is possible.

Summary

Nutritional status and dietary habits can affect and be affected by specific oral conditions. Comprehensive patient care requires that nutritional factors be considered in the etiology, progression, and sequelae of oral problems.[128, 129]

Dental-team members should routinely screen patients for nutritional issues, provide dentally-oriented counseling, and refer patients to dietitians for further care. The nutritional implications in dental conditions are many and complex. No longer can nutrition in dentistry be summarized as "sugar is bad, and fluoride is good."

Answers and Explanations

1. b, c, and d—correct.

a—incorrect. It is not appropriate or possible for the dental team to attempt to assess actual nutritional status. This requires sophisticated laboratory testing under the supervision of a qualified medical professional.

2. d—correct.

a—incorrect. The minimum amount of a nutrient needed to prevent deficiency is not considered an appropriate standard of adequacy

b—incorrect. The maximum amount of a nutrient that will not cause toxicity is the UL or upper tolerable limit

c—incorrect. The average estimated requirement for healthy people would mean that half of the population would require more. Thus it is not used as the criteria for healthy populations

3. c—correct.

a—incorrect. Vitamin C-deficient wounds have poorer healing ability

b—incorrect. Vitamin-C deficiency affects all epithelial tissues including those in the oral cavity

d—incorrect. It is not ethical to conduct such studies in humans

4. b, c, and f—correct.

a—incorrect. The diet assessment process can be used to screen patients for possible nutrition risk, but cannot be used for true nutritional assessment

d—incorrect. Daily calorie intake cannot be determined using a diet screening tool. Patient's daily calorie intake is best assessed by a registered dietitian using an assessment tool designed for that purpose.

e—incorrect. The dental team can use screening information to refer the patient to a registered dietitian who is qualified to provide therapeutic diets. The dental team can provide nutrition information about healthy diet and diet/oral health relationships.

5. d—correct

a—incorrect. Sucrose is not the only cariogenic factor, and the amount is not as important as the distribution in the diet.

b—incorrect. The amount of sticky sweets is not as relevant as the frequency of usage of these items.

c—incorrect. The nutrient quality of the diet is only related to the caries process after tooth eruption through remineralization effects.

e—incorrect. Dessert is only one of many contributing factors to dental caries.

6. e—correct.

Tooth erosion can be caused by acid from vomiting, sugar-containing carbonated

beverages, gastroesophageal reflux, and sugar-free carbonated beverages to name but a few factors.

7. d—correct.

Periodontal disease is not *caused* by dietary deficiencies. However, calcium deficiency is thought to be a contributing factor to alveolar bone loss in humans.

8. a, c, d, e—correct.

b—incorrect. Orange-stained teeth are not necessarily caused by eating disorders. The staining can come from food, beverages, or other sources.

9. c—correct.

a—incorrect. Dry mouth is associated primarily with the use of medications and is not inevitable with aging.

b—incorrect. Dentures can impair taste perception if they cover taste buds on the upper palate.

d—incorrect. Calcium is important for all age groups. Calcium intake is associated with bone density in general and may be a factor in alveolar bone health as well.

10. a, b, and d—correct

d—incorrect. Patients with immune compromising conditions should *not* be told to reduce the frequency of eating to reduce caries risk. These patients are at high risk for nutritional deficiency and must eat high calorie foods on a frequent basis throughout the day. Oral risk should be reduced by having patients rinse the mouth and clean the teeth as best they can after each eating period and use remineralizing rinses.

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Chapter 15. Nutrition, Diet, and Oral Conditions - *Carole A. Palmer Linda D. Boyd*

Objectives

At the end of this chapter it will be possible to

1. Explain the underlying rationale for the Reference Daily Intakes, Food Guide Pyramid, and food labels.

2. Discuss the potential oral effects of severe malnutrition during organogenesis.

3. Discuss why foods with equal amounts of sugar are not necessarily equally cariogenic.

4. Describe how dietary patterns and food composition affects cariogenic potential.

5. Discuss the effects of food on buffering capacity.

6. Discuss the role of nutrition in periodontal disease.

7. Explain why elderly patients are at higher nutritional risk than other age groups.

8. Discuss the relevant nutritional considerations for patients who have diabetes, immunocompromising conditions, or head and neck surgery.

Introduction

Oral health, diet, and nutritional status are closely linked (Figure 15-1). Nutrition is an essential for the growth, development, and maintenance of oral structures and tissues. During periods of rapid cellular growth, nutrient deficiencies can have an *irreversible* effect on the developing oral tissues. Prior to tooth eruption, nutritional status can influence tooth enamel maturation and chemical composition as well as tooth morphology and size.[1] Early malnutrition increases a child's susceptibility to dental caries in the deciduous teeth.[2] Throughout life, nutritional deficiencies or toxicities can affect *host resistance*, healing, oral function, and oral-tissue integrity. For example, immune response to local irritants and healing of periodontal tissues may be impaired when nutritional status is compromised. Because the oral epithelium has more rapid cell turnover than most other tissues in the body, clinical signs of malnutrition are often manifest first in the oral cavity.

After tooth eruption, the effects of diet on the dentition are topical rather than systemic. Dietary factors and eating patterns can initiate exacerbate or minimize dental decay. Fermentable carbohydrates are *essential* for the implantation, colonization, and metabolism of bacteria in dental plaque. Factors such as eating frequency and retentiveness of carbohydrates influence the progression of carious lesions, while foods containing calcium and phosphorus, such as cheese, enhance remineralization. Frequent intake of *acidic foods* or beverages can cause enamel erosion. Conversely, impaired dental function may lead to poor nutritional health. Older adults with loose or missing teeth, or ill-fitting dentures often reduce their

intake of foods that require chewing, such as fresh fruits, vegetables, meats, and breads.[3] When the variety of foods in a diet is reduced, there is greater risk of nutrient inadequacies. The patient who undergoes oral or periodontal surgery may require dietary guidance to prevent deleterious changes in the diet. Patients with diabetes mellitus, oral cancer, or depressed immune function may suffer from oral conditions that compromise nutritional status. The dental clinician needs to *understand* how diet and nutrition can affect oral health, and how oral conditions can affect food choices and ultimately nutritional status. This chapter provides an overview of the relationships between diet, nutrition and dental practice, and offers appropriate suggestions for patient guidance.

Figure 15-1 Relationships between Nutrition and Health.

Importance of Diet Assessment and Counseling in Dentistry

The modern dental practitioner is not only concerned with educating patients for the prevention of caries and periodontal disease, but also plays an important role in screening patients for other health risks. Just as a medical history and blood pressure evaluation are used to screen for underlying medical conditions, a dietary assessment and screening can help pinpoint potential nutritional problems that may affect or be affected by dental care. Because of the large number of patients seen regularly in dental practice, the *dental team* is in an excellent position to recognize areas of *nutritional risk.* The role of the dental team should be to *screen patients* for nutritional risk, provide *dietary guidance* related to oral health, and *refer patients* to nutrition professionals for treatment of other nutrition-related systemic conditions.[3]

Question 1

What are appropriate nutrition interventions for dental clinicians?

A. Assess patients' nutritional status using laboratory and other biochemical assessment tools.

B. Screen patients for nutritional risk.

C. Recognize dietary problems in denture patients.

D. Provide diet guidance related to oral health.

The Basis for a Healthy Diet

Dietary Reference Intakes

Daily food intake must be sufficient to meet metabolic requirements for energy and provide the essential nutrients that the body cannot synthesize in sufficient quantities to meet physiologic needs. Since the 1940s, the *Food and Nutrition Board* (FNB) of the National Academy of Sciences has published the *Recommended Dietary Allowances* (RDA), which were recommendations for daily nutrient intake that would support growth and maintenance of body tissues, and prevent deficiency diseases. (4-Food and Nutrition Board). Beginning in 1997, the Food and Nutrition Board began

to make major changes to the format and purpose of the nutrition recommendations. *The Dietary Reference Intakes (DRI) expands and replaces the RDA*[5] by addressing the prevention of *chronic degenerative diseases* and the risk of *excess intake of nutrients.*[5]

The DRI are quantitative estimates of nutrient values to be used for planning and assessing diets for healthy people.[5] These reference values vary by *gender and life stage group.* DRI consist not only of RDA but also three other types of reference values shown in Table 15-1.

Evaluation of the true nutritional status of an individual requires a *combination* of clinical, biochemical, and anthropometric data.[5] So if an individual reports an intake of a nutrient below the RDA, more information would be necessary to determine if an actual deficiency exists. Conversely, nutrient intakes that meet the RDA over time have a low probability of being inadequate.

Dietary Guidelines for Americans

The *Dietary Guidelines for Americans* were first published in 1980, and are revised every 5 years.[6] The guidelines are designed to complement the DRIs by making recommendations for food choices to promote health. The 2000 Dietary Guidelines for Americans contain 10 recommendations, grouped into *three areas called the ABC* of good health. They are shown in Table 15-2.[6] These *newest* guidelines place more emphasis on *physical activity* and *healthy weight* compared to previous editions. The focus on preventing obesity is caused by the increased risk it presents for many chronic and degenerative diseases, such as heart disease, stroke, diabetes, arthritis, high blood pressure, and some kinds of cancer. The recommendations emphasize balance, moderation, and variety in food choices, and promote increased use of whole grains, fruits and vegetables, and decreased use of saturated fat, cholesterol, and salt. In addition, for the first time, the guidelines address food safety in an effort to combat food-borne illness, an important public health concern.[6]

The 2000 Dietary Guidelines for Americans define a healthy weight according to the *Body Mass Index (BMI).* The BMI is a medical standard for defining obesity that not only is highly correlated with independent measures of body fat, but is also used to determine if a person is at increased health risk due to excess weight[7] (Table 15-3). A healthy BMI of 19 to 25 is associated with the lowest statistical health risk [8-Meisler, 1996]. Persons with BMI above 25 are considered obese, and the recommendation is to lose 1 to 2 BMI units (10 to 15 pounds) to reduce their risk for chronic disease.[7]

Food Guide Pyramid

To help people select nutrient-rich foods and to follow the Dietary Guidelines, the *Food Guide Pyramid* was developed by the U.S. Department of Agriculture.[9] The Food Guide Pyramid displays foods in five categories based on their nutrient composition (Figure 15-2). Whole grains, such as rice, pasta, cereals, and breads, found at the broad base of the Pyramid should form the foundation of a healthful diet. They are good sources of carbohydrate (including fiber) and minerals. Fruits and vegetables form the next level of the Pyramid. The meat group contains good sources of protein, vitamins, and minerals. Meat alternates, legumes, eggs, nuts, and tofu, are

included in the meat group. The dairy group is comprised primarily of good calcium sources. The small triangle at the top of the Pyramid is for the fats, oils, and sweets that provide primarily added calories and, thus, should be eaten in small amounts. No single food group is more important than another; each group provides some, but not all, of the essential nutrients.

Standardized serving sizes and the recommended number of servings for various age groups are specified. However, the caloric content of foods varies widely within a food group. The desirable number of servings from each food group depends not only upon age and sex, but also the calorie goal. For example, if 1,600 calories were the daily energy goal, an individual would choose the minimum number of servings of low-fat food choices from each group. If additional calories are needed, increased servings should come from the grain, fruit, and vegetable groups, rather than the top of the pyramid.

Food Labels

The Nutrition Facts panel found on most processed food packages helps the consumer select foods that meet the Dietary Guidelines (Figure 15-3). The *National Labeling and Education Act of 1990* requires that comprehensive nutrition information *must* appear on the labels of most processed foods and processed meats and poultry products. In *addition,* nutrition information at point of purchase is *voluntary* for fresh fruits, vegetables, and raw fish. In accord with the mandatory food labeling regulations published by the Food and Drug Administration in 1994,[10] the nutrition panel on processed foods must include the following:

- *A standardized portion size* (designed to make nutritional comparisons of similar products easier, and reflects the serving sizes that people actually eat).
- The number of *servings per container.*
- The amounts of *total calories* and *calories from fat* per serving.
- The *number of grams* per serving of total fat, saturated fat, cholesterol, sodium, total carbohydrates, dietary fiber, sugars, and protein.

In addition, the nutritional contribution of *one* serving of the product must be stated as a *percentage of the Daily Values.* The Daily Values are based on the RDA for protein, vitamins, and minerals and on standards designed especially for food labels for nutrients not covered in the RDA such as fat, cholesterol, total carbohydrates, dietary fiber, and sodium. The calculations to determine the percents of Daily Values are based on a *2,000-calorie diet.* Depending on a person's age, gender, and activity level, a person may need more or less than 100% of a Daily Value. The Daily Value also helps consumers see how a food fits into an overall daily diet.

Other information, such as the amounts of polyunsaturated or monounsaturated fats or other vitamins and minerals, is optional. In addition, descriptors such as "free," "low," "high," "light," "lean," or "reduced," may be used on the label as long as a standard portion *meets defined criteria.* For example, to be labeled "low-calorie" a serving must have no more than 40 calories. To be labeled "low-fat," no more than 3 grams of fat per serving is allowed.

Health claims for the potential benefit of a nutrient or food in relation to a disease or

health condition will be allowed on labels if they are supported by scientific evidence and are approved by the Food and Drug Administration (FDA). The 12 health claims currently allowed to be placed on food labels are shown in Table 15-4.[11]

Figure 15-2 The Food Guide Pyramid: A Guide to Daily Food Choices is an outline of what to eat each day. Not a rigid prescription but a general guide that lets each person choose a healthful diet, the Pyramid calls for eating a variety of foods to get the needed nutrients while consuming the right amount of calories to maintain a healthy weight. (Courtesy U.S. Department of Agriculture, Human Nutrition Information Service.)

Figure 15-3 Food Label.

Question 2

The Daily Reference Intakes (DRI) are set at:

A. the minimum amount of a nutrient needed to prevent deficiency.

B. the maximum amount that will not cause toxicity.

C. the average estimated requirement for healthy people.

D. the average requirement plus a margin of safety.

Nutrition in the Development and Integrity of Oral Tissues and Structures

Nutrition plays an important role in the initial growth and development or oral tissues and in their continuous integrity through the lifespan. Optimal nutrition during periods of hard and soft tissue development allow these tissues to reach their optimal potential for growth and resistance to disease. Malnutrition (either over or under-nutrition) during critical periods of *organogenesis* can have *irreversible* effects on developing tissues. Examples of this effect can be seen in the *tetracycline staining* of teeth, in dental *fluorosis*, and in the fever-induced *enamel hypoplasia* seen in the primary teeth.[12] In the dentition, malnutrition is less well documented in humans than in animals, but it appears that during the "critical periods," malnutrition can result in dentition with increased caries susceptibility.[13] Malnutrition *after* initial organ and tissue development is *usually reversible*, but can still compromise tissue regeneration and healing and increase susceptibility to oral diseases. Nutrients for which deficiencies or excesses have been directly associated with oral conditions are protein; energy; vitamins C, A, D; iodine; and fluoride.

Protein/Calorie Malnutrition

Protein is the most abundant organic compound in the body and is required for the synthesis of virtually all body tissues and structures. Proteins account for the structure of DNA, the tensile strength of collagen, and the viscosity of saliva. Thus, aberrations in protein nutriture can have far reaching oral and systemic effects.

The normal turnover of epithelial tissue in the oral cavity requires a continual supply of nutrients. For example, *every 3 to 6 days, the basal epithelium of the gingiva*

undergoes renewal.[14] Thus, any severe deficiency of protein/calorie intake will result in a decrease in mitotic activity in the crevicular epithelium, as well as elsewhere throughout the body.[15] In a comparison of periodontal involvement in patients with severe malnutrition (kwashiorkor) with that of healthy controls in South India,[16] fewer caries and more periodontal disease was found among the undernourished group. Since the oral hygiene indices of both groups were similar, it was assumed that the difference was due to nutritional factors. (It should be noted that any malnutrition of the severity of kwashiorkor represents a multi-nutrient deficiency). Impaired protein synthesis has been found if protein malnutrition occurs during the developmental stage in animals.[17] In animal models, short-term fasting (4 days) resulted in a 40% reduction in collagen production.[18] In the same study, a 10% decrease in collagen synthesis was noted with a reduced dietary intake meeting 20% of requirements.[18] These findings suggest that even short-term states of undernutrition may impact collagen synthesis.

In *chronically malnourished* children, several studies have shown delays in tooth eruption patterns, and increased tooth enamel solubility, leading to increased caries susceptibility.[19-25]

The *linear hypoplasia* reported in the enamel of primary teeth of children in underprivileged populations is thought to contribute to their high prevalence of dental caries. This type of hypoplasia appears to be related to the severity of the malnutrition.[26]

With the exception of the *cleansing* and *diluting* effects of saliva, oral defense mechanisms depend on an adequate supply of proteins. The *glycoproteins* that result in aggregation of bacteria arise from the salivary glands. *Lysozyme, salivary peroxidase,* and *lactoferrin* are also *glycoproteins. Secretory IgA* (sIgA) arises mainly from the labial and buccal glands and is an immunoglobulin. The cell types involved in cellular immunity (polymorphonuclear lymphocytes and macrophages and the enzymes used in phagocytosis) also require protein for their production.[27]

Probably one of the *most deleterious* effects of protein/calorie deficiency is the depletion of the *cellular and immunocellular defenses* of both the oral and the connective sides of the barrier epithelial cells lining the gingival crevice. In general, the severity of the impaired immunologic response parallels the severity of the protein or calorie deficiency.[28]

Minerals

Calcium, in association with vitamin D and phosphorus is essential for proper development and maintenance of mineralized tissues (teeth and alveolar bone). A deficiency of these nutrients during critical phases of tooth development in children results in hypo-mineralization of developing teeth, and possible delayed eruption patterns.[29] Enamel hypoplasia may be seen in prematurely born very low birth-weight (VLBW) infants due to the higher needs for calcium and phosphorus in these infants.[30] In addition, VLBW infants have immature kidneys and may not metabolize adequate levels of vitamin D.[30]

Iron is of interest since iron deficiency is the *most common deficiency* in the United

States. Iron deficiency anemia is manifest in the oral cavity by pallor of oral tissues, especially the tongue. The tongue may appear shiny, with blunted filiform papillae. The effects of iron deficiency on mineralized tissues are less clear. In rats, even a marginal deficiency of iron in the rat diet predisposes the rats to caries. Conversely, supplementing a caries-promoting diet with iron produced a major reduction in caries with the greatest effect shown in the neonatal period.[31] In addition, iron serves as a cofactor with ascorbic acid in collagen synthesis, as is copper.[32]

Zinc regulates *function in inflammation* by inhibiting the release of lysosomal enzymes and histamines. A zinc deficiency can inhibit collagen formation and reduce cell-mediated immunity.[33] The effect of zinc in modifying periodontal defense mechanisms has been shown in rabbits,[35] but has yet to be clearly delineated in humans.[36, 37]

Vitamins

Vitamin A is essential for the development and continued integrity of all body organs and tissues, including the *epithelial mucosa* of the oral cavity. In vitamin-A deficiency, cell differentiation is impaired: *Mucus-secreting cells* are replaced with keratin-producing cells. The result is defective tissue formation, and impaired healing. Vitamin-A deficiency also results in impairment of *both specific and nonspecific immunoprotective mechanisms.* Deficiency can affect tissue response to bacterial infection, mucosal immunity, parasitic and viral infection, natural killer-cell activity, and phagocytosis.[38] Vitamin-A toxicity can show similar effects, with impaired healing response being the most direct affect on the oral cavity.[39] Effects include proliferation of oral epithelium, reduction of the keratin layer, thickening of the basal membrane, and increase in the granular layer. A patient who took 200,000 IU of vitamin A daily for over 6 months presented with painful gingival lesions, along with nausea, vomiting, xerostomia, and headaches. Clinical examination revealed gingival erosions, ulcerations, bleeding, swelling, loss of keratinization, color changes, and desquamation of the lips.[39] All pathologic manifestations disappeared within 2 months of the elimination of the vitamin A supplements when oral hygiene habits were unchanged.

Vitamin C (ascorbic acid) is essential to oral health. Synthesis of *hydroxyproline*, an essential component of *collagen*, requires ascorbic acid. Defects in collagen synthesis are responsible for the many manifestations of vitamin-C deficiency (*scurvy*). In the oral cavity these include spontaneous bleeding, infusions of blood into interdental papillae, loosening and exfoliation of teeth, detachment of oral epithelial tissue, and impaired wound healing.

The effects of vitamin-C deficiency are best studied in animal models, where all factors can be controlled. Acute scurvy can be produced by placing monkeys on a vitamin-C deficient diet for 12 weeks. The hydroxyproline content of the gingiva started to decline in the first four weeks and occurred at a faster rate than in skin.[40] By the end of the 8th week, the synthesis of hydroxyproline was totally impaired.[40] The results are extensive gingival pocket formation and tooth mobility due to degradation of the collagen making up periodontal ligament fibers.[25]

Although frank scurvy is rare, even marginal deficiencies may result in alterations in

collagen synthesis. Thus deficient or marginal ascorbic acid intakes may be a conditioning factor in the development of gingivitis and one of the early manifestations of vitamin-C deficiency.[41] The most recent epidemiologic data from NHANES III (National Health and Nutrition Examination Survey) suggests that the odds of having periodontal disease are 1.2 times greater in those with low dietary vitamin-C intakes.[42] In the same study, *smokers and former smokers* with low vitamin-C intake are at 1.6 times greater risk of having periodontal disease.[42] Research findings suggest that people with marginal vitamin-C deficiency supplemented with ascorbic acid have a statistically significant increase in hydroxyproline in periodontal tissues.[43]

Ascorbic acid is essential to immune related functions, such as resistance to oral infection, via its role in leukocyte formation and subsequent *phagocytosis.*

Conversely, chronic vitamin C excess may precipitate a scurvy-like condition (rebound scurvy) upon cessation of the vitamin. Because the impact of deficient levels of vitamin C is first observed in gingival tissues, dentists and dental hygienists in clinical practice may be the first to diagnose the phenomenon.[44] The B-complex vitamins primarily function as *co-enzymes in energy metabolism.* B-complex vitamins are found widely in foods, and usually together. With the exception of B_{12} in the elderly and folic acid in pregnant women, deficiencies of single B vitamins are uncommon. Oral signs and symptoms of B-complex vitamin deficiencies include cracks in the corners of the mouth (cheilosis), inflammation, burning, redness, pain and swelling of the tongue.[45]

Question 3

Which is *true* about vitamins and oral health?

A. Vitamin-C-deficient wounds heal as well as non-vitamin-C-deficient wounds

B. Vitamin A-toxicity does not have oral effects

C. The oral manifestations of vitamin-C deficiency are related to defects in collagen formation

D. Effects of deficiency and toxicity are best studied in humans

Diet and Nutrition in Oral Conditions: Background and Counseling Strategies

Who Needs Diet Guidance Caries Prevention

Dietary *education and guidance* are important for the prevention and control of dental caries. Patients should be carefully assessed to determine the level of prevention and nutrition guidance needed following these Institute of Medicine prevention guidelines:[46]

Selective Prevention: This strategy targets subset of the total population that are deemed to be at risk for caries for a variety of reasons. Examples include:

Adolescents at risk of caries because of high intake of soft drinks and snack foods.

Caries-prevention counseling for patients with xerostomia or cariogenic diet patterns.

Proactive diet suggestions for new denture wearers or those having jaw fixation.

Diet advice prior to radiation or chemotherapy.

Using current diet patterns as a basis for discussion, patients should be taught the role of diet in caries, what are cariogenic and noncariogenic eating patterns, and how to adapt current diet to lower cariogenic risk.

Indicated Prevention: This strategy targets individuals showing early danger signs of caries, such as extensive cervical demineralization. These individuals need the immediate aforementioned interventions as well as more detailed guidance on how to reduce cariogenicity of their current diet. This will involve determining the factors influencing current habits, and working with the patient to develop appropriate and acceptable strategies for improvement. Patients need to be followed up on a regular basis to promote long-term change.

Question 4

The diet assessment process in dentistry is designed to:

A. diagnose nutrient deficiencies

B. help screen patients for oral-health risk factors

C. serve as a teaching tool

D. determine patients' daily caloric intake

E. provide a therapeutic diet prescription for patients

F. be part of total preventive assessment

Dental Caries: Role of Carbohydrates in Caries Development

Dental caries is a common plaque-dependent bacterial infection that is strongly affected by diet. Development of clinical caries is contingent upon the interaction of three local factors in the mouth: *a susceptible tooth, cariogenic bacteria, and fermentable carbohydrate* (Figure 15-4). Absence of one of these factors dramatically reduces caries risk. Mutans streptococci are the predominant oral bacteria that initiate the caries process. Newly erupted teeth with a thin enamel layer are very caries susceptible. Tooth morphology, especially the presence of deep pits and fissures, influences the likelihood that mutans streptococci will attach to and colonize the tooth's surface. Plaque bacteria ferment starches and sugars, producing organic acids. These acids demineralize dental enamel.[47]

Other dietary factors *counteract* the damaging effects of carbohydrates. The presence

of protective minerals and ions such as *fluoride, calcium, and phosphorus* in *plaque and saliva,* promote remineralization of incipient lesions. In addition to transporting minerals, saliva contains *buffering agents, bicarbonate and phosphates,* that neutralize organic acids. Thus, the amount and composition of saliva affect the caries process. Other host factors that influence caries risk include: genetic predisposition, immune status, malnutrition during tooth formation, education level, and income status.

In the most recent national health and examination survey (NHANES III, Phase I) 94% of adults showed evidence of coronal caries and 22.5% of adults had root caries.[48] In the same survey, 25% of the children and teens aged 5 to 17 had 80% of the dental caries detected in the permanent teeth.[49] For these caries-prone children and adults, *nutrition counseling* about the damaging effects of fermentable carbohydrates on teeth is essential.

Through epidemiological and clinical studies discussed in Chapter 14, the causal relationship between sugar consumption and dental caries has been established. Animal studies suggest that an increase in the concentration of sucrose in the diet reduces dental plaque formation and increases the incidence of dental caries.[50,51] People with very low sugar intakes have low-caries scores. People in nations that have high sugar intakes have high rates of caries.[52] It is unclear if this is primarily the topical effect of sugar consumption or systemic effects on dentin formation. However, the amount of sugar consumed is not the sole dietary variable associated with caries development. Sucrose plays a more dominant role than other sugars in the development of smooth surface caries. One of sucrose's metabolic by-products, an extracellular polysaccharide called *glucan,* enables the mutans streptococci to adhere to the smooth enamel surfaces.[53] However, the amount of sucrose necessary for the implantation of mutans streptococci is very low.

Although sugar intake is high among most persons in industrialized countries, it is more difficult to demonstrate a *correlation* between caries prevalence and the amount of sugar consumed than in developing countries where sugar intake is lower. Three recent clinical trials of English, United States, and Canadian schoolchildren examined the relationship between sugar intake and dental caries. In England, 405 children with a mean age of 11.6 years were followed for 2 years. Total sugar intake (118 grams per day or 21% of total calorie intake) had the highest significant correlation with caries rates.[54] Intake of sugary foods before bedtime was highly correlated with caries incidence. In the United States, 499 children aged 11 to 15 years, living in nonfluoridated rural Michigan communities, were followed for 3 years. The average increase in decayed, missing, and filled surfaces (DMFS) over the 3 years was 3.1 in girls and 2.7 for boys. The daily average sugar intake was 142 grams, this represented 26.5% of their total energy intake. Children who obtained a higher percent of their total calories from sugars had more proximal surface caries. The average number of eating occasions and the number of sugary between-meal snacks consumed were not related to caries increment.[55]

Fifty percent of 232 11-year-old children in a Canadian study had *inadequate diets.* Children with superior diets tended to develop fewer caries; however, the association was not statistically significant.[56] Differences in eating patterns and intake of caries-promoting foods among the children in these studies may have been too small to

result in significant differences in caries experience. Other factors contributing to the caries decline in western countries are: fluoride intake from water, the use of fluoridated dentifrices, improved plaque control, the use of dental sealants, and more frequent visits to the dentist.[57]

The use of *sugar alcohols and alternative sweeteners* in foods also has had a role in *reducing* caries. Perhaps one of the most promising sugar substitutes to be studied is *xylitol*, a sugar alcohol that has been demonstrated to be non-cariogenic as well as promoting remineralization.[58] Xylitol's ability to inhibit metabolic acid production by mutans streptococci results in minimal depression of plaque pH. Maintenance of the *plaque pH close to the saliva pH* also fosters remineralization of teeth.[59] In addition, the substitution of xylitol for fermentable sugars in the diet results in a less cariogenic bacterial flora. The importance of other non-fermentable sweeteners in caries control is detailed in Chapter 14.

Simple sugars are not the only carbohydrate that influences the development of a carious lesion. Highly refined *cooked starch-sugar combinations* such as doughnuts, cookies, potato chips, and some ready-to-eat breakfast cereals *produce a prolonged acidogenic response* when retained in interproximal spaces.[60] When starches are cooked, they are partially degraded. This allows the salivary alpha-amylase to convert starch particles retained on the tongue, oral mucosa, and teeth to *maltose.* Making maltose available to plaque bacteria extends the length of time the plaque pH will remain low and permit enamel demineralization to occur. Thus, retentive high starch foods may be more acidogenic than high-sugar-low-starch foods that are rapidly eliminated from the mouth.[61]

Effects of Eating Patterns and Physical Form of Foods

Other dietary factors that may hinder or enhance caries development include: the frequency of eating, the *physical form* of the carbohydrate (liquid vs. solid), *retentiveness* of a food on the tooth surface, the *sequence* in which foods are consumed (e.g., cheese eaten before a sweet food limits the pH drop), and the presence of minerals in a food.

Frequent between-meal snacking on sugar or processed starch-containing foods increases plaque formation and extends the length of time that bacterial acid production can occur. When total daily sugar intake was held constant, increasing the frequency of sugar intake for groups of rats resulted in increased number of *Streptococci mutans* in plaque and the amount of caries experienced.[62] The positive relationship between frequency of sugar intake and caries in humans was first demonstrated in the *Vipeholm* study.[63] Subjects who consumed candies *between meals* developed *more* caries than those who were fed equal amounts of sugars *with* meals. Frequent snacking between meals keeps the plaque pH low and extends the time for enamel and dentin demineralization to occur.

Bacterial fermentation can continue as long as carbohydrate adheres to the enamel and exposed dentinal tooth surfaces. Even though starchy foods vary in their cariogenic potential, the highly refined starchy foods, such as soft bread and potato chips, that are retained on tooth surfaces for prolonged periods of time, result in a lowered pH which may last *up to 60 minutes.*[64,61] High-sucrose confectionery foods deliver high

levels of sugar to the oral bacteria immediately after the foods are consumed, whereas high-starch foods deliver progressively increasing concentrations of sugars over a considerably longer period of time.

The *sequence* in which foods are eaten affects how much the plaque pH falls. Sugared coffee consumed at the *end* of a meal will cause the plaque pH to remain *low for a longer time* than when an unsweetened food is eaten *following* intake of sugared coffee.[65] If peanuts are eaten before or after sugar-containing foods, the plaque pH is less depressed.[66]

Some components of foods *are protective* against dental caries. Protein, fat, phosphorus, and calcium inhibit caries in rats.[67] Aged natural cheeses have been shown to be cariostatic.[68] When cheese is eaten following a sucrose rinse, the plaque pH remains higher than when no cheese follows a sucrose rinse. In addition, enamel demineralization, measured using the intraoral cariogenicity test, is reduced. The protective effect of cheeses is attributed to *their texture* that *stimulates salivary flow*, and their protein, calcium, and phosphate content that *neutralizes plaque acids.* Fluoride found in drinking water, foods, and dentifrices increases a tooth's resistance to decay and enhances remineralization of carious lesions.

Lipids seem to accelerate oral clearance of food particles. Some fatty acids, linoleic and oleic, in low concentration, inhibit growth of mutans streptococcus. Lectins, proteins found in plants, appear to interfere with microbial colonization and may affect salivary function.[69]

Figure 15-4 Factors Required for Caries Development.

Question 5

In the diet of a patient with rampant dental caries, which is *most relevant* to the problem?

A. total amount of sucrose consumed

B. total amount of sticky sweets consumed

C. nutrient quality of the meals and snacks

D. number of meals and snacks

E. what is eaten for desert in the evening

Measuring the Cariogenic Potential of Foods

Since it is unethical to conduct human experiments to measure the true cariogenic potential of foods, other indirect tests have been developed. These tests enable researchers to classify foods into at least three categories: protective, low, and high cariogenic potential. Currently the cariogenic potential or the ability to induce caries in humans may be assessed indirectly by measuring the ability of a test food to cause: caries formation in animals, acid production in dental plaque, or demineralization of

enamel.[1]

Animal studies have been conducted using a *programmed feeding machine*. In one study, 20 common snack foods were presented to rats at specified intervals during the day.[70] After sulcal and smooth surface caries were scored in the animals, cariogenic potential indices (CPI's) were computed for each food (the sucrose group had a CPI value of one) (Table 15-5). A food with a CPI of 0.4 had low cariogenic potential. Those snack foods with high cariogenic potential had 1% or more hydrolyzable starch in combination with sucrose or other sugars.

Acid production in the mouth during bacterial fermentation of a food is predictive of the contribution of that food to the caries process. Measurement of plaque acidogenicity can be measured by determining the pH of a plaque sample taken from the mouth or *in situ.*[1] Foods that cause the plaque pH to fall below the critical demineralization level (*pH 5.5 to 5.0*) are considered acidogenic. Measurement of oral plaque pH requires placement of a wire-*telemetric appliance* containing a pH microelectrode in the space where a tooth is missing in the mouth. As the test food is chewed, the pH under undisturbed plaque at the site of the indwelling electrode is continually transmitted to an external receiver. The rate of the fall and rise of the pH at an interproximal site can be recorded continuously using plaque telemetry. Foods found to have low acidogenic potential using this method include: aged cheeses, some vegetables, meats, fish, and nuts.[71]

To assess the ability of a food to demineralize dental enamel, an *intraoral cariogenicity test* has been developed. Bovine or human dental enamel slabs are imbedded in a prosthesis and placed in the mouth where a tooth is missing. After ingesting a test food, changes in surface microhardness or enamel porosity are determined.[72] Since each test measures a different aspect of cariogenicity, foods will be ranked differently. It is recommended that two testing methods be used to determine food acido/cariogenicity potential.[73,74] Table 15-6 shows the acidogenic potential of foods. Table 15-7 provides diet suggestions for caries prevention.

Question 6

Tooth erosion can be caused by

A. acid from vomiting

B. sugar-containing carbonated beverages

C. gastro-esophageal reflux

D. sugar-free carbonated beverages

E. all of the above

Early Childhood Caries

One of the most severe forms of caries occurs in infants. Inappropriate feeding practices may result in progressive dental caries on the buccal and lingual surfaces of

newly erupted primary maxillary anterior teeth of infants and toddlers. The overall prevalence of early childhood caries (also called *baby bottle tooth* decay or *nursing caries*) is estimated to be 5%.[75] However, a much higher prevalence has been seen among Alaskan and Oklahoma Native American children (53%) and Navajo (72%) and Cherokee (55%) Head Start children attending Head Start programs.[76, 77]

Primary risk factors for early childhood caries include putting a child to sleep at naptime or bedtime with a bottle containing a liquid *other than plain water,* allowing an infant to breast-feed at will during the night, and extended use of the nursing bottle or sippy cup beyond 1 year of age. Results of the 1991 National Health Interview Survey show that 16.7% or 3.5 million children between 6 months and 5 years of age are put to sleep with a liquid in the bottle other than plain water.[78] Inappropriate feeding practices were reported more often by parents with *less than a high school education,* low incomes, Hispanic backgrounds, and those parents whose children had not been to a dentist in the past year.

Children who develop maxillary anterior caries are at increased risk of developing posterior caries in the future.[79] To prevent early childhood caries, dentists, pediatricians, and other health care professionals should ask parents about their infant feeding practices. Those parents who report inappropriate feeding practices should receive counseling. Programs serving low-income families, such as the Special Supplemental Food Program for Women, Infant, and Children (WIC), can play a major role in providing education to parents at higher risk for using inappropriate feeding practices.

Nutrition and Periodontal Disease

Like caries, periodontal disease is an infectious disease, multifactorial in etiology, and occurs when virulence of the bacterial challenge is *greater than the host defense and repair capability.* The course of periodontal disease involves periods of progression and remission. Unlike the direct causative relationship between carbohydrates and caries, nutritional factors seem to play a much more subtle role in periodontal status. Nutritional factors *can alter host susceptibility* to periodontal disease and/or modulate its progress.[80] The nutritional factors related to *preventing infection* and *enhancing wound healing* in general applies to the prevention and management of periodontal disease as well.[81] *If* both the challenge to and the defense and repair capabilities of the periodontal tissues are in balance, nutrition could be the deciding factor in whether health or disease results. Even when the periodontium is healthy, there is continual need for nutrients to maintain the tissues. Once inflammation is established, the need for nutrients *increases.* There is a close relationship between malnutrition and infection, with infection aggravating malnutrition and malnutrition abetting infection. Defense in the gingival crevice and connective tissue all require an adequate intake of all nutrients to ensure adequate production and function of defense and supporting cells.[82-86] With the increased needs of cellular immunity and the additional demands by the tissue cells attempting to maintain and repair damaged areas, a greater supply of all nutrients is needed. This has led to evidence showing that nutrient requirements may be higher at local sites of increased stress than in the rest of the body. Such localized challenges may result in *end-organ nutrient deficiencies.*[87, 88]

Diet Guidelines

Whenever routine scaling, prophylaxis, and oral plaque control procedures fail to reverse gingivitis and before any treatment for periodontitis is attempted, a thorough diet evaluation and patient counseling session is indicated. The patient should be informed about the importance of systemic nutrition in the defense and repair of oral tissues. Recommendations should be made to help ensure optimal nutrition to help prevent and manage periodontal disease. These include:

- Eat a *nutritionally adequate diet* following the food pyramid guidelines.
- Increase the use of saliva-stimulating fibrous foods.
- Multivitamin/mineral supplements should be in doses *no higher* than one to two times Recommended Dietary Allowance levels.
- *Avoid fad diets* which could be deficient in nutrients.
- Avoid *single* vitamin supplements.
- *Avoid potentially detrimental megadoses* of vitamins and minerals (10× RDA or higher).

Question 7

Periodontal disease is caused by dietary deficiencies. Calcium deficiency is thought to be a contributing factor in alveolar bone loss in humans.

A. both statements are true.

B. both statements are false.

C. the first statement is true: the second is false.

D. the first statement is false the second is true.

Eating Disorders

Eating disorders, especially *bulimia,* are often first diagnosed in the dental office. Patients, usually young females, present with severe erosion of the lingual tooth surfaces. The oral tissues are often red, sore, and painful. The esophagus may be inflamed, and parotid salivary glands are often swollen. Bulimia is characterized by recurrent episodes of *binge eating* (consumption of large amounts of foods at a time) followed by self-induced regurgitation (purging). The average intake of food during a binge is 3,400 calories over an hour, with some individuals ingesting as much as 50,000 calories in 24 hours.[89] Patients may also use laxatives and/or diuretics to induce malabsorption and fluid loss. The acid from stomach regurgitation irritates the esophagus and the oropharyngeal soft tissues. The regurgitated acid in combination with xerostomia, results in rapid and extensive destruction of tooth enamel.[90]

Patients often first deny having an eating disorder. However, when confronted with the oral evidence, they often admit to the disorder. The dentist should refer the patient to an eating-disorder management program and elicit patient agreement to undergo treatment. The diagnosis of this disorder by the dentist and the realization of the dental destruction caused by the disorder, often convince patients to agree to treatment. A *multidisciplinary* approach to treatment is needed, including physicians,

psychiatrists, psychologists, nutritionists, and social workers. The patient must be cautioned that for dental rehabilitation to be successful, the underlying problem (the eating disorder and its causes) must be resolved.

Question 8

Oral problems that may be seen in patients with eating disorders include:

A. swollen salivary glands

B. orange-stained teeth

C. decreased salivary flow

D. decreased oral pH

E. severe enamel demineralization

The Aging Patient

The aging patient is often faced with a variety of challenges that can undermine both oral health and nutritional status.[91] As a result, the elderly are considered particularly susceptible to malnutrition.[92] Compared to younger individuals, elders have a significantly decreased ability to respond to physiologic challenges. Sensory function decreases leading to *impaired taste and smell.*[93] Changes in the gastrointestinal system can affect the ability to digest, absorb and utilize food properly. Functional problems, such as arthritis or vision difficulties can affect the ability to prepare and eat food. Psychosocial problems such as loneliness, depression, lack of money, and poor access to food can all undermine good eating habits.

Problems in the oral cavity, such as xerostomia and loose teeth, have been considered major contributors to the poor eating habits of the elderly and may be a major contributor to malnutrition.[92,105,94,95] Several studies have shown that dentate status can affect eating ability[96] and subsequent diet quality.[97, 98, 99] Individuals with one or two complete dentures had a 20% decline in diet quality compared to those with at least partial dentition in one or both arches.[100] Another study showed that compared to those with 25 or more teeth, edentulous individuals consumed less fiber and carotene, fewer vegetables, and more cholesterol, saturated fat and calories.[101] Dentures can affect *taste and swallowing ability*, especially if they are maxillary dentures. The denture covers those taste buds found on the upper palate. And when the upper palate is covered, it becomes difficult to detect the location of food in the mouth. For this reason, dentures are considered to be the *major cause of choking* in adults.[102]

Dry mouth (xerostomia) is common in the older population, in part because of xerostomic medications commonly taken. Xerostomia makes eating more difficult and increases the cariogenic potential of the diet.[103,104] It has also been associated with burning mouth syndrome and inadequate diet.[105]

Conversely, nutrition is an important factor in oral status.[106] In a sample population of 843 elderly people, there was a significant association between low ascorbic acid

levels and the prevalence of oral mucosal lesions.[107] Low calcium intake throughout life has been shown to *contribute to osteoporosis.* In turn, osteoporosis in alveolar bone is thought to be an important contributing factor to the resorption of alveolar bone that ultimately results in tooth loss.[108] The alveolar process is composed primarily of trabecular bone, which is more labile to calcium imbalances than is cortical bone. Thus, the alveolar bone provides a potential labile source of calcium available to meet *other* tissue needs. Since the alveolar process is thought to undergo resorption *prior* to other bones; it is projected that changes detected in the alveolar process may eventually be used for early detection of osteoporosis.[109] Mandibular bone mass was correlated with total body calcium and bone mass of the radius and vertebrae in dentate and edentulous postmenopausal women with osteoporosis,[110] with the highest correlation between total body and mandibular bone mass. Thus, the mandible reflects the mineral status of the entire skeleton. Calcium intake in postmenopausal osteoporotic women was also correlated with mandibular density; supporting the hypothesis that low calcium intake may contribute to reduced bone density.[111,112] In a study of 329 healthy post-menopausal women, an inverse relationship was shown between bone mineral density and number of existing teeth, with those women who received dentures after the age of forty having the lowest bone mineral density.[113]

Older patients should be carefully screened for nutritional risk factors, and should be educated about the importance of good nutrition to general and oral health.[114, 115] If major nutritional problems are suspected the patient should be referred to a nutritionist.[116] When new dentures are provided, patients should be counseled on how to adapt their usual diet to a softer consistency for the first few days after denture insertion.

Question 9

Which is true about aging?

A. Dry mouth always occurs with aging.

B. Dentures improve taste perception.

C. Taste and smell acutely tend to decrease.

D. Calcium intake is not of concern with this group.

The Diabetic Patient

The *diabetic* dental patient is at *greater risk* for developing oral infections and periodontal disease than the nondiabetic patient.[117,118] The dental team needs to be aware of current approaches to diabetes management and carefully monitor the patient's health status prior to initiating dental treatment.

The nutrition care plan generally requires that patients have meals and snacks of specific nutrient composition *at regularly scheduled intervals,* coordinated with medications (insulin or oral agents) and exercise. Dietary management has changed from the high fat, low carbohydrate diets of past decades to the more liberal use of

complex carbohydrates and the reductions in fat recommended today.[119,107] A well-balanced diabetic diet should be low in cariogenicity, since the use of cariogenic fermentable carbohydrates should be infrequent. Frequent use of hard candies or other foods taken to counteract hypoglycemia are an indication that the diabetes is not well controlled. Patients with uncontrolled diabetes should be referred to their physician for further management. *In the dental office, quickly assimilated carbohydrate sources such as juices, milk, and crackers, should be kept readily available in the event that a diabetic patient develops symptoms of hypoglycemia.*

Patients with Immunocompromising Conditions (Cancer, AIDS)

Immunocompromised patients, such as those with cancer or AIDS, often have *increased* requirements for nutrients while having major physiologic and psychosocial impediments to eating. Cancer often sets up a syndrome of weight loss and wasting in which both metabolism and nutrient losses *increase.* The cancer often causes severe anorexia, taste changes, and early satiety. The pain and discomfort of oral infections such as the herpes simplex and oral candidiasis found in AIDS and chemotherapy patients, can also impair the desire and ability to eat.[120] *Over half of all head and neck cancer patients are nutritionally compromised at initial diagnosis.*[121] Radiation therapy increases eating difficulty by causing painful oral *mucositis, dysphagia,* and severe *xerostomia.*[122]

When providing dental treatment to patients suffering from cancer or AIDS, team members need to understand the nutrition principles underlying the care, so that dental services provided can be coordinated effectively with total care. The nutrition care plan initially focuses on providing *high caloric intake in frequent small meals.* Liquid supplements may be used if optimal nutriture cannot be achieved via food alone. In more serious cases, patients may need enteral (tube) feedings or more advanced nutritional support. A high calorie diet will likely be high in sugars and total calories.[123] In these cases, the dental team should *not* caution patients to reduce the frequency of eating, since this will contradict nutritional management goals. Rather, thorough cleaning after each eating period, and use of fluoride mouth rinses and topical fluoride trays before bed should be stressed. This approach is standard protocol for immunocompromised patients as part of an aggressive preventive dental program.[124] Cancer patients should be cautioned, however, about the potential oral sequelae of an increased frequency of eating. Patients should also be cautioned to avoid the use of slowly dissolving hard candy often used to assuage the xerostomia. The most important *monitoring* tool for these patients is *weight status.* The patient should be queried at each visit about how their weight is being maintained. Involuntary weight loss of 10 pounds or more is a warning for the need for more intensive care.

Oral Surgery and Intermaxillary Fixation

The patient who has had oral surgery, whether therapeutic or as a result of trauma, needs special nutritional consideration (125-Kendall, 1982). An adequate diet *before surgery is needed to support adequate post-surgical response.* If food consumption will be impaired for a short period of time, the risk of nutritional deficiency is low. The risk of deficiency increases with length of eating impairment. The surgery itself can result in an anorexia, inability to chew, and increased metabolic requirements.[126]

After surgery, a patient may need a *liquid diet for 1 or 2 days*, but should progress as soon as possible to a soft diet of high nutritional quality, until a normal diet can be resumed. In some cases, nutritionally complete liquid supplements may be appropriate and should be prescribed in consultation with the patient's dietitian and physician. Often patients prefer purees of normal foods over commercial liquid supplements.[127] Multivitamin/ mineral supplements may be appropriate as well.

Question 10

Which of the following is/are true?

A. A well-controlled diabetic diet should be low in caries risk.

B. Patients with cancer often have increased nutrient needs.

C. Patients with immune-compromising conditions should be told to reduce the frequency of eating to reduce caries risk.

D. The oral surgery patient may require a liquid diet for 1 to 2 days after surgery but should return to a normal diet as soon as it is possible.

Summary

Nutritional status and dietary habits can affect and be affected by specific oral conditions. Comprehensive patient care requires that nutritional factors be considered in the etiology, progression, and sequelae of oral problems.[128, 129]

Dental-team members should routinely screen patients for nutritional issues, provide dentally-oriented counseling, and refer patients to dietitians for further care. The nutritional implications in dental conditions are many and complex. No longer can nutrition in dentistry be summarized as "sugar is bad, and fluoride is good."

Answers and Explanations

1. b, c, and d—correct.

a—incorrect. It is not appropriate or possible for the dental team to attempt to assess actual nutritional status. This requires sophisticated laboratory testing under the supervision of a qualified medical professional.

2. d—correct.

a—incorrect. The minimum amount of a nutrient needed to prevent deficiency is not considered an appropriate standard of adequacy

b—incorrect. The maximum amount of a nutrient that will not cause toxicity is the UL or upper tolerable limit

c—incorrect. The average estimated requirement for healthy people would mean that half of the population would require more. Thus it is not used as the criteria for

healthy populations

3. c—correct.

a—incorrect. Vitamin C-deficient wounds have poorer healing ability

b—incorrect. Vitamin-C deficiency affects all epithelial tissues including those in the oral cavity

d—incorrect. It is not ethical to conduct such studies in humans

4. b, c, and f—correct.

a—incorrect. The diet assessment process can be used to screen patients for possible nutrition risk, but cannot be used for true nutritional assessment

d—incorrect. Daily calorie intake cannot be determined using a diet screening tool. Patient's daily calorie intake is best assessed by a registered dietitian using an assessment tool designed for that purpose.

e—incorrect. The dental team can use screening information to refer the patient to a registered dietitian who is qualified to provide therapeutic diets. The dental team can provide nutrition information about healthy diet and diet/oral health relationships.

5. d—correct

a—incorrect. Sucrose is not the only cariogenic factor, and the amount is not as important as the distribution in the diet.

b—incorrect. The amount of sticky sweets is not as relevant as the frequency of usage of these items.

c—incorrect. The nutrient quality of the diet is only related to the caries process after tooth eruption through remineralization effects.

e—incorrect. Dessert is only one of many contributing factors to dental caries.

6. e—correct.

Tooth erosion can be caused by acid from vomiting, sugar-containing carbonated beverages, gastroesophageal reflux, and sugar-free carbonated beverages to name but a few factors.

7. d—correct.

Periodontal disease is not *caused* by dietary deficiencies. However, calcium deficiency is thought to be a contributing factor to alveolar bone loss in humans.

8. a, c, d, e—correct.

b—incorrect. Orange-stained teeth are not necessarily caused by eating disorders. The staining can come from food, beverages, or other sources.

9. c—correct.

a—incorrect. Dry mouth is associated primarily with the use of medications and is not inevitable with aging.

b—incorrect. Dentures can impair taste perception if they cover taste buds on the upper palate.

d—incorrect. Calcium is important for all age groups. Calcium intake is associated with bone density in general and may be a factor in alveolar bone health as well.

10. a, b, and d—correct

d—incorrect. Patients with immune compromising conditions should *not* be told to reduce the frequency of eating to reduce caries risk. These patients are at high risk for nutritional deficiency and must eat high calorie foods on a frequent basis throughout the day. Oral risk should be reduced by having patients rinse the mouth and clean the teeth as best they can after each eating period and use remineralizing rinses.

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Chapter 16. Understanding Human Motivation for Behavior Change - *Mary Kaye Sawyer-Morse Alexandra Evans*

Objectives

At the end of this chapter it will be possible to

1. Define motivation.

2. List reasons why individuals may not be motivated to receive regular oral care.

3. Describe two different approaches to motivate individuals to change behavior.

4. Describe elements of three common behavioral health promotion theories.

5. Explain the importance of appropriate health provider communication.

6. Describe four common client-provider communication styles.

7. Describe motivational interviewing and FRAMES.

Introduction

The mouth represents an area of the body of special importance and value. According to Horowitz and coworkers,[1] the mouth is associated with the development of (1) a healthy personality, (2) perceptions, and (3) the overall experience of pleasure. Many areas of the mouth, especially the gingival tissues, are easily accessible for self-diagnosis and primary preventive treatment. Individuals can easily detect gums that are red or bleeding. In addition, the tongue, with its highly developed neurosensory feedback system, can be useful in helping people to assess their own plaque levels and resultant need for improved oral hygiene behavior. As a result, dental professionals should devise strategies for motivating oral self-care behavior by teaching clients how to recognize their own signs of dental distress or neglect.

In this chapter the interrelationship of motivation, education, and behavioral modification are considered—all with the objective of helping dental professionals develop more effective interpersonal skills, thereby becoming more effective health educators and counselors.[2] The task of educating the client can be greatly simplified by a knowledge of and the application of a few basic constructs of educational and health promotion and human motivation. These same constructs apply equally to either private or public health practices.

The Problem: Oral health is an essential component of health throughout life. Poor oral health and untreated oral diseases can have a significant effect on quality of life. The mouth is the entry point for food and the beginning of the gastrointestinal tract. The ability to chew and swallow is a critical function required to obtain essential nutrients for the body—the building blocks of good health.[3] However, millions of individuals in the United States have dental caries and periodontal disease, resulting in unnecessary pain, difficulty in chewing, swallowing, and speaking, increased medical costs, loss of self-esteem, decreased economic productivity through lost work and school days, and, in extreme cases, death.[4] The Healthy People 2010 document recognizes the importance of oral health and includes 17 specific objectives related to the overall goal: To prevent and control oral and craniofacial diseases, conditions, and injuries and improve access to related services.[5]

Regular and timely dental visits provide an opportunity for the early diagnosis, prevention, and timely treatment of oral diseases and conditions, as well as for the assessment of self-care practices. However, approximately 66% of people in the United States do not see a dentist regularly,[5] and among specific subpopulations, such as certain ethnic groups or low-income groups, the proportion not receiving regular care is even higher.[6] For example, the Medical Expenditure Panel Survey in 1996 indicated that 44% of the total population visited a dentist in the past year, while 50% of non-Hispanic whites, 30% of Hispanics, and 27% of non-Hispanic blacks had a visit. In addition, 55% of those individuals with some college education had a past-

year visit compared to 24% of those with less than a high school education.[7]

The reasons individuals may not be motivated to seek regular and timely care include: high cost of dental care, lack of dental insurance, lack of providers from underserved racial and ethnic groups, fear of dental visits, habitual personal neglect, lack of knowledge, limited oral-health literacy, and negative feedback or unflattering statements about dentistry received from friends or relatives.[5] Other factors that have contributed to people losing confidence in dentists include prior negative experienced with dentists (poorly executed or ineffective treatment and unnecessary or questionable extractions or other treatments), dental treatment that did not last long enough, and lack of access to appropriate dental care. Previous painful experiences and perceived negative dentist behaviors (e.g. arrogance, sarcasm, or inconsideration) appear to be especially important to the anxious individual who is mentally preparing for dental treatment.[8] See Table 16-1. Most of these barriers can be overcome by effective client education and motivation programs and more effective interpersonal communication by the dental professional.

Dental Education and Motivational Programs

In previous chapters, it is stated that primary preventive dentistry can be effectively implemented by using the following five actions: 1) plaque control, 2) reduction of sugar in the diet, 3) fluoride therapy, 4) use of pit-and-fissure sealants, and 5) client education. The successful use of any of these actions requires effective relations between dental professionals and clients to achieve and maintain a maximum level of oral health. Three major enabl-ing factors are necessary to perform the above listed actions—appropriate skill-based education, client self-motivation, and appropriate psychomotor skills.

For any preventive dentistry program to succeed, information about what needs to be done and how it is to be accomplished must be available to both the dental professional and the client. For the client, this information (and sometimes misinformation) is often learned through school-based health programs; the dentist, media, and advertising; and from peers, friends, neighbors, or relatives. On the other hand, dental professionals learn preventive dentistry as part of the curriculum in dental and dental hygiene schools, through reading professional dental journals, by attending professional meetings and conferences, and through participation in continuing education programs. In some cases, the gap between the information possessed by the clients and the dental professionals is great. This gap in knowledge poses a problem because people tend to seek what they already believe and avoid exposure to anything that mandates changes.

In general, the personality characteristics of dentists indicate that technical proficiency and attention to detail may be more common than strong interpersonal communication skills.[9] For this reason dental professionals may need to cultivate specific knowledge and expertise in the area of human behavior and motivation techniques. Because the skills to accomplish these tasks are not commonly taught in dental school, many dental professionals do not have adequate skills to provide information to clients appropriately.

In addition, many dental professionals are taught that providing knowledge to a client

is sufficient to change the client's behavior. However, extensive research indicates that information by itself is necessary, but not sufficient. Human behavior is a product of the interaction of multiple factors such as attitude, self-efficacy, knowledge, or perceived risk and benefits. Any one factor can be powerful but none acts independently.[10] Therefore, not only do many dental professionals need to acquire or strengthen skills on how to provide information to clients, they also need to learn how to appropriately motivate clients so that behavior change can occur. Many health behaviors theories explain health behavior and can guide effective behavior change. For further description of three common health behavior theories, see section Health Promotion Approach to Behavior Change.

Motivation

What is motivation? Everyone is motivated to action or inaction. To not be motivated is to be dead. Some argue that humans are primarily instinctual in nature. This argument is difficult to accept because of the varied nature of human behavior. If the "instinct theory" was valid, all humans would show a uniformity of behavior across all cultures.[11] This, of course, is not the case. Others believe that behavior is learned and that our environment determines our actions. Indeed, no one should downplay the importance of environmental forces on human behavior. Motivation may be described as the interaction between the environment, personal and behavioral factors.[12] Despite the fact that human behavior is highly variable and at times unpredictable, one thing is certain: Individuals' performances or behaviors are based on the degree to which they are motivated. Motivation makes the difference.

Human motivation is complex. It is based on a blending of expectations, ideas, feelings, desires, hopes, attitudes, values, and other factors that initiate, maintain, and regulate behavior toward achieving a given goal or outcome. Other factors, such as previous adverse experiences, educational insufficiency, nonacceptance by peers, a poor self-image, and impoverished socioeconomic circumstances can significantly influence behavior. Motivation factors can change with the passage of time. Humans are strongly goal-oriented and can demonstrate a tremendous drive to achieve their personal ambitions. For some, however, a significant part of the pleasure is derived from working toward a goal; after they have "arrived," their pleasure is somewhat diminished. For these individuals, getting there is not only half the fun, it is possibly all the fun. For example, some individuals periodically become intensely motivated to upgrade their oral health status. Appointments are made with the dentist, all restorative work is completed, preventive programs are developed with a great amount of client participation until all dental care has been completed, at which time the individual appears to lose interest until another sudden flurry of interest may occur at a later date.

Motivation then is seen not as a personality problem or trait but rather as a state of readiness or eagerness to change. This readiness may fluctuate from one time or situation to another and can be influenced by the dental professional.[13]

Question 1

Which of the following statements, if any, are correct?

A. The layperson who is undereducated in dental health readily accepts suggested changes in preventive programs that are directed to better oral health.

B. Perceived negative dentist behaviors may deter patients from seeking necessary dental treatment.

C. Primarily, human motivation can be explained and understood as being instinctual in nature.

D. In general, providing patients with knowledge is sufficient to facilitate behavior change.

Educational Approach to Behavior Change

The Learning Process

Because information transmittal involves learning, it is desirable to turn to the teaching profession for how information is best imparted to ensure long-term retention. Ensuring that a client adheres with a home care regimen can be the most difficult part of therapy.[14] According to Bloom's taxonomy of educational objectives, a hierarchy of six levels of learning attainment progresses from a complete lack of information to goal attainment (see Figure 16-1).[15] These successive levels are knowledge, comprehension, application, analysis, synthesis, and evaluation. Most teaching today is at the lowest knowledge stage. After mastery of this stage, the learner can only define, repeat, or name facts; it is only partial learning at best. Possible verbs used in stating cognitive outcomes of teaching programs starting with the knowledge level up to evaluation are listed in Figure 16-2. If material is only taught at the lower levels of the taxonomy, learning is incomplete.

The implication of partial learning is apparent when applied to plaque control methods. The average person knows and comprehends that brushing and flossing clean the teeth. They can even demonstrate that they can brush their teeth in some fashion. But how many people can evaluate the effectiveness of their efforts? How many can analyze where problems lie, and how many can propose innovations to their personal oral hygiene program that might make it more effective?

Teaching at the higher levels of Bloom's taxonomy is necessary to accomplish this type of learning. At each cognitive level the teaching should feature an explanation of the subject, followed in sequence by demonstration, applications, feedback, and reinforcement. The use of these sequential steps in all teaching helps to ensure a mastery of the desired topic or skill. In moving from one level of complexity to the next, the learner is exposed to an organized continuum of interrelated facts. Even after successfully mastering all levels of Bloom's hierarchy, however, it is very possible that a skill or subject area learned in an academic or clinical environment is not applied at home, in a more informal environment on a routine basis. Day-to-day application occurs only after an individual has learned sufficient information to determine that a specific benefit accrues to him or her from its use and thus has become motivated. Education involves learning; practical application involves self-motivation.[16] At this point, the knowledge needs to be incorporated into the client's existing value systems.

Incorporating Knowledge into Value Systems

Personal belief systems and values strongly influence an individual's behavior. Values are developed through the application of knowledge, which thus requires that an individual has enough facts to develop concepts and then a sufficient number of concepts to develop a value.[17] This concept is portrayed graphically in Figure 16-3. The base of the pyramid consists of facts, which are the building blocks of all learning. Sometimes great voids or even misinformation occur in this body of information. Yet, regardless of its completeness or accuracy, this substratum of information is where concepts are formed by use of one's reasoning power. Concepts, less numerous than facts, represent the organization and classification of facts into meaningful personal habits or patterns. The greater number of correct facts arising from different inputs, the greater the possibility of developing correct concepts. On top of these supporting facts and concepts rest values—beliefs and bodies of knowledge important to the individual.

These values are only as strong as the supporting information. It should be noted that not all dental values are positive. For example, for individuals living under impoverished conditions who do not appreciate the value of teeth from a health or social viewpoint or where the loss of teeth is considered as normal, facts, concepts, and values are often negative. These negative perceptions can motivate nonparticipation in dental programs.[18] It has also been noted that a client's relationship with the dental professional influences their anxiety level and resulting compliance with suggested oral health-care practices.[8] The dental professional must carefully consider the possible myriad facts and concepts that can make up this pyramid when trying to change a client's value system—a value system that is valid to only the individual client.

Values are not neutral but are held with personal feeling.[17] When they are challenged, they frequently generate an emotional, defensive response. Making changes in one's behavior is often very difficult and involves dealing with conflict. Hayakawa[19] expands this idea when he writes, "the process of learning, which is also the process of growth, is essentially a means of resolving conflicts . . . a conflict must always be present before learning can occur . . . conflict then is a necessary accompaniment of personality development, and the progressive assimilation of disturbing stimuli is the only practical means by which a stable organization can be obtained. Without conflict, no learning results."

Therefore it is necessary that the dental professional understand that because of the client's value system, resistance is normal and permanent changes in some forms of behavior are difficult to achieve. This same resistance is met from the client in the dental office, or from many in the community, when new health programs are proposed. For example, sugar discipline is difficult to instill because of concepts and values shaped early in childhood by the media and candy-laden shelves in the supermarkets; water fluoridation efforts have failed in some areas because of a barrage of misinformation and distorted facts, leading to strongly held values by those voting against fluoridation. Such resistance to change should not prevent the continual education and pressure for more effective oral disease control programs. In this quest, however, we must be careful how we approach the value systems of our clients or of

the community. We must respect the fact that others have their own value systems tied to their own set of expectations that may be quite different from ours.

Can human values be changed? The answer is yes, but this statement must be qualified. Values are slow to form and slow to change.[17] Even if the factual information is complete and adequate, time is required for concepts to evolve and mature; even more time is required before other additional facts and concepts are acquired to support a new value. Stated another way, a dental professional should not expect dramatic and immediate changes in client behavior as a result of only one or two counseling sessions. Thus to attain a behavioral change, a health education program is often confronted with the imposing requirement to modify or reconstruct completely the facts and concepts making up an existing value structure. No wonder so many health education programs fail. A good example is smoking behavior. Virtually all smokers have enough facts necessary to develop the concept that the behavior, cigarette smoking, is harmful. Yet many have not accepted this concept into their own value systems to the point of behavioral change, namely of not smoking. It is also seen in caries and periodontal disease control programs in which clients are unwilling to conduct lifelong programs of plaque control.

Figure 16-1 Bloom's Taxonomy of Educational Objectives.

Figure 16-2 Some possible verbs for use in stating cognitive outcomes. (Courtesy of Marybelle Savage.)

Figure 16-3 The interrelationship between values, concepts, and facts using oral health as a positive end value. Learning on all three levels helps individuals discern facts, make sense of them and, finally, to live by the meaning they perceive.

Question 2

Which of the following statements, if any, are correct?

A. Different groups of individuals presented with the same facts can develop different concepts.

B. Once facts and concepts are a part of an individual's life, values fall in place.

C. Most education results in the learner being able to attain the cognitive level of evaluation on Bloom's hierarchy.

D. The dental professional must acknowledge that values are slow to change and that resistance to changing values is normal.

Health Promotion Approach to Behavior Change

An alternative way of examining human motivation draws from the health education and health promotion literature. Although many definitions for health promotion exist, one of the more common ones states that health promotion is "any combination of health education and related organizational, political, and economic interventions designed to facilitate behavioral and environmental changes conducive to health."[20]

Central to all health promotion definitions is the concept of health behavior. Positive informed changes in health behavior are usually the ultimate goals of health promotion activities. Health behavior refers to "those personal attributes such as beliefs, expectations, motives, values, perceptions, and other cognitive elements; personality characteristics, including affective and emotional states and traits; and overt behavioral patterns, actions, and habits that relate to health maintenance, to health restoration, and to health improvement."[21] Specific to the field of dental care, health behaviors include getting regular dental check-ups, regular brushing and flossing, and reducing sugar intake.

Identifying the personal attributes most significant for certain health behaviors is critical for the development of successful interventions. For example, to increase the number of individuals who obtain regular and timely dental check-ups, dental health-care providers need to be aware of the personal attributes, or the predisposing factors, that contribute to people getting regular check-ups. This information can come from two sources: empirical data and health promotion theories. Empirical data can provide us with data obtained through epidemiological studies. Health-promotion theories can explain and predict why people behave the way they do.

Health Behavior Change Theories

Three prominent theories that will be discussed in the following sections include the Health Belief Model (HBM),[22] Social Cognitive Theory (SCT),[12] and the Transtheoretical Model (TTM).[23] These theories share the central assumptions that people are capable of forethought, planning, and rational decision making. People are goal oriented and self-regulating beings. All of these theories explicitly or implicitly recognize that people experience their decision making and self-regulation as part of a dynamic social-learning process.[12] While the HBM mainly predicts behavior, SCT and TTM address the processes of behavior change and allow for the identification of appropriate strategies to facilitate behavior change.

Individuals' motivation is central to most health behavior theories for either prediction or behavior change purposes. As will be noted below, most of these theories include the assumption that individuals are interested in planning and controlling their actions and are not passive "lumps of clay."

Health Belief Model

The Health Belief Model (HBM) is a commonly used theory to predict individual's behavior regarding preventive health care. Originally developed in the 1950s to explain widespread failure of people to participate in interventions to prevent tuberculosis,[24] HBM has been extended to apply to people's responses to symptoms and to their compliance with medical regimens.[22]

HBM includes five main components: *perceived susceptibility*, *perceived severity*, *perceived benefits*, *perceived barriers*, and *self-efficacy*. *Perceived susceptibility* refers to a person's subjective perception of the risk of becoming sick, while perceived severity refers to the person's feelings of the seriousness of becoming sick or leaving the illness untreated (both medical and clinical and social consequences). The

combination of susceptibility and severity is often labeled *perceived threat*. Before a person will take action and change behavior, the perceived threat needs to high. For example, before a person will consider flossing every day, he or she needs to believe that not flossing will lead to periodontal disease *and* that periodontal disease can have serious negative consequences for him or her.

When an individual has a high *perceived threat*, that person will analyze the *perceived benefits and barriers* of performing a certain behavior. *Perceived benefits* refer to the beliefs regarding the effectiveness of the available actions in reducing the disease threat. Thus, a person who believes that flossing every day will reduce the risk of developing periodontal disease will be more likely to perform this behavior than a person who does not have this belief. Contrary to perceived benefits, *perceived barriers* (e.g. painful, difficult, upsetting, inconvenient, time-consuming) can act as impediments to engaging in the health behavior. Thus a sort of cost-benefit analysis occurs when individuals decide whether the perceived benefits override the perceived barriers. If they do, those individuals will most likely perform the behavior. If the barriers outweigh the benefits, the behavior will probably not occur. Thus, even if a person feels a high threat for periodontal disease, he or she may not change his current behavior to daily flossing when the perceived barriers for flossing every day (e.g., time-consuming, painful, inconvenient) are stronger than the benefits.

Determining their client's perceived threat, perceived benefits, and barriers can be very helpful for a dental professional who wants to encourage a client to change behaviors. By asking the right type of questions, all health professionals can obtain this information. The dental professional can then address any perceived misconceptions and, consequently, facilitate behavior change.

Social Cognitive Theory

HBM, is a theory that focuses on psychosocial factors within the individual that can affect behavior change. Social Cognitive Theory (formerly known as Social Learning Theory) includes both individual as well as environmental influences. Thus, SCT explains human behavior in terms of a triadic, and reciprocal model, in which personal factors, environmental influences, and behavior interact continuously.[12] In addition to explaining why a person behaves in a certain manner, SCT can facilitate behavior change by providing specific learning strategies (e.g., modeling). For a more detailed description of the various SCT constructs, please see *Health Behavior and Education* (1997) by Glanz et al.

Reciprocal determinism is the underlying assumption of SCT. It explains that behavior, environmental factors, and individual influences are continuously interacting and each one affects the other. For example, a person who has high dental anxiety (a personal factor) and receives no reinforcement to see a dentist regularly (environmental factor) is not likely to go for preventive dental check ups. However, if this person receives positive feedback for seeing a dentist (environmental factor), and has a role model who visits a dentist every 6 months (environmental factor), her level of dental anxiety may actually decrease. As a result, she may be more likely to go see a dentist. SCT underscores the importance of avoiding simplistic "single direction of change" thinking. Behaviors do not occur in isolation and interventions should focus both on the individual and the environment.[10]

Modeling, one of the key learning strategies proposed by SCT, has been successfully used with dental clients to decrease dental fear and anxiety. A study performed by Bernstein (1982) looked at the effectiveness of different strategies to reduce fear of dentistry in adult clients who had avoided dental treatment for from 1 to 10 years. The strategies studies included participant modeling (a SCT strategy), symbolic modeling, and graduated exposure. Results suggested that even though the strategies were equally effective for the short-term, participant modeling was most effective for reducing fear for long-term period.[25]

Stages of Change Model

Oral health care providers have sought to understand and create those conditions that would lead to beneficial and helpful behavior changes for their clients. The Transtheoretical Model (TTM), developed by two psychologists, Drs. Prochaska and DiClemente[23] is a powerful and widely accepted model for understanding how and why people change, either on their own or with the assistance of others. The model is based on the individual's state of readiness or willingness to change, which may fluctuate from one time or situation to another.

The Transtheoretical Model is composed of three main constructs, one of which is the Stages of Change. The stages of change construct describes a series of five progressive stages through which individuals pass in the course of changing a behavior. The "wheel of change" derived from the Prochaska-DiClemente model (Figure 16-4) reflects the reality that in almost any change process, it is possible for a person to go around the "wheel" or relapse several times before achieving a stable change. For example, an individual who is willing and ready to start flossing once a day may begin this practice receiving information from his dentist, then relapse after several weeks, and then start the daily flossing routine again after another dental visit. Thus, according to the Stages of Change, relapses or slips to previous behaviors is normal and a realistic occurrence.

The five stages of change as linked to the development of health behaviors, including optimal oral hygiene habits, are described below. Daily flossing will be used as the specific example to illustrate this theory.

Precontemplation—Individuals in this stage are not aware of the positive consequences of daily flossing and have no conscious intentions of starting to floss daily within the next 6 months.

Contemplation—Individuals in the Contemplation stage are aware of the positive consequences of changing their current behaviors and plan to start flossing within the next 6 months (near future).

Preparation—Individuals in this stage are making concrete steps to adopting oral hygiene practices. They may have bought new floss or scheduled dental appointments.

Action—Individuals in the Action stage are actually flossing every day but have done so less than 6 months.

Maintenance—Individuals have flossed daily for over 6 months.

As discussed earlier, at any time an individual may relapse to a previous stage, thus an individual in the Action stage could relapse to the Preparation or even the Contemplation stage.

Corresponding to each stage are appropriate counseling techniques. Thus, by understanding the specific stages of behavior change and the corresponding emotions that may accompany them, oral health care providers can better understand the actions, or inactions, of their clients. With a better understanding, they will be more able to meet the immediate needs of their clients and counsel them appropriately. For example, precontemplators are not ready to change their behavior and they do not want to hear threatening messages. They have a very strong preponderance of "pros" about their current behavior and have a poor acknowledgement of the "cons." These individuals should be given balanced information about the current behavior, handled with kindness and care, and left alone. It is not reasonable to blame these individuals for being unmotivated to change their current oral hygiene practices.

Individuals who are in the contemplation stage tend to have a balance between the positive and negative feelings about their current behaviors. They are often still ambivalent about changing. Even when contemplators move into the preparation stage, when the strengths of the pros for changing behaviors have increased over the cons, they may still have positive feelings about their current behaviors that are strong.

The Stages of Change model indicates that the goal of the oral health care provider is not necessarily one of action. Because many individuals tend to be in the precontemplation or contemplation stages, it is very worthwhile to try to "move" these individuals to the next stage.

Figure 16-4 Prochaska and DiClemente's six stages of change (as modified by Christen, et al., 1994).

Question 3

Which of the following statements, if any, are correct?

A. Affecting knowledge is central to most health behavior theories.

B. The stages of change model suggest that there is a one-way, linear progression through five stages: precontemplation, contemplation, preparation, action, and maintenance.

C. SCT explains that behavior is reciprocally affected by personal and environmental factors.

D. All health promotion theories can predict health behavior and address processes of change.

Approaches for Different Levels of Client Motivation and Adherence

Plaque-control measures are difficult to accomplish and require considerable time, skill, and perseverance. In fact, current measures of oral hygiene requiring fastidious removal of all supragingival plaque may be beyond the average individual.[26] Thus a blend of education, motivation, and psychomotor skills are necessary to ensure good personal oral hygiene measures. No good evidence supports the fact that mass education alters individual behavior. Instead, individualized approaches are usually necessary, and even these are not always successful.

For a dentist entrusted with the preventive care of a moderately motivated individual, the recall program should be at sufficiently frequent intervals to compensate for lapses in client self-care routines. At the same time, the educational and motivation phases of client education should be emphasized to improve the participation and effectiveness in self-care programs. In this way, the dental professional assumes the task of caring for the client to the extent that compensates for the shortcomings of the client while preparing the client to adopt a greater role in maintaining personal oral health status. Ultimately, it is the client who must assume as much responsibility for self-care as possible and to seek out the dental professional for evaluation (examination) and reinforcement when deficiencies are noted or suspected.

Once an individual becomes sufficiently motivated and changes his or her behavior, the next important issue is adherence. Adherence implies that people choose freely to undertake behavioral plans, have input to them, and have collaborative involvement in adjusting their plans.[27] What makes an individual continue to follow dental recommendations and adhere to practice oral hygiene? Although there is a paucity of literature in the area of adherence to oral hygiene practices, literature related to other health behaviors can provide some information.

Although there are some common factors, potential determinants of adherence are not consistently detected. A clinically-oriented framework by Meichenbaum and Turk (1987) may be useful for oral health-care professionals. This framework divides factors related to adherence into characteristics of the individual (e.g., knowledge, attitudes, beliefs, expectancies about health, treatment), disease (e.g., complexity, duration, side effects), the treatment regimen (e.g., complexity, duration, type, cost), relationship to the health-care-provider clinic staff (e.g., client-provider staff), and clinic organization (e.g., staff enthusiasm).[28]

This framework can be used to provide order to a list of determinants and can help identify categories of potential moderators of adherence to treatments. Thus, to improve adherence to specific regimen, oral health care professionals can use this framework to examine their clients and clinics to determine potential areas of improvement.

Selecting Methods of Influencing Behavior Change

Client-Provider Relationship/Communication Styles

Determining the most appropriate type of client-physician relationship is extremely important for the practicing dentist. While some health-care professionals prefer to be the expert and authority, others understand that not all clients respond well to this type

of relationship. When a client does not respond well to the type of relationship practiced by the health care provider, important information may be lost. On the other hand, the positive benefits derived from good doctor-client communication include both immediate effects during the visit and long term effects following the visits and involve compliance with prescribed regimen, pain experience, physiologic changes, speed of recovery, and functional state.[29]

There are four archetypal forms of the doctor-client relationship: paternalism, consumerism, mutuality, and default. Paternalism is regarded as the more traditional and probably the most common form of the doctor-client relationship.[30] The paternalistic model provides a social control function in that the health care provider is seen as the expert and dominant, controlling figure, while the client is passive and free from social responsibilities. The physician maintains emotional detachment and acts only in his or her sphere of expertise. Although some may view this type relationship as negative, some clients may actually draw comfort and support from a doctor-father figure. The supportive nature of paternalism seems to be very important when a client is in need of extensive services and therefore is vulnerable. In times of emergency, when correct decisions must be made quickly to avoid life-threatening events, the health-care provider must take control and the paternalistic form is usually necessary.

The consumerism prototype is the opposite of paternalism. In this type of relationship, the power relationship between the client and the physician are reversed: the client or the consumer has more power or control than the physician.[10] Especially when trying to "sell" prevention to the client, the physician's role is to convince the client of the necessity of non-curative services such as regular dental checkups or daily brushing. Several authors have defined consumerism as a client challenge to unilateral decision making by physicians when reaching closure on diagnosis and treatment plans.[31] In this prototype, the health provider and client co-jointly explore the various options and planning objectives. This type of relationship appeals to higher order means of acceptance, including reasoning, nonthreatening persuasion, and rewards. The healthcare provider typically talks less, listens more, questions, reacts, and synthesizes when necessary.[32]

Compared to consumerism, the mutuality prototype offers a more moderate alternative. The client still has a great deal of power but so does the physician. In mutuality, both individuals (client and physician) bring recognized strengths and resources to the relationship. In this model, the client recognizes his or her role as part of a joint venture while the physician understands the centrality of the client in his or her care.[10]

In some cases, the client and physician remain at odds and cannot negotiate a change in the relationship due to poor fit. In this case, a total lack of control exists and the default prototype occurs.[29] Although the client and physician may still see each other during regular visits, the client may fail to make a commitment to prescribed regimens and the physician may cease to be engaged or try to educate the client.

Motivational Interviewing in the Change Process

Motivational interviewing, introduced by Miller and Rollnick (1991) is a particular

method to help people recognize and do something about their present and potential behavioral problems.[33] It is particularly useful for those clients who are reluctant to change and ambivalent about changing. This technique attempts to help resolve ambivalence and to move the individual along the path to change. Ambivalence is a state of mind in which a person has coexisting but conflicting feelings about some issue. This "I want to but I don't want to" dilemma is at the heart of the problem of all change. Ambivalence is a type of conflict within an individual that has the potential for keeping people "stuck" and creating stress. Ambivalent smokers who have been told by their periodontist that tobacco use can cause periodontal disease, might readily acknowledge that their oral health is endangered, yet may feel equally concerned about their ability to cope with stressful situations without smoking.

Oral-health-care providers must understand that ambivalence is not merely a "bad sign." It should be regarded as normal, acceptable, common, and understandable part of the change process. What is highly valued by some (e.g., having good oral health) will be of little importance to others.

Five broad clinical principles underlie motivational interviewing.[33] These principles emphasize that the clinician should: 1) express empathy (through skillful reflective listening, the clinician seeks to understand and accept the client's feelings and perspective without judging, criticizing or blaming, and realizes that ambivalence is normal); 2) develop discrepancy (help the client understand the discrepancy between their present behavior and their ability to reach their important goals; clients should discover and present their own arguments for and against change); 3) avoid argumentation (a gently persuasive/soft confrontation approach should be used—one that asserts that clients have the freedom to do as they please; avoid sending the message that "I'm the expert and I'm going to tell you how to run your life"; do not accuse clients of being "in denial" or label their behavior); 4) roll with resistance (invite the client to consider new information and offer new perspectives, without being imposing); 5) support self-efficacy (it is essential to support the client's self-esteem and their general self-regard; the client is responsible for choosing and carrying out personal change and the overall message is of hope and faith to the client; "You can do it. You can succeed.").

The FRAMES Brief Counseling Elements

Miller and Rollnick[34] have described six practical counseling elements that are active ingredients in effective and brief counseling interventions. They are summarized in the acronym "FRAMES."

- Feedback—The client is given feedback of their current status. The importance of conducting a thorough assessment provides the client an opportunity to reflect in detail upon their situation.
- Responsibility—There is an emphasis on the individual's personal responsibility for change. "It's up to you to decide what to do with this information. Nobody can decide for you, and no one can change your habit patterns if you don't want to change."
- Advice—Simple, clear advice to the client to make a change in their lifestyle is given.
- Menu—By offering clients a menu of alternative strategies for changing their problem behavior, the clinician provides a range of options, which allows clients to

select strategies that match their particular needs and situations.

- Empathy—Understand another's meaning through the use of reflective listening, whether you have had similar experiences yourself. Use of warmth, respect, supportiveness, caring, concern, sympathetic understanding, commitment, and active interest to convey this element.
- Self-efficacy—Reinforcing the client's hope or optimism in their ability to make changes promotes self-efficacy. Remember that your belief in the client's ability to change is often a significant determinant of outcomes.

Basic Philosophy

A basic philosophy of prevention is itself a value. One basic philosophy concerning preventive dentistry is that clients deserve to know the cause of their dental diseases and how they can prevent them. This is a responsibility for the health educator. Once armed with the knowledge, however, the client reserves the right to remain sick. This is a problem of self-motivation. Clients are ultimately responsible for their own dental health. In the final analysis, prevention is a shared responsibility between the practitioner and the client.

Summary

The maintenance of good oral health requires a partnership between the dental professional and the patient. No preventive program can be a success unless the patient participates in a home self-care program to supplement office care programs, with the level of success being proportionate to the amount of participation. Maximum participation can be expected when the patient knows what to do, how to do it, and above all has the motivation to adhere to recommended procedures. Educational strategies can be used to teach facts and skills, but these are useless without motivation. Motivation can be initiated by an individual based on some need or desire, or it can be facilitated by persuasion from external sources. With or without motivation, learning is best achieved in sequential steps, as described by Bloom's hierarchy of cognitive levels. As an individual accumulates facts, the facts merge into concepts and ultimately into values, which in turn engender motivation. At times motivation provides the drive to alter lifestyle to attain habit patterns necessary to maintain good oral health. The dental professional can exert a direct or indirect influence on such a change by providing appropriate behavior modeling, by taking a more active role as an authoritarian, or by participating as a nonauthoritarian in developing a program of planned change with the patient. All health education requires learning, but the successful application of all health knowledge requires motivation.

Answers and Explanations

1. B—correct.

A—incorrect. The average layperson does not accept change without considerable persuasion.

C—incorrect. Human motivation is complex in nature and best described as the interaction between the environment, personal, and behavioral factors.

D—incorrect. Knowledge is rarely sufficient to change behavior.

2. A, D—correct.

B—incorrect. Facts and concepts represent unorganized and organized thoughts, respectively; values represent the acceptance and personal application of facts and concepts.

C—incorrect. Most education is directed to the initial level—facts; very little learning ends up at the evaluation level.

3. C—correct.

A—incorrect. Most health-behavior theories attempt to explain or predict behavior.

B—incorrect. The stages of change model suggest that behavior change does not typically follow a linear progression but rather is cyclical as an individual experiences relapse and adopts new behaviors.

D—incorrect. Health-promotion theories *attempt* to explain or predict behavior with varying degrees of accurateness.

Self-Evaluation Questions

1. Health promotion can be defined as _____.

2. An individual, through reasoning, organizes facts into _____; which in turn are the basis for a(n) _____.

3. The central assumption underlying health promotion theories is _____.

4. The five main concepts of the Health Belief Model include: _____, _____, _____, _____, _____.

5. The six cognitive levels of Bloom's hierarchy of learning are knowledge, _____, _____, _____, _____, _____, and _____.

6. The one main difference between HBM and Social Cognitive Theory is _____.

7. _____ is the underlying assumption of SCT.

8. _____ implies that an individual chooses freely to undertake behavioral plans, have input to them, and has a collaborative involvement in modifying the plan.

9. In the dentist-patient partnership, it is the _____ who must assume responsibility for home care programs, whereas the _____ must assume responsibility of identifying and correcting deficiencies that occur in a home care program.

10. In the development of optimal oral hygiene habits, patients encounter five progressive stages of change. They are: _____, _____, _____, _____, and _____.

11. In the process of applying motivational interviewing, the clinician should apply five principles, which are: _____, _____, _____, _____, and _____.

12. The process whereby the clinician seeks to understand and accept the patient's feelings and perspectives without judgment, criticizing, or blaming is called: _____.

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Chapter 16. Understanding Human Motivation for Behavior Change - *Mary Kaye Sawyer-Morse Alexandra Evans*

Objectives

At the end of this chapter it will be possible to

1. Define motivation.

2. List reasons why individuals may not be motivated to receive regular oral care.

3. Describe two different approaches to motivate individuals to change behavior.

4. Describe elements of three common behavioral health promotion theories.

5. Explain the importance of appropriate health provider communication.

6. Describe four common client-provider communication styles.

7. Describe motivational interviewing and FRAMES.

Introduction

The mouth represents an area of the body of special importance and value. According to Horowitz and coworkers,[1] the mouth is associated with the development of (1) a healthy personality, (2) perceptions, and (3) the overall experience of pleasure. Many areas of the mouth, especially the gingival tissues, are easily accessible for self-diagnosis and primary preventive treatment. Individuals can easily detect gums that are red or bleeding. In addition, the tongue, with its highly developed neurosensory feedback system, can be useful in helping people to assess their own plaque levels and resultant need for improved oral hygiene behavior. As a result, dental professionals should devise strategies for motivating oral self-care behavior by teaching clients how to recognize their own signs of dental distress or neglect.

In this chapter the interrelationship of motivation, education, and behavioral modification are considered—all with the objective of helping dental professionals develop more effective interpersonal skills, thereby becoming more effective health educators and counselors.[2] The task of educating the client can be greatly simplified by a knowledge of and the application of a few basic constructs of educational and health promotion and human motivation. These same constructs apply equally to either private or public health practices.

The Problem: Oral health is an essential component of health throughout life. Poor oral health and untreated oral diseases can have a significant effect on quality of life. The mouth is the entry point for food and the beginning of the gastrointestinal tract. The ability to chew and swallow is a critical function required to obtain essential nutrients for the body—the building blocks of good health.[3] However, millions of individuals in the United States have dental caries and periodontal disease, resulting in unnecessary pain, difficulty in chewing, swallowing, and speaking, increased medical costs, loss of self-esteem, decreased economic productivity through lost work and school days, and, in extreme cases, death.[4] The Healthy People 2010 document recognizes the importance of oral health and includes 17 specific objectives related to the overall goal: To prevent and control oral and craniofacial diseases, conditions, and injuries and improve access to related services.[5]

Regular and timely dental visits provide an opportunity for the early diagnosis, prevention, and timely treatment of oral diseases and conditions, as well as for the assessment of self-care practices. However, approximately 66% of people in the United States do not see a dentist regularly,[5] and among specific subpopulations, such as certain ethnic groups or low-income groups, the proportion not receiving regular care is even higher.[6] For example, the Medical Expenditure Panel Survey in 1996 indicated that 44% of the total population visited a dentist in the past year, while 50% of non-Hispanic whites, 30% of Hispanics, and 27% of non-Hispanic blacks had a visit. In addition, 55% of those individuals with some college education had a past-year visit compared to 24% of those with less than a high school education.[7]

The reasons individuals may not be motivated to seek regular and timely care include: high cost of dental care, lack of dental insurance, lack of providers from underserved racial and ethnic groups, fear of dental visits, habitual personal neglect, lack of

knowledge, limited oral-health literacy, and negative feedback or unflattering statements about dentistry received from friends or relatives.[5] Other factors that have contributed to people losing confidence in dentists include prior negative experienced with dentists (poorly executed or ineffective treatment and unnecessary or questionable extractions or other treatments), dental treatment that did not last long enough, and lack of access to appropriate dental care. Previous painful experiences and perceived negative dentist behaviors (e.g. arrogance, sarcasm, or inconsideration) appear to be especially important to the anxious individual who is mentally preparing for dental treatment.[8] See Table 16-1. Most of these barriers can be overcome by effective client education and motivation programs and more effective interpersonal communication by the dental professional.

Dental Education and Motivational Programs

In previous chapters, it is stated that primary preventive dentistry can be effectively implemented by using the following five actions: 1) plaque control, 2) reduction of sugar in the diet, 3) fluoride therapy, 4) use of pit-and-fissure sealants, and 5) client education. The successful use of any of these actions requires effective relations between dental professionals and clients to achieve and maintain a maximum level of oral health. Three major enabl-ing factors are necessary to perform the above listed actions—appropriate skill-based education, client self-motivation, and appropriate psychomotor skills.

For any preventive dentistry program to succeed, information about what needs to be done and how it is to be accomplished must be available to both the dental professional and the client. For the client, this information (and sometimes misinformation) is often learned through school-based health programs; the dentist, media, and advertising; and from peers, friends, neighbors, or relatives. On the other hand, dental professionals learn preventive dentistry as part of the curriculum in dental and dental hygiene schools, through reading professional dental journals, by attending professional meetings and conferences, and through participation in continuing education programs. In some cases, the gap between the information possessed by the clients and the dental professionals is great. This gap in knowledge poses a problem because people tend to seek what they already believe and avoid exposure to anything that mandates changes.

In general, the personality characteristics of dentists indicate that technical proficiency and attention to detail may be more common than strong interpersonal communication skills.[9] For this reason dental professionals may need to cultivate specific knowledge and expertise in the area of human behavior and motivation techniques. Because the skills to accomplish these tasks are not commonly taught in dental school, many dental professionals do not have adequate skills to provide information to clients appropriately.

In addition, many dental professionals are taught that providing knowledge to a client is sufficient to change the client's behavior. However, extensive research indicates that information by itself is necessary, but not sufficient. Human behavior is a product of the interaction of multiple factors such as attitude, self-efficacy, knowledge, or perceived risk and benefits. Any one factor can be powerful but none acts independently.[10] Therefore, not only do many dental professionals need to acquire or

strengthen skills on how to provide information to clients, they also need to learn how to appropriately motivate clients so that behavior change can occur. Many health behaviors theories explain health behavior and can guide effective behavior change. For further description of three common health behavior theories, see section Health Promotion Approach to Behavior Change.

Motivation

What is motivation? Everyone is motivated to action or inaction. To not be motivated is to be dead. Some argue that humans are primarily instinctual in nature. This argument is difficult to accept because of the varied nature of human behavior. If the "instinct theory" was valid, all humans would show a uniformity of behavior across all cultures.[11] This, of course, is not the case. Others believe that behavior is learned and that our environment determines our actions. Indeed, no one should downplay the importance of environmental forces on human behavior. Motivation may be described as the interaction between the environment, personal and behavioral factors.[12] Despite the fact that human behavior is highly variable and at times unpredictable, one thing is certain: Individuals' performances or behaviors are based on the degree to which they are motivated. Motivation makes the difference.

Human motivation is complex. It is based on a blending of expectations, ideas, feelings, desires, hopes, attitudes, values, and other factors that initiate, maintain, and regulate behavior toward achieving a given goal or outcome. Other factors, such as previous adverse experiences, educational insufficiency, nonacceptance by peers, a poor self-image, and impoverished socioeconomic circumstances can significantly influence behavior. Motivation factors can change with the passage of time. Humans are strongly goal-oriented and can demonstrate a tremendous drive to achieve their personal ambitions. For some, however, a significant part of the pleasure is derived from working toward a goal; after they have "arrived," their pleasure is somewhat diminished. For these individuals, getting there is not only half the fun, it is possibly all the fun. For example, some individuals periodically become intensely motivated to upgrade their oral health status. Appointments are made with the dentist, all restorative work is completed, preventive programs are developed with a great amount of client participation until all dental care has been completed, at which time the individual appears to lose interest until another sudden flurry of interest may occur at a later date.

Motivation then is seen not as a personality problem or trait but rather as a state of readiness or eagerness to change. This readiness may fluctuate from one time or situation to another and can be influenced by the dental professional.[13]

Question 1

Which of the following statements, if any, are correct?

A. The layperson who is undereducated in dental health readily accepts suggested changes in preventive programs that are directed to better oral health.

B. Perceived negative dentist behaviors may deter patients from seeking necessary dental treatment.

C. Primarily, human motivation can be explained and understood as being instinctual in nature.

D. In general, providing patients with knowledge is sufficient to facilitate behavior change.

Educational Approach to Behavior Change

The Learning Process

Because information transmittal involves learning, it is desirable to turn to the teaching profession for how information is best imparted to ensure long-term retention. Ensuring that a client adheres with a home care regimen can be the most difficult part of therapy.[14] According to Bloom's taxonomy of educational objectives, a hierarchy of six levels of learning attainment progresses from a complete lack of information to goal attainment (see Figure 16-1).[15] These successive levels are knowledge, comprehension, application, analysis, synthesis, and evaluation. Most teaching today is at the lowest knowledge stage. After mastery of this stage, the learner can only define, repeat, or name facts; it is only partial learning at best. Possible verbs used in stating cognitive outcomes of teaching programs starting with the knowledge level up to evaluation are listed in Figure 16-2. If material is only taught at the lower levels of the taxonomy, learning is incomplete.

The implication of partial learning is apparent when applied to plaque control methods. The average person knows and comprehends that brushing and flossing clean the teeth. They can even demonstrate that they can brush their teeth in some fashion. But how many people can evaluate the effectiveness of their efforts? How many can analyze where problems lie, and how many can propose innovations to their personal oral hygiene program that might make it more effective?

Teaching at the higher levels of Bloom's taxonomy is necessary to accomplish this type of learning. At each cognitive level the teaching should feature an explanation of the subject, followed in sequence by demonstration, applications, feedback, and reinforcement. The use of these sequential steps in all teaching helps to ensure a mastery of the desired topic or skill. In moving from one level of complexity to the next, the learner is exposed to an organized continuum of interrelated facts. Even after successfully mastering all levels of Bloom's hierarchy, however, it is very possible that a skill or subject area learned in an academic or clinical environment is not applied at home, in a more informal environment on a routine basis. Day-to-day application occurs only after an individual has learned sufficient information to determine that a specific benefit accrues to him or her from its use and thus has become motivated. Education involves learning; practical application involves self-motivation.[16] At this point, the knowledge needs to be incorporated into the client's existing value systems.

Incorporating Knowledge into Value Systems

Personal belief systems and values strongly influence an individual's behavior. Values are developed through the application of knowledge, which thus requires that an

individual has enough facts to develop concepts and then a sufficient number of concepts to develop a value.[17] This concept is portrayed graphically in Figure 16-3. The base of the pyramid consists of facts, which are the building blocks of all learning. Sometimes great voids or even misinformation occur in this body of information. Yet, regardless of its completeness or accuracy, this substratum of information is where concepts are formed by use of one's reasoning power. Concepts, less numerous than facts, represent the organization and classification of facts into meaningful personal habits or patterns. The greater number of correct facts arising from different inputs, the greater the possibility of developing correct concepts. On top of these supporting facts and concepts rest values—beliefs and bodies of knowledge important to the individual.

These values are only as strong as the supporting information. It should be noted that not all dental values are positive. For example, for individuals living under impoverished conditions who do not appreciate the value of teeth from a health or social viewpoint or where the loss of teeth is considered as normal, facts, concepts, and values are often negative. These negative perceptions can motivate nonparticipation in dental programs.[18] It has also been noted that a client's relationship with the dental professional influences their anxiety level and resulting compliance with suggested oral health-care practices.[8] The dental professional must carefully consider the possible myriad facts and concepts that can make up this pyramid when trying to change a client's value system—a value system that is valid to only the individual client.

Values are not neutral but are held with personal feeling.[17] When they are challenged, they frequently generate an emotional, defensive response. Making changes in one's behavior is often very difficult and involves dealing with conflict. Hayakawa[19] expands this idea when he writes, "the process of learning, which is also the process of growth, is essentially a means of resolving conflicts . . . a conflict must always be present before learning can occur . . . conflict then is a necessary accompaniment of personality development, and the progressive assimilation of disturbing stimuli is the only practical means by which a stable organization can be obtained. Without conflict, no learning results."

Therefore it is necessary that the dental professional understand that because of the client's value system, resistance is normal and permanent changes in some forms of behavior are difficult to achieve. This same resistance is met from the client in the dental office, or from many in the community, when new health programs are proposed. For example, sugar discipline is difficult to instill because of concepts and values shaped early in childhood by the media and candy-laden shelves in the supermarkets; water fluoridation efforts have failed in some areas because of a barrage of misinformation and distorted facts, leading to strongly held values by those voting against fluoridation. Such resistance to change should not prevent the continual education and pressure for more effective oral disease control programs. In this quest, however, we must be careful how we approach the value systems of our clients or of the community. We must respect the fact that others have their own value systems tied to their own set of expectations that may be quite different from ours.

Can human values be changed? The answer is yes, but this statement must be qualified. Values are slow to form and slow to change.[17] Even if the factual

information is complete and adequate, time is required for concepts to evolve and mature; even more time is required before other additional facts and concepts are acquired to support a new value. Stated another way, a dental professional should not expect dramatic and immediate changes in client behavior as a result of only one or two counseling sessions. Thus to attain a behavioral change, a health education program is often confronted with the imposing requirement to modify or reconstruct completely the facts and concepts making up an existing value structure. No wonder so many health education programs fail. A good example is smoking behavior. Virtually all smokers have enough facts necessary to develop the concept that the behavior, cigarette smoking, is harmful. Yet many have not accepted this concept into their own value systems to the point of behavioral change, namely of not smoking. It is also seen in caries and periodontal disease control programs in which clients are unwilling to conduct lifelong programs of plaque control.

Figure 16-1 Bloom's Taxonomy of Educational Objectives.

Figure 16-2 Some possible verbs for use in stating cognitive outcomes. (Courtesy of Marybelle Savage.)

Figure 16-3 The interrelationship between values, concepts, and facts using oral health as a positive end value. Learning on all three levels helps individuals discern facts, make sense of them and, finally, to live by the meaning they perceive.

Question 2

Which of the following statements, if any, are correct?

A. Different groups of individuals presented with the same facts can develop different concepts.

B. Once facts and concepts are a part of an individual's life, values fall in place.

C. Most education results in the learner being able to attain the cognitive level of evaluation on Bloom's hierarchy.

D. The dental professional must acknowledge that values are slow to change and that resistance to changing values is normal.

Health Promotion Approach to Behavior Change

An alternative way of examining human motivation draws from the health education and health promotion literature. Although many definitions for health promotion exist, one of the more common ones states that health promotion is "any combination of health education and related organizational, political, and economic interventions designed to facilitate behavioral and environmental changes conducive to health."[20]

Central to all health promotion definitions is the concept of health behavior. Positive informed changes in health behavior are usually the ultimate goals of health promotion activities. Health behavior refers to "those personal attributes such as beliefs, expectations, motives, values, perceptions, and other cognitive elements;

personality characteristics, including affective and emotional states and traits; and overt behavioral patterns, actions, and habits that relate to health maintenance, to health restoration, and to health improvement."[21] Specific to the field of dental care, health behaviors include getting regular dental check-ups, regular brushing and flossing, and reducing sugar intake.

Identifying the personal attributes most significant for certain health behaviors is critical for the development of successful interventions. For example, to increase the number of individuals who obtain regular and timely dental check-ups, dental health-care providers need to be aware of the personal attributes, or the predisposing factors, that contribute to people getting regular check-ups. This information can come from two sources: empirical data and health promotion theories. Empirical data can provide us with data obtained through epidemiological studies. Health-promotion theories can explain and predict why people behave the way they do.

Health Behavior Change Theories

Three prominent theories that will be discussed in the following sections include the Health Belief Model (HBM),[22] Social Cognitive Theory (SCT),[12] and the Transtheoretical Model (TTM).[23] These theories share the central assumptions that people are capable of forethought, planning, and rational decision making. People are goal oriented and self-regulating beings. All of these theories explicitly or implicitly recognize that people experience their decision making and self-regulation as part of a dynamic social-learning process.[12] While the HBM mainly predicts behavior, SCT and TTM address the processes of behavior change and allow for the identification of appropriate strategies to facilitate behavior change.

Individuals' motivation is central to most health behavior theories for either prediction or behavior change purposes. As will be noted below, most of these theories include the assumption that individuals are interested in planning and controlling their actions and are not passive "lumps of clay."

Health Belief Model

The Health Belief Model (HBM) is a commonly used theory to predict individual's behavior regarding preventive health care. Originally developed in the 1950s to explain widespread failure of people to participate in interventions to prevent tuberculosis,[24] HBM has been extended to apply to people's responses to symptoms and to their compliance with medical regimens.[22]

HBM includes five main components: *perceived susceptibility*, *perceived severity*, *perceived benefits*, *perceived barriers*, and *self-efficacy*. *Perceived susceptibility* refers to a person's subjective perception of the risk of becoming sick, while perceived severity refers to the person's feelings of the seriousness of becoming sick or leaving the illness untreated (both medical and clinical and social consequences). The combination of susceptibility and severity is often labeled *perceived threat*. Before a person will take action and change behavior, the perceived threat needs to high. For example, before a person will consider flossing every day, he or she needs to believe that not flossing will lead to periodontal disease *and* that periodontal disease can have serious negative consequences for him or her.

When an individual has a high *perceived threat*, that person will analyze the *perceived benefits and barriers* of performing a certain behavior. *Perceived benefits* refer to the beliefs regarding the effectiveness of the available actions in reducing the disease threat. Thus, a person who believes that flossing every day will reduce the risk of developing periodontal disease will be more likely to perform this behavior than a person who does not have this belief. Contrary to perceived benefits, *perceived barriers* (e.g. painful, difficult, upsetting, inconvenient, time-consuming) can act as impediments to engaging in the health behavior. Thus a sort of cost-benefit analysis occurs when individuals decide whether the perceived benefits override the perceived barriers. If they do, those individuals will most likely perform the behavior. If the barriers outweigh the benefits, the behavior will probably not occur. Thus, even if a person feels a high threat for periodontal disease, he or she may not change his current behavior to daily flossing when the perceived barriers for flossing every day (e.g., time-consuming, painful, inconvenient) are stronger than the benefits.

Determining their client's perceived threat, perceived benefits, and barriers can be very helpful for a dental professional who wants to encourage a client to change behaviors. By asking the right type of questions, all health professionals can obtain this information. The dental professional can then address any perceived misconceptions and, consequently, facilitate behavior change.

Social Cognitive Theory

HBM, is a theory that focuses on psychosocial factors within the individual that can affect behavior change. Social Cognitive Theory (formerly known as Social Learning Theory) includes both individual as well as environmental influences. Thus, SCT explains human behavior in terms of a triadic, and reciprocal model, in which personal factors, environmental influences, and behavior interact continuously.[12] In addition to explaining why a person behaves in a certain manner, SCT can facilitate behavior change by providing specific learning strategies (e.g., modeling). For a more detailed description of the various SCT constructs, please see *Health Behavior and Education* (1997) by Glanz et al.

Reciprocal determinism is the underlying assumption of SCT. It explains that behavior, environmental factors, and individual influences are continuously interacting and each one affects the other. For example, a person who has high dental anxiety (a personal factor) and receives no reinforcement to see a dentist regularly (environmental factor) is not likely to go for preventive dental check ups. However, if this person receives positive feedback for seeing a dentist (environmental factor), and has a role model who visits a dentist every 6 months (environmental factor), her level of dental anxiety may actually decrease. As a result, she may be more likely to go see a dentist. SCT underscores the importance of avoiding simplistic "single direction of change" thinking. Behaviors do not occur in isolation and interventions should focus both on the individual and the environment.[10]

Modeling, one of the key learning strategies proposed by SCT, has been successfully used with dental clients to decrease dental fear and anxiety. A study performed by Bernstein (1982) looked at the effectiveness of different strategies to reduce fear of dentistry in adult clients who had avoided dental treatment for from 1 to 10 years. The

strategies studies included participant modeling (a SCT strategy), symbolic modeling, and graduated exposure. Results suggested that even though the strategies were equally effective for the short-term, participant modeling was most effective for reducing fear for long-term period.[25]

Stages of Change Model

Oral health care providers have sought to understand and create those conditions that would lead to beneficial and helpful behavior changes for their clients. The Transtheoretical Model (TTM), developed by two psychologists, Drs. Prochaska and DiClemente[23] is a powerful and widely accepted model for understanding how and why people change, either on their own or with the assistance of others. The model is based on the individual's state of readiness or willingness to change, which may fluctuate from one time or situation to another.

The Transtheoretical Model is composed of three main constructs, one of which is the Stages of Change. The stages of change construct describes a series of five progressive stages through which individuals pass in the course of changing a behavior. The "wheel of change" derived from the Prochaska-DiClemente model (Figure 16-4) reflects the reality that in almost any change process, it is possible for a person to go around the "wheel" or relapse several times before achieving a stable change. For example, an individual who is willing and ready to start flossing once a day may begin this practice receiving information from his dentist, then relapse after several weeks, and then start the daily flossing routine again after another dental visit. Thus, according to the Stages of Change, relapses or slips to previous behaviors is normal and a realistic occurrence.

The five stages of change as linked to the development of health behaviors, including optimal oral hygiene habits, are described below. Daily flossing will be used as the specific example to illustrate this theory.

Precontemplation—Individuals in this stage are not aware of the positive consequences of daily flossing and have no conscious intentions of starting to floss daily within the next 6 months.

Contemplation—Individuals in the Contemplation stage are aware of the positive consequences of changing their current behaviors and plan to start flossing within the next 6 months (near future).

Preparation—Individuals in this stage are making concrete steps to adopting oral hygiene practices. They may have bought new floss or scheduled dental appointments.

Action—Individuals in the Action stage are actually flossing every day but have done so less than 6 months.

Maintenance—Individuals have flossed daily for over 6 months.

As discussed earlier, at any time an individual may relapse to a previous stage, thus an individual in the Action stage could relapse to the Preparation or even the

Contemplation stage.

Corresponding to each stage are appropriate counseling techniques. Thus, by understanding the specific stages of behavior change and the corresponding emotions that may accompany them, oral health care providers can better understand the actions, or inactions, of their clients. With a better understanding, they will be more able to meet the immediate needs of their clients and counsel them appropriately. For example, precontemplators are not ready to change their behavior and they do not want to hear threatening messages. They have a very strong preponderance of "pros" about their current behavior and have a poor acknowledgement of the "cons." These individuals should be given balanced information about the current behavior, handled with kindness and care, and left alone. It is not reasonable to blame these individuals for being unmotivated to change their current oral hygiene practices.

Individuals who are in the contemplation stage tend to have a balance between the positive and negative feelings about their current behaviors. They are often still ambivalent about changing. Even when contemplators move into the preparation stage, when the strengths of the pros for changing behaviors have increased over the cons, they may still have positive feelings about their current behaviors that are strong.

The Stages of Change model indicates that the goal of the oral health care provider is not necessarily one of action. Because many individuals tend to be in the precontemplation or contemplation stages, it is very worthwhile to try to "move" these individuals to the next stage.

Figure 16-4 Prochaska and DiClemente's six stages of change (as modified by Christen, et al., 1994).

Question 3

Which of the following statements, if any, are correct?

A. Affecting knowledge is central to most health behavior theories.

B. The stages of change model suggest that there is a one-way, linear progression through five stages: precontemplation, contemplation, preparation, action, and maintenance.

C. SCT explains that behavior is reciprocally affected by personal and environmental factors.

D. All health promotion theories can predict health behavior and address processes of change.

Approaches for Different Levels of Client Motivation and Adherence

Plaque-control measures are difficult to accomplish and require considerable time, skill, and perseverance. In fact, current measures of oral hygiene requiring fastidious removal of all supragingival plaque may be beyond the average individual.[26] Thus a blend of education, motivation, and psychomotor skills are necessary to ensure good

personal oral hygiene measures. No good evidence supports the fact that mass education alters individual behavior. Instead, individualized approaches are usually necessary, and even these are not always successful.

For a dentist entrusted with the preventive care of a moderately motivated individual, the recall program should be at sufficiently frequent intervals to compensate for lapses in client self-care routines. At the same time, the educational and motivation phases of client education should be emphasized to improve the participation and effectiveness in self-care programs. In this way, the dental professional assumes the task of caring for the client to the extent that compensates for the shortcomings of the client while preparing the client to adopt a greater role in maintaining personal oral health status. Ultimately, it is the client who must assume as much responsibility for self-care as possible and to seek out the dental professional for evaluation (examination) and reinforcement when deficiencies are noted or suspected.

Once an individual becomes sufficiently motivated and changes his or her behavior, the next important issue is adherence. Adherence implies that people choose freely to undertake behavioral plans, have input to them, and have collaborative involvement in adjusting their plans.[27] What makes an individual continue to follow dental recommendations and adhere to practice oral hygiene? Although there is a paucity of literature in the area of adherence to oral hygiene practices, literature related to other health behaviors can provide some information.

Although there are some common factors, potential determinants of adherence are not consistently detected. A clinically-oriented framework by Meichenbaum and Turk (1987) may be useful for oral health-care professionals. This framework divides factors related to adherence into characteristics of the individual (e.g., knowledge, attitudes, beliefs, expectancies about health, treatment), disease (e.g., complexity, duration, side effects), the treatment regimen (e.g., complexity, duration, type, cost), relationship to the health-care-provider clinic staff (e.g., client-provider staff), and clinic organization (e.g., staff enthusiasm).[28]

This framework can be used to provide order to a list of determinants and can help identify categories of potential moderators of adherence to treatments. Thus, to improve adherence to specific regimen, oral health care professionals can use this framework to examine their clients and clinics to determine potential areas of improvement.

Selecting Methods of Influencing Behavior Change

Client-Provider Relationship/Communication Styles

Determining the most appropriate type of client-physician relationship is extremely important for the practicing dentist. While some health-care professionals prefer to be the expert and authority, others understand that not all clients respond well to this type of relationship. When a client does not respond well to the type of relationship practiced by the health care provider, important information may be lost. On the other hand, the positive benefits derived from good doctor-client communication include both immediate effects during the visit and long term effects following the visits and involve compliance with prescribed regimen, pain experience, physiologic changes,

speed of recovery, and functional state.[29]

There are four archetypal forms of the doctor-client relationship: paternalism, consumerism, mutuality, and default. Paternalism is regarded as the more traditional and probably the most common form of the doctor-client relationship.[30] The paternalistic model provides a social control function in that the health care provider is seen as the expert and dominant, controlling figure, while the client is passive and free from social responsibilities. The physician maintains emotional detachment and acts only in his or her sphere of expertise. Although some may view this type relationship as negative, some clients may actually draw comfort and support from a doctor-father figure. The supportive nature of paternalism seems to be very important when a client is in need of extensive services and therefore is vulnerable. In times of emergency, when correct decisions must be made quickly to avoid life-threatening events, the health-care provider must take control and the paternalistic form is usually necessary.

The consumerism prototype is the opposite of paternalism. In this type of relationship, the power relationship between the client and the physician are reversed: the client or the consumer has more power or control than the physician.[10] Especially when trying to "sell" prevention to the client, the physician's role is to convince the client of the necessity of non-curative services such as regular dental checkups or daily brushing. Several authors have defined consumerism as a client challenge to unilateral decision making by physicians when reaching closure on diagnosis and treatment plans.[31] In this prototype, the health provider and client co-jointly explore the various options and planning objectives. This type of relationship appeals to higher order means of acceptance, including reasoning, nonthreatening persuasion, and rewards. The healthcare provider typically talks less, listens more, questions, reacts, and synthesizes when necessary.[32]

Compared to consumerism, the mutuality prototype offers a more moderate alternative. The client still has a great deal of power but so does the physician. In mutuality, both individuals (client and physician) bring recognized strengths and resources to the relationship. In this model, the client recognizes his or her role as part of a joint venture while the physician understands the centrality of the client in his or her care.[10]

In some cases, the client and physician remain at odds and cannot negotiate a change in the relationship due to poor fit. In this case, a total lack of control exists and the default prototype occurs.[29] Although the client and physician may still see each other during regular visits, the client may fail to make a commitment to prescribed regimens and the physician may cease to be engaged or try to educate the client.

Motivational Interviewing in the Change Process

Motivational interviewing, introduced by Miller and Rollnick (1991) is a particular method to help people recognize and do something about their present and potential behavioral problems.[33] It is particularly useful for those clients who are reluctant to change and ambivalent about changing. This technique attempts to help resolve ambivalence and to move the individual along the path to change. Ambivalence is a state of mind in which a person has coexisting but conflicting feelings about some

issue. This "I want to but I don't want to" dilemma is at the heart of the problem of all change. Ambivalence is a type of conflict within an individual that has the potential for keeping people "stuck" and creating stress. Ambivalent smokers who have been told by their periodontist that tobacco use can cause periodontal disease, might readily acknowledge that their oral health is endangered, yet may feel equally concerned about their ability to cope with stressful situations without smoking.

Oral-health-care providers must understand that ambivalence is not merely a "bad sign." It should be regarded as normal, acceptable, common, and understandable part of the change process. What is highly valued by some (e.g., having good oral health) will be of little importance to others.

Five broad clinical principles underlie motivational interviewing.[33] These principles emphasize that the clinician should: 1) express empathy (through skillful reflective listening, the clinician seeks to understand and accept the client's feelings and perspective without judging, criticizing or blaming, and realizes that ambivalence is normal); 2) develop discrepancy (help the client understand the discrepancy between their present behavior and their ability to reach their important goals; clients should discover and present their own arguments for and against change); 3) avoid argumentation (a gently persuasive/soft confrontation approach should be used—one that asserts that clients have the freedom to do as they please; avoid sending the message that "I'm the expert and I'm going to tell you how to run your life"; do not accuse clients of being "in denial" or label their behavior); 4) roll with resistance (invite the client to consider new information and offer new perspectives, without being imposing); 5) support self-efficacy (it is essential to support the client's self-esteem and their general self-regard; the client is responsible for choosing and carrying out personal change and the overall message is of hope and faith to the client; "You can do it. You can succeed.").

The FRAMES Brief Counseling Elements

Miller and Rollnick[34] have described six practical counseling elements that are active ingredients in effective and brief counseling interventions. They are summarized in the acronym "FRAMES."

- Feedback—The client is given feedback of their current status. The importance of conducting a thorough assessment provides the client an opportunity to reflect in detail upon their situation.
- Responsibility—There is an emphasis on the individual's personal responsibility for change. "It's up to you to decide what to do with this information. Nobody can decide for you, and no one can change your habit patterns if you don't want to change."
- Advice—Simple, clear advice to the client to make a change in their lifestyle is given.
- Menu—By offering clients a menu of alternative strategies for changing their problem behavior, the clinician provides a range of options, which allows clients to select strategies that match their particular needs and situations.
- Empathy—Understand another's meaning through the use of reflective listening, whether you have had similar experiences yourself. Use of warmth, respect, supportiveness, caring, concern, sympathetic understanding, commitment, and active interest to convey this element.

• Self-efficacy—Reinforcing the client's hope or optimism in their ability to make changes promotes self-efficacy. Remember that your belief in the client's ability to change is often a significant determinant of outcomes.

Basic Philosophy

A basic philosophy of prevention is itself a value. One basic philosophy concerning preventive dentistry is that clients deserve to know the cause of their dental diseases and how they can prevent them. This is a responsibility for the health educator. Once armed with the knowledge, however, the client reserves the right to remain sick. This is a problem of self-motivation. Clients are ultimately responsible for their own dental health. In the final analysis, prevention is a shared responsibility between the practitioner and the client.

Summary

The maintenance of good oral health requires a partnership between the dental professional and the patient. No preventive program can be a success unless the patient participates in a home self-care program to supplement office care programs, with the level of success being proportionate to the amount of participation. Maximum participation can be expected when the patient knows what to do, how to do it, and above all has the motivation to adhere to recommended procedures. Educational strategies can be used to teach facts and skills, but these are useless without motivation. Motivation can be initiated by an individual based on some need or desire, or it can be facilitated by persuasion from external sources. With or without motivation, learning is best achieved in sequential steps, as described by Bloom's hierarchy of cognitive levels. As an individual accumulates facts, the facts merge into concepts and ultimately into values, which in turn engender motivation. At times motivation provides the drive to alter lifestyle to attain habit patterns necessary to maintain good oral health. The dental professional can exert a direct or indirect influence on such a change by providing appropriate behavior modeling, by taking a more active role as an authoritarian, or by participating as a nonauthoritarian in developing a program of planned change with the patient. All health education requires learning, but the successful application of all health knowledge requires motivation.

Answers and Explanations

1. B—correct.

A—incorrect. The average layperson does not accept change without considerable persuasion.

C—incorrect. Human motivation is complex in nature and best described as the interaction between the environment, personal, and behavioral factors.

D—incorrect. Knowledge is rarely sufficient to change behavior.

2. A, D—correct.

B—incorrect. Facts and concepts represent unorganized and organized thoughts, respectively; values represent the acceptance and personal application of facts and concepts.

C—incorrect. Most education is directed to the initial level—facts; very little learning ends up at the evaluation level.

3. C—correct.

A—incorrect. Most health-behavior theories attempt to explain or predict behavior.

B—incorrect. The stages of change model suggest that behavior change does not typically follow a linear progression but rather is cyclical as an individual experiences relapse and adopts new behaviors.

D—incorrect. Health-promotion theories *attempt* to explain or predict behavior with varying degrees of accurateness.

Self-Evaluation Questions

1. Health promotion can be defined as _____.

2. An individual, through reasoning, organizes facts into _____; which in turn are the basis for a(n) _____.

3. The central assumption underlying health promotion theories is _____.

4. The five main concepts of the Health Belief Model include: _____, _____, _____, _____, _____.

5. The six cognitive levels of Bloom's hierarchy of learning are knowledge, _____, _____, _____, _____, _____, and _____.

6. The one main difference between HBM and Social Cognitive Theory is _____.

7. _____ is the underlying assumption of SCT.

8. _____ implies that an individual chooses freely to undertake behavioral plans, have input to them, and has a collaborative involvement in modifying the plan.

9. In the dentist-patient partnership, it is the _____ who must assume responsibility for home care programs, whereas the _____ must assume responsibility of identifying and correcting deficiencies that occur in a home care program.

10. In the development of optimal oral hygiene habits, patients encounter five progressive stages of change. They are: _____, _____, _____, _____, and _____.

11. In the process of applying motivational interviewing, the clinician should apply five principles, which are: _____, _____, _____, _____, and _____.

12. The process whereby the clinician seeks to understand and accept the patient's feelings and perspectives without judgment, criticizing, or blaming is called: _____.

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Chapter 17. Dental Public-Health Programs - *Mark D. Macek and Harold S. Goodman*

Objectives

At the end of this chapter, it will be possible to

1. List the core functions of public health.

2. Define dental public health and relate this definition to dental public-health programs.

3. Compare the methods of public health-care practitioners and personal health-care practitioners.

4. Describe the seven-step model for assessing oral-health-care needs and relate this model to a planning cycle for public-health programs.

5. Outline the scope of traditional dental public-health programs.

6. Describe recent changes in the United States that are relevant to dental public-health practice.

7. List the various organizations that maintain and support public health programs.

8. Describe how the Surgeon General's report on oral health in America has impacted dental public-health programs.

Introduction

In 1994, the *Core Functions of Public Health Steering Committee*, co-chaired by Drs. Philip R. Lee (Assistant Secretary for Health) and M. Joycelyn Elders (Surgeon General of the U.S. Public Health Service), produced a consensus statement outlining the essential services of public health in the United States.[1] The new statement provided a vision for public health—*Healthy People in Healthy Communities*—and defined its mission: *Promote physical and mental health and prevent disease, injury, and disability.* The consensus statement also provided broader description of the core functions of public health—*assessment, policy development*, and *assurance.*[2] According to the statement, the *purpose of public health* included: 1) preventing epidemics and the spread of disease; 2) protecting against environmental hazards; 3) preventing injuries; 4) promoting and encouraging healthy behaviors and mental health; 5) responding to disasters and assisting communities in recovery; and 6) assuring the quality and accessibility of health services. The *practice of public health* included: 1) monitoring health status to identify and solve community-health problems; 2) diagnosing and investigating health problems and health hazards in the community; 3) informing, educating, and empowering people about health issues; 4) mobilizing community partnerships and action to identify and solve health problems; 5) developing policies and plans that support individual and community health efforts; 6) enforcing laws and regulations that protect health and ensure safety; 7) linking people to needed personal-health services and assuring the provision of health care when otherwise unavailable; 8) assuring a competent public and personal health-care workforce; 9) evaluating effectiveness, accessibility, and quality of personal and population-based health services; 10) researching for new insights and innovative solutions to health problems.

In 1976, the American Dental Association adopted a definition of *dental public health*, stating that it was:

. . . the science and art of preventing and controlling dental diseases and promoting dental health through organized community efforts. It is that form of dental practice which serves the community as a patient rather than the individual. It is concerned with the dental education of the public, with applied dental research, and with the administration of group dental care programs as well as the prevention and control of dental diseases on a community basis. . .[3].

Given this definition, *dental public-health programs* refer to organized efforts that strive to prevent and control oral and craniofacial diseases at the community level. Dental public-health programs are highly varied and include activities that cover a wide spectrum, from small-scale local projects to large-scale national and international ventures. Given that a community is the focus, dental public-health programs must satisfy the criteria of practicality, feasibility, acceptability, safety, effectiveness, and efficiency.

Historic Perspective

Dental disease has been a significant problem for Americans since the nation's early history.[6] Between 1862 and 1864, loss of teeth was the fourth most frequent cause for rejection of young men for draft into the Union Army during the Civil War.[7] In 1918, military draftees for World War I were rejected—because of defective and deficient teeth—at a rate that exceeded 10% in some states.[8] During the conscription period of World War II, the *U.S. War Department Mobilization Regulation* required that a recruit have a minimum of three serviceable, natural anterior and posterior teeth in opposition, per arch, to be acceptable for military service. Fifteen percent of recruits were rejected, because they could not pass these rather liberal criteria.[6] During the 1920s, the Metropolitan Life Insurance Company conducted one of the earliest epidemiological studies of the dental condition of a large, heterogeneous, adult, civilian population.[9] Oral examinations of more than 12,000 adults revealed that, among 20- to 24-year-olds, more than half of the teeth had been affected by dental caries, and this proportion increased steadily in older age groups.

During the next several decades, the number of epidemiological surveys conducted among civilians increased dramatically.[10-15] It was not surprising that these studies reflected the high dental caries prevalence levels noted in earlier studies and conducted among military recruits. The surveys showed that dental caries was a serious health problem among young adults, and often resulted in tooth loss. The studies also showed that dental caries began early in life and affected young children.

Between 1933 and 1934, the U.S. Public Health Service (USPHS) sponsored a survey conducted among thousands of 6- to 14-year-old children in 26 states across the United States.[16] The study revealed high dental caries prevalence levels in children, as well. In 1937, the classic Hagerstown, Maryland, study,[17] which introduced the Decayed, Missing and Filled index for teeth (DMFT) and tooth surfaces (DMFS), showed moderately high caries prevalence levels among the examined children. The study also showed that children with the highest dental-caries index scores received only 2% of the treatment time rendered by dentists.

The dental-caries experience of children and the progression of the disease in adults provided the rationale for the application of dental public-health programs to address the problem. The efforts, cooperation, and interactions of a number of individuals and agencies led to one such dental public-health program, the implementation of adjusted water fluoridation.

Fluoridation—A Monumental Public-Health Success Story

Fluoridation is the principal dental public health preventive program available in the control of dental caries in the population. During a national health conference in 1966, former Surgeon General Dr. Luther L. Terry stated, "Controlled fluoridation is one of the four great mass preventive health measures of all time. The four horsemen of health are: the pasteurization of milk, the purification of water, immunization against disease, and controlled fluoridation of water."[18] The Centers for Disease Control and Prevention recently listed fluoridation among the top ten public health triumphs of the 20th century.[19]

The historic development of fluoridation in the United States serves as an example of

the contributions of individuals of varied backgrounds representing personal and public segments of the profession. For example, Dr. H. Trendley Dean, considered the "father of fluoridation," had a prominent role in the early developing story of the importance of fluoride to tooth enamel.[20] Dean was an officer in the USPHS who led extensive studies that later established that 1 part per million (ppm) of fluoride in a community water supply reduced dental-caries prevalence.[21]

As important as the contributions of Dean and the USPHS were to the subsequent implementation of community fluoridation, one should not lose sight of the roles played by Dr. Frederick McKay, a personal health-care practitioner in Colorado Springs, Colorado, and Dr. G. V. Black, a practitioner and prominent dental educator. McKay and Black conducted numerous investigations of *Colorado brown stain*, a condition indicative of excessive amounts of naturally occurring fluoride ion during tooth development, and found that dental caries was less prevalent among those afflicted.[22] In addition, one should consider the influence of an industrial chemist, H. V. Churchill, who developed the analytic method that could detect minute quantities of fluoride in water, a critical step necessary to establish the link between the level of fluoride ions in water and the dental caries experience of the population consuming the water.[23] At the same time, Smith and Smith,[24] agricultural researchers, also linked mottled enamel with water fluoride concentrations. Following these and other studies,[25] independent researchers conducted controlled trials of the effect that fluoride ion in a community water system might have on dental caries experience in children. Beginning in 1945 and proceeding through the mid-1950s, researchers added fluoride to the water systems of four test communities (Grand Rapids, Michigan; Newburgh, New York; Evanston, Illinois; and Brantford, Ontario) and observed the dental caries experience of their residents. These trials successfully demonstrated that adjusted water fluoridation, at concentrations of 1.0 to 1.2 ppm, could dramatically reduce dental caries experience in children.[26-29]

According to the most recent national data available, approximately 162 million persons, or approximately 65.8% of the total U.S. population, drink adjusted or naturally occurring fluoridated water.[30] However, this represents a nearly 4% increase since 1992. Efforts to increase the proportion of the world population drinking fluoridated water still have been thwarted, in part, because of the continuing political activities of the anti-fluoridation movement. Supporters of this movement continue to oppose adjusted fluoridation for many reasons, the vast majority of which are equivocal.[31]

Despite the efforts of opposition groups, community-water fluoridation continues to receive widespread support from both the personal and public health-care sectors. Numerous health professional organizations, consumer and advocacy groups, and the Surgeon General continue to endorse community-water fluoridation.[32-35] Adjustment of water fluoride concentrations to optimal levels is an example of a successful dental public-health program—groups working together to prevent and control oral and craniofacial diseases in the community.

Current Problem

Burt and Eklund[36] define a *public-health problem* as meeting two criteria: a) a condition or situation that is a widespread actual or potential cause of morbidity or

mortality; and b) an existing perception the condition is a public-health problem on the part of the public, government, or public health authorities. A number of oral and craniofacial diseases and conditions represent public health problems in the United States today, and are briefly discussed below. These are the principal concerns that need to be addressed by both the personal and public healthcare sectors to improve oral health at the community level.

Dental Caries

Dental caries is one of the most prevalent diseases in the United States. About 17% of children aged 2 to 4 years have had a carious lesion in a primary tooth during their lifetime, and the prevalence jumps to 49.7% among children aged 5 to 9 years.[37] Among permanent teeth, 26.0% of children aged 5 to 11 years have had a carious lesion and 67.3% of children aged 12 to 17 years have had a carious lesion.[37] Dental caries is also highly prevalent among U.S. adults, as approximately 94% of dentate adults aged 18 years or older have had a carious lesion during their lifetime.[38]

Dental-caries prevalence and severity also is associated with race/ethnicity and socioeconomic status (Figure 17-1). Certain minority children exhibit a higher prevalence of primary tooth decay than do their peers, as 34.2% of non-Hispanic white children aged 2 to 9 years have had a carious lesion, whereas 38.8% of non-Hispanic black children and 53.0% of Mexican-American children have had a carious lesion.[37] Among adolescents aged 12 to 17 years, lower poverty status is associated with higher mean dental-caries experience scores and a greater percentage of untreated disease.[39]

Periodontal Diseases

Gingivitis, one of the periodontal diseases, is moderately prevalent in persons aged 13 years or older. On average, 62.9% of persons in this age range exhibit gingival bleeding, and 12.0% of sites are involved.[40] Gingivitis, as measured by gingival bleeding, is also more prevalent among Mexican-Americans than it is among non-Hispanic blacks and non-Hispanic whites aged 30 years or older.[41] Calculus, a contributing factor in gingivitis, is present in 89.9% of persons aged 13 years or older.[40] Although most persons would not consider gingivitis a serious threat to one's health, it receives a great deal of attention in the appearance-conscious United States, given the condition's effect on esthetics and gingivitis precedes, but does not necessarily progress to periodontitis.

Periodontitis is the second of the periodontal diseases and is associated with greater morbidity than is gingivitis, and as such, is considered a more serious public-health problem. On average, 27.0% of males and 17.5% of females aged 13 years or older have at least one site with 5+mm loss of periodontal attachment.[40] This gender difference is statistically significant. The prevalence of attachment loss is also significantly higher among minority groups, as 24.9% of non-Hispanic blacks and 17.1% of non-Hispanic whites aged 13 years or older exhibit the condition.[40]

Oral and Pharyngeal Cancer

There are approximately 30,200 cases of oral and pharyngeal cancer detected in the

United States each year, and this number accounts for some 2.4% of all cancers. Of persons with oral and pharyngeal cancer, approximately 7,800 die each year. The overall 5-year survival rate for persons with oral and pharyngeal cancer is 52%, which is lower than that for cancers of the prostate, breast, bladder, larynx, cervix, colon, and rectum.[42] Persons diagnosed with oral and pharyngeal cancer at an early stage have a much better prognosis than do those diagnosed at a later stage, as the 5-year survival rate is 81.3% for early-stage diagnosis and 21.6% for advanced-stage diagnosis. Only 35% of individuals with oral and pharyngeal cancer are diagnosed at an early stage of the disease.[42]

Craniofacial Birth Defects

Oral clefts are among the most common classes of congenital malformations in the United States. On average, there are 1.2 cases of cleft lip (with or without cleft palate) per 1,000 live births and 0.56 cases of cleft palate per 1,000 live births in the general population (Figure 17-2).[43] These defects may affect facial appearance throughout life. Cleft palate occurs more frequently in females, whereas cleft lip or cleft lip/palate occurs more frequently in males.[44-47] The oral cleft incidence rate for whites is more than 3 times the incidence rate for blacks.

Intentional and Unintentional Injuries

It is assumed that injuries to the head, face, and teeth are relatively common, however the majority of our knowledge regarding the number of injuries comes from emergency department data and more severe injuries. The leading causes of such injuries include falls, assaults, sports injuries, and motor-vehicle collisions.[48-50] According to data collected in 1993 and 1994, there were approximately 20 million visits to emergency departments per year for craniofacial injuries. Falls and assaults each accounted for about 31% of visits and sports-related injuries accounted for approximately 19% of injuries.[51] Injuries resulting from bicycles and tricycles accounted for 5% of head and 19% of face injuries.[52] Overall, 24.9% of persons aged 6 to 50 years have had an injury that resulted in damage to one or more incisor teeth.[53] According to data collected in 1991, personal health-care dentists treated more than 5.9 million craniofacial injuries.[54]

Figure 17-1 Disparities in prevalence of unrestored dental caries exist between poor and non-poor. (From U.S. Department of Health and Human Services. *Oral Health in America: A Report of the Surgeon General.* Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Dental and Craniofacial Research, 2000(35):63.)

Figure 17-2 Incidence of selected congenital defects. (From Schulman et al., 1993.[43])

Dental Public-Health Methods

Personal oral-health-care practitioners serve the oral health needs of individual patients, and the personal health-care delivery system requires a one-on-one interaction between practitioner and individual patient. Public-health dentistry focuses on the community and, as such, does not necessarily require a one-on-one interaction between practitioner and individual patient. When a dental public-health program such as water fluoridation is successfully implemented in a community, a much

broader cross section of the community benefits — much broader than could be expected by personal health-care practitioners, alone.

Knutson[55] contrasted the methods employed by personal and public healthcare practitioners. Each consisted of six, sequential steps that permit a logical progression from identification of a problem to its solution (Table 17-1). For the individual patient, a personal healthcare practitioner initiates treatment with a careful examination and history, which leads to an accurate diagnosis of the problem. Afterwards, the personal healthcare practitioner plans a course of treatment. Once treatment services have been provided, and fees paid, subsequent visits provide for evaluation and follow-up. The methods employed in public-health practice parallel those of the personal health-care practitioner, but involve the total community instead of an individual patient. Dental public-health methods are discussed in greater detail below.

Examination versus Survey

When a personal health-care practitioner begins the examination process, he or she collects subjective information from the patient and objective information, such as visual and tactile data, radiographic images, and other signs of disease. By contrast, when a public health-care practitioner assesses the extent of disease in a community, he or she must rely on descriptive information, such as existing survey data or other epidemiological assessments.

Question 1

Which of the following statements, if any, are correct?

A. As a general rule, children with the greatest oral-health treatment needs are also the children who receive priority care.

B. The overwhelming majority of Americans die with at least one carious or restored tooth.

C. The incidence of oral clefts is higher among blacks than it is among whites.

D. Core functions of public health include assessment, policy development, and assurance.

E. Dental public-health programs do not necessarily require a one-on-one interaction between practitioner and individual patient.

Some descriptive survey data have been collected and reported previously. At the national level, surveys such as the National Health Examination Survey, National Health and Nutrition Examination Survey, National Health Interview Survey, and surveys conducted by the National Institute of Dental and Craniofacial Research[56] have provided assessments at the community level regarding the distribution of diseases, such as dental caries and periodontitis, as well as oral health knowledge and behavioral practices. At the state level, surveys such as the Behavioral Risk Factor Surveillance System or cancer registries have provided useful descriptive information

regarding oral health care utilization practices and incidence of oral and pharyngeal cancer. Selected states have also administered surveys to assess the oral-health status of their citizens (Figure 17-3).

For the dental public-health-care practitioner, the focus of the survey step is to compile all of the descriptive information that exists in a state, county, region, or local area. When descriptive data do not exist, the dental public-health-care practitioner must find a way to collect useful information. During the mid-1990s, prompted by the newly defined essential functions of public health, the Association of State and Territorial Dental Directors (ASTDD) developed a model for the collection of oral health data at state and local levels,[57] referred to as the *Seven-step Model for Assessing Oral Health Needs.* The seven steps included:

1. Identifying partners and forming an advisory committee.

2. Conducting self-assessment to determine goals and resources.

3. Planning the needs assessment.

- Conduct inventory of available primary and secondary data
- Determine need for primary data collection
- Identify resources
- Select methods
- Develop work plan

4. Collect data.

5. Organize and analyze data.

6. Report findings and utilize the data for program planning, advocacy, and education.

7. Evaluate needs assessment and return to first step, as necessary.

ASTDD intended that the collected data be used as part of a planning cycle (Figure 17-4) that would lead to the implementation of necessary dental public-health programs. Just as a personal health-care practitioner would not consider initiating treatment on a patient without subjective and objective data at hand, the public health-care practitioner would not consider initiating a dental public-health program without descriptive data regarding the needs of the community.

Diagnosis versus Analysis

Once a personal health-care practitioner has gathered sufficient subjective and objective information from the patient, he or she uses professional judgment and experience to diagnosis a disease or condition, if one exists. Once a public health-care practitioner has collected sufficient survey data, he or she analyzes the information in order to answer specific questions. Is there a dental public health problem? If so, what is the extent of the problem? Are there appropriate solutions available to address the problem?

The analysis step helps the public health-care practitioner assess when a problem exists and helps to quantify its extent. Public health-care practitioners rely on standard statistical methods to summarize survey data findings during the analysis step. For example, state-specific survey data may show that 45% of schoolchildren have unrestored dental caries, and that this percentage is significantly higher than would be expected at a national level. The significant difference in percentages may point to a dental public health problem in that state. In addition, survey data may show that oral and pharyngeal cancer incidence in one county is significantly higher than is the rate in a neighboring county. One would expect the first county to receive special attention or a targeted dental public health program. Without the analysis step, however, the difference between the two counties might be less obvious.

In order for a public health-care practitioner to compare analytical findings to other survey data, or transmit analytical findings to other public health-care practitioners, he or she uses standard measurement tools and descriptive guidelines, called dental indexes. A variety of dental indexes have been developed for specific oral and craniofacial diseases and conditions. Some of the more common indexes are listed below.

Dental Indexes

An important tool used in examinations of a population group is a *dental index,* a numeric score that quantifies the magnitude of the disease measured. A number of indexes have been developed for the purpose of providing the objective measurement of the oral health status of a population group. The number of *teeth* that are decayed, missing, or filled—the DMFT index[17]—is a total score of all affected teeth and provides a dental caries experience score for an individual. A count of *tooth surfaces* that are decayed, missing, or filled is a DMFS index and provides greater precision regarding the dental caries history of an individual or population. The mean DMFT score for a population group is the total average dental caries experience at a particular time. Dental caries experience in the primary dentition is denoted by the use of *lower case letters* to represent the number of decayed, extracted, or filled primary *teeth* and *surfaces; deft* and *defs.*[58] This index has recently been modified to *dft* and *dfs,* because of the difficulty in distinguishing a primary tooth that has been extracted from one that has been lost to the natural process of exfoliation.

The status of periodontal tissues has been evaluated using several indexes. The *Gingival Index* (GI) of Loe and Silness[59] is particularly suited for assessing changes in gingival health that might be observed during the evaluation period of an oral hygiene program. Several plaque indexes have also been developed to assess the status of oral hygiene in population groups. The *Plaque Index* (P) of Silness and Loe[60] quantifies the extent of plaque on defined areas of specific tooth surfaces. The *Oral Hygiene Index—Simplified* (OHI-S) of Greene and Vermillion[61] measures oral debris and calculus on specific tooth surfaces.

The *Periodontal Index* (PI) of Russell[62] and the *Periodontal Disease Index* (PDI) of Ramfjord[63] were once used for assessing the severity of periodontitis, but are no longer considered valid. When these indexes were developed, it was believed that gingivitis and periodontitis were on a continuum; as gingivitis became more severe, periodontitis resulted. Consequently, the PI and PDI were developed as composite

indexes, assessing gingivitis and periodontitis together. Today, it is well established that gingivitis does not necessarily lead to periodontitis, and that the two diseases are unique. Although the PI and PDI are no longer used, the PDI left behind a measurement component that is valid for assessing tissue destruction. The surviving measurement component, sometimes referred to as *loss of attachment* or *LOA*, calculates the loss of periodontal attachment that has occurred adjacent to a tooth. The *Community Periodontal Index of Treatment Need* (CPITN) is not an index of periodontitis, but a measure of the necessity for periodontal treatment.[64] The CPITN has been used by nations around the world.

When public healthcare practitioners employ a dental index during the analysis step, they must pay particular attention to the training of examiners. Consistency in the application of scoring criteria is paramount to the validity of index scores. A comparison of DMFT scores from one county to another would be of little value, for example, if the examiners in the two counties applied the scoring criteria in different ways.

Treatment Planning versus Program Planning

Once a personal health-care practitioner has identified a disease or condition, and assessed its extent, he or she is ready to transmit the information to the patient and plan a treatment strategy. Once a public health-care practitioner has identified the existence of a dental public-health problem and assessed its extent, he or she is ready to transmit the information to concerned individuals and community partners. Together, the public health-care practitioner and partners develop a public-health program that is tailored to the needs of the community.

During the treatment planning and program planning steps, decisions must take into consideration such factors as available time, finances, knowledge, experience, attitudes, and willingness to complete the plan. Just as an individual patient must consider his or her personal circumstances when selecting treatment options, community leaders must consider community resources and priorities when selecting appropriate public health program options.

Treatment versus Program Operation

Once the patient and personal health-care practitioner have decided on an appropriate treatment plan, treatment of the disease or condition begins. Once the community and public healthcare practitioner have decided on an appropriate program plan, the public health program is set in motion. Program operation usually includes three features, including health education, disease prevention, and provision of services.[65] Given that administrations change, resources shift, and attitudes and motivations evolve, the program operation step is never static. Community interventions are generally more difficult to orchestrate than are plans that address an individual, because more factors must be taken into consideration at the community level.

Figure 17-3 Surveys designed to establish the oral health needs of children frequently take place in a school setting. (Courtesy of Dr. Arthur Benito, Research Triangle Institute, North Carolina.)

Figure 17-4 Dental public-health program planning cycle.

Question 2

Which of the following statements, if any, are correct?

A. *Treatment planning* requires the input of the personal healthcare practitioner and informed consent of the patient, whereas *program planning* requires the input of the public-health dentist and informed consent of involved community leaders.

B. Individual State Health Departments operate under the administrative control of the U.S. Department of Health and Human Services.

C. The *examination* step of personal health care is analogous to the *analysis* step of public health care.

D. Community-water fluoridation campaigns often fail because of political issues—not because of health department decisions.

E. The *Community Periodontal Index of Treatment Need* (CPITN) is a valid measure of periodontal tissue destruction.

Payment for Services versus Financing

For the individual patient facing a treatment plan, the scope and extent of treatment services depend on personal resources and/or the existence of third-party payment plans. For the community looking forward to the initiation of a dental public-health program, the scope and extent of the program depend on the existence of available public and personal health-care funds. In most cases, public programs are funded via the federal government or the state. Program administration and funding typically originate from state-level health departments, or county-level or local area-level health departments, when they exist.

Evaluation versus Appraisal

When a personal health-care practitioner completes an individual patient's treatment plan, he or she evaluates the individual during periodic intervals, to assure that the oral health is maintained and any arising treatment needs are identified and met. The responsibilities of the public health-care practitioner are comparable. During the appraisal step, the public health-care practitioner first needs to assess whether the program has adequately addressed the needs of the community. As such, all public-health programs should have a measurable set of objectives against which success or failure may be appraised. If, for example, a dental public-health program were initiated to reduce the oral-cancer incidence in a county experiencing unusually high rates, then the program should contain a target incidence rate that would signify success. Once the public health-care practitioner has assessed whether an objective has been met, he or she must monitor the existence of a public health program on a regular basis. If new survey data are required, the public health-care practitioner should secure them. If the standard against which success is judged should change, the public health-care practitioner should reassess whether the program would be considered a success.

For both the personal and public health-care practitioners, the evaluation and appraisal steps represent the link between the end of a treatment plan or public-health program and the beginning of a new plan or program. As long as individual patients have treatment needs or communities have public-health problems, the six steps of the personal and public health-care practitioner can be applied.

An Example of a Dental Public-Health Program

A *dental sealant* is a plastic material that is applied to the pit-and-fissure surfaces of the teeth by oral health-care professionals. Dental sealants function as a primary preventive agent against dental caries by obstructing the pit-and-fissure surface from bacteria. Dental sealants also may serve as a secondary preventive agent against dental caries when applied to incipient lesions. Dentists and dental hygienists apply dental sealants in private health-care facilities, however this means of providing the preventive agent is limited by access to the facilities and the personal circumstances of the patients in need.

This section of the chapter presents a dental public health problem in a fictitious community called *Yourtown* and proceeds through the six steps of the public healthcare practitioner's method, in order to illustrate how a dental sealant campaign might be employed as an effective dental public health program (Figure 17-5). Although this exercise describes a specific problem and program solution, the principles may be applied more broadly to other problems and solutions.

Survey

The survey step of the process encompasses the seven-step model of assessing oral health needs. Dr. Sally Sealem, the county health officer in Yourtown, began the process by identifying partners and forming an advisory committee of interested parties. Dr. Sealem asked administrators from the school board to join her, as well as staff members from the health department, the director of the dental hygiene training program at Yourtown Community College, the state dental director, and the administrator of a nonprofit health-care facility.

During the advisory committee's first meeting, the members discussed their resources and limitations. Dr. Sealem knew that the most cost-effective dental sealant program would involve school-aged children. The school administrators assured the health officer that a dental sealant program would be welcomed into the local schools. The administrators also said that the principals, teachers, and school nurses would be willing to coordinate communication with parents and students. Dr. Sealem also recognized that oral health-care professionals would need to participate in the program. The dental director told her that he would discuss the proposed program with the state and local dental society. He was fairly sure that the dental society would embrace the program and provide the names of a few retired practitioners who might be interested in volunteering their time to the program. The director of the training program in dental hygiene also offered the assistance of her faculty and students.

The dental director also said that there was little money in the budget for a dental sealant program. Upon hearing that the dental director's budget did not allow for a

dental sealant program, the administrator of the non-profit healthcare facility said that their treatment clinic would be willing to donate some money and supplies to the program and the attendees from the health department said that they would look into the existence of grant money from private corporations, community groups, and the federal government.

Prior to the meeting, Dr. Sealem compiled demographic data for Yourtown and all of the relevant data regarding dental caries and preventive oral health programs. She discovered that the community contained approximately 10,000 school-aged children. Most of these children lived within 10 miles of their respective schools, however a few were transported via bus from neighboring rural areas. The socioeconomic status (SES) profile of the community was relatively low, with approximately 56% of children qualifying for free or reduced meals at school. Dr. Sealem also learned that Yourtown did not have access to fluoridated community water. Dr. Sealem had no data describing the dental caries prevalence or the prevalence of dental sealants in Yourtown, however she did have access to data from several national surveys.

The advisory committee recognized that survey data from Yourtown would have provided a more complete picture of the oral-health conditions in their community than the national data, but they also recognized that in order to collect such data, they would have to conduct a survey for which they had limited resources. Given the circumstances, the advisory committee ultimately decided that they would rely on the national data to draw conclusions about their community.

Analysis

From studies of national data,[39] Dr. Sealem knew that dental caries prevalence was higher among poor children than it was among their non-poor counterparts. She also knew that the percentage of unrestored disease was higher among the poor children. In addition, national studies showed that only 18.5% of children aged 5 to 17 years had one or more sealed teeth.[66] Given that there was a sizeable proportion of poor children in Yourtown and given that national survey data showed that poor children had greater needs, the advisory committee concluded that there was good reason to initiate a dental sealant program in their community.

Program Planning

During the program-planning stage, the advisory committee listed all of the possible ways to implement a dental-sealant program in Yourtown. Some of the options included use of a mobile dental van, visits to churches and other meeting places, expansion of services at the health department, expansion of services at the nonprofit health-care facility, and a school-based program. In deciding on the best approach, the advisory committee considered available resources and potential advantages and disadvantages of each option. Given that school administrators provided ready access to schools, and because this was where the majority of children could be found, the advisory committee decided that they would use a school-based dental-sealant program. They also decided that they would use students from the dental-hygiene training program at the community college to educate parents and teachers about the benefits of this preventive oral-health measure, and they would use the retired dentists from the community to administer the dental sealants.

In recognition of the budgetary constraints, the nonprofit health-care facility provided disposable gloves, masks, dental mirrors, and tongue blades to the program. In addition, staff members from the health department were able to procure grant funding from a local philanthropic organization and dental sealant materials from a national dental supply distributor. The advisory committee used the grant funds to purchase a portable dental chair, generator, and light source.

Program Operation

After thorough consideration of the problem and analysis of its severity, careful planning, and procurement of funding, the school-based dental sealant program was put into operation. In preparation for the initiation of the program, in-service training programs were conducted for all participants to affirm goals and standardize treatment protocols. The application of dental sealants to the school children progressed well, because the advisory committee had paid such careful attention during the previous stages.

Financing

Although the advisory committee was able to solicit the necessary funds for the first year of the dental sealant program, they realized that in order for the program to have a lasting impact, they would need to procure new funding over time. The great success of the program made this step relatively easy. The advisory committee created press releases and gave them to the print media. Dr. Sealem asked the local television stations to interview her during "health spots" on the local news. The advisory committee capitalized on the popularity of the program among parents and community leaders by asking them to request additional funds from their legislative representatives for the state's health budget. Staff members at the health department wrote new grant applications and continued to solicit funds of other agencies and organizations.

Appraisal

The advisory committee used national data to determine whether their community would be a good candidate for a dental sealant program. This approach was satisfactory for the initiation of the program, but it would not suffice during the appraisal stage. In order for the advisory committee to evaluate whether the dental sealant program had been successful in reducing dental caries experience in Yourtown, they would need new data; a baseline assessment and periodic assessments of dental-caries and dental-sealant prevalence among the school children.

The appraisal stage is arguably one of the most difficult components of a dental public health program. It requires careful delineation of measurable goals and objectives and a detailed plan to collect evaluation data over many years. The appraisal stage must take into consideration the benefits of the program and weigh them against the cost. It must also consider alternative preventive and treatment regimens as they develop, and assess whether these new strategies might be a better option.

Enthusiasm and excitement frequently drive the first few years of a new program,

however funding agencies and legislators will eventually demand that their resources are being applied to an efficient and effective program. Without a valid appraisal plan in place, the ability for an administrator such as Dr. Sealem to demonstrate efficiency and effectiveness is all but impossible.

Although this may be the most difficult component of a dental public health program, administrators have a number of resources at their disposal. Health departments typically have epidemiologists and survey researchers available for consultation. ASTDD and the Division of Oral Health at the Centers for Disease Control and Prevention also have consultants available.

Figure 17-5 School-based dental sealant programs have been found to be an effective approach to reducing dental caries in pit-and-fissure tooth surfaces. (Courtesy of Ohio Division of Dental Health.)

Levels of Dental Public Health Operation

There are numerous international and national organizations that have as a primary or secondary focus, the prevention and control of oral and craniofacial diseases at the community level. At the international level, the World Health Organization (WHO) has accepted the responsibility of coordinating the efforts of all member organizations in developing and improving oral and medical health programs throughout the world. WHO has several regional offices located throughout the world that aid in administering programs on a local level.

Based in Washington, D.C., the Pan American Health Organization (PAHO) is one such regional office for the Americas. Member States of PAHO include all 35 countries in the Americas and Puerto Rico is an Associate Member. France, the Netherlands, and the United Kingdom of Great Britain and Northern Ireland are Participating States, and Portugal and Spain are Observer States. The mission of PAHO is to strengthen national and local health systems and improve the health of the peoples of the Americas. It works in collaboration with Ministries of Health, other government and international agencies, nongovernmental organizations, universities, social security agencies, community groups, and many others. PAHO targets the most vulnerable groups, including mothers and children, workers, the poor, the elderly, and refugees and displaced persons. It focuses on access issues and a Pan-American approach, encouraging nations to work collab- oratively on common issues.

The World Dental Federation (FDI) is an independent, professional organization for dentistry. The activities of the FDI cover all aspects of personal and public oral healthcare and take place all over the world. Among its varied responsibilities, FDI contributes to the development and dissemination of statements regarding policies, standards, and information related to oral health care. In addressing this responsibility, FDI produces the statements via its Scientific Commission or in collaboration with other professional organization throughout the world.

At the national level, the U.S. Department of Health and Human Services (DHHS) is the Cabinet-level branch of the federal government that is responsible for the planning and implementation of a broad array of health programs, from support for and protection of Americans of all ages, to aid for persons with disabilities, as well as assistance and new opportunities for those in need. In short, DHHS is responsible for

public health in the United States, supporting the world's largest medical research effort, assuring the safety of foods and health care products, and fighting the ravages of drug and alcohol abuse. Planning begins in Washington, D.C., with objectives evolving as health needs shift. For example, at one time there was a need to finance new dental and medical schools to increase the output of health professionals; more recently there has been a need for specifically focused programs to accelerate development of control measures for either caries or periodontal disease, and continually there are efforts to refine programs offering better access to, or less cost for, medical and oral healthcare. DHHS responsibilities in the United States are divided into 10 geographic regions (I to X), each one having a central office. These offices facilitate administration by providing consultation and monitoring expertise for regional and local health programs involving federal funds.

DHHS oversees 12 major organizations, each with a different influence over public health issues and dental public health programs (Figure 17-6). The *Administration for Children and Families* (ACF) is responsible for numerous programs that provide services and assistance to needy children and families, administers the new state-federal welfare program (*Temporary Assistance to Needy Families*), administers the Head Start program, provides funds to assist low-income families in paying for child care, and supports state programs to provide for foster-care and adoption assistance. The *Health Resources and Services Administration* (HRSA) helps provide health resources for medically underserved populations, supports a nationwide network of community and migrant health centers and primary care programs for the homeless and residents of public housing, works to build the health-care workforce, maintains the National Health Service Corps, works to improve child health, and provides services to persons with AIDS through the Ryan White CARE Act programs. The *Agency for Healthcare Research and Quality* (AHRQ) supports investigator-initiated research designed to improve the outcomes and quality of health care, reduce its costs, address patient safety and medical errors, and broadens access to effective services. The *Centers for Disease Control and Prevention* (CDC) administer a health surveillance system designed to monitor and prevent outbreaks of disease. It also guards against international disease transmission, maintains national-health statistics and provides for immunization services and supports research into disease and injury prevention. The CDC's Division of Oral Health maintains and reports on national and local oral-health surveillance data, consults with states and local health departments regarding oral-health assessments and survey techniques, administers the Water Fluoridation Reporting System, and publishes policy statements regarding control of infection. The *Agency for Toxic Substances and Disease Registry* (ATSDR) works with states and other federal agencies to prevent exposure to hazardous substances from waste sites. The Substance Abuse and Mental Health Services Administration (SAMSHA) strives to improve the quality and availability of substance abuse prevention, addiction treatment, and mental health services. The *Administration on Aging* (AoA) provides and supports ombudsman services for elderly, and provides policy leadership on aging issues. The *Food and Drug Administration* (FDA) assures the safety of foods and cosmetics, and the safety and efficacy of pharmaceuticals, biological products, and medical devices, including those used in personal oral health care settings and dental public health programs. The *Centers for Medicare and Medicaid Services* (CMS), formerly *Health Care Financing Administration* (HCFA), serves the needs of Medicaid and Medicare beneficiaries. The *Indian Health Service* (IHS) oversees and supports a network of hospitals, health centers, school-based

health centers, health stations, and urban Indian health centers that provide services to nearly 1.5 million Native Americans and Alaska Natives. The *National Institutes of Health* (NIH), the world's premier medical research organization, supports research projects nationwide in diseases like heart ailments, diabetes, cancer, HIV, Alzheimer's Disease, and asthma. The *National Institute for Dental and Craniofacial Research* (NIDCR), one of the NIH institutes, supports intramural and extramural research regarding dental caries, periodontitis, oral and pharyngeal cancer, facial clefts, oral health disparities, and preventive oral health therapies. The *Program Support Center* (PSC) provides, for a fee, solution- and customer-oriented support for administrative operations, financial management and human resources throughout DHHS, as well as other departments and federal agencies.

The USPHS encompasses the Commissioned Corps, the uniformed service of the DHHS. Dental officers in the Commissioned Corps serve the oral health treatment needs of Native Americans and Alaska Natives as part of the IHS; active duty members, dependents, and retirees of the U.S. Coast Guard; and persons incarcerated under the Federal Bureau of Prisons. The Surgeon General leads the Commissioned Corps of the USPHS.

Each state has a health department that may or may not include an oral-health division. Of those states with an oral health focus, many divide their jurisdictional operation into regions to better administer and monitor state-administered oral-health programs. The regional programs include operation of clinics for needy populations, state prison systems, and in some cases, school systems. Consultations with communities desiring to establish or to improve community oral health, public health education programs, and fluoride initiatives receive major emphasis.

Within each state, populous counties and cities may administer community oral health treatment clinics through local health departments. These clinics usually operate in schools, economically underprivileged areas, or among population subgroups that do not otherwise have access to routine personal oral-health care. Federal, state, and local tax funds are intermixed in the delivery of care at all levels.

Figure 17-6 Organizational chart for the U.S. Department of Health and Human Services.

Dental Public Health Programs

Health Promotion and Health Education

Health promotion and health education are integral components of most successful dental public-health programs (Table 17-2). *Health promotion* consists of any planned combination of educational, political, regulatory, and organizational supports for actions and conditions conducive to the health of a community or group of individuals in a defined geographic location (67). Projects designed to be administered in schools, such as fluoride mouthrinse programs and dental-sealant programs, have been particularly successful, because dental caries is prevalent in children and those with the greatest needs may reside with parents/guardians who are otherwise unable to provide for their treatment needs in personal healthcare facilities. School-based health care programs are discussed elsewhere in this textbook. Health-promotion activities do not require active participation of its recipients, however. Public sanitation

measures, for example, promote health among humans around the world, yet most of these persons enjoy the benefits without action or awareness. Consequently, health-promotion activities are vitally important to dental public-health programs, because they do not usually depend on recipient awareness or cooperation for success.

Question 3

Which of the following statements, if any, are correct?

A. The American Dental Association is under the auspices of the World Health Organization.

B. The health policies of the U.S. Department of Health and Human Services are administrated from the 10 Regional Offices of the Centers for Disease Control and Prevention.

C. Approximately 144 million persons in the United States drink fluoridated water.

D. Health promotion frequently yields more immediate effects on the public than does education of the public.

E. The Surgeon General's Report on Oral Health in America, released in 2000, was one of many such reports dedicated to dentistry.

Health education includes any combination of learning experiences designed to enable the voluntary adoption of behaviors or actions that are conducive to health and healthful living (Figure 17-7).[68] Whereas health-promotion activities do not require the active participation of its recipients, health education does. For this reason, dental public-health programs that rely heavily on health education are subject to the attitudes, beliefs, and other motivating factors of the recipients. In addition, although knowledge is an important element of empowerment, knowledge does not guarantee that appropriate actions or behavioral changes will follow.[69] Health-promotion activities in dental public health programs frequently include health-education components, but health-education, alone, is not sufficient to prevent oral diseases or conditions.

Community-Water Fluoridation

Community-water fluoridation, or the addition of appropriate concentrations of fluoride compounds into water systems to prevent dental caries, is a health promotion activity within a dental public health program. As beneficial as water fluoridation is in the battle against dental caries, the addition of fluoride to water is not an automatic condition, however, and frequently requires the savvy and careful coordination of dental public-health professionals, water engineers, legislators, and organized dentistry. In order to conduct a successful fluoridation campaign, one must understand political realities and recognize available resources in the community that can be used to assist in securing a favorable outcome.[70] Successful campaigns require dedicated and enthusiastic persons who are coordinated by an individual with good political skills. Support from all segments of the population, not just health professionals, is crucial. The best method to achieve fluoridation in a small community is through city

council action if state laws do not require a referendum. Endorsements offered by strategic role models, such as a mayor, city council member, or other community leaders, play an important role in the process.

Coordination of activities is also important once a water fluoridation program has been initiated. Studies show that water distribution centers frequently maintain aqueous fluoride concentrations that are lower than recommended levels.[71] In order to combat this reality, authority should rest with an administrator who is dedicated to the dental public health program and who is in a position to manage the system. Frequently, the dental director of the state health department is an ideal choice for the administrative position. When a dental director is unavailable, dental public health professionals should assign a person who has the responsibility of water fluoridation surveillance and management.

Special Population Groups

Selected dental public-health programs include projects that focus on particular population subgroups. Certain groups, for example, because of health status, position in society, attitudinal barriers, or geographic location, do not have ready access to personal healthcare providers and must receive care in special clinics, supported by public or private funds. The oral health care needs of these groups, which include Native Americans and Alaska Natives, long-term-care populations, migrant groups, medically compromised individuals, beneficiaries of the Department of Veterans Affairs, persons with developmental disabilities, homeless individuals, the elderly, and persons with low socioeconomic status, are usually significantly greater than they are for the general population.[56,72-85] For example, medically compromised individuals and persons with Acquired Immunodeficiency Syndrome (AIDS) are frequently predisposed to rapidly progressing periodontitis and other oral problems.[86,87] Alzheimer's disease and other dementias compro- mise the ability of many older persons to take care of their mouths.[88, 89]

The abilities and limited experience levels of some personal oral health-care professionals, as well as the conditions within which they work may stand in the way of effective provision of care for some of these special population groups.[90-92] For example, routine treatment facilities are frequently inaccessible to a person who is homebound because of physical or mental disabilities or limitations. Dentists and auxiliaries, trained in the use of mobile treatment equipment and management of the disabled patient, are necessary in order to provide oral-health care to the homebound.[93] In this example, the removal of barriers to care is an example of an effective dental public-health program.

Dental public-health professionals in public-health agencies, local or state health departments, and academic institutions are called upon to provide consultation or initiate programs for individuals with particular diseases or conditions. Examples may include educational programs aimed toward mothers and designed to address feeding behaviors leading to early childhood caries, programs designed to produce mouth guards for high school athletes, programs designed to assess the function of removable prostheses in a geriatric population, programs designed to provide fluoride therapy to cancer patients undergoing head and neck radiation, programs designed to

screen low-income children for oral diseases, or programs designed to provide information regarding oral and pharyngeal cancer prevention.

Figure 17-7 Classroom dental-education programs are important, but it is critical to evaluate their effect. (Courtesy of the National Institute of Dental and Craniofacial Research.)

New Strategies Needed

Changing Disease Patterns

During the early 1900s, acute infectious diseases were more prevalent than they are today and accounted for greater morbidity and higher mortality among the general population. During the 1950s and 1960s, with the advent of immunizations and antibiotics, public health professionals began to shift their attention to chronic diseases, such as heart ailments, cancer, strokes, and diabetes. Dental public health programs have had to adapt to changing disease prevalence, as well.

One of the truly significant developments in dental public health has been the decline in dental-caries prevalence during the past 15 years.[56, 94-102] Reduced susceptibility to dental caries, particularly among children and young adults, is altering the oral-health status of the population. NIH estimated that the United States saved approximately $100 billion in dental expenditures during the 1980s as a result of this improvement in oral health.[103] The change in dental-caries prevalence represents a major success for personal oral health preventive and treatment services and dental public health programs, but it also presents new challenges to the profession.

During the early-1900s, dental caries was highly prevalent across age groups and population sub-groups. Everyone required treatment services.[104] Today, as a result of effective prevention and improved treatment regimens, dental caries is concentrated in a substantially smaller proportion of the population. The challenge to dental public-health professionals is to concentrate on identifying high-risk individuals and expanding services for those who have not had access to care. Current trends to decrease spending for public programs as well as reduce health-care costs should favor preventive programs that are targeted to those who have higher unmet levels of oral disease.[105-106]

Changing Public-Health Practices

Dental public-health programs should be organized to meet the needs of the population. As needs change, dental public health efforts should evolve to address these changing needs.[107] An accepted characteristic of a profession is that it shall be willing to respond to changing needs as a result of its own successful preventive and treatment activities.[108] Concern over the current ability of the public-health profession to adapt to change is addressed in an Institute of Medicine report, entitled *The Future of Public Health.*[109] The report contends that public health in the United States is disorganized, splintered, and unprepared to accommodate and address future challenges. The report goes on to state that the means to maintain and expand public health programs and meet the demands of a changing environment is via assessment, policy development, and assurance.

Contrary to the report's recommendations, political and economic forces in the United States have served to reduce or discontinue many dental public health programs. The decline of dental public health programs at the national, state and local levels is, in part, a result of the perception that oral health is not a major concern.[110] Neighborhood, rural, migrant, and homeless health centers have suffered severe cutbacks in federal outlays for oral health care services, personnel, and scope of programs.[110] Public-health dentistry curricula in many schools of public health are experiencing major reductions or dissolution. Many community-dentistry programs in dental schools are only modest in scope, relative to the concentration of resources devoted to these programs when first initiated.

Why has the downsizing of dental public-health programs progressed with relatively few challenges? One answer may be the lack of an organized constituency or advocacy group for dental public-health issues. A partnership between the public and personal health-care dental sectors is essential if oral health concerns are to be effectively promoted. Often the aims of professional groups within dentistry tend to be compartmentalized and narrowly defined. Public dental programs may also be seen as competitive with personal healthcare practitioners. Preventive approaches are apt to be erroneously classified as public sector or personal healthcare sector programs. Yet the efforts of both should reinforce common goals. Fluoridation, for example, may be seen as an effective public health measure but the promotion of fluoride dentifrices may not be. Yet they complement each other and both are public-health measures.[111]

Cooperation between dental public-health organizations, such as, the American Association of Public Health Dentistry (AAPHD), the American Public Health Association (APHA) Oral Health Section, and the American Dental Association (ADA) could help resolve the differing perspectives of the personal healthcare and public sectors. Cooperation could also foster an influential alliance in local and national campaigns addressing dental public health issues. Collaboration with a multitude of national and local voluntary non-dental health and educational organizations, such as, the Children's Defense Fund, American Association of Retired Persons, or the National Health Education Coalition is equally important to promote oral health as essential to overall health and to integrate oral health issues within the health, educational, and policy directives of these organizations. By working together on certain broad-based popular issues (i.e. access to health services), these separate partnerships can evolve into a coalition, such as the National Oral Health Alliance that can be recruited to actively support specific oral health issues.

In 1998, 53.8 billion dollars were spent on oral health care services, representing about 4.7% of the total health expenditures budget for that year.[112] Expenditures for oral health care services increased between 1997 and 1998 at approximately the same rate as expenditures for medical health care (5.3 versus 5.6%). Although these figures suggest that oral health-care services were adequately funded, comparisons with funding levels from earlier decades paint a different picture. In 1960, for example, 2 billion dollars were spent on oral health-care services, but this represented 7.3% of the total budget.[35] With reductions in funds to support oral health-care services, public-health program administrators will have to become more opportunistic and adaptive in order to conduct effective programs.

Other advocacy measures that can be pursued in support of dental public-health

programs may be advanced through regulatory and legislative routes. An area of activity often entered with some reluctance is the political arena. Those in dental public-health programs characteristically go about their duties quietly, content to live within the constraints imposed by citizens who, for example, vote against fluoridation. Niessen believes that there are community regulatory roles for dental public health regarding compliance with fluoridation and infection control standards.[113] If successful, efforts to educate and persuade others of the importance of these issues could pay big dividends. The preventive benefit provided to a community by initiating and/or monitoring fluoridation or a practice act that addresses infection control may be greater than the benefit attained from a lifetime of practice by a dozen dentists.

Successful public-health workers need to be opinion leaders and community decision makers regarding oral health programs and services. Gaupp expands this notion when stating that, "It is opportune for the oral health interest groups to strike out on their own by working toward a national, comprehensive, oral health bill."[114] Resource development could also be expedited if dental public-health programs attained influence in the regulatory and legislative arenas.

National Oral-Health Objectives

The USPHS recognizes that an effective means to expand advocacy and regulatory activities and generate support for oral-health programs is via the setting of measurable and achievable, national health objectives. In 1980, the federal government established a program, entitled *Promoting Health/Preventing Disease: Objectives for the Nation, 1990,*[115] to identify and monitor a variety of health objectives, including 12 that addressed oral health and fluoridation. Although this program provided an early opportunity to promote oral health alongside other national health priorities, it did not adequately address the means by which states and localities could meet the objectives. Subsequent national-health objectives for 2000[116] built upon the previous framework, by providing strategies that would be helpful in meeting the new objectives. Toward that end, the USPHS outlined twenty-nine measurable oral health objectives and indicators in another document, entitled *Healthy Communities 2000: Model Standards,*[117] and called for periodic reports[118-200] and consortia[121] to promote the national health objectives for 2000.

In 2000, the USPHS released national health objectives for 2010, which included an oral health focus area[122] (Table 17-3). These oral health objectives differed from previous ones, in that they incorporated a "better than the best" standard for setting goals, as opposed to setting disparate goals for certain population sub-groups. For example, the best value attained for any single population subgroup in 2000 was used to determine the goal for all population sub-groups in 2010. The rationale behind this standard-setting method was to establish a single high goal for all groups, rather than to perpetuate disparities over time.

Special Populations

During the last two decades, the United States has experienced an increase in the number of special population groups, including persons in long-term care,[74] medically compromised individuals,[78] and the homeless.[81,82] Higher unmet needs in these special population groups has been hampered by limited financial resources at the federal and

state levels. The proportion of older persons in the population has also increased[123] and will continue to increase, as the "baby-boom" generation ages. The increased oral health care needs of older Americans could have dramatic effects on the oral health-care delivery system[5,124-126] and the ability to meet the national health objectives for 2010 if personal and public health-care programs are not developed to address the demand.

Limited access to oral health-care services for the special population groups also could affect the ability to meet the national-health objectives for 2010. Only a small proportion of the special population groups have personal dental-insurance coverage, and oral health-care benefits via public programs has not kept pace with changing demands.[127,128] Medicaid expenditures for oral health-care services have decreased by almost 30% since 1987, far more than any other health-care service.[112,129] In 1998, Medicaid expenditures for oral health care represented only 1.3% of the total Medicaid expenditures budget.[112]

Other Trends Affecting Oral Health

Other trends could influence the attainment of national-health objectives, including advances in technology, personnel requirements, and professional education. Advances in implant materials, restorative methods, chemotherapeutic agents, genetics, and the identification of risk markers for disease,[130] for example, should affect personal and public healthcare delivery systems well into the future. Advances in computer technology should lead to developments in all areas of biomedical research, innovative ways to manage and retrieve data, and the provision of health care services.

Human resources are a critical factor in any dental public health program. Changes in the distribution of oral health-care personnel certainly could impact meeting the national health objectives for 2010. Recent data have suggested that the number of dentists will decline during the next 15 to 20 years,[131] however the prediction models used to determine "appropriate" levels of personnel frequently have suffered from a lack of data and generally have been unable to account for epidemiological, social, economic, and political variability over time.[132] Consequently, whether the nation as a whole faces an undersupply of oral health care professionals remains unclear, however, unless actions are taken to address the lack of personal and public healthcare professionals in designated "dental health manpower shortage areas,"[133] it is fairly certain that these parts of the country will find it difficult meeting the national-health objectives.

The professional educational curricula is evolving continuously, as a result of budgetary constraints and redistributions in enrollment, distributions of disease, treatment and health-care delivery systems, information transfer, and demographics. Changes in the curricula generally require additional interdisciplinary research, preventive modalities, and community-based initiatives.[134]

Emerging Public Concerns

Public and professional reactions to perceived risks in the oral health care delivery system affect treatment modalities, service utilization, and ultimately oral health

status. Well publicized reports of individuals contracting a number of conditions from fluoride, and amalgam restorations have prompted the dental research community to review the risks associated with the use of these fundamental components of dental prevention and treatment.[135,136] Of even greater threat to the practice of dentistry and the recruitment of future dental personnel is the fear of contracting an HIV infection/AIDS in the dental office by both health-care providers and patients.[137,138] While dental public-health professionals have been at the forefront in ensuring access for patients infected with the AIDS virus, many dental practitioners are still reluctant to treat known AIDS patients. On the other hand, the revelation of the probable occupational transmission of the AIDS virus from a dentist to five of his patients has generated a high level of concern and anxiety about receiving dental care among the public.[138-140]

Dental public-health activities have been directed at preventing transmission of infectious diseases in the dental office by requiring dentists to comply with recommended ADA and CDC infection control guidelines and the Occupational Safety and Health Administration (OSHA) Bloodborne Pathogens Standard. However, implementation of these edicts is already dramatically changing the scope and cost of delivering oral health-care services in personal and public health-care settings.[141-143]

Surgeon General's Report

In 1997, Donna Shalala, then Secretary of DHHS, commissioned the Office of the Surgeon General to create a report to, "Define, describe, and evaluate the interactions between oral health and general health and well-being (quality of life), through the life span, in the context of changes in society."[144] During the next three years, under the direction of the National Institute of Dental and Craniofacial Research, Project Director Dr. Caswell A. Evans supervised an impressive list of contributing authors and content experts. On May 25, 2000, at Shepherd Elementary School in Washington, D.C., Assistant Secretary for Health and Surgeon General, David Satcher, released *Oral Health in America: A Report of the Surgeon General,*[35] the first-ever Surgeon General's report exclusively dedicated to oral health issues. In his presentation to the Nation that day, Surgeon General Satcher summarized key themes of the report: 1) oral health means much more than healthy teeth, 2) oral health is integral to general health, 3) safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease, and 4) general health-risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health.

The Surgeon General's Report was divided into five parts, each relating to a particular question. Part One asked *what is oral health,* Part Two asked *what is the status of oral health in America,* Part Three asked *what is the relation between oral health and general health and well-being,* Part Four asked *how is oral health promoted and maintained* and *how are oral diseases prevented,* and Part Five asked *what are the needs and opportunities to enhance oral health.* In answering these questions, the Surgeon General's Report listed several findings that reflected the four principal themes:

- Oral diseases and disorders, in and of themselves, affect health and well-being

throughout life.
- Safe and effective measures exist to prevent the most common dental diseases—dental caries and periodontal diseases.
- Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use, and poor dietary choices affect oral and craniofacial health, as well.
- There are profound and consequential oral health disparities within the U.S. population.
- Additional information is needed to improve America's oral health and eliminate health disparities.
- The mouth reflects general health and well-being.
- Oral diseases and conditions are associated with other health problems.
- Scientific research is key to further reduction in the burden of diseases and disorders that affect the face, mouth, and teeth.

The Surgeon General's Report summarized dramatic changes in oral health issues during the last century, and it also brought to light some serious challenges for the future. It stated that, although oral health has improved in the United States, disparities in health still exist. Specific population groups, such as infants and young children, the poor, those residing in rural locations, the homeless, persons with disabilities, racial and ethnic minorities, the institutionalized, and the frail elderly, continue to experience a greater burden of oral and craniofacial diseases. The Surgeon General's Report also stated that there were great disparities in access to oral health care and utilization of preventive services, each crucial to the establishment and maintenance of optimal oral and general health. Finally, the report recognized that there were insufficient data to describe the population subgroups in greatest need for oral health-care services and dental public-health programs. The lack of data will make the development of relevant and effective dental public health programs a more difficult task.

By publishing the Surgeon General's Report, the Office of the Surgeon General has made available important and timely information to health-care practitioners, public-health professionals, policy makers, and the public. For access to the report, the Office of the Surgeon General provides an electronic version of the document and offers a free hardcopy of the report to all who request one.

Summary

The core functions of public health include assessment, policy development, and assurance. These functions are also essential components of dental public health, which is defined as the science and art of preventing and controlling dental diseases and promoting health through organized community efforts. It follows, then, that dental public-health programs are any organized efforts that strive to prevent and control oral and craniofacial diseases at the community level.

Dental disease has been a significant problem for Americans since the nation's early history. Arguably, one of the most successful dental public health programs ever created to address these problems has been community water fluoridation. As successful as fluoridation has been, however, new dental public-health programs need to be developed to meet the needs of population subgroups who have suffered from higher burdens of disease and have had poorer access to timely preventive and

treatment services. The Surgeon General's Report on Oral Health in America highlighted some of these concerns and placed them in the context of existing programs and political realities. In addition, the federal government recognized that one way to address some of the oral health disparities that exist is to establish realistic national health objectives for 2010.

The initiation and implementation of any dental public-health program follows an established planning cycle, the first stage of which involves assessing the oral-health needs of the community. Once a problem is tentatively identified, it is addressed through the use of six sequential steps of the public healthcare practitioner's method—survey, analysis, program planning, program operation, financing, and appraisal. ASTDD established a seven-step model for needs assessment which functions well during the first step.

When traditional dental public-health programs prove ineffective, they must be replaced by more cost-effective approaches. The combination of less disease, more effective use of personnel, and improved technology and preventive methods, particularly dental sealants, provides opportunities to create dental public-health programs for those who have been traditionally neglected. In order to fulfill these opportunities, however, a constituency of public and personal dental and non-dental advocacy groups is required.

Dental public-health programs play a critical role in the promotion and maintenance of oral health in America. The challenge for dental public-health practitioners is to devise programs that are effective, yet incorporate the principles of sound planning and implementation. The oral health of the public depends on it.

Answers and Explanations

1. B, D, and E—correct.

A—incorrect. Children with the greatest treatment needs are usually at the bottom of the economic scale and have fewer resources available. Until access to care for these children is improved, they will continue to be in great need of oral-health treatment services.

C—incorrect. In the United States, the incidence of oral clefts is three times higher among whites than it is among blacks.

2. A and D—correct.

B—incorrect. The State Health Departments are under the administrative control of State government. There is often cooperation between the U.S. Department of Health and Human Services and State Health Departments, however, because many health programs are financed by the federal government.

C—incorrect. The *examination* step of personal-health care is analogous to the *survey* step of public-health care.

E—incorrect. The CPITN is a valid measure of treatment need. A valid measure of

tissue destruction is an assessment of loss of periodontal attachment (LOA).

3. C and D—correct.

A—incorrect. The American Dental Association is not under the auspices of the World Health Organization, however it is a member of the World Dental Federation (FDI).

B—incorrect. The ten Regional Offices are of the U.S. Department of Health and Human Services, not the CDC.

E—incorrect. The Surgeon General's Report on Oral Health in America was the first ever report of its kind.

Self-Evaluation Questions

1. The core functions of public health include _____, _____, and _____.

2. By definition, dental public-health programs are _____.

3. According to 1998 estimates, approximately $_____ was spent on oral health-care services in the United States.

4. *DMFS* represents decayed, missing, and filled tooth surfaces, whereas, _____ represents a caries experience index for primary teeth.

5. By definition, a public-health problem is one that meets the following criteria: _____ and _____.

6. The following are comparative methods used in personal and public healthcare practice:

Six steps of personal-health care Six steps of public-health care

Examination Survey

_____ _____

Treatment planning Program planning

Treatment _____

Payment for services Financing

_____ Appraisal

7. Two dental public-health program strategies for primary prevention of oral and pharyngeal cancer are: _____ and _____.

8. List three national oral health objectives for 2010: _____, _____, and _____.

9. List the four principle themes of the Surgeon General's Report on Oral Health: _____, _____, _____, and _____.

10. Health promotion consists of any: _____.

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Chapter 17. Dental Public-Health Programs - *Mark D. Macek and Harold S. Goodman*

Objectives

At the end of this chapter, it will be possible to

1. List the core functions of public health.

2. Define dental public health and relate this definition to dental public-health programs.

3. Compare the methods of public health-care practitioners and personal health-care practitioners.

4. Describe the seven-step model for assessing oral-health-care needs and relate this model to a planning cycle for public-health programs.

5. Outline the scope of traditional dental public-health programs.

6. Describe recent changes in the United States that are relevant to dental public-health practice.

7. List the various organizations that maintain and support public health programs.

8. Describe how the Surgeon General's report on oral health in America has impacted dental public-health programs.

Introduction

In 1994, the *Core Functions of Public Health Steering Committee,* co-chaired by Drs. Philip R. Lee (Assistant Secretary for Health) and M. Joycelyn Elders (Surgeon General of the U.S. Public Health Service), produced a consensus statement outlining the essential services of public health in the United States.[1] The new statement provided a vision for public health—*Healthy People in Healthy Communities*—and defined its mission: *Promote physical and mental health and prevent disease, injury, and disability.* The consensus statement also provided broader description of the core functions of public health—*assessment, policy development,* and *assurance.*[2] According to the statement, the *purpose of public health* included: 1) preventing epidemics and the spread of disease; 2) protecting against environmental hazards; 3) preventing injuries; 4) promoting and encouraging healthy behaviors and mental health; 5) responding to disasters and assisting communities in recovery; and 6) assuring the quality and accessibility of health services. The *practice of public health* included: 1) monitoring health status to identify and solve community-health problems; 2) diagnosing and investigating health problems and health hazards in the community; 3) informing, educating, and empowering people about health issues; 4) mobilizing community partnerships and action to identify and solve health problems; 5) developing policies and plans that support individual and community health efforts; 6) enforcing laws and regulations that protect health and ensure safety; 7) linking

people to needed personal-health services and assuring the provision of health care when otherwise unavailable; 8) assuring a competent public and personal health-care workforce; 9) evaluating effectiveness, accessibility, and quality of personal and population-based health services; 10) researching for new insights and innovative solutions to health problems.

In 1976, the American Dental Association adopted a definition of *dental public health*, stating that it was:

. . . the science and art of preventing and controlling dental diseases and promoting dental health through organized community efforts. It is that form of dental practice which serves the community as a patient rather than the individual. It is concerned with the dental education of the public, with applied dental research, and with the administration of group dental care programs as well as the prevention and control of dental diseases on a community basis. . .[3].

Given this definition, *dental public-health programs* refer to organized efforts that strive to prevent and control oral and craniofacial diseases at the community level. Dental public-health programs are highly varied and include activities that cover a wide spectrum, from small-scale local projects to large-scale national and international ventures. Given that a community is the focus, dental public-health programs must satisfy the criteria of practicality, feasibility, acceptability, safety, effectiveness, and efficiency.

Historic Perspective

Dental disease has been a significant problem for Americans since the nation's early history.[6] Between 1862 and 1864, loss of teeth was the fourth most frequent cause for rejection of young men for draft into the Union Army during the Civil War.[7] In 1918, military draftees for World War I were rejected—because of defective and deficient teeth—at a rate that exceeded 10% in some states.[8] During the conscription period of World War II, the *U.S. War Department Mobilization Regulation* required that a recruit have a minimum of three serviceable, natural anterior and posterior teeth in opposition, per arch, to be acceptable for military service. Fifteen percent of recruits were rejected, because they could not pass these rather liberal criteria.[6] During the 1920s, the Metropolitan Life Insurance Company conducted one of the earliest epidemiological studies of the dental condition of a large, heterogeneous, adult, civilian population.[9] Oral examinations of more than 12,000 adults revealed that, among 20- to 24-year-olds, more than half of the teeth had been affected by dental caries, and this proportion increased steadily in older age groups.

During the next several decades, the number of epidemiological surveys conducted among civilians increased dramatically.[10-15] It was not surprising that these studies reflected the high dental caries prevalence levels noted in earlier studies and conducted among military recruits. The surveys showed that dental caries was a serious health problem among young adults, and often resulted in tooth loss. The studies also showed that dental caries began early in life and affected young children.

Between 1933 and 1934, the U.S. Public Health Service (USPHS) sponsored a survey conducted among thousands of 6- to 14-year-old children in 26 states across the

United States.[16] The study revealed high dental caries prevalence levels in children, as well. In 1937, the classic Hagerstown, Maryland, study,[17] which introduced the Decayed, Missing and Filled index for teeth (DMFT) and tooth surfaces (DMFS), showed moderately high caries prevalence levels among the examined children. The study also showed that children with the highest dental-caries index scores received only 2% of the treatment time rendered by dentists.

The dental-caries experience of children and the progression of the disease in adults provided the rationale for the application of dental public-health programs to address the problem. The efforts, cooperation, and interactions of a number of individuals and agencies led to one such dental public-health program, the implementation of adjusted water fluoridation.

Fluoridation—A Monumental Public-Health Success Story

Fluoridation is the principal dental public health preventive program available in the control of dental caries in the population. During a national health conference in 1966, former Surgeon General Dr. Luther L. Terry stated, "Controlled fluoridation is one of the four great mass preventive health measures of all time. The four horsemen of health are: the pasteurization of milk, the purification of water, immunization against disease, and controlled fluoridation of water."[18] The Centers for Disease Control and Prevention recently listed fluoridation among the top ten public health triumphs of the 20th century.[19]

The historic development of fluoridation in the United States serves as an example of the contributions of individuals of varied backgrounds representing personal and public segments of the profession. For example, Dr. H. Trendley Dean, considered the "father of fluoridation," had a prominent role in the early developing story of the importance of fluoride to tooth enamel.[20] Dean was an officer in the USPHS who led extensive studies that later established that 1 part per million (ppm) of fluoride in a community water supply reduced dental-caries prevalence.[21]

As important as the contributions of Dean and the USPHS were to the subsequent implementation of community fluoridation, one should not lose sight of the roles played by Dr. Frederick McKay, a personal health-care practitioner in Colorado Springs, Colorado, and Dr. G. V. Black, a practitioner and prominent dental educator. McKay and Black conducted numerous investigations of *Colorado brown stain*, a condition indicative of excessive amounts of naturally occurring fluoride ion during tooth development, and found that dental caries was less prevalent among those afflicted.[22] In addition, one should consider the influence of an industrial chemist, H. V. Churchill, who developed the analytic method that could detect minute quantities of fluoride in water, a critical step necessary to establish the link between the level of fluoride ions in water and the dental caries experience of the population consuming the water.[23] At the same time, Smith and Smith,[24] agricultural researchers, also linked mottled enamel with water fluoride concentrations. Following these and other studies,[25] independent researchers conducted controlled trials of the effect that fluoride ion in a community water system might have on dental caries experience in children. Beginning in 1945 and proceeding through the mid-1950s, researchers added fluoride to the water systems of four test communities (Grand Rapids, Michigan; Newburgh, New York; Evanston, Illinois; and Brantford, Ontario) and observed the

dental caries experience of their residents. These trials successfully demonstrated that adjusted water fluoridation, at concentrations of 1.0 to 1.2 ppm, could dramatically reduce dental caries experience in children.[26-29]

According to the most recent national data available, approximately 162 million persons, or approximately 65.8% of the total U.S. population, drink adjusted or naturally occurring fluoridated water.[30] However, this represents a nearly 4% increase since 1992. Efforts to increase the proportion of the world population drinking fluoridated water still have been thwarted, in part, because of the continuing political activities of the anti-fluoridation movement. Supporters of this movement continue to oppose adjusted fluoridation for many reasons, the vast majority of which are equivocal.[31]

Despite the efforts of opposition groups, community-water fluoridation continues to receive widespread support from both the personal and public health-care sectors. Numerous health professional organizations, consumer and advocacy groups, and the Surgeon General continue to endorse community-water fluoridation.[32-35] Adjustment of water fluoride concentrations to optimal levels is an example of a successful dental public-health program—groups working together to prevent and control oral and craniofacial diseases in the community.

Current Problem

Burt and Eklund[36] define a *public-health problem* as meeting two criteria: a) a condition or situation that is a widespread actual or potential cause of morbidity or mortality; and b) an existing perception the condition is a public-health problem on the part of the public, government, or public health authorities. A number of oral and craniofacial diseases and conditions represent public health problems in the United States today, and are briefly discussed below. These are the principal concerns that need to be addressed by both the personal and public healthcare sectors to improve oral health at the community level.

Dental Caries

Dental caries is one of the most prevalent diseases in the United States. About 17% of children aged 2 to 4 years have had a carious lesion in a primary tooth during their lifetime, and the prevalence jumps to 49.7% among children aged 5 to 9 years.[37] Among permanent teeth, 26.0% of children aged 5 to 11 years have had a carious lesion and 67.3% of children aged 12 to 17 years have had a carious lesion.[37] Dental caries is also highly prevalent among U.S. adults, as approximately 94% of dentate adults aged 18 years or older have had a carious lesion during their lifetime.[38]

Dental-caries prevalence and severity also is associated with race/ethnicity and socioeconomic status (Figure 17-1). Certain minority children exhibit a higher prevalence of primary tooth decay than do their peers, as 34.2% of non-Hispanic white children aged 2 to 9 years have had a carious lesion, whereas 38.8% of non-Hispanic black children and 53.0% of Mexican-American children have had a carious lesion.[37] Among adolescents aged 12 to 17 years, lower poverty status is associated with higher mean dental-caries experience scores and a greater percentage of untreated disease.[39]

Periodontal Diseases

Gingivitis, one of the periodontal diseases, is moderately prevalent in persons aged 13 years or older. On average, 62.9% of persons in this age range exhibit gingival bleeding, and 12.0% of sites are involved.[40] Gingivitis, as measured by gingival bleeding, is also more prevalent among Mexican-Americans than it is among non-Hispanic blacks and non-Hispanic whites aged 30 years or older.[41] Calculus, a contributing factor in gingivitis, is present in 89.9% of persons aged 13 years or older.[40] Although most persons would not consider gingivitis a serious threat to one's health, it receives a great deal of attention in the appearance-conscious United States, given the condition's effect on esthetics and gingivitis precedes, but does not necessarily progress to periodontitis.

Periodontitis is the second of the periodontal diseases and is associated with greater morbidity than is gingivitis, and as such, is considered a more serious public-health problem. On average, 27.0% of males and 17.5% of females aged 13 years or older have at least one site with 5+mm loss of periodontal attachment.[40] This gender difference is statistically significant. The prevalence of attachment loss is also significantly higher among minority groups, as 24.9% of non-Hispanic blacks and 17.1% of non-Hispanic whites aged 13 years or older exhibit the condition.[40]

Oral and Pharyngeal Cancer

There are approximately 30,200 cases of oral and pharyngeal cancer detected in the United States each year, and this number accounts for some 2.4% of all cancers. Of persons with oral and pharyngeal cancer, approximately 7,800 die each year. The overall 5-year survival rate for persons with oral and pharyngeal cancer is 52%, which is lower than that for cancers of the prostate, breast, bladder, larynx, cervix, colon, and rectum.[42] Persons diagnosed with oral and pharyngeal cancer at an early stage have a much better prognosis than do those diagnosed at a later stage, as the 5-year survival rate is 81.3% for early-stage diagnosis and 21.6% for advanced-stage diagnosis. Only 35% of individuals with oral and pharyngeal cancer are diagnosed at an early stage of the disease.[42]

Craniofacial Birth Defects

Oral clefts are among the most common classes of congenital malformations in the United States. On average, there are 1.2 cases of cleft lip (with or without cleft palate) per 1,000 live births and 0.56 cases of cleft palate per 1,000 live births in the general population (Figure 17-2).[43] These defects may affect facial appearance throughout life. Cleft palate occurs more frequently in females, whereas cleft lip or cleft lip/palate occurs more frequently in males.[44-47] The oral cleft incidence rate for whites is more than 3 times the incidence rate for blacks.

Intentional and Unintentional Injuries

It is assumed that injuries to the head, face, and teeth are relatively common, however the majority of our knowledge regarding the number of injuries comes from emergency department data and more severe injuries. The leading causes of such

injuries include falls, assaults, sports injuries, and motor-vehicle collisions.[48-50] According to data collected in 1993 and 1994, there were approximately 20 million visits to emergency departments per year for craniofacial injuries. Falls and assaults each accounted for about 31% of visits and sports-related injuries accounted for approximately 19% of injuries.[51] Injuries resulting from bicycles and tricycles accounted for 5% of head and 19% of face injuries.[52] Overall, 24.9% of persons aged 6 to 50 years have had an injury that resulted in damage to one or more incisor teeth.[53] According to data collected in 1991, personal health-care dentists treated more than 5.9 million craniofacial injuries.[54]

Figure 17-1 Disparities in prevalence of unrestored dental caries exist between poor and non-poor. (From U.S. Department of Health and Human Services. *Oral Health in America: A Report of the Surgeon General.* Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Dental and Craniofacial Research, 2000(35):63.)

Figure 17-2 Incidence of selected congenital defects. (From Schulman et al., 1993.[43])

Dental Public-Health Methods

Personal oral-health-care practitioners serve the oral health needs of individual patients, and the personal health-care delivery system requires a one-on-one interaction between practitioner and individual patient. Public-health dentistry focuses on the community and, as such, does not necessarily require a one-on-one interaction between practitioner and individual patient. When a dental public-health program such as water fluoridation is successfully implemented in a community, a much broader cross section of the community benefits — much broader than could be expected by personal health-care practitioners, alone.

Knutson[55] contrasted the methods employed by personal and public healthcare practitioners. Each consisted of six, sequential steps that permit a logical progression from identification of a problem to its solution (Table 17-1). For the individual patient, a personal healthcare practitioner initiates treatment with a careful examination and history, which leads to an accurate diagnosis of the problem. Afterwards, the personal healthcare practitioner plans a course of treatment. Once treatment services have been provided, and fees paid, subsequent visits provide for evaluation and follow-up. The methods employed in public-health practice parallel those of the personal health-care practitioner, but involve the total community instead of an individual patient. Dental public-health methods are discussed in greater detail below.

Examination versus Survey

When a personal health-care practitioner begins the examination process, he or she collects subjective information from the patient and objective information, such as visual and tactile data, radiographic images, and other signs of disease. By contrast, when a public health-care practitioner assesses the extent of disease in a community, he or she must rely on descriptive information, such as existing survey data or other epidemiological assessments.

Question 1

Which of the following statements, if any, are correct?

A. As a general rule, children with the greatest oral-health treatment needs are also the children who receive priority care.

B. The overwhelming majority of Americans die with at least one carious or restored tooth.

C. The incidence of oral clefts is higher among blacks than it is among whites.

D. Core functions of public health include assessment, policy development, and assurance.

E. Dental public-health programs do not necessarily require a one-on-one interaction between practitioner and individual patient.

Some descriptive survey data have been collected and reported previously. At the national level, surveys such as the National Health Examination Survey, National Health and Nutrition Examination Survey, National Health Interview Survey, and surveys conducted by the National Institute of Dental and Craniofacial Research[56] have provided assessments at the community level regarding the distribution of diseases, such as dental caries and periodontitis, as well as oral health knowledge and behavioral practices. At the state level, surveys such as the Behavioral Risk Factor Surveillance System or cancer registries have provided useful descriptive information regarding oral health care utilization practices and incidence of oral and pharyngeal cancer. Selected states have also administered surveys to assess the oral-health status of their citizens (Figure 17-3).

For the dental public-health-care practitioner, the focus of the survey step is to compile all of the descriptive information that exists in a state, county, region, or local area. When descriptive data do not exist, the dental public-health-care practitioner must find a way to collect useful information. During the mid-1990s, prompted by the newly defined essential functions of public health, the Association of State and Territorial Dental Directors (ASTDD) developed a model for the collection of oral health data at state and local levels,[57] referred to as the *Seven-step Model for Assessing Oral Health Needs.* The seven steps included:

1. Identifying partners and forming an advisory committee.

2. Conducting self-assessment to determine goals and resources.

3. Planning the needs assessment.

- Conduct inventory of available primary and secondary data
- Determine need for primary data collection
- Identify resources
- Select methods
- Develop work plan

4. Collect data.

5. Organize and analyze data.

6. Report findings and utilize the data for program planning, advocacy, and education.

7. Evaluate needs assessment and return to first step, as necessary.

ASTDD intended that the collected data be used as part of a planning cycle (Figure 17-4) that would lead to the implementation of necessary dental public-health programs. Just as a personal health-care practitioner would not consider initiating treatment on a patient without subjective and objective data at hand, the public health-care practitioner would not consider initiating a dental public-health program without descriptive data regarding the needs of the community.

Diagnosis versus Analysis

Once a personal health-care practitioner has gathered sufficient subjective and objective information from the patient, he or she uses professional judgment and experience to diagnosis a disease or condition, if one exists. Once a public health-care practitioner has collected sufficient survey data, he or she analyzes the information in order to answer specific questions. Is there a dental public health problem? If so, what is the extent of the problem? Are there appropriate solutions available to address the problem?

The analysis step helps the public health-care practitioner assess when a problem exists and helps to quantify its extent. Public health-care practitioners rely on standard statistical methods to summarize survey data findings during the analysis step. For example, state-specific survey data may show that 45% of schoolchildren have unrestored dental caries, and that this percentage is significantly higher than would be expected at a national level. The significant difference in percentages may point to a dental public health problem in that state. In addition, survey data may show that oral and pharyngeal cancer incidence in one county is significantly higher than is the rate in a neighboring county. One would expect the first county to receive special attention or a targeted dental public health program. Without the analysis step, however, the difference between the two counties might be less obvious.

In order for a public health-care practitioner to compare analytical findings to other survey data, or transmit analytical findings to other public health-care practitioners, he or she uses standard measurement tools and descriptive guidelines, called dental indexes. A variety of dental indexes have been developed for specific oral and craniofacial diseases and conditions. Some of the more common indexes are listed below.

Dental Indexes

An important tool used in examinations of a population group is a *dental index,* a numeric score that quantifies the magnitude of the disease measured. A number of indexes have been developed for the purpose of providing the objective measurement

of the oral health status of a population group. The number of *teeth* that are decayed, missing, or filled—the DMFT index[17]—is a total score of all affected teeth and provides a dental caries experience score for an individual. A count of *tooth surfaces* that are decayed, missing, or filled is a DMFS index and provides greater precision regarding the dental caries history of an individual or population. The mean DMFT score for a population group is the total average dental caries experience at a particular time. Dental caries experience in the primary dentition is denoted by the use of *lower case letters* to represent the number of decayed, extracted, or filled primary *teeth* and *surfaces; deft* and *defs.*[58] This index has recently been modified to *dft* and *dfs*, because of the difficulty in distinguishing a primary tooth that has been extracted from one that has been lost to the natural process of exfoliation.

The status of periodontal tissues has been evaluated using several indexes. The *Gingival Index* (GI) of Loe and Silness[59] is particularly suited for assessing changes in gingival health that might be observed during the evaluation period of an oral hygiene program. Several plaque indexes have also been developed to assess the status of oral hygiene in population groups. The *Plaque Index* (P) of Silness and Loe[60] quantifies the extent of plaque on defined areas of specific tooth surfaces. The *Oral Hygiene Index—Simplified* (OHI-S) of Greene and Vermillion[61] measures oral debris and calculus on specific tooth surfaces.

The *Periodontal Index* (PI) of Russell[62] and the *Periodontal Disease Index* (PDI) of Ramfjord[63] were once used for assessing the severity of periodontitis, but are no longer considered valid. When these indexes were developed, it was believed that gingivitis and periodontitis were on a continuum; as gingivitis became more severe, periodontitis resulted. Consequently, the PI and PDI were developed as composite indexes, assessing gingivitis and periodontitis together. Today, it is well established that gingivitis does not necessarily lead to periodontitis, and that the two diseases are unique. Although the PI and PDI are no longer used, the PDI left behind a measurement component that is valid for assessing tissue destruction. The surviving measurement component, sometimes referred to as *loss of attachment* or *LOA*, calculates the loss of periodontal attachment that has occurred adjacent to a tooth. The *Community Periodontal Index of Treatment Need* (CPITN) is not an index of periodontitis, but a measure of the necessity for periodontal treatment.[64] The CPITN has been used by nations around the world.

When public healthcare practitioners employ a dental index during the analysis step, they must pay particular attention to the training of examiners. Consistency in the application of scoring criteria is paramount to the validity of index scores. A comparison of DMFT scores from one county to another would be of little value, for example, if the examiners in the two counties applied the scoring criteria in different ways.

Treatment Planning versus Program Planning

Once a personal health-care practitioner has identified a disease or condition, and assessed its extent, he or she is ready to transmit the information to the patient and plan a treatment strategy. Once a public health-care practitioner has identified the existence of a dental public-health problem and assessed its extent, he or she is ready to transmit the information to concerned individuals and community partners.

Together, the public health-care practitioner and partners develop a public-health program that is tailored to the needs of the community.

During the treatment planning and program planning steps, decisions must take into consideration such factors as available time, finances, knowledge, experience, attitudes, and willingness to complete the plan. Just as an individual patient must consider his or her personal circumstances when selecting treatment options, community leaders must consider community resources and priorities when selecting appropriate public health program options.

Treatment versus Program Operation

Once the patient and personal health-care practitioner have decided on an appropriate treatment plan, treatment of the disease or condition begins. Once the community and public healthcare practitioner have decided on an appropriate program plan, the public health program is set in motion. Program operation usually includes three features, including health education, disease prevention, and provision of services.[65] Given that administrations change, resources shift, and attitudes and motivations evolve, the program operation step is never static. Community interventions are generally more difficult to orchestrate than are plans that address an individual, because more factors must be taken into consideration at the community level.

Figure 17-3 Surveys designed to establish the oral health needs of children frequently take place in a school setting. (Courtesy of Dr. Arthur Benito, Research Triangle Institute, North Carolina.)

Figure 17-4 Dental public-health program planning cycle.

Question 2

Which of the following statements, if any, are correct?

A. *Treatment planning* requires the input of the personal healthcare practitioner and informed consent of the patient, whereas *program planning* requires the input of the public-health dentist and informed consent of involved community leaders.

B. Individual State Health Departments operate under the administrative control of the U.S. Department of Health and Human Services.

C. The *examination* step of personal health care is analogous to the *analysis* step of public health care.

D. Community-water fluoridation campaigns often fail because of political issues—not because of health department decisions.

E. The *Community Periodontal Index of Treatment Need* (CPITN) is a valid measure of periodontal tissue destruction.

Payment for Services versus Financing

For the individual patient facing a treatment plan, the scope and extent of treatment

services depend on personal resources and/or the existence of third-party payment plans. For the community looking forward to the initiation of a dental public-health program, the scope and extent of the program depend on the existence of available public and personal health-care funds. In most cases, public programs are funded via the federal government or the state. Program administration and funding typically originate from state-level health departments, or county-level or local area-level health departments, when they exist.

Evaluation versus Appraisal

When a personal health-care practitioner completes an individual patient's treatment plan, he or she evaluates the individual during periodic intervals, to assure that the oral health is maintained and any arising treatment needs are identified and met. The responsibilities of the public health-care practitioner are comparable. During the appraisal step, the public health-care practitioner first needs to assess whether the program has adequately addressed the needs of the community. As such, all public-health programs should have a measurable set of objectives against which success or failure may be appraised. If, for example, a dental public-health program were initiated to reduce the oral-cancer incidence in a county experiencing unusually high rates, then the program should contain a target incidence rate that would signify success. Once the public health-care practitioner has assessed whether an objective has been met, he or she must monitor the existence of a public health program on a regular basis. If new survey data are required, the public health-care practitioner should secure them. If the standard against which success is judged should change, the public health-care practitioner should reassess whether the program would be considered a success.

For both the personal and public health-care practitioners, the evaluation and appraisal steps represent the link between the end of a treatment plan or public-health program and the beginning of a new plan or program. As long as individual patients have treatment needs or communities have public-health problems, the six steps of the personal and public health-care practitioner can be applied.

An Example of a Dental Public-Health Program

A *dental sealant* is a plastic material that is applied to the pit-and-fissure surfaces of the teeth by oral health-care professionals. Dental sealants function as a primary preventive agent against dental caries by obstructing the pit-and-fissure surface from bacteria. Dental sealants also may serve as a secondary preventive agent against dental caries when applied to incipient lesions. Dentists and dental hygienists apply dental sealants in private health-care facilities, however this means of providing the preventive agent is limited by access to the facilities and the personal circumstances of the patients in need.

This section of the chapter presents a dental public health problem in a fictitious community called *Yourtown* and proceeds through the six steps of the public healthcare practitioner's method, in order to illustrate how a dental sealant campaign might be employed as an effective dental public health program (Figure 17-5). Although this exercise describes a specific problem and program solution, the principles may be applied more broadly to other problems and solutions.

Survey

The survey step of the process encompasses the seven-step model of assessing oral health needs. Dr. Sally Sealem, the county health officer in Yourtown, began the process by identifying partners and forming an advisory committee of interested parties. Dr. Sealem asked administrators from the school board to join her, as well as staff members from the health department, the director of the dental hygiene training program at Yourtown Community College, the state dental director, and the administrator of a nonprofit health-care facility.

During the advisory committee's first meeting, the members discussed their resources and limitations. Dr. Sealem knew that the most cost-effective dental sealant program would involve school-aged children. The school administrators assured the health officer that a dental sealant program would be welcomed into the local schools. The administrators also said that the principals, teachers, and school nurses would be willing to coordinate communication with parents and students. Dr. Sealem also recognized that oral health-care professionals would need to participate in the program. The dental director told her that he would discuss the proposed program with the state and local dental society. He was fairly sure that the dental society would embrace the program and provide the names of a few retired practitioners who might be interested in volunteering their time to the program. The director of the training program in dental hygiene also offered the assistance of her faculty and students.

The dental director also said that there was little money in the budget for a dental sealant program. Upon hearing that the dental director's budget did not allow for a dental sealant program, the administrator of the non-profit healthcare facility said that their treatment clinic would be willing to donate some money and supplies to the program and the attendees from the health department said that they would look into the existence of grant money from private corporations, community groups, and the federal government.

Prior to the meeting, Dr. Sealem compiled demographic data for Yourtown and all of the relevant data regarding dental caries and preventive oral health programs. She discovered that the community contained approximately 10,000 school-aged children. Most of these children lived within 10 miles of their respective schools, however a few were transported via bus from neighboring rural areas. The socioeconomic status (SES) profile of the community was relatively low, with approximately 56% of children qualifying for free or reduced meals at school. Dr. Sealem also learned that Yourtown did not have access to fluoridated community water. Dr. Sealem had no data describing the dental caries prevalence or the prevalence of dental sealants in Yourtown, however she did have access to data from several national surveys.

The advisory committee recognized that survey data from Yourtown would have provided a more complete picture of the oral-health conditions in their community than the national data, but they also recognized that in order to collect such data, they would have to conduct a survey for which they had limited resources. Given the circumstances, the advisory committee ultimately decided that they would rely on the national data to draw conclusions about their community.

Analysis

From studies of national data,[39] Dr. Sealem knew that dental caries prevalence was higher among poor children than it was among their non-poor counterparts. She also knew that the percentage of unrestored disease was higher among the poor children. In addition, national studies showed that only 18.5% of children aged 5 to 17 years had one or more sealed teeth.[66] Given that there was a sizeable proportion of poor children in Yourtown and given that national survey data showed that poor children had greater needs, the advisory committee concluded that there was good reason to initiate a dental sealant program in their community.

Program Planning

During the program-planning stage, the advisory committee listed all of the possible ways to implement a dental-sealant program in Yourtown. Some of the options included use of a mobile dental van, visits to churches and other meeting places, expansion of services at the health department, expansion of services at the nonprofit health-care facility, and a school-based program. In deciding on the best approach, the advisory committee considered available resources and potential advantages and disadvantages of each option. Given that school administrators provided ready access to schools, and because this was where the majority of children could be found, the advisory committee decided that they would use a school-based dental-sealant program. They also decided that they would use students from the dental-hygiene training program at the community college to educate parents and teachers about the benefits of this preventive oral-health measure, and they would use the retired dentists from the community to administer the dental sealants.

In recognition of the budgetary constraints, the nonprofit health-care facility provided disposable gloves, masks, dental mirrors, and tongue blades to the program. In addition, staff members from the health department were able to procure grant funding from a local philanthropic organization and dental sealant materials from a national dental supply distributor. The advisory committee used the grant funds to purchase a portable dental chair, generator, and light source.

Program Operation

After thorough consideration of the problem and analysis of its severity, careful planning, and procurement of funding, the school-based dental sealant program was put into operation. In preparation for the initiation of the program, in-service training programs were conducted for all participants to affirm goals and standardize treatment protocols. The application of dental sealants to the school children progressed well, because the advisory committee had paid such careful attention during the previous stages.

Financing

Although the advisory committee was able to solicit the necessary funds for the first year of the dental sealant program, they realized that in order for the program to have a lasting impact, they would need to procure new funding over time. The great success of the program made this step relatively easy. The advisory committee created

press releases and gave them to the print media. Dr. Sealem asked the local television stations to interview her during "health spots" on the local news. The advisory committee capitalized on the popularity of the program among parents and community leaders by asking them to request additional funds from their legislative representatives for the state's health budget. Staff members at the health department wrote new grant applications and continued to solicit funds of other agencies and organizations.

Appraisal

The advisory committee used national data to determine whether their community would be a good candidate for a dental sealant program. This approach was satisfactory for the initiation of the program, but it would not suffice during the appraisal stage. In order for the advisory committee to evaluate whether the dental sealant program had been successful in reducing dental caries experience in Yourtown, they would need new data; a baseline assessment and periodic assessments of dental-caries and dental-sealant prevalence among the school children.

The appraisal stage is arguably one of the most difficult components of a dental public health program. It requires careful delineation of measurable goals and objectives and a detailed plan to collect evaluation data over many years. The appraisal stage must take into consideration the benefits of the program and weigh them against the cost. It must also consider alternative preventive and treatment regimens as they develop, and assess whether these new strategies might be a better option.

Enthusiasm and excitement frequently drive the first few years of a new program, however funding agencies and legislators will eventually demand that their resources are being applied to an efficient and effective program. Without a valid appraisal plan in place, the ability for an administrator such as Dr. Sealem to demonstrate efficiency and effectiveness is all but impossible.

Although this may be the most difficult component of a dental public health program, administrators have a number of resources at their disposal. Health departments typically have epidemiologists and survey researchers available for consultation. ASTDD and the Division of Oral Health at the Centers for Disease Control and Prevention also have consultants available.

Figure 17-5 School-based dental sealant programs have been found to be an effective approach to reducing dental caries in pit-and-fissure tooth surfaces. (Courtesy of Ohio Division of Dental Health.)

Levels of Dental Public Health Operation

There are numerous international and national organizations that have as a primary or secondary focus, the prevention and control of oral and craniofacial diseases at the community level. At the international level, the World Health Organization (WHO) has accepted the responsibility of coordinating the efforts of all member organizations in developing and improving oral and medical health programs throughout the world. WHO has several regional offices located throughout the world that aid in administering programs on a local level.

Based in Washington, D.C., the Pan American Health Organization (PAHO) is one such regional office for the Americas. Member States of PAHO include all 35 countries in the Americas and Puerto Rico is an Associate Member. France, the Netherlands, and the United Kingdom of Great Britain and Northern Ireland are Participating States, and Portugal and Spain are Observer States. The mission of PAHO is to strengthen national and local health systems and improve the health of the peoples of the Americas. It works in collaboration with Ministries of Health, other government and international agencies, nongovernmental organizations, universities, social security agencies, community groups, and many others. PAHO targets the most vulnerable groups, including mothers and children, workers, the poor, the elderly, and refugees and displaced persons. It focuses on access issues and a Pan-American approach, encouraging nations to work collab- oratively on common issues.

The World Dental Federation (FDI) is an independent, professional organization for dentistry. The activities of the FDI cover all aspects of personal and public oral healthcare and take place all over the world. Among its varied responsibilities, FDI contributes to the development and dissemination of statements regarding policies, standards, and information related to oral health care. In addressing this responsibility, FDI produces the statements via its Scientific Commission or in collaboration with other professional organization throughout the world.

At the national level, the U.S. Department of Health and Human Services (DHHS) is the Cabinet-level branch of the federal government that is responsible for the planning and implementation of a broad array of health programs, from support for and protection of Americans of all ages, to aid for persons with disabilities, as well as assistance and new opportunities for those in need. In short, DHHS is responsible for public health in the United States, supporting the world's largest medical research effort, assuring the safety of foods and health care products, and fighting the ravages of drug and alcohol abuse. Planning begins in Washington, D.C., with objectives evolving as health needs shift. For example, at one time there was a need to finance new dental and medical schools to increase the output of health professionals; more recently there has been a need for specifically focused programs to accelerate development of control measures for either caries or periodontal disease, and continually there are efforts to refine programs offering better access to, or less cost for, medical and oral healthcare. DHHS responsibilities in the United States are divided into 10 geographic regions (I to X), each one having a central office. These offices facilitate administration by providing consultation and monitoring expertise for regional and local health programs involving federal funds.

DHHS oversees 12 major organizations, each with a different influence over public health issues and dental public health programs (Figure 17-6). The *Administration for Children and Families* (ACF) is responsible for numerous programs that provide services and assistance to needy children and families, administers the new state-federal welfare program (*Temporary Assistance to Needy Families*), administers the Head Start program, provides funds to assist low-income families in paying for child care, and supports state programs to provide for foster-care and adoption assistance. The *Health Resources and Services Administration* (HRSA) helps provide health resources for medically underserved populations, supports a nationwide network of community and migrant health centers and primary care programs for the homeless and residents of public housing, works to build the health-care workforce, maintains

the National Health Service Corps, works to improve child health, and provides services to persons with AIDS through the Ryan White CARE Act programs. The *Agency for Healthcare Research and Quality* (AHRQ) supports investigator-initiated research designed to improve the outcomes and quality of health care, reduce its costs, address patient safety and medical errors, and broadens access to effective services. The *Centers for Disease Control and Prevention* (CDC) administer a health surveillance system designed to monitor and prevent outbreaks of disease. It also guards against international disease transmission, maintains national-health statistics and provides for immunization services and supports research into disease and injury prevention. The CDC's Division of Oral Health maintains and reports on national and local oral-health surveillance data, consults with states and local health departments regarding oral-health assessments and survey techniques, administers the Water Fluoridation Reporting System, and publishes policy statements regarding control of infection. The *Agency for Toxic Substances and Disease Registry* (ATSDR) works with states and other federal agencies to prevent exposure to hazardous substances from waste sites. The Substance Abuse and Mental Health Services Administration (SAMSHA) strives to improve the quality and availability of substance abuse prevention, addiction treatment, and mental health services. The *Administration on Aging* (AoA) provides and supports ombudsman services for elderly, and provides policy leadership on aging issues. The *Food and Drug Administration* (FDA) assures the safety of foods and cosmetics, and the safety and efficacy of pharmaceuticals, biological products, and medical devices, including those used in personal oral health care settings and dental public health programs. The *Centers for Medicare and Medicaid Services* (CMS), formerly *Health Care Financing Administration* (HCFA), serves the needs of Medicaid and Medicare beneficiaries. The *Indian Health Service* (IHS) oversees and supports a network of hospitals, health centers, school-based health centers, health stations, and urban Indian health centers that provide services to nearly 1.5 million Native Americans and Alaska Natives. The *National Institutes of Health* (NIH), the world's premier medical research organization, supports research projects nationwide in diseases like heart ailments, diabetes, cancer, HIV, Alzheimer's Disease, and asthma. The *National Institute for Dental and Craniofacial Research* (NIDCR), one of the NIH institutes, supports intramural and extramural research regarding dental caries, periodontitis, oral and pharyngeal cancer, facial clefts, oral health disparities, and preventive oral health therapies. The *Program Support Center* (PSC) provides, for a fee, solution- and customer-oriented support for administrative operations, financial management and human resources throughout DHHS, as well as other departments and federal agencies.

The USPHS encompasses the Commissioned Corps, the uniformed service of the DHHS. Dental officers in the Commissioned Corps serve the oral health treatment needs of Native Americans and Alaska Natives as part of the IHS; active duty members, dependents, and retirees of the U.S. Coast Guard; and persons incarcerated under the Federal Bureau of Prisons. The Surgeon General leads the Commissioned Corps of the USPHS.

Each state has a health department that may or may not include an oral-health division. Of those states with an oral health focus, many divide their jurisdictional operation into regions to better administer and monitor state-administered oral-health programs. The regional programs include operation of clinics for needy populations, state prison systems, and in some cases, school systems. Consultations with

communities desiring to establish or to improve community oral health, public health education programs, and fluoride initiatives receive major emphasis.

Within each state, populous counties and cities may administer community oral health treatment clinics through local health departments. These clinics usually operate in schools, economically underprivileged areas, or among population subgroups that do not otherwise have access to routine personal oral-health care. Federal, state, and local tax funds are intermixed in the delivery of care at all levels.

Figure 17-6 Organizational chart for the U.S. Department of Health and Human Services.

Dental Public Health Programs

Health Promotion and Health Education

Health promotion and health education are integral components of most successful dental public-health programs (Table 17-2). *Health promotion* consists of any planned combination of educational, political, regulatory, and organizational supports for actions and conditions conducive to the health of a community or group of individuals in a defined geographic location (67). Projects designed to be administered in schools, such as fluoride mouthrinse programs and dental-sealant programs, have been particularly successful, because dental caries is prevalent in children and those with the greatest needs may reside with parents/guardians who are otherwise unable to provide for their treatment needs in personal healthcare facilities. School-based health care programs are discussed elsewhere in this textbook. Health-promotion activities do not require active participation of its recipients, however. Public sanitation measures, for example, promote health among humans around the world, yet most of these persons enjoy the benefits without action or awareness. Consequently, health-promotion activities are vitally important to dental public-health programs, because they do not usually depend on recipient awareness or cooperation for success.

Question 3

Which of the following statements, if any, are correct?

A. The American Dental Association is under the auspices of the World Health Organization.

B. The health policies of the U.S. Department of Health and Human Services are administrated from the 10 Regional Offices of the Centers for Disease Control and Prevention.

C. Approximately 144 million persons in the United States drink fluoridated water.

D. Health promotion frequently yields more immediate effects on the public than does education of the public.

E. The Surgeon General's Report on Oral Health in America, released in 2000, was one of many such reports dedicated to dentistry.

Health education includes any combination of learning experiences designed to enable the voluntary adoption of behaviors or actions that are conducive to health and healthful living (Figure 17-7).[68] Whereas health-promotion activities do not require the active participation of its recipients, health education does. For this reason, dental public-health programs that rely heavily on health education are subject to the attitudes, beliefs, and other motivating factors of the recipients. In addition, although knowledge is an important element of empowerment, knowledge does not guarantee that appropriate actions or behavioral changes will follow.[69] Health-promotion activities in dental public health programs frequently include health-education components, but health-education, alone, is not sufficient to prevent oral diseases or conditions.

Community-Water Fluoridation

Community-water fluoridation, or the addition of appropriate concentrations of fluoride compounds into water systems to prevent dental caries, is a health promotion activity within a dental public health program. As beneficial as water fluoridation is in the battle against dental caries, the addition of fluoride to water is not an automatic condition, however, and frequently requires the savvy and careful coordination of dental public-health professionals, water engineers, legislators, and organized dentistry. In order to conduct a successful fluoridation campaign, one must understand political realities and recognize available resources in the community that can be used to assist in securing a favorable outcome.[70] Successful campaigns require dedicated and enthusiastic persons who are coordinated by an individual with good political skills. Support from all segments of the population, not just health professionals, is crucial. The best method to achieve fluoridation in a small community is through city council action if state laws do not require a referendum. Endorsements offered by strategic role models, such as a mayor, city council member, or other community leaders, play an important role in the process.

Coordination of activities is also important once a water fluoridation program has been initiated. Studies show that water distribution centers frequently maintain aqueous fluoride concentrations that are lower than recommended levels.[71] In order to combat this reality, authority should rest with an administrator who is dedicated to the dental public health program and who is in a position to manage the system. Frequently, the dental director of the state health department is an ideal choice for the administrative position. When a dental director is unavailable, dental public health professionals should assign a person who has the responsibility of water fluoridation surveillance and management.

Special Population Groups

Selected dental public-health programs include projects that focus on particular population subgroups. Certain groups, for example, because of health status, position in society, attitudinal barriers, or geographic location, do not have ready access to personal healthcare providers and must receive care in special clinics, supported by public or private funds. The oral health care needs of these groups, which include Native Americans and Alaska Natives, long-term-care populations, migrant groups, medically compromised individuals, beneficiaries of the Department of Veterans Affairs, persons with developmental disabilities, homeless individuals, the elderly,

and persons with low socioeconomic status, are usually significantly greater than they are for the general population.[56,72-85] For example, medically compromised individuals and persons with Acquired Immunodeficiency Syndrome (AIDS) are frequently predisposed to rapidly progressing periodontitis and other oral problems.[86,87] Alzheimer's disease and other dementias compro- mise the ability of many older persons to take care of their mouths.[88, 89]

The abilities and limited experience levels of some personal oral health-care professionals, as well as the conditions within which they work may stand in the way of effective provision of care for some of these special population groups.[90-92] For example, routine treatment facilities are frequently inaccessible to a person who is homebound because of physical or mental disabilities or limitations. Dentists and auxiliaries, trained in the use of mobile treatment equipment and management of the disabled patient, are necessary in order to provide oral-health care to the homebound.[93] In this example, the removal of barriers to care is an example of an effective dental public-health program.

Dental public-health professionals in public-health agencies, local or state health departments, and academic institutions are called upon to provide consultation or initiate programs for individuals with particular diseases or conditions. Examples may include educational programs aimed toward mothers and designed to address feeding behaviors leading to early childhood caries, programs designed to produce mouth guards for high school athletes, programs designed to assess the function of removable prostheses in a geriatric population, programs designed to provide fluoride therapy to cancer patients undergoing head and neck radiation, programs designed to screen low-income children for oral diseases, or programs designed to provide information regarding oral and pharyngeal cancer prevention.

Figure 17-7 Classroom dental-education programs are important, but it is critical to evaluate their effect. (Courtesy of the National Institute of Dental and Craniofacial Research.)

New Strategies Needed

Changing Disease Patterns

During the early 1900s, acute infectious diseases were more prevalent than they are today and accounted for greater morbidity and higher mortality among the general population. During the 1950s and 1960s, with the advent of immunizations and antibiotics, public health professionals began to shift their attention to chronic diseases, such as heart ailments, cancer, strokes, and diabetes. Dental public health programs have had to adapt to changing disease prevalence, as well.

One of the truly significant developments in dental public health has been the decline in dental-caries prevalence during the past 15 years.[56, 94-102] Reduced susceptibility to dental caries, particularly among children and young adults, is altering the oral-health status of the population. NIH estimated that the United States saved approximately $100 billion in dental expenditures during the 1980s as a result of this improvement in oral health.[103] The change in dental-caries prevalence represents a major success for personal oral health preventive and treatment services and dental public health programs, but it also presents new challenges to the profession.

During the early-1900s, dental caries was highly prevalent across age groups and population sub-groups. Everyone required treatment services.[104] Today, as a result of effective prevention and improved treatment regimens, dental caries is concentrated in a substantially smaller proportion of the population. The challenge to dental public-health professionals is to concentrate on identifying high-risk individuals and expanding services for those who have not had access to care. Current trends to decrease spending for public programs as well as reduce health-care costs should favor preventive programs that are targeted to those who have higher unmet levels of oral disease.[105-106]

Changing Public-Health Practices

Dental public-health programs should be organized to meet the needs of the population. As needs change, dental public health efforts should evolve to address these changing needs.[107] An accepted characteristic of a profession is that it shall be willing to respond to changing needs as a result of its own successful preventive and treatment activities.[108] Concern over the current ability of the public-health profession to adapt to change is addressed in an Institute of Medicine report, entitled *The Future of Public Health.*[109] The report contends that public health in the United States is disorganized, splintered, and unprepared to accommodate and address future challenges. The report goes on to state that the means to maintain and expand public health programs and meet the demands of a changing environment is via assessment, policy development, and assurance.

Contrary to the report's recommendations, political and economic forces in the United States have served to reduce or discontinue many dental public health programs. The decline of dental public health programs at the national, state and local levels is, in part, a result of the perception that oral health is not a major concern.[110] Neighborhood, rural, migrant, and homeless health centers have suffered severe cutbacks in federal outlays for oral health care services, personnel, and scope of programs.[110] Public-health dentistry curricula in many schools of public health are experiencing major reductions or dissolution. Many community-dentistry programs in dental schools are only modest in scope, relative to the concentration of resources devoted to these programs when first initiated.

Why has the downsizing of dental public-health programs progressed with relatively few challenges? One answer may be the lack of an organized constituency or advocacy group for dental public-health issues. A partnership between the public and personal health-care dental sectors is essential if oral health concerns are to be effectively promoted. Often the aims of professional groups within dentistry tend to be compartmentalized and narrowly defined. Public dental programs may also be seen as competitive with personal healthcare practitioners. Preventive approaches are apt to be erroneously classified as public sector or personal healthcare sector programs. Yet the efforts of both should reinforce common goals. Fluoridation, for example, may be seen as an effective public health measure but the promotion of fluoride dentifrices may not be. Yet they complement each other and both are public-health measures.[111]

Cooperation between dental public-health organizations, such as, the American Association of Public Health Dentistry (AAPHD), the American Public Health

Association (APHA) Oral Health Section, and the American Dental Association (ADA) could help resolve the differing perspectives of the personal healthcare and public sectors. Cooperation could also foster an influential alliance in local and national campaigns addressing dental public health issues. Collaboration with a multitude of national and local voluntary non-dental health and educational organizations, such as, the Children's Defense Fund, American Association of Retired Persons, or the National Health Education Coalition is equally important to promote oral health as essential to overall health and to integrate oral health issues within the health, educational, and policy directives of these organizations. By working together on certain broad-based popular issues (i.e. access to health services), these separate partnerships can evolve into a coalition, such as the National Oral Health Alliance that can be recruited to actively support specific oral health issues.

In 1998, 53.8 billion dollars were spent on oral health care services, representing about 4.7% of the total health expenditures budget for that year.[112] Expenditures for oral health care services increased between 1997 and 1998 at approximately the same rate as expenditures for medical health care (5.3 versus 5.6%). Although these figures suggest that oral health-care services were adequately funded, comparisons with funding levels from earlier decades paint a different picture. In 1960, for example, 2 billion dollars were spent on oral health-care services, but this represented 7.3% of the total budget.[35] With reductions in funds to support oral health-care services, public-health program administrators will have to become more opportunistic and adaptive in order to conduct effective programs.

Other advocacy measures that can be pursued in support of dental public-health programs may be advanced through regulatory and legislative routes. An area of activity often entered with some reluctance is the political arena. Those in dental public-health programs characteristically go about their duties quietly, content to live within the constraints imposed by citizens who, for example, vote against fluoridation. Niessen believes that there are community regulatory roles for dental public health regarding compliance with fluoridation and infection control standards.[113] If successful, efforts to educate and persuade others of the importance of these issues could pay big dividends. The preventive benefit provided to a community by initiating and/or monitoring fluoridation or a practice act that addresses infection control may be greater than the benefit attained from a lifetime of practice by a dozen dentists.

Successful public-health workers need to be opinion leaders and community decision makers regarding oral health programs and services. Gaupp expands this notion when stating that, "It is opportune for the oral health interest groups to strike out on their own by working toward a national, comprehensive, oral health bill."[114] Resource development could also be expedited if dental public-health programs attained influence in the regulatory and legislative arenas.

National Oral-Health Objectives

The USPHS recognizes that an effective means to expand advocacy and regulatory activities and generate support for oral-health programs is via the setting of measurable and achievable, national health objectives. In 1980, the federal government established a program, entitled *Promoting Health/Preventing Disease: Objectives for the Nation, 1990*,[115] to identify and monitor a variety of health

objectives, including 12 that addressed oral health and fluoridation. Although this program provided an early opportunity to promote oral health alongside other national health priorities, it did not adequately address the means by which states and localities could meet the objectives. Subsequent national-health objectives for 2000[116] built upon the previous framework, by providing strategies that would be helpful in meeting the new objectives. Toward that end, the USPHS outlined twenty-nine measurable oral health objectives and indicators in another document, entitled *Healthy Communities 2000: Model Standards,*[117] and called for periodic reports[118-200] and consortia[121] to promote the national health objectives for 2000.

In 2000, the USPHS released national health objectives for 2010, which included an oral health focus area[122] (Table 17-3). These oral health objectives differed from previous ones, in that they incorporated a "better than the best" standard for setting goals, as opposed to setting disparate goals for certain population sub-groups. For example, the best value attained for any single population subgroup in 2000 was used to determine the goal for all population sub-groups in 2010. The rationale behind this standard-setting method was to establish a single high goal for all groups, rather than to perpetuate disparities over time.

Special Populations

During the last two decades, the United States has experienced an increase in the number of special population groups, including persons in long-term care,[74] medically compromised individuals,[78] and the homeless.[81,82] Higher unmet needs in these special population groups has been hampered by limited financial resources at the federal and state levels. The proportion of older persons in the population has also increased[123] and will continue to increase, as the "baby-boom" generation ages. The increased oral health care needs of older Americans could have dramatic effects on the oral health-care delivery system[5,124-126] and the ability to meet the national health objectives for 2010 if personal and public health-care programs are not developed to address the demand.

Limited access to oral health-care services for the special population groups also could affect the ability to meet the national-health objectives for 2010. Only a small proportion of the special population groups have personal dental-insurance coverage, and oral health-care benefits via public programs has not kept pace with changing demands.[127,128] Medicaid expenditures for oral health-care services have decreased by almost 30% since 1987, far more than any other health-care service.[112,129] In 1998, Medicaid expenditures for oral health care represented only 1.3% of the total Medicaid expenditures budget.[112]

Other Trends Affecting Oral Health

Other trends could influence the attainment of national-health objectives, including advances in technology, personnel requirements, and professional education. Advances in implant materials, restorative methods, chemotherapeutic agents, genetics, and the identification of risk markers for disease,[130] for example, should affect personal and public healthcare delivery systems well into the future. Advances in computer technology should lead to developments in all areas of biomedical research, innovative ways to manage and retrieve data, and the provision of health

care services.

Human resources are a critical factor in any dental public health program. Changes in the distribution of oral health-care personnel certainly could impact meeting the national health objectives for 2010. Recent data have suggested that the number of dentists will decline during the next 15 to 20 years,[131] however the prediction models used to determine "appropriate" levels of personnel frequently have suffered from a lack of data and generally have been unable to account for epidemiological, social, economic, and political variability over time.[132] Consequently, whether the nation as a whole faces an undersupply of oral health care professionals remains unclear, however, unless actions are taken to address the lack of personal and public healthcare professionals in designated "dental health manpower shortage areas,"[133] it is fairly certain that these parts of the country will find it difficult meeting the national-health objectives.

The professional educational curricula is evolving continuously, as a result of budgetary constraints and redistributions in enrollment, distributions of disease, treatment and health-care delivery systems, information transfer, and demographics. Changes in the curricula generally require additional interdisciplinary research, preventive modalities, and community-based initiatives.[134]

Emerging Public Concerns

Public and professional reactions to perceived risks in the oral health care delivery system affect treatment modalities, service utilization, and ultimately oral health status. Well publicized reports of individuals contracting a number of conditions from fluoride, and amalgam restorations have prompted the dental research community to review the risks associated with the use of these fundamental components of dental prevention and treatment.[135,136] Of even greater threat to the practice of dentistry and the recruitment of future dental personnel is the fear of contracting an HIV infection/AIDS in the dental office by both health-care providers and patients.[137,138] While dental public-health professionals have been at the forefront in ensuring access for patients infected with the AIDS virus, many dental practitioners are still reluctant to treat known AIDS patients. On the other hand, the revelation of the probable occupational transmission of the AIDS virus from a dentist to five of his patients has generated a high level of concern and anxiety about receiving dental care among the public.[138-140]

Dental public-health activities have been directed at preventing transmission of infectious diseases in the dental office by requiring dentists to comply with recommended ADA and CDC infection control guidelines and the Occupational Safety and Health Administration (OSHA) Bloodborne Pathogens Standard. However, implementation of these edicts is already dramatically changing the scope and cost of delivering oral health-care services in personal and public health-care settings.[141-143]

Surgeon General's Report

In 1997, Donna Shalala, then Secretary of DHHS, commissioned the Office of the Surgeon General to create a report to, "Define, describe, and evaluate the interactions

between oral health and general health and well-being (quality of life), through the life span, in the context of changes in society."[144] During the next three years, under the direction of the National Institute of Dental and Craniofacial Research, Project Director Dr. Caswell A. Evans supervised an impressive list of contributing authors and content experts. On May 25, 2000, at Shepherd Elementary School in Washington, D.C., Assistant Secretary for Health and Surgeon General, David Satcher, released *Oral Health in America: A Report of the Surgeon General,*[35] the first-ever Surgeon General's report exclusively dedicated to oral health issues. In his presentation to the Nation that day, Surgeon General Satcher summarized key themes of the report: 1) oral health means much more than healthy teeth, 2) oral health is integral to general health, 3) safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease, and 4) general health-risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health.

The Surgeon General's Report was divided into five parts, each relating to a particular question. Part One asked *what is oral health,* Part Two asked *what is the status of oral health in America,* Part Three asked *what is the relation between oral health and general health and well-being,* Part Four asked *how is oral health promoted and maintained* and *how are oral diseases prevented,* and Part Five asked *what are the needs and opportunities to enhance oral health.* In answering these questions, the Surgeon General's Report listed several findings that reflected the four principal themes:

- Oral diseases and disorders, in and of themselves, affect health and well-being throughout life.
- Safe and effective measures exist to prevent the most common dental diseases—dental caries and periodontal diseases.
- Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use, and poor dietary choices affect oral and craniofacial health, as well.
- There are profound and consequential oral health disparities within the U.S. population.
- Additional information is needed to improve America's oral health and eliminate health disparities.
- The mouth reflects general health and well-being.
- Oral diseases and conditions are associated with other health problems.
- Scientific research is key to further reduction in the burden of diseases and disorders that affect the face, mouth, and teeth.

The Surgeon General's Report summarized dramatic changes in oral health issues during the last century, and it also brought to light some serious challenges for the future. It stated that, although oral health has improved in the United States, disparities in health still exist. Specific population groups, such as infants and young children, the poor, those residing in rural locations, the homeless, persons with disabilities, racial and ethnic minorities, the institutionalized, and the frail elderly, continue to experience a greater burden of oral and craniofacial diseases. The Surgeon General's Report also stated that there were great disparities in access to oral health care and utilization of preventive services, each crucial to the establishment and maintenance of optimal oral and general health. Finally, the report recognized that

there were insufficient data to describe the population subgroups in greatest need for oral health-care services and dental public-health programs. The lack of data will make the development of relevant and effective dental public health programs a more difficult task.

By publishing the Surgeon General's Report, the Office of the Surgeon General has made available important and timely information to health-care practitioners, public-health professionals, policy makers, and the public. For access to the report, the Office of the Surgeon General provides an electronic version of the document and offers a free hardcopy of the report to all who request one.

Summary

The core functions of public health include assessment, policy development, and assurance. These functions are also essential components of dental public health, which is defined as the science and art of preventing and controlling dental diseases and promoting health through organized community efforts. It follows, then, that dental public-health programs are any organized efforts that strive to prevent and control oral and craniofacial diseases at the community level.

Dental disease has been a significant problem for Americans since the nation's early history. Arguably, one of the most successful dental public health programs ever created to address these problems has been community water fluoridation. As successful as fluoridation has been, however, new dental public-health programs need to be developed to meet the needs of population subgroups who have suffered from higher burdens of disease and have had poorer access to timely preventive and treatment services. The Surgeon General's Report on Oral Health in America highlighted some of these concerns and placed them in the context of existing programs and political realities. In addition, the federal government recognized that one way to address some of the oral health disparities that exist is to establish realistic national health objectives for 2010.

The initiation and implementation of any dental public-health program follows an established planning cycle, the first stage of which involves assessing the oral-health needs of the community. Once a problem is tentatively identified, it is addressed through the use of six sequential steps of the public healthcare practitioner's method—survey, analysis, program planning, program operation, financing, and appraisal. ASTDD established a seven-step model for needs assessment which functions well during the first step.

When traditional dental public-health programs prove ineffective, they must be replaced by more cost-effective approaches. The combination of less disease, more effective use of personnel, and improved technology and preventive methods, particularly dental sealants, provides opportunities to create dental public-health programs for those who have been traditionally neglected. In order to fulfill these opportunities, however, a constituency of public and personal dental and non-dental advocacy groups is required.

Dental public-health programs play a critical role in the promotion and maintenance of oral health in America. The challenge for dental public-health practitioners is to

devise programs that are effective, yet incorporate the principles of sound planning and implementation. The oral health of the public depends on it.

Answers and Explanations

1. B, D, and E—correct.

A—incorrect. Children with the greatest treatment needs are usually at the bottom of the economic scale and have fewer resources available. Until access to care for these children is improved, they will continue to be in great need of oral-health treatment services.

C—incorrect. In the United States, the incidence of oral clefts is three times higher among whites than it is among blacks.

2. A and D—correct.

B—incorrect. The State Health Departments are under the administrative control of State government. There is often cooperation between the U.S. Department of Health and Human Services and State Health Departments, however, because many health programs are financed by the federal government.

C—incorrect. The *examination* step of personal-health care is analogous to the *survey* step of public-health care.

E—incorrect. The CPITN is a valid measure of treatment need. A valid measure of tissue destruction is an assessment of loss of periodontal attachment (LOA).

3. C and D—correct.

A—incorrect. The American Dental Association is not under the auspices of the World Health Organization, however it is a member of the World Dental Federation (FDI).

B—incorrect. The ten Regional Offices are of the U.S. Department of Health and Human Services, not the CDC.

E—incorrect. The Surgeon General's Report on Oral Health in America was the first ever report of its kind.

Self-Evaluation Questions

1. The core functions of public health include _____, _____, and _____.

2. By definition, dental public-health programs are _____.

3. According to 1998 estimates, approximately $_____ was spent on oral health-care services in the United States.

4. *DMFS* represents decayed, missing, and filled tooth surfaces, whereas, _____ represents a caries experience index for primary teeth.

5. By definition, a public-health problem is one that meets the following criteria: _____ and _____.

6. The following are comparative methods used in personal and public healthcare practice:

Six steps of personal-health care Six steps of public-health care

Examination Survey

_____ _____

Treatment planning Program planning

Treatment _____

Payment for services Financing

_____ Appraisal

7. Two dental public-health program strategies for primary prevention of oral and pharyngeal cancer are: _____ and _____.

8. List three national oral health objectives for 2010: _____, _____, and _____.

9. List the four principle themes of the Surgeon General's Report on Oral Health: _____, _____, _____, and _____.

10. Health promotion consists of any: _____.

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Chapter 18. Preventive Oral Health in Early Childhood - *Stephen J. Goepferd Franklin Garcia-Godoy*

Objectives

At the end of this chapter, it will be possible to

1. Understand the rationale for professional preventive dental intervention for infants and toddlers.

2. Explain the type and process of early infant caries.

3. Provide appropriate recommendations for infant feeding that minimize the child's risks for developing *early-childhood caries* (formerly called "nursing caries").

4. Explain why it is so important that the mother and other members of the immediate family have a very high level of oral health, and especially a low *Streptococcus mutans* count from before birth until a mature, nonpathologic plaque is established in the infant.

5. Describe the six major areas to discuss with parents during the interview process.

6. Provide appropriate counseling on feeding/diet management, tooth cleaning, and fluoride management for parents of infants and toddlers.

7. Describe the timing, location, positioning, and steps for examining infants and toddlers.

8. Provide a rationale for determining the frequency of recall examinations.

9. Describe the process of anticipatory guidance and the age-specific information to be discussed during the dental visit.

Introduction

The dental profession possesses the knowledge and technology to assist parents in raising children free of dental disease. Dentistry's goal is to help infants and toddlers avoid the pain and devastation that accompanies early childhood caries (formerly called "nursing caries"), provide them with a pleasant, nonthreatening introduction to dentistry, and to establish and reinforce the foundation of preventive dental habits. Although this potential exists, *the preventive process must begin early in infancy* (birth to 1 year of age),[1] to ensure a successful outcome.

As health professionals, it is necessary to identify the potential for the development of disease, and institute effective measures for preventing the initiation of the disease; it is then a sound and logical practice *to intervene prior* to the onset of the disease, rather than treat the effects of the disease. Examples exist in pediatric medicine with *"well-baby"* evaluations and immunization programs. Pediatricians recommend that the infant be evaluated five times during the first year, and three times during the second year of life. Although these visits are aimed at evaluating development, prevention, or early detection of disease, physicians are not trained to provide a thorough dental evaluation or proper preventive dental health counseling. *It is the dental profession that must be proactive and assume the responsibility.*

Prenatal Considerations

In the short period after the diagnosis of pregnancy, an expectant mother is exposed to a barrage of information applicable to her health and that of her unborn child. Dentistry should be included in this routine. At the time of the dental counseling session, a knowledgeable dental professional should be the source of the essential information.

Dental counseling should come early, since the first trimester of pregnancy is a critical time. All organ systems are forming during this period. Tooth buds begin formation at the fourth to fifth week of gestation followed by the initial mineralization of bones and teeth from the ninth to twelfth week. Stress experienced by the unborn child at this time can produce dento-oral deformities. For example, a cleft lip or palate results when the maxillae fail to unite between the fourth to sixth weeks. These changes can result from a variety of etiologic factors affecting the mother such as genetics, stress of an injury, severe virus infection, alcohol toxicity, or smoking. An excessive stress to the fetus at any critical time in development can result in a temporary but often irreparable arrest in cellular growth.

Proper nutrition during pregnancy is essential. Although nutritional deficiencies in the mother usually must be severe to affect the unborn child, a daily balanced diet provides the necessary proteins, fats, carbohydrates, vitamins, and minerals. This requirement can usually be met by the adequate intake of the *four basic food groups,* although the obstetrician may desire to prescribe nutritional supplements. Bones of the maternal system form a large mineral reserve for use by the developing child.

All obstetric services should develop a positive referral system to assure that expectant mothers receive an early dental examination, preventive dentistry counseling for themselves and the future child, as well as necessary treatment. The referral may be to a private practice, a hospital dental service, or to a public-health facility.

If available, the mother-to-be should be encouraged to seek a flexible dental program where prevention, monitoring, and therapy is commensurate with the severity of the dental condition. Many women who become pregnant are already long overdue for treatment, and to postpone needed care for nine more months could cause severe oral problems. Dental radiographs for emergencies may be necessary, but *should be avoided whenever possible during the first trimester.* If radiographs *are necessary, careful gonadal and abdominal shielding* is required as with all dental patients. All dental treatment should be completed by the end of the second trimester, since the position of the baby by the third trimester affects the woman's posture, making long dental appointments quite uncomfortable.[2] Once the treatment is completed, the attending dentist should supply *feedback to the obstetrician,* indicating completion of the primary, secondary, and tertiary preventive dentistry treatment plans.

This emphasis on excellent maternal oral health is required for three reasons: (1) to reduce the possibility of onset and/or progression of caries and periodontal disease throughout the pregnancy; (2) because of the personal involvement of the mother with dental treatment, prevention, and counseling, there is a greater possibility of better care for the expected child; and (3) to *reduce the number of cariogenic organisms in the mother's mouth.*

Question 1

Which of the following statements, if any, are correct?

A. Dental counseling should be initiated in the first trimester of pregnancy.

B. Proper nutrition during pregnancy is not essential for the infant's oral health.

C. Mothers-to-be should seek a flexible dental program.

D. Dental radiographs should be avoided during the first trimester of pregnancy.

Dental Caries—An Infectious Disease

Evidence suggests that *dental caries is an infectious disease* process initiated via the transmission of *S. mutans* from parents to their infants.[3-5] The specific plaque hypothesis suggests microbial specificity in dental caries, and longitudinal evidence supports the role of *S. mutans* in caries initiation.[6,7] The following characteristics of *S. mutans* are important relative to dental caries in children.

- Permanent *S. mutans* colonization of the oral cavity in infants occurs only after the eruption of teeth.[8]
- *S. mutans* has difficulty colonizing in an oral cavity already colonized by mature oral flora.[9]

- Sucrose facilitates the adherence of *S. mutans* to the tooth surface.[10,11]
- The source of infection of the infant with *S. mutans* is from within the family, most likely the mother.[3-5,12-14]
- A minimum threshold level of maternal *S. mutans* is necessary for transmission of the microorganism to the infant.[12-14]

Transmission of *S. mutans* to the infant most likely occurs *during the first year of life, after the eruption of teeth.*[12-15] If the infant has a high sucrose diet in the presence of *S. mutans* the conditions are favorable for the initiation of caries. *The early establishment of oral hygiene measures and the adoption of a low cariogenic diet and low-risk feeding patterns should begin in infancy.*

With the above bulleted points as a backdrop, it is possible to develop the guidelines that minimize the possibility of transmittal of a cariogenic flora from the members of the family that will be most closely associated with the child. The most important goal is to reduce the bacterial challenge to the point that the *potential for transmission of S. mutans is minimal.* For the mother especially, this will require the continual maintenance of a high level of oral hygiene. Preferably, such a program should commence no later than the sixth month of pregnancy and continue throughout the time of the eruption of teeth and onward until a mature, stable, nonpathogenic plaque has been established (very low *S. mutans* count) on the child's erupted primary teeth.[15] Such a program includes appropriately spaced professional visits for prophylaxis, bacterial counts, and monitoring oral health. For the mother, the manual and chemical plaque control procedures may include, in addition to the toothbrush, irrigation devices and use of such antiplaque rinses as chlorhexidine, which can specifically target *S. mutans.* For the child, the most important procedure from time of birth would be a restriction of cariogenic foods, and oral hygiene attention, such as discussed later in this chapter.

Question 2

Which of the following statements, if any, are correct?

A. Caries is an infectious disease transmitted to the infant mainly by the mother.

B. The most important bacteria associated with caries initiation is *Streptococcus mutans.*

C. *Streptococcus mutans* colonization occurs immediately after the child is born.

D. Sucrose facilitates the adherence of *Streptococcus mutans* to the tooth surface.

E. *Streptococcus mutans* can colonize any tooth with a mature plaque.

The Mother-at-Risk

A pregnant woman is often at considerable risk of caries development. The mother's teeth do not lose calcium as postulated in a number of myths; instead, the risk of dental caries probably increases because of changes in eating habits. For example, the sucking of hard candy to reduce nausea, dietary cravings, and frequent between-meal

snacks of refined carbohydrates can raise the caries potential of the dental plaque. In addition, the mother often *experiences* nausea or "morning sickness," causing vomiting with a regurgitation of stomach acid which may cause *erosion* and *demineralization* of the lingual surfaces of the teeth. Many times, only a toothbrush or a sudsy dentifrice is needed to trigger a gag reflex.

Avoidance of aberrant eating habits and snacking, and exercise of sugar discipline can greatly minimize the possibility of caries development. If the mother is living in a nonfluoridated area, fluoride supplements should be considered. There are few reports available that indicate a decrease in caries prevalence of children born to mothers taking fluoride.[16-18] Since the supplement brings the level of fluoride intake to one part per million—a level ingested daily by over 100 million other Americans—there is no danger of excessive intake by the mother. There is the *possibility* of some benefit to the child. In addition, the expectant mother should be provided with appropriate treatment and recall appointments during the period of pregnancy. Professionally applied topical fluorides and systemic fluoride supplementation can benefit from the additional daily home use of fluoride dentifrices and mouth rinses. These measures will both prevent demineralization of the teeth, as well as facilitate remineralization in the event of the development of an incipient lesion.

Question 3

Which of the following statements, if any, are correct?

A. During pregnancy, the mother's teeth lose calcium.

B. If the pregnant woman lives in a nonfluoridated area, fluoride supplements should be considered.

C. The ideal amount of fluoride in water should range from 0.7 to 1 part per million.

D. Fluoride ingestion by the pregnant woman offers the possibility of some benefit to the child.

E. The use of fluorides both by mother and child prevents demineralization and facilitates remineralization of the teeth.

Rationale for Early Preventive Intervention

Early Childhood Caries

Dental caries can and does occur in infants and toddlers *well before 3 years of age.* Early infant caries has been observed in children as young as *12 months of age.*[19-21]

One of the first major hazards to the child's primary dentition is early childhood caries. This condition has also been referred to as "nursing caries," nursing bottle caries, *nursing bottle mouth, baby-bottle syndrome, baby-bottle tooth decay* (BBTD), and *bottle-mouth caries.* The caries pattern of this condition is highlighted by *rampant* dental caries *initially* involving the maxillary primary incisors,[6] and progressing to the first primary molars in later stages[22-25,27] (Figure 18-1). It is caused by continual,

prolonged exposure of the primary teeth to milk, infant formula, fruit juices, soft drinks, or other sugar/carbohydrate-containing fluids placed in the nursing bottle.

Once teeth erupt, the practice of offering a child a bottle filled with cariogenic fluid as a pacifier or at naptime or bedtime should be discouraged. Once teeth erupt and plaque accumulates, the ingestion of sugar-containing fluids *during bedtime or naptime* places the child at considerable risk for dental caries since salivary flow decreases during sleep and the fluid pools around the teeth, creating a highly acidic environment. This permits the pooling of the oral fluids around the maxillary anterior teeth.[6] Not all primary teeth are equally attacked. During sucking of the nipple of either the bottle or the breast, the tongue overlies the lower incisors, which directs the sweetened liquid against the *maxillary incisors* and to the back of the palate. The mandibular incisors often are either completely intact or only slightly affected, while the maxillary incisors bear the brunt of the repeated acid attacks. The other primary teeth are involved to various degrees, depending on the suckling habits of the infant.

The caries attack begins with the appearance of white areas of demineralization around the gingival third of the teeth (see Figure 18-1). With time, these incipient lesions begin to turn brown as active caries progresses. Eventually, the carious lesions that ring the cervical areas of the teeth can result in entire crowns being lost, either by fracture of the undermined enamel or by the continuous action of the caries. In either event, only the exposed root is left in the alveolous (see Figure 18-1).

If a bottle is to be used as a pacifier, it should be filled with *water.*

Early childhood caries can also occur in some breast-fed children who are nursed every time the infant indicates a desire for feeding (demand feeding, with 10 or more nursing events over a 24-hour period).[26,27]

Figure 18-1 Different stages of early childhood caries according to Garcia-Godoy, et al.[24,25]

Question 4

Which of the following statements, if any, are correct?

A. Dental caries may occur in infants and toddlers well before 3 years of age.

B. Early childhood caries is mainly due to continual, prolonged exposure of the primary teeth to milk, infant formula, fruit juices, soft drinks, or other sugar-containing fluids placed in the nursing bottle.

C. Once teeth erupt, the practice of offering a child a bottle filled with cariogenic fluid as a pacifier or at naptime or bedtime should be discouraged.

D. Early childhood caries does not occur in breast-fed children.

E. If a bottle is used as a pacifier, it should be filled with water.

The loss of teeth resulting from early childhood caries can have far-reaching effects on the child's eventual face growth.[18]

On occasion, there will be a pattern of multiple, severe caries in a toddler without a substantiated history of early nursing patterns that placed the infant at an increased risk. *The caries process is certainly multifactorial* and at times, a definite cause may not be identifiable. Nevertheless, sound primary preventive strategies early on will provide the appropriate environment for the prevention of dental caries.

Disease Prevention—A Proactive Approach

As health professionals can identify the potential for the development of disease and have effective measures available for preventing the initiation of disease, it is a sound and logical practice to *intervene* prior to the onset of disease whenever possible rather than to wait and treat the effects of the disease. Examples of primary prevention exist in pediatric medicine with "well-baby" evaluations and immunization programs. Pediatricians recommend that the infant be evaluated five times during the first year and three times during the second year of life. Although these visits are aimed at evaluating development and prevention or early detection of disease, physicians are not adequately trained to provide a thorough dental evaluation or proper preventive dental health counseling. *The dental profession must be proactive and assume this responsibility.*

Public and Professional Attitudes

Because of events such as early childhood caries, there is a *growing desire by parents* of infants and toddlers to receive an early dental evaluation and obtain information on the prevention of dental diseases in their children. According to parents, the major reasons for them seeking early dental evaluations are:

- Desire for information on preventing tooth decay for their child
- Desire to avoid unpleasant experiences that the parents had suffered
- Desire to learn what their role is in their child's oral health
- Recommended by their pediatrician or family physician.

The education process can probably best start with the obstetrician and pediatrician explaining to the expectant or new parents the cause and consequences of continued intake of sugar fluids. The physician can further aid in reducing the problem by prescribing those bottle formulae that contain the least sugar. For instance, there is a considerable range in the amount of sugar found in the various commercially available baby foods. Finally, the dental profession should emphasize the need for high school and community dental education programs to alert would-be parents of their responsibilities in dental care for their infant.

Early Dental Care

The newborn should become accustomed to oral care early. After feeding, the ridges where the teeth will later appear and the palate should be gently wiped with gauze or a soft washcloth. This removes leftover food, and establishes a routine for the mother to clean inside the child's mouth. Children need directly supervised oral hygiene care throughout childhood. It was traditionally recommended that a child should visit the dental office no later than *21/2 years of age.* Ideally, the child's first dental visit

should occur at *6 months of age and no later that at 1 year of age.*[17] The purpose of this initial visit is to permit an evaluation of the mouth and jaws for proper formation and alignment of structures. A second objective of this visit is to allow the child to become familiar with the dental office and its personnel under pleasant circumstances and forestall future apprehension.

Infant Oral-Health Education

According to the United Nations' Convention on "The Rights of the Child," articles 2 and 24, all children should have the same rights and have right to health and medical service.[28] Early childhood caries is a lifestyle disease with biologic, behavioral, and social determinants. An early screening of all children at around 1 year of age is an excellent opportunity for early detection of risk factors and risk indicators that may increase the possibilities for its prevention. The caries risk evaluation should form the base for appropriate recommendations of preventive measures.[28]

The American Academy of Pediatric Dentistry states: "*Infant dental care begins with dental health counseling for the newborn, which should include a dental office visit for preventive oral health counseling no later than 12 months of age.*[17] However, for those children who are delayed in erupting teeth, the first visit may be postponed, but should occur *within 6 months following the eruption of the first tooth.*"

The American Society of Dentistry for Children also recommends in *The Answer Book* that children should visit the dentist between 6 and 12 months of age.[18] Recently, a federal program called "Early and Periodic Screening, Diagnosis, and Treatment" (EPSDT), which *mandates* that medical and dental services be provided to children from low-income families, adopted the policy that children in the EPSDT program receive a dental screening by *12 months of age.* A recent survey among 54 dental schools pediatric dentistry programs showed that 86% teach students to see infants at 12 months of age or younger.[29]

One study evaluated an oral-health promotion program involving health visitors and mothers of 8-month-old babies in order to address some of the risk factors associated with nursing caries.[30] The oral-health promotion program significantly improved mothers recall of advice given by health visitors encouraging the use of a feeder cup, brushing their babies' teeth with fluoride toothpaste and restricting sugary foods and drinks. Significant improvements were also found in recall of advice regarding the use of sugar-free medicine and registering babies with a dentist. The program encouraged a higher proportion of the mothers to bring their children to clinics for a hearing check.[30]

The advantages of the infant oral-health approach are:

- Identifying and modifying detrimental feeding habits, reducing potential caries risk
- Assisting parents in establishing low caries-risk snacking and dietary patterns for their child
- Explaining and demonstrating tooth cleaning procedures for infants and toddlers
- Determining fluoride status and recommending an optimum fluoride program;
- Introducing dentistry to the child in a pleasant, nonthreatening manner
- Preparing parents for upcoming dental events for their child (anticipatory guidance).

Question 5

Which of the following statements, if any, are correct?

A. Two major reasons for parents to seek early dental evaluations for their children are a desire for information on preventing tooth decay and a desire to learn what their role should be in their child's oral health.

B. After feeding the newborn child, the ridges where the teeth will later appear should be gently wiped with gauze or a soft washcloth.

C. The child's first dental visit should occur at 21/2 years of age.

D. Some advantages of an infant oral-health approach are: identifying and modifying detrimental feeding habits and explaining and demonstrating to the parents tooth cleaning procedures for infants and toddlers.

E. The dental profession should emphasize the need for high school and community dental-education programs to alert would-be parents of their responsibilities in dental care for their infants.

A Protocol for Early Preventive Intervention

The Interview

The interview process and counseling session should be thorough and specific, yet concise. The attention span of the infant is limited and once the child becomes bored and seeks attention from the parent(s) their attentiveness to your discussion will be limited at best. Experience shows that the interview and preventive counseling are best accomplished *before* the examination of the infant for the following reasons:

- Specific parental concerns can be identified and addressed during the examination.
- Should the infant fuss during the examination (normal behavior) the parent(s) usually direct their attention toward the child during the ensuing discussion and not toward the dentist.
- The child can be kept busy with toys, etc., before the examination in a nonthreatening environment and the parent(s) will be better able to direct their attention toward the dentist.

The interview should begin with a discussion of the parents' reason for seeking care. Historical information gathered at the initial interview would assist the practitioner in developing the most appropriate and individualized preventive program for the family. Categories of helpful information are discussed in the following paragraphs.

1. GROWTH AND DEVELOPMENT. An abnormal pattern of development may be discovered or suspected, prompting a referral for further evaluation. Also, the date of the eruption of the first tooth will provide a baseline for determining dental development patterns and assist in answering future questions from parents regarding their child's dental development.

2. FEEDING HISTORY. Knowledge of the feeding patterns during infancy is critical to assist the dentist in assessing the child's risk for developing early childhood caries by discovering *potentially harmful feeding habits* and to help form a basis for recommendations regarding proper feeding practices that minimize the potential for dental disease.

3. MEDICAL HISTORY. A complete medical history is important. Knowledge of any systemic conditions that may adversely affect dental health will assist in developing appropriate preventive strategies. For example, long-term, frequent intake of sucrose-based medications may require additional recommendations for tooth cleaning to offset the increased caries risk from the sucrose intake.

4. PREVENTIVE ASSESSMENT. Information regarding dental development, dental health attitudes, and current oral hygiene practices will serve as a starting point for counseling parents regarding an appropriate preventive program for their child. A history of tooth decay in the family ("soft teeth") will provide insight into the environmental influences as well as parental attitudes regarding dental health and serve to guide the dentist's discussion regarding preventive strategies.

5. FLUORIDE SUPPLEMENTATION. It is important to know if the child has access to fluoride in drinking water. It is not sufficient to establish that a family lives in a fluoridated community. On occasion, the family may drink bottled water, which contains an unknown quantity of fluoride. On the other hand, a family drinking well water may or may not be receiving systemic fluoride depending upon the concentration of fluoride in the water. Before any fluoride supplements are prescribed, the water should be tested for fluoride concentration and supplements prescribed accordingly. Some families live in rural settings with well water, but the child spends the majority of the day in a location with fluoridated water such as a day care facility or school. Therefore, an accurate assessment of all potential sources of fluoride intake should be explored before making any recommendations regarding fluoride supplementation.

If the daily intake of fluoride is insufficient, parents should be informed that small daily dosages are beneficial to a child's teeth. Appendix 18-1 will aid in determining the amount of fluoride supplementation needed. This can initially be best accomplished by the use of *fluoride drops.* Around the age of 3, the drops can be replaced by *fluoride tablets,* which are swallowed. Later, as the child gains skill in chewing the tablet, the fluoride-laden saliva can be swished around the mouth and then swallowed to provide a topical application as well as systemic benefits. The practice of using a tablet a day should continue until the child is at least 12 years old, although many believe that fluoride supplementation should be considered as long as the individual—child or adult—has a fluoride-deficient intake.

6. ORAL HYGIENE. An assessment of current tooth-cleaning activities is important to establish the parents' role in oral hygiene for their child. Many parents think that allowing an infant or toddler to brush their own teeth is adequate. If the infant's teeth are being brushed, it is important to *establish how, when, and by whom,* and inquire whether the parents experience any difficulties during the process.

In one study, almost half of the parents interviewed had started toothbrushing programs for their infants at 12 months and 75% had done so by 18 months. With such infant and toddler toothbrushing programs, only a small amount, approximately the size of a pea, of a fluoridated dentifrice should be used in order to avoid the possibility of the child ingesting an excess of fluoride. Around the age of 6, daily fluoride mouth rinses may be initiated as part of the total lifelong oral health program.

Question 6

Which of the following statements, if any, are correct?

A. In a preventive oral-health program, the interview and preventive counseling are best accomplished before the examination of the infant.

B. The interview should begin with a discussion of the parents' reason for seeking dental care.

C. A complete medical history is not important in the initial visit.

D. A history of tooth decay is important to establish environmental issues as well as parental attitudes towards dental health.

E. It is important to know if the child has access to fluoride in the drinking water before prescribing any fluoride supplement.

Counseling

Based upon the information gathered to this point, the practitioner is ready to provide recommendations on *how parents can play an active role* in preventing dental disease in their child by assuming the responsibility for the following procedures.

Oral Hygiene

Parents should be educated regarding the following tooth-cleaning recommendations.

- A parent, other adult, or older sibling must assume total responsibility for tooth cleaning in infants and young children. Many children are unable to perform adequate plaque removal until 6 to 8 years of age.
- Tooth cleaning should be done in a comfortable location and pleasant environment. Positioning will be demonstrated during the examination.
- A dentifrice is not necessary for infants. In many cases, it may be a source for objection because of the taste and foaming action.
- If a dentifrice is used, only a pea-sized amount should be placed on the brush to avoid ingestion of excess fluoride.
- Tooth cleaning should be accomplished with a small, soft-bristled toothbrush.
- Tooth cleaning should be accomplished at least once daily.
- The evening tooth cleaning may be easier to accomplish following the infant's last feeding instead of waiting until just before bedtime since a tired infant can frequently be fussy during the procedure.

Question 7

Which of the following statements, if any, are correct?

A. Tooth cleaning of infant's teeth should be done by a parent, other adult, or older sibling.

B. A dentifrice is not necessary in infants.

C. If a dentifrice is used, the entire length of the brush head should be filled.

D. In infants, tooth cleaning should be accomplished at least once a day.

E. A large toothbrush is adequate for cleaning infant's teeth.

Diet Management

Parents have control, for the most part, over their child's diet during the early years. The exceptions include time spent with babysitters and in day care settings. Parents can have some influence in those situations, however, if they make their wishes known. The following information should be shared with parents.

- Infants should be weaned from the bottle around 12 months of age.
- The bottle should not be used as a pacifier nor given during bedtime or naptime.
- Only formula or milk should be offered in the bottle.
- Frequent, prolonged episodes of breast-feeding could be a caries risk.
- Sleeping with the child and allowing nursing through the night should be avoided.
- Infants and young children generally will eat more frequently than three times daily.
- Between-meal snacks should consist of foods that have a low cariogenic potential.
- Total amount of cariogenic foods is not the issue, rather the frequency of ingestion and retentiveness of the food are the factors that contribute to the caries risk.

Question 8

Which of the following statements, if any, are correct?

A. During the infant's oral health first visit, the parents should be informed that the infant should be weaned from the bottle around 12 months of age.

B. Only formula or milk should be offered in the bottle.

C. Between-meal snacks should consist of foods that have a low cariogenic potential.

D. Frequent, prolonged episodes of breast- feeding could be a caries-risk situation.

E. Total amount of cariogenic food consumption is more important than the frequency of cariogenic food consumption.

The Examination

Once the interview and counseling aspects of the visit are completed, the dentist is ready to proceed with the examination of the infant or toddler. The dental chair and overhead light are neither required nor very useful for examining children this young. Since one of the prime objectives is to provide a dental examination in a pleasant, nonthreatening manner, the procedure is best accomplished in the *knee-to-knee* position for children under 3 years of age (Figure 18-2). This position provides a stable, yet comfortable environment that incorporates the security of parental involvement, which may produce a calming effect on infants and toddlers who lack the cognitive ability to cooperate. Should the child offer resistance, the dentist can easily and gently stabilize the child's mouth and head cradled in the lap while the parent holds the child's hands and can stabilize the legs by cradling them with the elbows. Many of the infants and toddlers accept the examination procedures in this position without resistance. It is important in those instances where the children resist or cry that the parents be assured that the behavior is normal (and expected) for the child's age and should not be considered "bad" or "uncooperative."

The examination should begin with a soft touch, evaluating the extraoral head and neck conditions first, allowing the child to become accustomed to the dentist's actions. The examination of the oral cavity should begin by using the fingers to palpate the oral structures before introducing the dental instruments. Illumination can be provided with a penlight or flashlight held by the dental assistant. Access and stabilization of the mouth can be obtained by placing a finger on the gum pad distal to the most posterior tooth in a maxillary quadrant. Following inspection of the oral soft and hard tissues, a dental cleaning (plaque removal) is accomplished with a soft bristled, moist, child-sized toothbrush. Rarely will a rubber cup and polishing paste be required for stain removal. The tooth-cleaning process is discussed and demonstrated as you remove the plaque. At this point, it is very important that the child be repositioned with the head cradled in the parent's lap and the parent given the opportunity to practice the tooth-cleaning process with the dentist's supervision and guidance. This will help some parents get over their reluctance to clean their child's teeth, especially when the child resists. Occasionally, some infants and toddlers exhibit tight contacts between the anterior as well as the posterior teeth, which accumulate considerable plaque. The parent can be shown *how* to clean these areas using dental floss in a holder with relative ease. The parents are advised that they need to perform tooth cleaning for their child at least once per day, but preferably following each meal. The most critical time to clean the teeth is following the last meal or snack of the day. It is emphasized that toothpaste is not required and is usually objectionable to the infant. If it is used, only a minimal quantity should be placed on the brush.

It should also be emphasized early that when the child is becoming accustomed to the routine of having a parent brush the teeth, the tooth cleaning should not become an unpleasant struggle for those infants and toddlers who initially resist the procedure. On those occasions where the child struggles considerably, the procedure should not be abandoned. Rather, less attention can be placed on performing thorough plaque removal, while maintaining a consistent effort to establish a routine with the child. A more thorough tooth cleaning can be performed another day when the child is more cooperative. Parents can be reminded of other routines that are accomplished in spite of the child's objections, such as washing the child's hair. If the tooth-cleaning routine is established during the first 12 months, strong objections and resistance to the procedure during the "terrible twos" can usually be avoided.

Concluding the Appointment

The appointment is concluded by addressing the following areas.

- Provide the parents with a summary of your clinical findings.
- Make appropriate recommendations based upon the clinical findings.
- Solicit and answer any remaining questions that the parents may have.
- Reinforce the parents' role and responsibilities in their child's oral-health care.
- Establish an optimal fluoride program (pending any water analysis). (See Appendix 18-1.)
- Distribute educational pamphlets/brochures as desired.
- Provide anticipatory guidance information.
- Establish an appropriate recall schedule.

Anticipatory Guidance

Anticipatory guidance is a process for preparing the parents for upcoming developmental changes and concerns that may arise before the next scheduled dental visit in order to minimize the negative effects that may arise. Wherever possible, *preventive oral health information* should be provided to expectant parents during prenatal education programs. Examples of such information are provided in Appendix 18-2, 18-3, and 18-4.

Establishing a Recall Schedule

The recall appointment may be scheduled for 3, 6, or 12 months depending upon the child's potential risk for developing dental disease based upon clinical findings, stage of dental development, and feeding or diet patterns. Examples for determining appropriate recall schedules are listed in Appendix 18-5.

Figure 18-2 Knee-to-knee examination position.

Summary

The potential exists today for dental-health professionals to assist parents in raising caries-free children. The knowledge and technology are available and the request for this service is growing. The dental professional has the opportunity to accept this role with enthusiasm and continue to be a leader among the health professions in disease prevention. The dental profession must not ignore the oral health needs of infants and toddlers under 3 years of age. We must instead, take advantage of our knowledge and technology and begin our disease prevention efforts with children as infants and educate parents-to-be and new parents regarding their important role in the oral health of their children. By doing so, we can provide a pleasant and logical introduction to dentistry and promote the profession in a most positive way.

Answers and Explanations

1. A, C, and D—correct.

B—incorrect. Although nutritional deficiencies in the mother must be severe to affect the unborn child, a daily balanced diet provides the necessary proteins, fats, carbohydrates, vitamins, and minerals.

2. A, B, and D—correct.

C—incorrect. *Streptococcus mutans* colonization of the oral cavity in infants occurs only after the eruption of teeth.

E—incorrect. *Streptococcus mutans* has difficulty colonizing in an oral cavity already colonized by a mature dental plaque.

3. B, C, D, and E—correct.

A—incorrect. The mother's teeth do not lose calcium. Instead, the risk of dental caries occurrence probably increases because of changes in eating habits.

4. A, B, C, and E—correct.

D—incorrect. Early childhood caries has been reported in several studies dealing with breast-fed children, although the prevalence is lower than in bottle-fed children.

5. A, B, D, and E—correct.

C—incorrect. The child's first dental visit should be at 6 months of age and no later than 12 months of age to permit a complete examination of the child's mouth and jaws, to allow the child to become familiar with the dental office procedures under a pleasant situation, and to provide parents with early preventive advice.

6. A, B, D, and E—correct.

C—incorrect. A complete medical history is important as some systemic conditions may adversely affect dental health.

7. A, B, and D—correct.

C—incorrect. If a dentifrice is used, only a pea-sized or smaller amount should be placed on the brush to avoid ingestion of excess fluoride.

E—incorrect. Although a large toothbrush may be carefully used to clean anterior teeth, a small soft-bristled toothbrush provides easier access to all areas of the teeth as well as more comfort to the infant's mouth.

8. A, B, C, and D—correct.

E—incorrect. Total amount of cariogenic food is not the issue; rather, the frequency of ingestion and retentiveness of the food are the factors that contribute to the caries risk.

Self-Evaluation Questions

1. Dental counseling to pregnant women should start _____.

2. Dental radiographs should be avoided during the _____ trimester of pregnancy.

3. The emphasis on excellent maternal oral health is required for three reasons: _____, _____, and _____.

4. Evidence suggests that dental caries is an _____ disease.

5. Dental caries is mainly produced by the bacteria _____.

6. The main source of transmission of *Streptococcus mutans* to the infant's mouth is mainly from _____.

7. Children of mothers taking fluoride at the time of the child's birth revealed less caries _____.

8. Three other terms that describe early childhood caries are _____, _____, and _____.

9. The caries process is _____ and at times a definite cause may not be identifiable.

10. Recently, it is recommended that a child should first visit the dental office at _____ of age.

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Chapter 19. Oral Health Promotion in Schools - *Alice M. Horowitz*

Norman O. Harris

Objectives

At the end of this chapter it will be possible to

1. Explain why general and oral-health school programs are needed.

2. Discuss why many teachers are concerned about the prospect of teaching oral health and of conducting daily toothbrushing as a primary prevention measure.

3. Explain why school-based health clinics (SBHCs) offer the potential of providing access and funding for prevention and treatment programs.

4. Describe an effective primary preventive program that can be accomplished by *existing* school staff personnel.

5. Identify the reasons that school-based sealant and fluoride regimens should target "high-risk" students; explain how a dental hygienist can contribute to a preventive program; and finally, state the benefits that a dentist can add to the school health team.

6. Justify the need for a school tobacco intervention program to help prevent student use of smoking and smokeless tobacco products.

7. Describe the role of football helmets and intraoral mouthguards, as well as what to do if a player's tooth has been knocked out.

8. Explain how an expansion of a school's mission to include teaching about broad

societal problems can be competitive with teaching a normal academic curriculum; suggest a solution to this dilemma.

We need to do a better job of weaving a safety net of understanding, appreciation and guidance in the family, in the community and school. We need to start thinking of health and education as interlocking spheres.

—C. Everett Koop, MD, Surgeon General

Introduction

Today we have the ability to prevent or control most oral diseases or conditions of school-age children. Thousands of children and youths in the United States have benefited from the use of these preventive procedures. For example, dental caries has been reduced dramatically among U.S. children 5 through 17 years of age.[1-3] In fact, recent data show that nearly 55% of these children are caries-free in their permanent dentition, although the percentage *decreases dramatically* with age. In addition, an overall impression is that most students' mouths are cleaner with little noticeable exogenous stain and reasonably healthy gingiva. Unfortunately, preventive procedures have neither been available nor affordable by all children. This inequality likely explains why approximately 80% of caries lesions are found among one quarter of the U.S. child population.[1-3] Moreover, sharp disparities still persist in oral health status and use of dental services.[1] Poor children are more likely than non-poor children to have a *higher proportion of decay and are least likely to receive treatment.*[1-3]

Daily more than 6,000 U.S. youths 18 years of age and younger try their first cigarette.[4] It is well established that the use of *tobacco products*—smoking and smokeless tobacco—is the primary risk factor for oral and pharyngeal cancers.[5] Yet, the use of "spit" tobacco (chewing tobacco and snuff) among U.S. youths is on the rise and is not limited to lower socioeconomic groups. Purportedly, some youths believe that "spit" tobacco is a safe alternative to smoking. Moreover, when youths use spit or chew tobacco they are likely to switch to cigarettes. Equally or more disconcerting is that smoking cigarettes remains high and is increasing among the young.[6] One report showed that in 1998, 30% of high-school-senior girls reported that they had smoked in the past 30 days.[4] Also it is noteworthy that few health education texts include information that the use of tobacco and alcohol products are primary risk factors for oral cancers.[7]

The good news is that investing in comprehensive tobacco control programs, which includes schools, *does work.*[8,9] Two major contributing factors include: preventing the initiation of tobacco use among youth and promoting quitting among young people and adults.[8,9]

Equally important is the fact that large numbers of youth, especially minorities, do not complete high school.[10] And, many become parents at an early age. Thus, health literacy is especially important for youths who drop out of high school. It is important that they gain the knowledge and skills to attain and maintain good health, including oral health, for themselves and others who may depend on them. For these reasons, promoting oral health in schools is not only desirable but also a necessity.

Beyond the family, no other institution in our society can do more for child and adolescent health.[11] In today's fast-paced society in which children often live in single-parent or dual-career families, the school may become the *only* bastion of constancy in a child's environment. But, oral health programs in schools *cannot be considered in isolation* from the maelstrom of conflicting needs and priorities of overall medical, dental, and social needs of children and youth.

Definitions

For mutual understanding the following definitions are provided for use in this chapter.

Diet refers to the oral *intake of all foods and beverages.*

Nutrition is the study of metabolism that occurs *following the ingestion of foods and beverages.*

Program is an *organized* group of procedures designed to solve a defined problem.

Health education is any combination of *planned learning experiences* designed to facilitate voluntary actions conducive to health.[12] Health education must be an integral part of any school health program. Health education is used to inform, educate, and reinforce previous health messages. It is essential to educate a broad spectrum of individuals and groups to gain acceptance and use of health measures. Health education *alone* cannot function as a preventive method.[13,14] Still, accurate information and knowledge are important because they enable individuals, groups, and agencies or institutions to make *informed decisions* regarding oral health.[13,14]

Health literacy is the capacity of an individual to *obtain, interpret,* and *understand* basic health information and services and the competence to use, or not to use, such information and services in ways that are health enhancing.[2]

Health promotion is any *planned combination* of educational, political, regulatory, and organizational supports for actions and conditions conducive to the health of individuals, groups, or communities.[12] This definition differs from a more common use of promotion that is generally regarded as public relations or marketing activities. Although these kinds of activities frequently are a part of health promotion, this definition refers to actions intended to modify an individual's environment in a way that will improve health *regardless of individual actions* or to enable individuals to take advantage of preventive and treatment procedures by removing existing barriers. For example, one health promotion strategy to influence oral health might include providing a community-based fluoride regimen such as fluoride mouthrinse or tablets in schools that includes appropriate education for parents, students, and school personnel. Another example, using a regulatory support, might include the *prohibition* of any tobacco products on school property. This type of regulation along with appropriate education about the health effects of tobacco use as well as an increase in the *state's tobacco tax* would constitute a health promotion strategy.

Comprehensive school health education is a *planned,* systematic, and ongoing learning opportunity that enables *all students* (K-12) to be productive learners and to

make well-considered health decisions throughout their lives.

Comprehensive school health program is an *organized* set of policies, procedures, and activities designed to protect and promote the health and well being of students and staff, which has traditionally included health services, a healthy school environment, and health education.

Who is Responsible for Teaching Oral Health?

School age children, especially younger students, largely depend on parents and/or school-based programs for oral-health information or for inclusion in preventive dentistry or treatment programs. Many teachers believe that oral-health instruction should be the responsibility of parents and health educators—not teachers of academic subjects. This belief might be legitimate if, universally, parents were able to care for their children's oral health. To illustrate, Mandel has pointed out that there has been a profound drop in caries for 17-year-olds, with 50% being free of decay; at the same time he expressed concern for the 50% still experiencing caries.[15] In addition, the continued growth of numbers of children living in poverty, coupled with *cuts in public health programs*, may not bode well for the underserved population's access to dental services.[16,17] In fact, recent reports indicate that there is a huge disparity between low-income children and their higher-income counterparts.[1,2] A child reared in a home where the parents are subject to economic and educational disadvantages is often dentally neglected. In these homes, parental intercession in the oral-health care of a child frequently begins with seeking help to relieve pain, often a difficult task because access to dental care is not easy for them. Too many working mothers and single parents find it very difficult to take time off from work to tend to their children's health, especially when there are additional barriers to access. Others may be apathetic or so overwhelmed with how to feed their family that oral health simply is not a priority in their lives.

Even with highly motivated and educated adults, their knowledge about oral health is often minimal.[18-21] In addition, behavioral change may be more difficult to influence at home under parental guidance than under the tutelage of a teacher. In other words, many parents themselves do not know how to help their children help themselves and need the support of a school health program. Still, whenever possible, the parent must be included in a school-based, oral health program. Parents can provide strong positive reinforcement, either through role-modeling or verbal messages that support the attitudinal and behavioral changes promulgated in the school setting. Ideally, parent education should parallel child education; in this way, parents can learn to improve their own oral health as well as have the guidelines to assist their children. This educational process of parents is often necessary to help overcome the barriers raised by *their* past adverse experiences with dental treatment and its financial hardships.

The bottom line is that all children have *the right to a good education* to enable them to be knowledgeable, productive adults. A healthy child is better able to learn. Schools and health are inextricably linked; thus, school health programs are the underpinnings of promoting health and preventing diseases among our children.[22,23]

Typically school health programs have had three major components: health *education*,

health *services*, and a healthful *environment.* More recently some have expanded this triad to an eight-component model that includes health education, physical education, health services, nutrition services, health promotion for school staff, counseling and psychological services, a healthy school environment, and parent and community involvement.[23] Those who advocate the eight-component model believe that it is more comprehensive to help solve complex problems now faced by communities. No individual model is best. Rather, school-based programs, like other health programs, should be comprehensive and *based on the needs of the population* they are designed to serve.

Roles of Oral-Health-Care Providers

Professional Volunteerism

In addition to parental support, *professional involvement* in school programs is desirable. The luxury of having salaried dentists or dental hygienists (or both) employed by school systems, however, is rare. Therefore, opportunities abound for dentists and dental hygienists to volunteer in school-based programs. Professional input is valuable for identifying teaching-learning resources, speaking to students, faculty, and parent groups, providing in-service training of faculty and administrators, and for assisting the schools on special occasions, such as career day and health fairs. The support of the professional community enhances a program's credibility, improves the image of dentistry and dental hygiene, and may be a *practice builder* for participating providers. A more consistent presence of the oral health professions throughout the academic year is needed to help strengthen school-community relationships.

Dentists, dental hygienists, and students of each provider group can and should play major roles in school health programs.[22] Their involvement may range from taking the lead in planning a comprehensive oral health program and implementing it, to participating by providing education to students, treatment or preventive services, or in-service training for members of the faculty. Or, they may simply play a role of being supportive of an appropriate procedure that is being recommended in a local school. Whatever the role one plays it requires that the provider be knowledgeable about the needs of students in the school(s), which necessitates conducting a needs assessment. And, it requires current scientific knowledge about how to prevent oral diseases and conditions identified among the children in the target school(s).[22]

Health care providers should support the use of valid procedures. That is, one should recommend, use, and provide accurate information about only those procedures known to be effective based on research.[22] For example in a school in which students have high-caries rates in chewing surfaces of posterior teeth, dental sealants should be recommended in addition to regular use of a regimen of fluoride (Figure 19-1).

Most important, oral-health-care providers must work with others who are involved in school-health programs. These groups include school nurses, health educators, physicians, nutritionists, school administrators, leaders of local religious organizations, members of the local and state health departments, parent-teacher associations, parents, students, and politicians. Ideally, oral-health-care providers should have solid community organization and communication skills.

Figure 19-1 A leaflet explaining what sealants are and who needs them is available from the NIDCR (1 NOHIC Way, Bethesda, MD 208892-3500).

School-Health Programs Past and Present

School-health programs (SHP) originated around the beginning of the 20th century to help *cope with contagion, screening needs* for physical disabilities, *nutritional deficiencies,* and *first aid* ministrations. Since their inception, school-health programs have varied in quality and content by state and community. In the 1930s and 1940s, children were provided nutritional supplements, eye examinations, health education, smallpox vaccinations, and in some cases oral-health services. In the early 1950s, school-based fluoride regimens were introduced in the form of multiple applications of operator-applied, neutral sodium fluoride and school water fluoridation. In the 1960s, the outstanding success of rapid immunization of total school populations against polio-myelitis, and the almost universal establishment of nursing services within school systems to deal with day-to-day accidents and illnesses, is a legacy to the attainment of early school health objectives.

But, the concepts and requirements of school health programs have been greatly broadened over the last three decades. At the onset of the 21st century, *school-health services now include or attempt to address major societal health issues* that have invaded the schools. These include: alcohol, drugs, and tobacco use (smoking and "spit"); safe-sex, HIV, AIDS, other sexually transmitted diseases; gang violence and child abuse; and self-esteem, depression, homicide, suicide, and terrorism. In addition, health problems of dysfunctional families, migratory workers, and the poverty stricken are often addressed.[24-28] When these problems are mixed with those of ethnicity, race, religion, and politics, planning school-health programs becomes a complex and challenging exercise.[29] Further, while attempting to address more issues, most school systems have had a sharp reduction in health-related personnel. Today, it is a luxury for a school to have a full-time nurse or health educator on the premises. Although dental hygienists originally were trained to work in schools to improve children's oral health, relatively few schools today have access to this type of oral-health-care provider.

There is little doubt that schools, especially public schools, must become more involved in a wide range of health education and promotion to help cope with overwhelming societal health problems. This need is enunciated clearly in two documents, *Healthy People 2010 and Healthy Schools 2000.*[2,30] Appendix 19-1 shows the oral-health objectives related to schools contained *in Healthy People 2010.* Many states have their own health objectives that include oral health. Each state department of health has at least one person responsible for directing health education. Unfortunately, these *state-level health educators and the state dental directors rarely work together* to achieve oral health objectives. Some states even require health educators in attendance in elementary, junior, and senior high schools. Further, some states provide written guidance in the form of curriculum guidelines for health education. Only a few states, however, elevate the status of health education to include *mandated testing.*[31]

In conflict with the expanding health role of the school system is the fact that *the primary mission of schools is education—not conserving health or preventing*

disease. School administrators are sensitive to any change or expansion of their basic mission. There are limits to just how far schools can continue to assume greater teaching responsibilities.[32] Probably the most important factor is the *lack of time* in the curriculum for new teaching requirements. Unless school days or years are extended, any expanded health program becomes *competitive* with basic education needs. This competition is exacerbated by legislative mandates that require additions of specific school health workloads without allocating additional specific health funding.[33] It is little wonder that, at times, teachers consider themselves dedicated, beleaguered, and underpaid professionals who are being called on to discharge duties *which "society" itself cannot solve.*[34]

The needs of a community and its schools are intricately interwoven. To foster cooperation between the two, School Health Councils often are developed to help mobilize the health resources of a community. In schools, nurses, health educators, physical education teachers, guidance counselors, food-service directors, principals, students, and parents share responsibility, while the greater community taps the expertise of existing civic organizations, health agencies, and business and professional leaders.[35] A recent trend of School Health Councils has been to recommend implementation of *school-based health clinics (SBHC)* both within and outside of school hours.[24,28,29] These SBHC are now in 45 states and the District of Columbia. They have grown from 200 in 1990 to 1,380 in 2000.[36] These clinics are now operational primarily in high-risk urban and rural areas with higher levels of poverty where there is a lack of access and funding to conventional health care.[37] Some of the services provided include: athletic and pre-employment physicals, laboratory and diagnostic screenings, prescriptions on a limited basis, first aid, family planning, prenatal and postpartum care, drug and alcohol counseling, nutrition and obesity counseling, and *dental care* (Figure 19-2). Sponsorship of SBHCs may include community organizations, health departments, medical centers, hospitals, private not-for-profit organizations, and school systems.[38] Funding may be from private foundations, local funding, insurance, or government sources such as Medicaid. Should this trend continue, it is conceivable that schools of the future will need an assistant school superintendent for health affairs.

Members of Congress and many health providers including oral-health providers, are interested in this SBHC approach to health care for children and youth as it would make *Medicaid and other appropriated funds available for this use.* The Department of Education and the Department of Health and Human Services have funding mechanisms in place to promote research and development for these types of clinics. According to proponents, the development of this nontraditional component of a school health service will: (1) permit the school nurse to return to a more traditional purpose of the nursing service to support the classroom teacher as well as acting as a liaison with the SBHC; (2) return to teachers the responsibility for classroom teaching of subjects that match their academic training; and (3) *provide a method of access and funding* for all health care for school-age children and youth.

Today there is a national concern for achieving both excellence of education and for improved health for children. School-based programs appear to be a natural site for addressing both concerns. Students can stay in class, parents can continue at work, and cost-effective, comprehensive school health programs, *including those for oral health*, can be made available.[28,38] It is important to note that one of the oral health

objectives in Healthy People 2010 is to increase the number of SBHC that include oral health (Appendix 19-1). A consensus exists that a student who is impaired by drugs or alcohol is as handicapped in learning as an individual who suffers from a sight or hearing deficiency. Thus, there is a need for schools to help shape social, psychological, and physical well being—*all in the name of school health.*

Figure 19-2 A student receives dental care in a school-based health clinic.

Question 1

Which of the following statements, if any, are correct?

A. Oral health education, promotion, prevention, and treatment can be best provided within the framework of an overall school medical-health program.

B. Teachers are well prepared academically and through continuing education courses to act as facilitators in solving *societal* problems.

C. Teachers are well prepared academically and through continuing education programs to act as facilitators in teaching *oral-health education.*

D. School-based health clinics (SBHC) that include oral-health services, are *now* in operation.

E. Approximately 80% of caries lesions are found in approximately 25% of schoolaged children.

Creating Effective, School-Based Oral-Health Programs

To involve communities, families, or individuals in assuming responsibility for their own oral health, many ingredients are necessary. These include but are not limited to: knowledge, skills, motivation, access to preventive agents and treatment services, and a safe, healthy environment. Further, decision makers and teachers must be willing to include health education and health promotion in schools. Finally, *policy* is important. For example, smoke-free schools are a result of a policy that bans the use of tobacco products—by all—on school campuses. Such policies help deter students and faculty from using these products.

It is impossible for the approximate 152,000 active dentists[39] and over 100,000 dental hygienists[40] in the United States to assume the tremendous task of imparting essential oral health education to the public and encourage appropriate behaviors that are requisite to optimal oral health. Much of this education can be accomplished through *schools*, *mass media*, and *industry*.

Planning—Key to Success

A successful school-based program does not just happen. An integrated plan of action for specified grades is essential, as is a clear delineation of program responsibilities and objectives.[20] It should be noted that, although including all grades—kindergarten through grade 12—is desirable and ideal, it rarely occurs regarding oral health. When

oral health is addressed in a school setting, it is more often confined to kindergarten through grade 6.

Judicious planning ultimately will result in meaningful, target-specific programs that are integrated throughout the school years. Planning must include a community needs assessment, establishing priorities, developing a budget, and a strong component of evaluation. Developing a written plan of action is necessary for a variety of reasons. The plan can serve as an educational tool to inform individuals and groups, especially those that are needed to "buy in" as a partner in the endeavor. A written plan also can be used to solicit funds to help underwrite the proposed program. And, of course, the plan serves as a guide for all who are actively involved to help maintain focus on the objectives.[22]

A diverse and committed work group helps to ensure a greater variety of input, a representative audience, and a broad-based sense of ownership. It is essential that cooperative working relationships be established in the *initial* planning stages among parents, community leaders—both business and political—teachers, school administrators, and oral health professionals. *A dental representative should be a member of all state or local school advisory councils to ensure professional as well as political input.*[11]

Educational Considerations

Traditionally, public schools have accepted the responsibility of teaching oral-health as a part of general health. Committed, knowledgeable teachers are the cornerstones of all effective, school-based oral-health education. However, the dental subject matter contained in many texts used in teachers' colleges relates mainly to the anatomy and physiology of the teeth, the supporting tissues, and salivary glands. Such a background enables schoolteachers to teach and promote basic information about the oral cavity. *Teachers cannot be expected to possess expertise* in a constantly changing pool of scientific knowledge relating to prevention and dental treatment options. As a result, they may be reluctant to teach what could be incorrect or obsolete information in these areas.

Periodic in-service training to upgrade teacher health knowledge, including oral health, would greatly help the faculty to develop greater competence and confidence.[41] To help attain this objective the state of Utah supplies their teachers with a biannual update of advances in dentistry.[42]

Teachers and the dental profession alike have questioned the priority given to school oral health education programs. Sometimes the questions are asked by the educators because the time taken for dental education competes with the time needed for other subjects. Is oral health instruction more important than, for instance, mathematics or vocational training? Health professionals also question the time devoted to school programs that feature mainly information transmittal, rather than a *combination of education and preventive/treatment regimens.* Education, alone, rarely suffices. For example, it is pointless to provide plaque removal instruction if toothbrushes are not provided and the instruction is not practiced in the classroom. Individuals, who are not provided assistance in shaping health attitudes, beliefs, and habits early in life, are likely to suffer the consequences of reduced productivity in later years. Health-related

knowledge and skills acquired in public school may become more meaningful in adult life when the importance of good health becomes more apparent.

The amount and quality of oral health taught in school is often too little to be assimilated by students, and not long-term enough to be amenable to good evaluation. A national school health evaluation conducted by the U.S. Public Health Service (USPHS), revealed that changes in health-related knowledge, practices and attitudes *increase* with the amount of instruction. The possibility of *securing that needed additional time is minimal* considering that in 1994, the annual average number of hours spent on health education, including oral health, was *13.8* hours![43]

Educational Principles

Oral-health education involves the use of many communication and organizational skills and processes. These include conducting a needs assessment, listening, planning, facilitating group and individual participation, informing all relevant individuals and groups, leading, writing, speaking, providing feedback and reinforcement, and providing in-service training. These activities enlist the help of others and foster cooperation and the adoption and maintenance of effective health measures and programs by individuals, institutions, organizations, and communities. Educational principles necessary for oral-health education include:

- Health education must be an *integral part* of any preventive or restorative service or regulation or legislation relevant to a health program. This education functions to introduce and reinforce understanding and acceptance of, and participation in, whatever the health program consists of.
- Educational materials can be used effectively to gain attention on specific topics and to reinforce or clarify a procedure or regimen. But, *educational materials alone do not constitute an oral-health program*; they can be useful aids.
- Education materials should be *accurate* and *consistent* with current scientific knowledge.
- Educational materials must be appropriately designed for specific *ages, level of literacy, and cultural groups* for whom they are designed. One leaflet, for example, cannot be expected to be suitable for all racial-ethnic groups in a given community.
- Educational materials should be evaluated *prior to* their final production and use with the intended target group.
- Interactive teaching in which there is active *participation* and *involvement* on the part of learners is essential for all age groups and content areas.[22]

Approaches for Health Education

Multiple channels of communication should be used to help ensure reaching as many of the target population as possible for purposes of informing and educating as well as for reinforcement. Oral health education should include *more than* one of the following approaches.

- *One-to-one communication* such as discussing the need for an oral-health program with a school superintendent.
- *Group presentations* of information and education, and demonstrations such as at parent- teacher meetings, in-service training for teachers, or organizational meetings.

• *Use of mass communication* such as newspapers, radio, television, web sites, and school newsletters to inform all groups about the need for an oral health program and plans to initiate a program, to solicit partners, and to provide feedback and reinforcement about the program's value and effectiveness.
• *Community organizational strategies*, such as coalitions, partnerships, councils, or committees to solve local health problems, gain commitment of resources, and conduct needs assessments.[22]

Student Participation

Philosophically, all children should be entitled to receive maximum information and primary preventive oral care. Providing maximum oral health treatment for all children, however, is not feasible. Poor children have a higher percentage of untreated decayed teeth than nonpoor children.[1] Hence, a school-based program, other than classroom education, fluoride mouth rinses, or tablet program, should be selective *in targeting the children most at risk* as eligible for a higher level of care. Once criteria are established defining "high risk," all students meeting the high-risk criteria should be eligible for the same preventive and treatment benefits. For example, some states base eligibility for sealant programs on whether a child is eligible for free school lunches. The economic status of individual students, however, *should not* be the only consideration because poor oral health occurs among the affluent as well as among the poor. Of importance in one study, it was found that following a few years of participation in an intensive caries prevention program, individuals were transformed *from high risk to low risk.*[44]

The extent of student involvement in program activities is an important issue. Personal involvement and participation tends to have a greater effect on behavior, attitudes, and beliefs. Active participation likely enhances interest in oral disease prevention measures and having a healthy mouth.

One opportunity for innovative student participatory learning comes during *Children's Dental Health Month held annually in February.* This activity, sponsored by the American Dental Association (ADA) and the American Dental Hygienists' Association (ADHA) was initiated in 1941 to provide an annual forum for oral health. Historically, both the dental and dental hygiene associations have participated in the program. Children's Dental Health Month has developed into the professions' *most widely supported and media-recognized oral-health event.* It is a time when schools, in cooperation with dental manufacturers, local dental and dental hygiene schools and associations, and other sponsors, organize oral health fairs for the public and for students. As a part of these health fairs, students engage in entertainment events, receive screening examinations, toothbrushes and toothpaste, watch demonstrations related to seat belt and air bag protection of the face and body, participate in discussions about the deadly effects of cigarette smoking and chewing tobacco or snuff, and learn about health-promoting behaviors (Figure 19-3).[45,46] During this time, students often develop group posters and exhibits, discuss nutritional information, and learn that dentistry is not a profession to be feared. The informality of these occasions may enhance student interest and fosters learning and development of desirable attitudes toward self-care and self-image.

Figure 19-3 Students learn how to remove plaque during *Children's Dental Health Month.*

Question 2

Which of the following statements, if any, are correct?

A. If the length of the present school year is maintained, any additional time commitments for teachers to adequately teach comprehensive health education and to address societal issues *must come from the teaching time used for other subjects.*

B. Teaching oral-health self-care is one of the most effective means for reducing oral disease.

C. Approximately *13.8 hours a year* is the average time devoted to *oral-health* teaching.

D. The annual Children's Dental Health Month is sponsored by the American Dental Association, and the American Dental Hygienists' Association.

E. Using multiple methods of communication is a better approach than using only one method in educating about oral health.

What To Teach?

The needs of students in a particular school or district coupled with available resources—human, equipment, and financial—will dictate necessary preventive and treatment services, education, and policies. Appendix 19-2 provides a list of topics that students should be taught regarding prevention and early detection of oral diseases and conditions.

Dental-Caries Prevention

Fluorides and Dental Sealants

Students need to learn that dental caries is an infectious disease with a multifactor etiology. And, they need to know that the disease can be *prevented, arrested and, reversed and how to do so.* Students should be taught that the appropriate use of fluorides and pit-and-fissure sealants are our best defense against this disease.[47] They also need to know about *systemic fluorides,* that is, fluoride intended for ingestion—community-water fluoridation, school-water fluoridation, and dietary-fluoride supplements—as well as *topical fluorides*—those fluoride products that are *not* intended to be ingested—toothpastes, mouthrinses, and operator- applied fluorides. Everyone needs to know that fluoride works both pre-eruptively and post-eruptively, but the primary ways that fluorides work are to *inhibit demineralization and to facilitate remineralization.*[48]

Pit-and-Fissure Sealants

Regarding dental sealants, students need to know how sealants protect occlusal surfaces of the teeth from decay; the ages at which teeth should be sealed and the need for possible reapplication of sealant material. Further, and very important, students

and parents need to know that teeth that have been sealed should not need to be restored with a filling (see Chapter 10).

Diet and Nutrition

The United States Department of Agriculture's food pyramid (see Chapter 15) makes it clear that sugars should be consumed only in moderation. Sweets not only are unhealthy for teeth but also are often in foods (cakes, pies, cookies, etc.) that are laden with unhealthy fats. Basic information on diet and nutrition should be a part of all health education. Students need to understand that consumption of carbohydrates, especially sugar is a *key* component of the caries process and that sugar must be present for caries to occur. Ideally, when discussing diet in relation to oral health, information should be taught with a parallel emphasis on the preventive benefits of fluoride and how they remineralize incipient lesions, which prevent them from becoming overt, and on the use of pit-and-fissure sealants and oral hygiene measures. In the United States, especially in recent years, more emphasis has been placed on making the tooth more resistant to decay with the use of *fluorides and dental sealants.* Perhaps the best message about sweets is that *if you must consume sugars, do so at meals and in moderation.* It is important that in our efforts to encourage consumption of nonsweet snacks that we do *not* encourage the use of high fat and high sodium foods.

Often, after a teacher has taught that sugar is one factor that causes tooth decay and suggests that the child avoid excessive and frequent ingestion of refined carbohydrates, the child then goes to the cafeteria. There, the child is confronted with attractive, sugar-laden desserts. The question then is, "What message do students really get—the message they hear in the classroom or the message they see, smell, taste, and enjoy in the cafeteria?"

Schools should provide an *environment* that promotes avoidance of an excessive intake of sugar consumption. One important method of meeting this objective is for the *school dietitian* to reduce the number of days a week in which confections are available. Instead, desserts such as fresh fruits can be offered that are nutritionally sound and limit the amount of sucrose consumed.

A second strategy for reducing student sucrose intake is for the *school principal or superintendent* to remove all vending machines that dispense candy and junk foods and soft drinks. Essentially, *the income from these machines uses the teeth of the children to subsidize nonbudgeted school expenses.*[49] Many schools have removed these kinds of machines or have substituted more nutritional snacks, including milk, fruit, and juices. Still, these machines remain a problem because selling sweets provides a major income for most schools in the United States. Recent evidence suggests that the consumption of fruits and vegetables may provide a protective effect against oral cancers (like several other cancers) and heart disease.[50] Further, several very successful 5-DAY programs have been initiated and evaluated.[50] These school-based programs are designed to increase student consumption of fruits and vegetables to at least five-a-day. Dietitians and school administrators can act as gatekeepers to better student health by exercising control of the menu and the items in the vending machines respectively.

Preventing Gingivitis

Classroom Toothbrushing

Thorough mechanical plaque removal on a routine basis will essentially *prevent and reverse gingivitis.* Thus, it is important for children and youth to know how to remove plaque using a toothbrush and dental floss without injuring their soft tissues. For some children daily toothbrushing in the classroom may be both needed and desirable; but, in most cases, *it is impractical.* Despite the need for emphasis on toothbrushing, some basic problems arise. Many teachers are willing to teach the mechanics of toothbrushing so long as they do not have to demonstrate the unfamiliar details of plaque control. In fact, the issue of toothbrushing is often played down since too-frequent repetition can be considered boring by students.[51]

Other than preschool teachers, very few teachers are willing to incorporate daily toothbrushing into their classroom schedule. The daily need for hygienic storage and continued replacement of worn-out and lost brushes also poses problems for a teacher. Unless replacement toothbrushes are made available to the children without cost, dedicating classroom time for activities in which several students may not benefit due to economic or other factors is resisted. Finally, few classrooms have the water supply and the sinks necessary for conveniently scheduling daily brushing as a classroom activity.

Despite these problems, some classroom brushing programs have been a success. (See Appendix 19-3 for a suggested method of teaching toothbrushing.) Although little or no evidence supports toothbrushing *alone* as a means of preventing caries, *overwhelming evidence supports the fact that toothbrushing with a fluoride dentifrice is beneficial.* Thus, one objective of toothbrush instruction is to encourage children to use a fluoride dentifrice when brushing teeth and not simply to teach toothbrushing as an exercise.

Some volunteers go into schools to teach plaque removal without providing toothbrushes and toothpaste for the students to practice. This approach, while well intended, may send very poor messages, especially among lower socioeconomic children who cannot afford toothbrushes and toothpaste. That is, the importance of this procedure will be lost if the child does not own a toothbrush. If toothbrushing instructions are provided in a classroom, *it is critical not only to demonstrate proper procedure but also to provide brushes and toothpaste so that students can be observed practicing to help correct inappropriate methods.*

Question 3

Which of the following questions, if any, are correct?

A. Topical fluorides can only be applied by a dentist or dental hygienist.

B. Sealants are most effective in preventing bicuspid and molar *occlusal* surface caries.

C. Sugar is the prime source of energy for cariogenic bacteria.

D. Daily toothbrushing in the classroom is *not* practical.

E. Toothbrushing alone does not significantly reduce the risk of caries; however, there is overwhelming evidence that brushing with a fluoride dentifrice does reduce caries incidence.

Oral-Cancer Prevention and Early Detection

The use of tobacco products and alcohol can be found among U.S. students as young as 8 or 9 years of age.[52] Recently there has been an increase in use of cigarettes and "spit" tobacco products among youths. Both females and males tend to smoke while the use of smokeless tobacco products is used primarily by males. Young white females smoke more than young black females, often as a means of weight control.[53-55] Intervention programs, therefore, need to be implemented at *early grade levels* and supportive regulations must be in place throughout all schools. That is, *all schools should be smoke- and drug-free.* Use of tobacco products should not be allowed on school premises or at any school events. Further, state and local regulations regarding the sale of tobacco products or alcohol to minors should be strongly enforced by all communities.[9,54]

Students must be taught about the risk factors for and signs and symptoms of oral cancers (Table 19-1, 19-2). Also, they need to know that an oral cancer examination exists and that they should have the exam if they smoke or chew tobacco or consume alcohol. In addition, students should be taught how to look in their own mouths for abnormal lesions, especially if they are at high risk. *Any oral or facial lesion that does not heal within 2 weeks should be seen by a health professional.*[56]

Tobacco Avoidance and Cessation

Tobacco use interventions—avoidance, refusal skills, and cessation—must be included in the curriculum of any comprehensive school-based oral-health program. Experimentation with smoking is occurring at earlier ages than ever; most users begin experimentation prior to adolescence.[6] Approximately 6,000 children and youth initiate smoking each day. Thirty thousand new cases of oral cancer are reported annually, with 8,000 people dying prematurely each year. People who use tobacco—smoke or smokeless—are at *several times greater risk* for oral cancers than are nonusers.[5,56]

The Healthy People 2010 objectives, 27-2, 27-3, 27-4, 27-7, and 27-11 (Appendix 19-1) address *youth and tobacco.* To respond to these objectives, school-based programs in elementary, junior, and senior high schools must include smoking and smokeless tobacco use interventions and provide students with tobacco-free environments.

A recent Surgeon General's Report states that educational strategies, conducted in conjunction with community- and media-based activities, can postpone or prevent smoking onset in 20 to 40% of adolescents.[4] This information is particularly important given the current decreasing age of smoking initiation especially among young girls. By delaying the onset of smoking, school programs have the potential to (1) prevent some students from ever starting; (2) reduce the possibility that young

students will become regular adult users; and (3) make it easier for those who do start tobacco use to stop. These same principles that apply to smoking *also can be applied to "spit" tobacco* intervention programs. The National Institute of Dental and Craniofacial Research and the National Cancer Institute have produced a guide for quitting spit tobacco (Figure 19-4).

Curriculum content must receive significant attention. Ideally, the topics of smoking and use of "spit" tobacco should be introduced in *primary school* with continuing reinforcement through middle or junior high school with *major reinforcement sessions in senior high school.* Tobacco subject matter may be integrated into preexisting curriculum units on drug abuse prevention and/or incorporated into health or physical education classes. *Coaches can be very influential* in persuading school athletes to cease use of tobacco of any kind as part of their training program.

Another pivotal ingredient for success is inclusion of content on *refusal skills.* Children and youth must be taught how to resist peer and media pressure by developing decision-making and problem-solving skills that facilitate refusal of undesirable habits and influences.[57] Children and youths also must be taught to realize that tobacco use is *not the norm* and has both immediate and long-range adverse physical and aesthetic effects.

A strong supportive network among students, parents, and teachers promotes program success. Student involvement is paramount. Although a teacher, health educator, or school nurse should lead sessions, students can assist in program delivery. Student role-playing and modeling exercises enhance student participation. Parental support is another critical component; the parental values opposing tobacco use and favoring antiuse programs enhance program credibility.[58] Finally, teachers, health educators, or school nurses must be properly trained and committed to the program.

Examples of successful programs are mentioned briefly to provide guidance to future program planners.

Frequently smoking and "spit" tobacco use is related in a positive way to star athletes who often serve as role models to adolescent males.[59] Appropriately, in 1993, major league baseball's executive council, the governing body of the sport, announced an on-the-field ban of all tobacco products for every uniformed employee of minor league baseball. Under this policy, use of tobacco products of any kind is not permitted in any team area. Violators are subject to fines ranging from $100 to $300 and face game ejections.[59] Unfortunately, this ban does not extend to the major league level of the sport. The urgent need for establishing effective tobacco avoidance and cessation programs early is iterated by Glynn: "*Today, as in every other day of the year, more than 3,000 adolescents will smoke their first cigarette on the way to regular smoking. During their lifetime, it can be expected that of these 3,000 children, about 23 will be murdered, 30 will die in traffic accidents, and nearly 750 will be killed by smoking-related disease.*"[60]

An outstanding article has been published that lends even greater *urgency* to achieving smoking avoidance and cessation by relating adverse changing in the body's immune system caused by smoking, to the onset, severity, and treatment of periodontitis (see also Chapter 13).[61]

Figure 19-4 Spit tobacco a guide for quitting is available from the NIDCR (1NOHIC Way, Bethesda, MD 20892-3500).

Preventive Dentistry in Sports

Sports are an important morale ingredient for both student athletes and the student body. During the 1989 to 1990 school year, in grades 9 through 11, the Ohio High School Athletic Association, which is the governing body for high school athletes in the state, listed 167,000 boys and 9,200 girls as competing in organized athletics with tournament play.[62] In many of the sports, the oral structures of these athletes are highly vulnerable to damage. The causative agents might be a hockey puck or a baseball speeding at 70 to 90 mph; a bone-crunching tackle or a thrown baseball bat; or an elbow following a spectacular basketball slam dunk. Yet, in a recent survey, results indicated that football was the only sport in which the majority of youth used mouthguards and headgear.[63]

In each session of competition, athletes face a 10% chance of orofacial injury, and a 33 to 56% possibility over a playing career. Protective equipment, rules, and regulations have been developed to reduce this toll. For instance, prototype leather football helmets with padding came into general use in the beginning of the 20th century, although were not required until 1939. By the 1950s, leather helmets had been replaced by a more protective hard plastic. Helmets protect the cranium and the ears, however, more than the lower face and mouth.

In 1960, the American Dental Association and the American Association of Physical Education issued a report that highlighted the fact that when high school players *did not* wear mouthguards *50% of all football injuries occurred in and around the mouth.*[64] The ADA House of Delegates soon passed a resolution urging that all athletes participating in contact sports wear intraoral mouthguards. This objective came to fruition in 1962, when several sports organizations mandated the use of mouthguards. It is estimated that as a result, from *100,000 to 200,000 oral injuries to football players are prevented annually.*[64]

Football is *not the only school sport* for which orofacial protection is needed. At the amateur level, sports that now mandate the use of mouthguards during practice sessions and in games are *boxing, football, ice hockey, lacrosse, and field hockey.* It is interesting that the Academy of Sports Dentistry has listed 40 different sports, including soccer, bicycling, skate boarding, and basketball in which cranial or orofacial protection should be considered.[64]

A mouthguard is constructed of soft plastic that is placed between the maxillary and mandibular dentitions. These devices help to prevent violent contact between the lips, cheeks, and teeth, and between the upper and lower teeth. It reduces the possibility of fractured jaws as well as lessening the likelihood of neck injuries, concussion, cerebral hemorrhage, unconsciousness, serious central nerve damage, and death. Although 87% of Division I colleges have team dentists, it is the *team trainer* that usually selects the mouthguards.[65]

In football, for example, prior to a game, it is the responsibility of game officials to check with coaches to ensure that players are using required protective equipment.[65]

When mouthguards and other protective equipment are not worn, penalties are levied, ranging from a reduction in the number of time outs to 5-yard penalties. It is the quarterbacks who are least compliant.[65] In the past decade, the thrust for protective equipment has been emphasized in an effort to reduce the possibility of *bleeding injuries.* In some states, any bleeding injuries are cause for officials to remove a player from the game due to the increased possibility of transmitting HIV.

When a mouthguard is not worn or fails, one of the most frequent orofacial injuries requiring emergency treatment by the dentist is the *avulsion of teeth.* Maxillary teeth are the most vulnerable. When avulsion occurs, a recovered tooth should be held by the crown to rinse and then *gently replaced at the site of origin.* If the tooth cannot be easily reinserted, it should not be allowed to dry. Milk is a good transport solution on the way for emergency treatment at a dental office. After replacement, a minimum 7- to 10-day splinting period is advised. All avulsed teeth with fully formed roots *eventually need endodontic treatment.*[66]

Question 4

Which of the following statements, if any, are correct?

A. The best grade level to introduce discussion of the health dangers of tobacco smoking, cessation, and avoidance is in *senior high school.*

B. The on-the-diamond use of tobacco products (especially chewing tobacco) by *minor league* baseball players provides a role model for aspiring high school athletes.

C. More people die from tobacco-associated diseases in the United States than from violent crimes and traffic accidents.

D. Five organized amateur sports that mandate the use of helmets during practice and games are football, boxing, ice hockey, field hockey, and lacrosse.

E. Mouthguards are more important than helmets in preventing avulsion of teeth.

Equally or more important to prevent serious facial injuries is that the use of seat belts should be strongly advocated across all age groups but perhaps especially among youths who tend to believe they are not vulnerable. Car accidents involving youths frequently result in orofacial, head, and neck injuries, which could be *reduced with* the appropriate use of seat belts.

Oral-Health-Programs for Preschool-Age Children

In 1965, Head Start, a national preschool program that provides early learning opportunities to poor and underprivileged children, was initiated under the Economic Opportunity Act of 1964. Health care and health education, including a dental component, have been a continual part of this program. The dental care component includes *annual examinations, preventive services, follow-up care, and classroom instruction.* Yet after four decades of existence, there is evidence that many Head Start centers *do not* comply uniformly with the U.S. Public Health Service Standards. As it now functions, Head Start is meeting the needs of *only a fraction* of the eligible

population.[67,68] However, some states have excellent Head Start oral health programs.

Today many other private and public preschool programs exist, with the number expected to increase dramatically if a national day care policy is established. The great majority of preschool programs will be under the auspices of private individuals or organizations with limited expertise in health education, specifically in oral health objectives and preventive practices.

Academically, preschool programs must include objectives, lesson plans, and the evaluation instruments needed to measure objective attainment. Even at preschool age, the important preventive dentistry related words, concepts, and skills can be introduced to young children to be reinforced later at higher grades. At preschool age, children are *especially eager to learn* and participate. Unlike most public schools, Head Start centers usually provide toothbrushes and toothpaste and the time to use both. Some Head Start programs that are located in communities without optimally fluoridated water also offer fluoride tablet programs.

Bright Futures is a health education innovation developed by the U.S. Department of Health and Human Services whose mission is to promote and improve the health, education, and well-being of children, families, and communities.[69] It offers expert guidance for providing health services to children and their families. This project includes an excellent section on oral health entitled, Bright Futures in Practice: Oral Health.[70]

Helping Teachers Teach

In a school curriculum, teaching mathematics becomes more sophisticated as a child progresses from grade-to-grade. In first grade, the child is taught to add 1 and 1; yet at the high-school level the young adult is able to perform problems of calculus. In many courses, such as chemistry, the didactic information is reinforced by laboratory assignments; the combination of information and experience helps develop student understanding and motor skills. This same approach could be used to teach oral health. Oral-health programs should be planned so that each higher-grade level receives a greater diversity and complexity of subject matter and practical experience. For example, elementary schoolchildren have less dexterity than junior-high students. Flossing, therefore, is more easily taught and practiced in the upper grades.

At the high-school level, students should have an advanced lay knowledge of terminology, anatomy, and functions of the oral cavity and the etiology and consequences of oral diseases. They also should have the understanding to accept responsibility for (1) preventing and controlling oral diseases, (2) identifying the presence of their own oral diseases at an early stage, and (3) seeking treatment once oral disease is suspected or identified. In other words, *students should be taught to open their eyes when they open their mouths in front of a mirror.*

The Tattletooth II Integrated Program

To construct an integrated curriculum for oral health that is applicable to a continuum of grade levels, it is first necessary to consider what information is relevant for each grade level. One example of a comprehensive grade-to-grade program developed for

teaching dental health is exemplified by *Tattletooth II, A New Generation program, developed by the Texas Department of Health.*

This comprehensive oral health curriculum targets students from *preschool through grade 12.* In 1993, with funds appropriated by the state legislature, the Texas Bureau of Dental Health Services employed approximately 20 dental hygienists and dental assistants to promote the program statewide. Annually, thousands of children are served by this program with aid from the Texas Dental Hygienists' Association.

Each grade level has five core lessons and two enrichment lessons. Background information *for the teacher* is provided as part of the lesson. Educational strategies are described for integrating oral health topics into discussions involving other subjects, such as languages, arts, mathematics, and science activities. To facilitate bilingual education, the program has been translated into Spanish.

In addition, the curriculum includes scope and sequence charts, a unit test, bulletin board suggestions, audiovisual lists, and suggested letters to be sent to parents. A videotape illustrating the appropriate techniques of brushing and flossing is available on loan to schools. The curriculum and training materials were designed, tested, evaluated, retested, reevaluated, and again revised. This program *matches subject matter with grade level,* provides teachers with guidance information, and is available in two languages to target major minority groups of the state. To obtain the program for their classroom, teachers must attend in-service training provided by a state regional dental hygienist who uses a multimedia approach in the training sessions. The program is copyrighted; however, *out-of-state educators may secure it gratis and reproduce it at their own expense.* Because the program has been in use for some time and still is, some of it will be replaced over the next few years with newer materials.

Putting it all Together—Comprehensive Oral Health Programs

Treatment is not the answer to solving children's oral health problems; rather *primary prevention is the key.* From an economic viewpoint, there is little rationale for treating a disease at great expense, when the disease can be prevented at a much lesser cost. From a humane viewpoint, there is even less reason not to develop strong preventive programs supported with treatment programs when prevention fails. Research has shown that incipient dental caries can be largely controlled by techniques now available, such as *sealant placement and remineralization therapy*—techniques that are little publicized and underused by the profession.[71] *Gingivitis also can be controlled by a combination of oral hygiene practices—prophylaxis, chemical, and manual plaque control.*

Many states and communities are focusing on how millions of underprivileged children can be provided with health care. As a keystone of this effort, care must be *acceptable, equitable, affordable, and accessible to all.* It is critical that the dental profession, health educators, and public health agencies take the steps necessary to ensure that oral health is represented in the planning and implementation of such national efforts. *State dental directors must play a major role in ensuring that dentistry's share of funds will be available.* Unfortunately, some states currently do not have state dental directors, and do not intend to fill these positions. At the same time some preventive activities have been curtailed or eliminated.[71]

There are several advantages to a comprehensive, school-based program: (1) students are available for preventive or treatment procedures; (2) school-based clinics may be less threatening than private offices; (3) school dental programs facilitate and increase the effectiveness of teaching oral health subjects; and (4) dental services supplement the school nursing services by providing total health care for schoolchildren.

A combined education, promotion, and preventive program in the school would greatly reduce the amount of classroom time lost in traveling to a treatment facility. Comprehensive school programs also would obviate the loss of study time due to pain and apprehension before and after treatment. This lost time can be considerable; for example, children missed more than 51 million hours of school in 1989 because of acute dental problems.[1] Combined school-based educational and preventive dentistry programs for all schools should be feasible and cost-effective in terms of staffing, money, and material. Most important, with comprehensive school programs the decayed, missing, and filled teeth (DMFT) of students *should demonstrate a substantial and steady decrease over time.* A few of these kinds of programs have been established. Others could be established at different levels, depending on available funds as described below and shown in Appendix 19-4.

Level 1

Level 1 should include the use of a comprehensive oral-health curriculum such as the *Tattletooth II program.* Providing such a curriculum minimizes the need for teachers or health educators to locate and organize lessons in an unfamiliar field. In addition, *the teacher with the help of a school nurse and or adult volunteer* can conduct weekly fluoride mouth rinses or administer daily fluoride tablet regimens. The nurse might be responsible for preparing the mouth rinses or making the tablets available on a schedule approved by the teachers. *Health educators,* if available, help coordinate and integrate all health education activities. They have extensive training in educational principles and health and are accustomed to working with school faculty. Fluoride mouth rinse or fluoride tablet regimens are easy to accomplish, economical, and effective. Table 19-3 compares the two school-based fluoride regimens. *Neither* of these self-applied, school-based fluoride regimens should be used if community or school water fluoridation is available.

School Water Fluoridation

Fluoridating a school's water supply is similar to community water fluoridation in that *no direct action is needed by individuals to accrue its benefits* other than consuming the water or foods prepared with it. School water fluoridation is used only when the school has an independent water supply, usually in consolidated rural schools. Consolidated rural schools are ideal for this approach because all grades from kindergarten through 12 are housed in the same complex using one water source. In the past, this preventive measure was used in over 600 U.S. schools in many states. Today, the need for this preventive measure has diminished both because the water supplies of many rural schools have been incorporated into major community water systems that are optimally fluoridated and because there are numerous other sources of fluoride available including dentifrices and mouth rinses. The recommended concentration of fluoride for school water supplies is *4.5 ppm.*[72] School water

fluoridation was developed and tested in the United States in the 1950s and 1960s. Researchers found up to a 40 percent reduction in dental caries after 12 years. Installation costs are relatively expensive and workers must be trained to operate, monitor, and maintain the fluoridation unit.

Dietary Fluoride Tablets

The use of fluoride tablets in schools is a method of administering systemic fluoride to children. This self-applied fluoride regimen is for use *only* in communities in which the water supply is fluoride-deficient and *has been used in the United States and abroad for over 40 years*.[47] All children who participate in self-applied fluoride programs must have parental consent. Usually, a classroom teacher who has been trained to supervise the procedure first dispenses the fluoride tablets to participating students. Students are then instructed to put the tablet in their mouth and to chew it for 30 seconds; the resultant solution is then vigorously swished between their teeth for another 30 seconds before the participants are told to swallow the solution. Using this approach, *both systemic and topical benefits* will accrue. The procedure is easy to perform, requires little time, and there are no waste products to dispose of. Studies conducted in the United States show that school-based fluoride tablet programs provide about a *20 to 30%* reduction in new caries lesions. A daily fluoride tablet appears to be *more effective* than a weekly mouth rinse, as well as being preferred by teachers.[73] The major drawback is that it is a daily procedure and some teachers object to it for this reason.

It is important to note that caries preventive effects of school fluoride regimens may not be permanent. After an 11-year follow-up study in Norway, it was concluded that the residual benefits of school-based fluoride programs decrease as the length of time between previous participation and follow-up increases.[74] It should be pointed out, that students should be educated to use a fluoride containing dentifrice during and following the school-based fluoride regimens. In contrast, Kobayashi and coworkers found good post treatment benefits after 11 years.[75]

Fluoride Mouth Rinsing

Fluoride mouth-rinsing programs are *the most widely used* school-based fluoride regimen in the United States and usually are *supervised by classroom teachers* or other adult volunteers. Caries reductions range from *20 to 25%*, although few recent studies have been conducted.[76] Fluoride mouth rinse regimens as originally conceived consisted of mixing a preweighed packet of fluoride powder with a specified amount of water in a container with a plastic pump calibrated to dispense 5 or 10 mL of solution that would yield a 0.2 percent neutral sodium fluoride rinse. After mixing, the solution is dispensed into paper cups for use by the students. Today, most schools order premixed solutions that come in individual containers. This latter approach is somewhat more expensive but it simplifies the procedure for use in classrooms. Using premixed solutions *reduces the time required* of paid or volunteer staff to simply dispensing a container and napkin to each student and then supervising the rinse procedure. Students are requested to put the solution in their mouth and to rinse vigorously for 60 seconds (Figure 19-5). When instructed, students are asked to empty the contents of their mouth back into the cups and blot their lips with the napkin. The waste products are then put into a plastic bag for disposal. Fluoride mouth rinse used

in schools is available in either a flavored or nonflavored and sweetened or nonsweetened varieties. Generally, this procedure is *not recommended for children before first grade* unless extensive training is conducted with the children to ensure that they do not swallow the contents of the cup. The weekly solution, if swallowed over time may contribute to fluorosis among children 6 years of age or younger because some of their permanent teeth are still developing. (See Appendix 19-5 for details on conducting a mouthrinse program.)

Level 2

Level-2 programs *include level-1 activities plus the addition of a dental hygienist* to the school health staff. The inclusion of a dental hygienist in a comprehensive school health program is critical. A dental hygienist is educated to plan and participate in school programs that include oral prophylaxes, use of a variety of methods of fluoride application to foster remineralization, teaching oral hygiene procedures, counseling on diet, placement of pit-and-fissure sealants, and screening and referral for suspected oral pathology for definitive diagnosis and treatment. A dental hygienist also serves as a school-resource person.

Many school districts or public-health agencies have dental trailers that are *used to provide prophylaxis and screening programs* for students. Others use portable equipment that is set up in a room designated by school authorities. Older, *teenaged students are more likely to present with gingivitis and calculus.* A periodic prophylaxis by a dental hygienist during the school years may help avoid the onset of periodontitis later on. In addition, the personal contact with a dental hygienist can help motivate teenagers to develop satisfactory plaque removal techniques and to understand the need to seek professional care when needed.

Dental trailers also may be used by hygienists *for placement of dental sealants.* Dental sealants are highly effective in protecting occlusal surfaces and lingual and buccal pits and fissures—sites where *up to 90% of all caries lesions occur.* In the 1988 to 1991 Third National Health and Examination Survey, phase 1 (NHANES III), *less than 19% of U.S. children and adolescents between 5 and 17 years of age had one or more sealants placed.*[76] In contrast, in Finland, so many of the occlusal surfaces are covered with sealant that these surfaces are often excluded in decayed, missing, and filled surfaces (DMFS) studies.[77]

The marked benefit of sealant placement in reducing caries incidence was reported by Sterritt and Frew in a study conducted in Guam.[78] Some 75,000 teeth of 15,000 children in grades 1 through 8 were sealed by *17 preventive dentistry technicians.* In a period of 2 years the average number of carious lesions per child *dropped from 5.35 to 2.92.* The first year retention rate for the self-curing sealant was 94% for first molars, 97% for premolars, and 75% for second molars. In one state program, it was demonstrated that, dental sealants reduced oral health disparities among school-aged children.[79] Sealant placement, when coupled with a *follow-up gel application of fluoride* helps provide protection to the *whole tooth.*[80] Ripa and colleagues have correctly pointed out that the combined use of sealants and exposure to a fluoride regimen in school *can result in a virtual elimination of dental decay in elementary schoolchildren.*[81]

Cost per child for sealant placement varies depending on whether dentists, dental hygienists, or dental assistants are used. A 1989 estimated cost for sealants ranged from $13.07 to $28.37.[71] In contrast, the restoration of an occlusal lesion averages about $51.00.[82] Most important, it must be recognized that once a restoration is placed it will continue to need to be replaced which further weakens and compromises the tooth because the restoration becomes larger at each replacement.

A guide, "Seal America: The Prevention Invention," has been developed for purchase at a nominal fee. It was supported by the Maternal and Child Health Bureau of the Health Resources and Services Administration. The kit was designed for use in developing and implementing dental sealant programs in communities.

Level 3

A level-3 program consists *of all of levels 1 and 2 requirements plus the addition of a treatment delivery option.* This level of a comprehensive school oral health program includes the ability to identify and refer all pathology for treatment as early as possible. To achieve this level, an annual screening is indicated for all children and a semiannual screening for children classified as high risk.

State practice acts permitting, *triage* with possible *referral* to a treatment facility can be accomplished by a dental hygienist.[83] During routine prophylaxis procedures and sealant placement, a dental hygienist can identify early pathology and refer the student for expeditious definitive diagnosis and treatment.

All too often, the present method of managing schoolchildren with oral problems is for the teacher or school nurse to send home a note indicating a need for treatment and recommending that a child be taken to a dentist. This approach *assumes* that the parent immediately seeks a private dentist or goes to a public-health clinic. In turn, it is *assumed* that when the dentist completes treatment, a postcard is returned to the school hygienist or nurse indicating that the referred pathology has been treated. Theoretically, this type of system has the advantage that it uses the professional delivery systems existing in the student's community. Unfortunately it does not always work. Not all parents respond positively because of lack of money or insurance, apathy, or lack of available time to take the child to a dentist. This formula of "no money, no priority, no dentist, equals no care" is an elementary equation repeated countless times each year in our schools.[84]

Another option for referral involves *contracting with local practitioners* to offer specific procedures for predetermined fees. In this case, the referral can be a direct transaction between the school system and dentist(s). The bill submitted by the dentist for completed work constitutes verification that the child received treatment. A third option often adopted is to *bus children to a public health clinic.* Or, in some communities dental societies have organized their dentists to volunteer to treat needy students.

The objective of whatever level of preventive care program is selected is that it be affordable, and accessible to all—*with a priority for high-risk students. Once the primary preventive dentistry procedures have reduced the incidence of oral disease to*

that of the annual treatment workload, the number of extractions for a school population should approach zero.

Figure 19-5 Students rinsing 60 seconds with fluoride under supervision of their teacher.

Foreign School-Based Programs

Sometimes we can learn other approaches to solving similar problems by looking at models in other countries. For example, the New Zealand School Dental Service reads like an exemplary level-3 program. It is *accessible* to all, being based in the schools, *equitable*, with all children being able to enroll, *affordable* because there is no charge for service, and *acceptable*, with 96% of the pupils being enrolled. The Service was formed in 1921 as a result of pressure on the government *by the dental profession* to help cope with the poor state of children's teeth. Originally, young women were trained to accomplish fillings and simple extractions. Today, both men and women are trained as dental therapists who can practice *only in state institutions.* They are not licensed for private practice. Supervision is provided by public health service officers and senior dental nurses. The Service is based in clinics located in larger schools and takes care of all preschool, primary, and intermediate students between ages 5 and 13.

Children are examined annually, although those considered at *high risk are examined semiannually*. The guidelines for high risk are:

1. Over 6 and under 9 years of age—Four or more deciduous teeth with full occlusal or compound fillings or new carious lesions other than buccal or lingual pits in permanent or deciduous teeth.

2. Over 9 years of age (permanent teeth only)—A full occlusal restoration or carious lesion on a first permanent molar, an interproximal cavity before age 9, or a new lesion in the previous 12 months other than a buccal or lingual pit.

The preventive program *focuses on high-risk children.* A 0.2 percent fluoride mouth rinse is used to prevent demineralization of "white spots," while fluoride varnish (Duraphat) is used for remineralization therapy of interproximal, enamel-limited lucencies. Finally, *fissure sealants are placed on all newly erupted vulnerable permanent molars to prevent occlusal caries.* This preventive emphasis and available treatment has resulted in a *drop* of DMFT of 10.7 in 1973 to 1.88 in 1992 (Figure 19-6), and a parallel drop in extractions from 18.20 per 100 in 1966, to 4.00 in 1992.[85]

Denmark also has an excellent and comprehensive school-based oral health care program. There has been a long tradition of providing oral health care to Danish children, in fact the first school dental clinic was established in 1909. The Child Oral Health Care Act of 1971 is the basis upon which the current Danish child oral health care service was established. Essentially, this legislation mandated communities to provide free dental care for children 6 to 16 years of age. The process was an incremental one with emphasis on primary prevention and oral health education of all parties. Concomitantly, national epidemiologic data was established and maintained, which has provided the scientific evidence of the successful reduction of dental caries among Danish children between 1972 and 1992. In 1986, a new national law was introduced to replace the 1971 Act. The long-range goal of the revised national law

was stated as: "The goal of the dental service is for the population to obtain healthy teeth, mouth and jaws, and to preserve them, in functional condition throughout life; this should be accomplished through a sufficient home dental care regimen, and a comprehensive preventive and curative dental health service." A major change in the 1972 Act was to include *all* children 16 years of age and younger. Again, the emphasis was on the use of fluorides, dental sealants, at least annual examinations and extensive educational interventions. Preventive regimens have been modified as new research has become available. The treatment of oral diseases and malocclusion also was included in the new legislation and by 1987 the system included all children. The law mandates oral health education for all children, parents and teachers. Education in schools is a part of the regular curriculum and, thus, is adjusted for age appropriateness and at the same time to maintain interest. Oral health instruction also is provided to gravid women and oral health instruction is included in home visits for newborn and young children. Older children are taught how to make dental appointments so that they can continue their oral health care as adults. The dental teams who provide these services include dentists, dental hygienists and dental assistants. Danish school children enjoy one of the lowest caries prevalence in the world.[86]

Figure 19-6 New Zealand Dental Service. Average DMF, ages 12-14 years, 1973-1991. Caries in permanent teeth has declined 78% in 18 years. (From internal data of the Department of Health, New Zealand, generated from annual returns prepared by principal dental officers.)

Question 5

Which of the following statements, if any, are correct?

A. Preventive care for all Head Start children is authorized; as a result, preventive services are now provided to the *majority* of Head Start children.

B. Both incipient caries and gingivitis can usually be *prevented, arrested, or reversed.*

C. No fluoride tablet program should be conducted in a school serviced by a water system containing optimally fluoridated water.

D. Levels 1, 2, and 3 school oral-health programs provide highly effective dental care options ranging from education- prevention to education-prevention- treatment.

E. An abrupt drop in occlusal caries incidence can be detected *within a year* after the initiation of an occlusal sealant program.

Research and Evaluation

There are approximately 48 million students in 110,000 elementary schools in the United States.[87] Many of these children currently benefit from preventive regimens previously described and supported by research. Many other children are not so fortunate, especially children from poor families. Research is needed to evaluate the success of currently used *programs* as well as the *feasibility* and *cost-effectiveness of future strategies* to promote oral health education, prevention, and treatment. Today, there is a need for research and evaluation on the best approaches to promoting oral

health in schools with a focus on school-based clinics. The primary objective of such research programs must focus on the 3 M's—*manpower*, *money*, and *material*, plus the *amount of classroom* time it will take from conventional classroom education.

To improve cost-effectiveness and oral health for all, there is a need to develop *validated techniques to identify high-risk students* for both dental caries and gingivitis. To secure such information, research and evaluation is necessary. Finally, the use of *standardized methods of recording data including the use of computerized recording* is necessary to permit a comparison of data from studies to accelerate administrative decisions.

Summary

A large segment of our children and adolescents are at risk for dropping out of school before completing high school as a result of a wide range of health, economic, and behavioral problems. Moreover, a large proportion of our school-age children do not have access to basic preventive and primary dental and medical care. With the challenges posed by overwhelming need and limited resources, children deserve to receive information and education that enables them to make informed choices about their health. Also, they deserve the opportunity to learn skills and develop attitudes that enable them to practice appropriate behaviors to enhance their oral and general health. Finally, they deserve to receive services that prevent and/or treat oral diseases. We have the ability to essentially eliminate most oral diseases among children. Less obvious are the political and administrative means needed to make these cost-efficient measures available to the children. School-based oral-health education and promotion programs give children and adolescents a chance to learn about their oral tissues in health and disease. Comprehensive, school-based oral-health prevention and treatment programs provide a means not only to learn about disease, but also to maintain the health of the oral tissues and structures. Dentists, dental hygienists, and students of each discipline have an opportunity and the responsibility to help make such comprehensive programs available.

The major successes of school-based oral-health programs have been achieved when education has been combined with active prevention and/or treatment programs.

Unfortunately, too few schools have routinely included active preventive dentistry regimens—fluoride mouth rinses and tablet programs, sealant applications, remineralization therapy, and a strong emphasis on the use of fluoride-containing dentifrices while brushing—all of which can reduce the DMFS of a school population. This reduction can be accomplished with only a minimal change in self-behavior or compliance required of the student. A few school systems have employed dental hygienists as resource personnel to aid in the teaching programs and/or to provide primary preventive services. The availability of a dental hygienist provides an effective and economic means for schools to plan and participate in higher-level preventive programs. Hygienists can function on the school staff as oral-health educators, conduct preventive programs, accomplish triage, and arrange for referral and follow up to ensure completeness of treatment.

The ubiquitous availability of sucrose-laden desserts and snacks can best be controlled by the school dietitian reducing the availability and frequency of foods and

desserts with high sugar content. The school administrator also can aid in the objective by prohibiting the on-campus placement of vending machines to dispense high-sucrose snacks.

Protecting the oral health of future generations is a commitment that must be shared by parents, teachers, school administrators, and all health professionals. This shared responsibility is especially relevant now that national health objectives for total child health care are established. Possibly the time is propitious to think in terms of a national school oral health policy—one that endorses universal access to oral-health education, health promotion, preventive regimens, triage, and treatment referral capabilities for discerned pathology.

As a final note, if oral-health promotion is to be accomplished through the school systems, it must be integrated with the general medical health program. In order not to detract from the teaching of the classic academic curriculum, the school year must be lengthened proportionate to the increased time demands of promoting health, and the school system's budget must be increased in order to meet the requirements for additional facilities, manpower, and materials.

Answers and Explanations

1. A, D, and E—correct.

B—incorrect. Teachers are certified by academic subject to be taught—English, mathematics, science, etc., but *definitely not* in in-depth comprehension of societal issues.

C—incorrect. Same answer as B; teachers are not trained or certified to impart oral-health education.

2. A, D, and E—correct.

B—incorrect. Substitute "least" for "most" and the answer becomes correct.

C—incorrect. The 13.8 hours figure is for the *total* amount of health education; of this time, one cannot expect sufficient time will be allowed for oral health education and promotion.

3. B, C, D, and E—correct.

A—incorrect. Every day, millions of people self-apply topical fluorides using fluoride dentifrices.

4. C, D, and E—correct.

A—incorrect. The health dangers of tobacco should be started at the elementary school level in order to intercept the publicity and peer pressure to use cigarettes and smokeless tobacco at an early age

B—incorrect. On-the-field minor-league baseball players and employees are not permitted to use tobacco products.

5. C, D, and E—correct.

A—incorrect. The first part of the statement is *correct*; the second part, which is the most important, *is incorrect* and should be corrected. In other words, prevention is authorized but not implemented.

B—incorrect. Since approximately 90% of the total lesions in the mouth occur on the occlusal surface, the reduction of caries incidence on this surface can be detected soon after the initiation of a sealant program.

Self-Evaluation Questions

1. The expanded eight-compartment model for school health programs includes, in addition to health education, health services, and a healthy environment, at least two additional health attributes which are: _____ and _____.

2. Give five health tasks which *might* be assumed, or are assigned to a school-based health clinic: _____, _____, _____, _____, and _____.

3. It is estimated that 80% of carious lesions in a school population are found in only _____ % of the students.

4. Two nations with comprehensive school dental programs are _____ and _____.

5. The two most important explanations for the cariostatic properties of fluoride are that it inhibits _____, and equally (or more) important, facilitates _____.

6. The two most important school-system officials who are in a position to limit the ingestion of sugar-containing foods, desserts, and snacks during school hours are the _____ and the _____.

7. Give three reasons why daily classroom toothbrushing is impractical: _____, _____, and _____.

8. Smokeless tobacco education includes: _____ techniques and _____ techniques.

9. One state school system that integrated an oral health teaching program that includes all 12 grades is the Texas _____ (name of program which may be obtained gratis by out-of-state health departments and major health providers).

10. Assuming that you are the coach, and an anterior tooth is avulsed by a hockey puck, describe how you would handle the situation _____.

11. One program that has been found to increase the number of fruits and vegetables consumed by school-aged children is the _____ program.

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Chapter 20. Preventive Oral Health Care for Compromised Individuals - *Roseann Mulligan Stephen Sobel*

Objectives

At the end of this chapter, it will be possible to:

1. Explain why patients with the same handicaps can respond differently, based on communication and patient treatment techniques used by the dentist.

2. Discuss how visual, auditory, speech, and cognitive deficiencies can be identified and at least partially compensated for in preventive dentistry planning and implementation.

3. Illustrate how some functional deficiencies can be identified that require consideration in prescribing preventive dentistry techniques and devices.

4. Name and describe how new or modified devices or aids can be used to stabilize or aid patients with neurologic or physical disorders.

5. Cite at least three examples of how fluorides, pit-and-fissure sealants, and sugar

discipline can be integrated into the preventive dental program for compromised individuals; also list possible exceptions.

6. Discuss the need to educate dental and dental-hygiene students, dentists, dental hygienists, and lay personnel to aid special patients in the home, in the office, and in institutional settings.

Introduction

A compromised individual is a person who has one or more physical, medical, mental, or emotional problems that result in a limitation of the ability to function normally in fulfilling the activities of daily living (ADLs). More than 36 million individuals in the United States are compromised.[1]

When a patient presents for care, the clinician's judgment of the patient's capabilities may be biased. Unrealistic expectations of the ravages of a specific disease may have been formed in advance from reading the scientific literature or from firsthand treatment of another patient similarly afflicted. These experiences may produce unconscious and inaccurate generalizations about a person's capabilities. Such labeling can undermine a patient's preventive oral-hygiene program, because it does not consider the individual's actual capabilities.

This chapter presents information on how to assess the capabilities of a patient of any age within any disease category and offers suggestions on the development of individualized oral disease prevention programs. Oral-hygiene aids and techniques applicable to a preventive program for the compromised patient, as well as special management techniques, are included.

Sensory Capabilities

Communication is compromised if the patient's hearing, vision, or speech is impaired. Communication is a critical factor in any attempt to engage the patient or caregiver in a behavioral change such as that required to improve the status of the patient's oral health.[2]

Visual Deficits

A number of factors, from harmful prenatal and perinatal environments to the normal aging process, can alter visual acuity. These changes may range from correctable deficiencies to total blindness. Other common visual deficits include a loss of peripheral vision as occurs with glaucoma[3] or visual field cuts resulting from a cerebrovascular accident.[4]

A patient with a significant visual disability may be carrying a white-tipped cane or may arrive with an escort. If the individual requests, the nondental staff escort—whether human or guide dog—should be allowed to accompany the patient into the treatment room. The escort may then be permitted to stay if space is available in the treatment area and if the presence of the escort contributes to the patient's comfort. An escort who functions as an attendant may be involved in any oral hygiene instructions and demonstrations, and this person may be the crucial element necessary

to a successful home care program.

Instructional materials to be used with patients who have decreased visual acuity could include commercial products that have been developed for pediatric dentistry programs, because such products have large pictures. Other commercially prepared pamphlets for self-instruction and information have limited value because the size of the print used in such pamphlets is too small for the visually impaired to see comfortably. Custom-made instructional sheets may be produced by the dental office using large black letters of at least 12-point type on off-white or white paper.[5] When appropriate, chairside instructions of tooth brushing and flossing should be demonstrated on oversized models of the dentition with a giant-sized toothbrush (Figure 20-1). These large models allow the patient with limited visual acuity to see and to understand some of the more subtle aspects of tooth brushing, such as the correct angulation of the bristles into the gingival crevice. Red floss can help when demonstrating flossing to those with visual impairment who have difficulty seeing white floss. Green floss is also available and can be used, but red is easier for the aging eye to see.[6] Once the flossing technique is understood and visual acuity permits, the patient may switch to white floss for regular home use. This allows the patient to check the color of the floss for possible gingival bleeding.

To demonstrate brushing and flossing techniques in the office, an inexpensive magnifying mirror should be employed to assist the patient in observing his or her own performance. A similar mirror should be recommended for the patient's use at home. These mirrors, commercially available, are usually used for applying cosmetics. With attachments provided, they can be adapted to be hung around the neck, affixed to a wall, or placed on a countertop, thus allowing patients to keep their hands free for performing oral hygiene and still use the magnifying mirror to enhance vision. Many also feature a lighted mirror, another aid for enhancing vision. Some patients who have experienced a cerebrovascular accident lack the spatial-perceptual skills necessary to use a mirror. For these people, using a mirror causes confusion and therefore is contraindicated. If a patient has visual problems so significant that a mirror cannot be used, he or she must be sensitized instead to the "feeling" and "smell" of a clean mouth to attest to the success of oral hygiene measures.[7]

For individuals with visual field cuts or decreases in peripheral vision, be sure that demonstrations are within the patient's visual field. To check the limits of a patient's vision, perform a visual assessment by positioning your hand in various locations around the patient's face while holding up one or more fingers. For each position, ask the patient how many fingers can be seen, and note whether the patient moves the head rather than just the eyes to see your fingers.

Many patients with visual problems experience an increased sensitivity to light or glare. Indiscriminate positioning of the dental light so that it shines in the eyes of a patient can result in significant discomfort for such a patient. This can be avoided by carefully focusing and positioning the operatory light. Also, it is advisable to have sunglasses available; these can be disinfected after use.

With loss of orbital fat during aging, the eye becomes "sunken" with an increase in the upper lid fold and a decrease in peripheral vision. Extra-ocular muscle weakness by the age of 70 inhibits the ability to rotate the eye upward greater than 15° from the

horizon.[8] Since arthritic changes in the cervical spine occur frequently among the elderly,[9] it may be difficult or impossible for some patients to tip the head back. Thus, to enhance communication with all patients, it is best to converse from a sitting position directly in front of the patient so that the clinician's eyes and the patient's eyes are at about the same level.

Hearing Disabilities

Hearing problems can occur in all age groups. The sound of dental equipment in another room, background music, and street noises can hinder communication with the hearing-impaired, as these sounds often mask the sound of speech. The most common problem in communicating with the hearing disabled, however, occurs when the speaker is not directly in front of the patient, at the same eye level, face to face.[10]

Most patients with hearing disabilities do some speech reading (formerly termed lip reading), but even the best speech reader is able to decipher only about one fourth of a message conveyed entirely through this method.[11] The hearing-disabled patient also relies on the communicator's facial expression and body language.[10] Speaking distinctly and slightly slower, without exaggeration and in a well-modulated voice, facilitates communication.[12] The progressive loss of hearing of high-frequency tones that commonly occurs among the elderly may make a female voice more difficult to hear than a male voice. Avoid back lighting[13] that places the speaker's face in a shadow. Shouting at a hearing-disabled patient is not recommended, because loud sounds are actually more difficult for the impaired ear to comprehend.[10] When speaking to a patient, other than while doing a procedure, lower or remove your face mask.

Pantomime and demonstration may be necessary when working with the hearing disabled. When writing information, use a clipboard and a red felt-tipped pen. After providing oral-hygiene instructions, have the patient demonstrate the suggested oral-hygiene skills on models or in his or her own mouth to assess how well the message was comprehended.

Hearing aids are becoming more difficult to detect, as new technologies allow for decreasing size or positioning entirely within the ear canal. Long hair may mask their presence; therefore, a conscious effort should be made to look for such aids (Figure 20-2). If preventive instructions are to be given to a patient with a hearing aid, be sure the patient's aid is in place and turned on. Often patients turn off or remove their hearing aids in anticipation of dental treatment, because the proximity of the speaker's body to the aid may cause it to emit high-pitched squeals called "feedback." Providing dental care with the clinician seated in the 12 o'clock position places the operator's arm nearly in contact with the patient's ear, and the clinician's sleeve may accidentally dislodge an over-the-ear hearing aid. Handpieces can also cause many types of hearing aids to produce feedback. Suggest to the patient that the aid be removed or turned off prior to treatment and replaced or turned back on prior to receiving instructions.

Hearing aids are expensive devices, and individuals who wear them usually prefer to remove them themselves. Once removed, the aids should be placed in a secure spot, such as the patient's pocket or purse, rather than on the bracket table where they could

be forgotten or gathered up and discarded. Individuals who have both hearing and visual deficiencies may have their hearing devices constructed as part of the frame of their glasses. In such cases it is recommended that the glasses be left in place but with the hearing device turned off.

For patients with visual or hearing deficiencies, keep distractions to a minimum (it is advisable to have office background music turned off at this time). This includes any interruption of the clinician at chairside as well as the distraction created by auxiliary personnel entering and leaving the room.

Speech and Language Disorders

Several conditions affect the motor or the cognitive components of speech or both. The patient with cerebral palsy may have speech impairment because of problems of the central nervous system affecting the muscular movements needed for speech.[14] With practice, a clinician who listens carefully and patiently to such speech can become adept at understanding much of it. This is the same sort of technique many dental providers have already achieved in learning to understand patients who attempt to speak with a rubber dam in place. Individuals, such as those who are mentally retarded, even though physiologically capable, may not use language because of their level of intellectual or emotional functioning. Those with neuromuscular diseases may have a weakness so severe that the muscles necessary to articulate sounds are unable to function. Deterioration of speech that once was normal may be due to progressive hearing loss. Individuals who are unable to hear the range of frequencies of the speech spectrum may develop a monotone voice. In addition, such a person often loses the ability to recognize how loudly he or she is speaking.

The patient who has suffered a stroke is particularly prone to language disorders. One type of disorder common to stroke patients is aphasia. In aphasia, the reception, integration, and expression of language are impaired. The aphasic patient, therefore, has difficulty finding the right words to communicate,[15] and this inability may be so pronounced that the patient may become very frustrated. This is especially true for those individuals who are otherwise cognitively intact. When dealing with these individuals, frame questions so that they can be answered with "yes," "no," or just a shake or nod of the head.

The aphasic individual may also omit or substitute sounds for words. The words then may be meaningless by themselves but convey intent by the way in which they are expressed. The speech may consist only of nouns, verbs, or a few adjectives. An extreme example of an aphasic patient was a woman who suffered a stroke so severe that her verbal communication was reduced to two exclamatory words that were always said together. With the help of her facial expression, body language, and the force and intonation used in saying these words, dental appointments were completed to the satisfaction of the patient and the provider.

Dysarthria is a speech disorder resulting from a motor dysfunction of the speech-producing elements. This dysfunction may be caused by lesions in the central nervous system, the peripheral nervous system, or in the muscles themselves. The symbolic language is intact; however, the coordination necessary to produce speech is impaired. This type of disturbance occurs in patients with amyotrophic lateral sclerosis (ALS),

Parkinson's disease, traumatic brain injury, myasthenia gravis, multiple sclerosis, and stroke.[16]

The substitution of written for verbal communication is a possible option for individuals in whom the recognition of language is still intact. Unfortunately, many of the causes of speech disorders result in slight or pronounced paralysis or tremors that prevent the patient from writing legibly. One solution is to provide the patient with a lapboard containing preprinted letters, common words, or pictures. Individuals with a knowledge of language but an inability to speak or to have their speech or writing understood can point to the letters or words or pictures to communicate. Another method is a sophisticated, small typewriter-like device in which a keyboard is used to type out a tickertape message. Quadriplegic patients may be outfitted with a tongue control that permits very subtle movements of the tongue against a toggle switch. This action causes letters to be printed on a television monitor mounted to a wheelchair or bed.

Today's society places great importance on verbal communication; therefore, an individual with poor verbal skills is sometimes incorrectly perceived as having poor cognitive and intellectual abilities. This is not necessarily the case.

Although nonverbal communication, such as smiling, hand holding, and shoulder touching, plays a role in the clinician-patient interaction, it becomes extremely significant when there is no alternative. In such a case, the clinician needs to enlist either the patient's attendant or a family member who has become attuned to "reading" the patient's needs. Usually, these constant companions can help interpret the underlying message of such nonverbal actions as a rolling of the eyeballs or a fixed stare.

Most autistic patients are known to be unable to use language appropriately, and many present significant management challenges. Visual pedagogy was used for a group of autistic children in Sweden. Pictures of the location and the activities to be anticipated were shown to the autistic patient including the dental office, outside and inside, the dental chair, a wide-open mouth, a mouth mirror, and a toothbrush. The pictures were shown to the patient at home before the appointment and explanations were given thus familiarizing the patient with what was to come. The technique proved quite successful for clinical care and can be used for preventive activities as well.[17]

In summary, both verbal and nonverbal techniques play roles in the communication process between a dental-care provider and a compromised dental patient. Speaking directly to the patient from a sitting position in front of the patient in a well-modulated, well-articulated voice and reinforcing each step of the communication with nonverbal cues are all techniques that should be used to produce a successful relationship with a patient who has impaired communication skills.

Figure 20-1 Large dental models and a helping hand are needed to perfect toothbrushing habits in the visually impaired.

Figure 20-2 When longer hair is combed over the ear, this hearing device can be very inconspicuous—and vulnerable to handpiece noise.

Question 1

Which of the following statements, if any, are correct?

A. Many specialized preventive techniques applicable to one handicapping condition are applicable to other handicapping conditions.

B. During periods of patient-dentist communications, it is better that the dentist be able to clearly see the patient's face than vice versa.

C. An aphasic patient is one who has difficulty in articulating personal thoughts and observations.

D. When a patient is unable to form words because of a motor dysfunction problem, the condition is known as dysarthria.

Cognitive Capacities

The functional capacity of a patient is of far greater importance than intelligence quotient (IQ) test results in determining the capability of benefiting from preventive dentistry instructions. For example, a patient who is mentally retarded is expected to have a low IQ, short attention span, and difficulty in understanding oral-hygiene instructions. Yet many of these patients, when properly taught and motivated, can successfully perform oral-hygiene procedures. To be successful, one must first determine the patient's level of cognitive ability and then direct all instruction to that level. Rather than attempting to analyze intelligence test scores, ask the patient a few simple questions to determine his or her functioning level. Questions of the type that might be asked of the patient, not the family member or attendant, could pertain to such everyday conversational topics as (1) What do you do in school (or at work, or in retirement)? (2) What hobbies do you like? (3) Why do you like a favorite television show? or (4) What has been the most difficult task you performed lately? One mentally retarded patient who responded to a similar series of questions confided that he enjoyed his job as a file clerk in a sheltered workshop environment, but he was sometimes confused trying to remember the order of letters in the alphabet. He added that the most difficult job he faced was paying the rent at the first of each month. Even though he knew he had sufficient money, the responsibility of ensuring that the funds reached his landlord in time caused him a great deal of anxiety.

Such information from the patient offers insights into levels of responsibility, understanding, attention span, usual level of dexterity, and memory for details. These facts greatly assist the dental-care provider in selecting the appropriate vocabulary, level of complexity of instruction, and reward system for adherence to a customized preventive dentistry program.

If it is determined that the patient is intellectually or cognitively impaired, the traditional education program for oral-hygiene techniques must be modified. Brushing the teeth is a complex task that needs to be broken down into very simple, discrete steps. This allows the impaired patient to follow the instructions and to succeed at each step toward the final goal, thereby integrating the simple tasks into a final complex task.[11] At the first appointment it may be possible to address only the

brushing of the occlusal surfaces of the teeth. This activity should be monitored and reinforced until a level of satisfactory compliance and incorporation into the patient's daily routine is achieved. Clinicians, in their quest to get the message across to the cognitively or intellectually impaired patient often tend to do and to say too much. It is important to keep instructional periods short with frequent repetition of the information. Using a level of language that is understood by the patient without being insulting is key. Written or tape-recorded reminders can be given for homework. At each appointment, the patient should be asked to state or show what he or she has been doing since the last visit. This provides feedback about the effectiveness of the previous instructional period and the patient's memory and mastery of the technique.[11] Of course, one must use judgment and an understanding of the patient's cognitive functional level in assessing the validity of the patient's answers.

Demonstrations of appropriate behavior by any dental patient, especially those with decreased cognitive functioning, should always be followed by immediate and positive feedback.[19] Reinforcement throughout the learning period should be supplemented with both verbal and nonverbal rewards; for example, a smile or a gift of a new toothbrush is often motivating. Development of rapport aids in reducing stress, which can be detrimental to an individual's ability to learn. For this reason all learning should occur in an environment in which the dental staff can reflect warmth and friendliness.

Family members or guardians, teachers, or other caregivers must assume responsibility for oral health care programs of patients with little cognitive ability.[20] The selected individuals should be thoroughly instructed by the dental staff in the proper techniques for that patient's oral health.

Functional Performance

Toothbrushing and flossing require the fine-motor skills or dexterity of the small muscles of the fingers and hands as well as the gross-motor skills of the larger muscle groups in the upper extremities.[21] The functioning of numerous muscles and nerves of the head, neck, and upper extremities are all involved, as is the range-of-motion capability of the joints, especially the shoulders and elbows. In many disabilities, one or more of these elements may be adversely affected or limited. Arthritis, for example, is a disease primarily of the joints and therefore often restricts the range of motion of the joint. Cerebral palsy is a central nervous system disorder. Because of aberrant neural signals transmitted to the muscle fibers, fine-motor skills are impaired. Muscle contractures also characteristically affect both the range and motion of the joints. A neuromuscular disease such as myasthenia gravis affects the nerve transmission process itself, resulting in a musculature so weak that dexterity, gross motor skills, and range of motion may all be affected.

An accurate assessment of a patient's ability to perform oral hygiene depends on the evaluation of each component of the task. Once a difficulty has been identified, either a device or a person is needed to compensate for the patient's inability. Gross motor skills such as grasping a toothbrush handle can often be improved by *orthotic*[a] appliances (Figure 20-3). Range-of-motion limitations may be altered by physical therapy, especially in the early stages of injury or disease, or in some cases by surgery. Dexterity necessary for the production of the small vibratory strokes

recommended in tooth brushing usually cannot be enhanced through physical medicine, surgery or orthotic remedies. For certain patients, an electric toothbrush may provide effective compensation for this lack of ability.

[a]Orthotic appliances are devices, such as splints and braces, that are used to provide support to deformed or weakened limbs.

Assessment

Specific hand function tests employed by occupational therapists to evaluate a patient's ability to use his or her hands can help dental clinicians assess a patient's potential ability to accomplish oral-hygiene techniques. If the patient shakes your hand when first greeted, pay attention to the strength of the patient's handclasp. Individuals with a weak grasp should be asked to firmly grip the index finger of the clinician's hand. If the grip is weak, the patient should again be asked to grip the finger "as hard as you can." Repeating this procedure several times using two, three, and four fingers of the practitioner's hand in place of the one finger enables the practitioner to decide at what diameter the patient has the strongest grip[22] (Figure 20-4). If it is determined that the patient would benefit from a manual toothbrush, the handle will need to be increased in diameter to match the number of fingers at which the patient had the strongest grasp.

The range of motion of the elbow and shoulder can be determined by having the patient extend and flex the lower arm, or rotate that arm about the shoulder. This information may be more quickly obtained by asking the patient, "Can you feed yourself?" Individuals who are able to do so, even if they use orthotic splints or other adaptive aids, are probably able to perform oral hygiene procedures. Patients who use special devices to aid in self-feeding should bring these devices to the dental office so that toothbrushes and other oral hygiene aids can be modified to fit them. The best way to assess whether a patient has sufficient dexterity and cognition to perform adequate oral hygiene is to offer the patient a toothbrush and to directly observe his or her success in plaque removal. This accomplishes two objectives: 1) The clinician can assess the patient's current level of ability and understanding prior to any intervention. 2) The clinician can establish a baseline level of achievement against which future improvements or modifications can be measured.

Noticing the ease or difficulty with which each movement is accomplished (grasping the brush, angling, and moving it) and the care needed to synchronize these actions into purposeful motion gives the dental professional insight into the capabilities, training, and education of the patient to date. An understanding of the patient's current abilities allows the health care provider to determine the type and number of educational interventions to be introduced. Patients with compromised motor skills often compensate for their deficiencies in ingenious ways. Therefore, their ability to perform their own oral hygiene should not be prejudged, and they should be given opportunities to demonstrate their proficiencies. Many patients who appear unable to handle a toothbrush or floss, because of deformed fingers or decreased motor functions, can compensate and function reasonably well.

Figure 20-3 An orthotic device that permits a firmer grasp on a modified toothbrush.

Figure 20-4 The muscular strength of a patient's hand can be assessed by handshake or by a grasp of the clinician's fingers.

Question 2

Which of the following statements, if any, are correct?

A. It is an inviolable rule that as the IQ decreases, so does the possibility of attaining cooperation from a mentally handicapped patient.

B. As an individual's disability increases, the need becomes greater for support from other individuals.

C. Explanations on primary preventive dentistry given to handicapped individuals should be detailed.

D. The hand strength and ability of a patient to use a toothbrush can often be determined by a handshake.

E. The best way to find out what a handicapped patient can do is to ask the person to accomplish a stated task.

Attendant Care

To ensure dental care and compliance with home self-care preventive programs, complete cooperation must be established among the family or caregivers, the health provider, and, to the extent possible, the patient.[23, 24] Many compromised individuals are unable to handle their own hygiene due to sensory, cognitive, or physical deficits. For these individuals, an attendant or family member should be instructed in the proper oral-health care for the patient.[25] If long-term compliance with instructions is the goal, the comfort of both the caregiver and the patient in performing the oral hygiene program is paramount. For this reason, a number of positions have been recommended for the caregiver to assume when providing oral hygiene care to the patient. Facts to be considered include the patient's size and strength, the attendant's size and strength, and the amount of control that needs to be exerted over the intentional or unintentional movements of the patient. One position that has proven to be successful when the patient is an adult is for the attendant to stand behind the patient, who is seated in a straight-backed chair or a wheelchair. In this position, it is easy to stabilize the patient's head by resting it against the body of the attendant. Brushing then proceeds with the attendant using the same kind of arm and brush positioning as when cleaning his or her own teeth. Performing this operation in front of a mirror takes further advantage of the attendant's own brushing habits, although a mirror is not a necessity. Other recommended positions include having the patient lie on a sofa or bed with the head in the caregiver's lap or sitting on the floor in front of a chair in which the caregiver is seated (Figure 20-5). As depicted in Figure 20-5 B, note that the caregiver's legs are used as an additional restraint to the arms of the patient. Caregivers and patients should both be advised that the bathroom is not the only location in which to brush teeth. In fact, it is often the least convenient room in the house because of its space limitations and the need to share its use with other members of the family. Water is not always necessary for toothbrushing, as salivary

flow is stimulated by brushing and thus provides moisture. If a patient has tender, friable gingival tissue that can easily become damaged by an initially dry toothbrush, the brush can be moistened beforehand to soften it. When no toothpaste is used, running water may not be needed. The elimination of the toothpaste increases visibility and decreases the possibility of gagging. In many cases, it has been found that when water and toothpaste were required, attendants or family members discontinued or decreased the number of toothbrushing sessions. Normally, a fluoride toothpaste is an important component of an oral-hygiene program in an uncompromised population. In compromised patients, however, if one must omit fluoride toothpaste from the routine, it can be compensated for by using fluorides in other forms.

Those patients who enjoy the taste or appreciate the aesthetic value of toothpaste can use a non-foaming, ingestible toothpaste[b] (originally developed for the astronauts). Because this toothpaste does not foam and can be swallowed, it is not necessary for the patient to be near a basin to expectorate.

If a patient likes to rinse with water or a mouthwash after brushing, a two-paper-cup technique can be used. One paper cup holds the rinse; the other is for the expectorate after the patient rinses. Because the cups are lightweight, patients can often hold both, bringing each of the cups up to their lips as needed. This two-cup technique provides a means of controlling dribbling or drooling, and it is valuable for an individual who is unable to lean over the basin, such as an arthritic patient, or for an individual who cannot purse the lips to expel the fluid, as is the case with muscular dystrophy patients.

[b]NASAdent, Scherer Laboratories, Inc., Dallas, TX.

Figure 20-5 Three different positions for a caregiver to use in aiding toothbrushing. (Courtesy of lonya Smith Ray and Gayla Hill Taylor.)

Specialized Equipment for Patient Management

Mouth Props

Several types of mouth props can be used to assist in opening and holding open the patient's mouth for oral hygiene procedures (Figure 20-6). A simple, effective mouth prop can be easily fabricated with two or three tongue blades wrapped together, padded on one end with 2 × 2 gauze squares, and secured in place with adhesive tape.[26] This prop can be used with patients who are unable to understand or to cooperate due to decreased cognitive functioning, as seen in mental retardation, mental deficiency, senile dementia, or in patients exhibiting neuromuscular dysfunction, such as occurs in cerebral palsy or muscular dystrophy. This prop may be used initially to help open the patient's mouth. If necessary, it can then be replaced by a custom-made finger cover[27] or several different types of commercially manufactured props, which would then be placed on the opposite side of the arch from where the original gauze wrapped prop is initially placed and then removed once the more compact prop is in position. Not only is the gauze and tongue-blade mouth prop useful for initial examinations and screenings, it is economical and disposable as well.

Commercially available intra-oral mouth props are frequently available in different sizes to accommodate adult and pediatric patients, or as a one-size-fits-all unit designed to accommodate a range of mouth sizes. When using a prop of the former design, the correct size must be chosen and placed far enough back in the oral cavity to be held in place by the force of the jaws attempting to close. Otherwise, on closure, the prop slides forward along the occlusal surfaces of the teeth. It is not as likely that this will occur with the second type of prop because the serrations and graduated size over the length of the prop better resist slippage. When using either of these prop types, one should tie floss through the hole in the prop and allow the floss to extend from the patient's mouth. If inadvertent swallowing of the prop occurs, an occluded airway results. In such an event, the prop can be retrieved by means of the floss ligature.

The most critical aspect of placing a mouth prop is to protect the caregiver's fingers. Those props that require the fingers to cross the occlusal plane as part of the placement process pose the greatest jeopardy of being bitten. Therefore newer devices that have positioning concavities may be of help (Figure 20-6 C). Another device is similar to a large thimble with flanges that fits over the thumb or one finger of the caregiver, freeing the other fingers and hand to stabilize the jaw during tooth brushing or a prophylaxis (Figure 20-6 B). Because the patient's jaw might suddenly snap closed upon removal of a prop, use of the gauze-wrapped tongue blades during removal of any mouth prop should be considered.

An alternative to the gauze-wrapped tongue blade is a disposable, handheld, Styrofoam mouth prop with graduated notches (Figure 20-6 A) that can be placed and controlled extra-orally. Another device controlled extra-orally is the Open Wide® Wraparound Mouth Prop with an extra-oral handle that can also be used with a suction device (Figure 20-6 D). It is possible to hyperextend the mandibular muscles with an oversized mouth prop or the overzealous placement of one. This can cause a muscle spasm, resulting in considerable discomfort to the patient. Bite blocks must be used with caution, as they have been known to cause hypoxemia.[28] The smaller the patient with regard to height and weight, the greater the risk that oxygen desaturation will occur when bite blocks are used, particularly when the bite blocks are too large.

Headrests

There are numerous ways of supporting and stabilizing the head and neck of compromised dental patients. For those individuals who remain in their conventional wheelchairs throughout treatment, a commercially available wheelchair headrest may be purchased and kept in the dental office. This headrest attaches to the hand grips of the wheelchair and adjusts to compensate for different chair widths and sitting heights of patients.[29] Other types of head stabilizers can be attached to the headrest of the dental chair with Velcro straps that extend around the back of the chair to secure the stabilizing device. Pillows designed for neck support are commercially available in retail stores. They can be used, with modifications if necessary, for patients with cervical spine deformities. A cerebral palsy head support consists of a block of foam with a depression in the center to stabilize the patient's head.[30] Pillows sold to airplane travelers that contain buckwheat hulls as the filling material and are shaped as enlarged neck collars can also be used to stabilize the head and neck of a patient during preventive procedures.

Soft Ties

Soft ties, which are cloth or soft leather straps, may be used to support and stabilize any part of the body, including the head.[31] Most commonly, soft ties are used to secure the upper and lower limbs to an appropriate leg or armrest. This prevents the limb from spasming, flailing, or hanging off the edge of the rest, a position that can compress nerves and lead to neural damage. Soft ties are not meant to be punitive or restraining devices. They are intended to provide positive support, stability, and security to the patient.

Body Wraps and Other Limb Stabilizers

Full-body wraps, such as pedo-wraps and papoose boards, are often used to immobilize smaller adult patients during dental treatment.[31] A plastic elbow stabilizer, that begins as a flat sheet and is curled into a tube around the arm, keeps the patient from being able to bend the elbow to push away a caregiver (Figure 20-7). These devices have limited usefulness in preventive programs where purposeful attempts are being made to actively involve patients in their own oral hygiene. Body wraps and stabilizers should be considered when others give care, and the patient is unable to cooperate. For some compromised patients full body wraps are welcomed as a source of security and comfort.[32]

Some developmentally disabled patients exhibit self-injurious behavior causing significant peri-oral trauma. Management of this behavior is often difficult as restraining devices applied extra-orally are only appropriate during active treatment periods and may not prevent intra-oral chewing of the tongue and/or lips that may occur at any time of the day or night. In selected cases, oral appliances can be effective in preventing trauma by deflecting tissues from the occlusal plane.[33]

Mouth props, soft ties, wraps, and elbow stabilizers are all considered forms of restraint, and communities continue to struggle with the issue of the appropriateness of restraints.[34] The use of restraints is controversial, and each jurisdiction may interpret what constitutes restraints differently. Practitioners and caregivers should research their state and local guidelines before employing such restraints.[35] The intent to use any of these items should be included in the informed consent provided to the patient's guardian.

Figure 20-6 A. Open Wide® Disposable Mouth Prop.[c] B. Dental Shield.[d] C. C-shaped mouth prop with positioning concavity.[e] D. Open Wide® Wraparand Mouth Prop[f] with extraoral handle and opening to accommodate suction device.

[c]Specialized Care Co., Hampton, NH.

[d]Athena Nordic, Falun, Sweden.

[e]Logi Bloc, COMMONSENSE Dental Products, Nunica, MI.

[f]Specialized Care Co., Hampton, NH.

Figure 20-7 A. Rainbow™ Elbow & Knee Stabilizer[g] being rolled to fit around

arm. B. Formed stabilizer in place.

[g]Specialized Care Co., Hampton, NH.

Oral-Hygiene Devices

Modifying Toothbrush Handles

In general, the principles and techniques of tooth brushing used for a compromised population are the same as for anyone else. In compromised individuals, however, good oral hygiene is much more difficult to achieve and maintain.[18] If it has been determined that the patient has adequate dexterity to produce the small strokes needed to brush properly, a manual toothbrush may produce satisfactory results. Toothbrush manufacturers are now providing a variety of different configurations[36] of brushes with increased handle dimensions, handles modifiable with hot water (Figure 20-8), angled brush heads, multiple brush heads, and curved bristles, all of which can be beneficial for special needs patients (Figure 20-9). One type of brush marketed for toddlers is designed with a large ovoid handle that prevents over insertion and potential intra-oral injury when a child is first learning to brush. Such a device may have application for an older compromised child (Figure 20-10). Even if the patient has a weakened hand grasp or uses orthotic splints or other adaptive appliances, a manual toothbrush can be modified to facilitate usage.[37,38] In a well-controlled study of children with cerebral palsy who received modified toothbrushes, plaque removal was increased by 28 to 35% over that achieved when conventional toothbrushes were used.[39] Figure 20-11 illustrates different methods of quickly augmenting toothbrush handles from commonly found materials. These include foam wrappings from packing materials, acrylic tray or bite registration material, the center foam piece from a hair curler, a bicycle grip with plaster anchoring the toothbrush inside, or a juice can with a slotted ball inside to hold the toothbrush.[22] Inexpensive, cylindrical, closed-cell foam can be obtained from orthotic or medical supply stores. This foam cylinder has significant advantages over other types of foam materials because it is composed of closed plastic cells that shed water. This eliminates the increase in weight and the need to squeeze out absorbed water on completion of a hygiene procedure.

Handles augmented with foam can be used by a wide range of compromised individuals. They can be easily adapted to orthotic appliances such as splints. Handles modified with heavier materials, however, such as the bicycle grip or the juice can, should not be used with arthritics or those with neuromuscular weaknesses. These latter two types of modifications are more appropriately used with mentally retarded individuals, including those with Down syndrome, and with cerebral palsy patients who typically have strong grips and limb musculature (see Figure 20-11).

Patients who are unable to flex their elbows because of joint involvement can be given a toothbrush with an extended handle. This can be fabricated by inserting a bicycle or wheelchair spoke into, and parallel to, the original toothbrush handle and fabricating a new acrylic handle out of orthodontic resin or a similar material. The handle may be further modified if the patient has grasp difficulties. Other simple modifications include reshaping the plastic handle of the toothbrush by heating it in warm water and bending to the desired configuration or gluing the handle of a nailbrush to the toothbrush handle.

Several devices have been developed to assist individuals with limited function to achieve independence. Often, products used to assist in feeding can be adapted for use in brushing the teeth, such as palmar cuffs or activities of daily living (ADL) cuffs.[n]

[n]An ADL cuff is a generic term for any kind of appliance adapted to the upper extremity to which various implements can be added, as, for example, a toothbrush, so that the patient might perform his or her own daily living tasks without assistance.

Figure 20-8 Commercially available toothbrush with handle that can be modified by immersion in hot water.[h]

[h]Shape It™ Toothbrush, John O. Bulter Co., Chicago, IL.

Figure 20-9 Commercially available toothbrushes designed for special needs patients. Clockwise from upper right: Curved bristle brush,[i] small triple brush head,[j] double brush head,[k] and large triple brush head.[l]

[i]Collis-Curve™ toothbrush, Collis-Curve, Brownsville, TX.

[j]SUPER-BRUSH® Junior, Denta-Co., Bergen, Norway.

[k]action 2. Action Hygiene Products, Inc., Toronto.

[l]SUPER-BRUSH, Denta-Co., Bergen, Norway.

Figure 20-10 Tooth brush with large handle to prevent overinsertion.[m]

[m]INFANT-TODDLER SAFETY TOOTHBRUSH®, Preventive Dental Specialties, Inc., Rothschild, WI.

Figure 20-11 Readily available foam tubes, bicycle handles, cans, or dental tray material can be used to modify the size of toothbrush handles.

Question 3

Which of the following statements, if any, are correct?

A. Dentifrices are essential to maintaining good oral-hygiene care among the handicapped.

B. Both mentally handicapped and individuals with neuromuscular dysfunction may need mouth props.

C. When it is necessary to constrain a neuromuscularly handicapped patient, it should be a nonpunitive action.

D. The appropriateness of using body wraps or pedo-wraps depends on the patient's size and stature.

E. A Bunsen burner flame is needed to modify a toothbrush handle.

Electric Toothbrushes

Electric toothbrushes are valuable aids in assisting compromised patients.[40] They are especially useful when the patient has the strength to grasp the handle and place the brush in the mouth but does not have the manual dexterity needed to perform the fine movements necessary for the cleaning function. The length and diameter of the handle of an electric toothbrush approximates those of manual toothbrushes that have been modified for individuals with compromised hand function.

Recent models of electric toothbrushes display on/off buttons that are user-friendly. Unlike previous models, which had switches that were difficult to manipulate,[41] most now have pressure plates that activate the brush head and are easy to use. The weight of the electric toothbrush is still a problem for some individuals to manage, especially patients with poor upper extremity muscle control or strength. This can be compensated for by positioning the patient at a table and demonstrating brushing while the patient's elbows on the table are used to support the increased weight. If the patient is in a wheelchair, a countertop can be used to support the toothbrush handle while activating it (Figure 20-12).

For patients able to perform their own oral hygiene, the effectiveness of electric toothbrushes in plaque removal has been well established. Much less has been accomplished in attempting to establish the effectiveness of electric toothbrush use in compromised patients. However, one study did compare the Interplak® to manual toothbrushes in a population of persons with mental retardation/developmental disabilities (MR/DD). Those using the Interplak® showed significant improvement in the Gingival Index over the twelve months of the study.[42] The few other studies involve caregivers who are typically in the dental field delivering care to nursing home patients. In one of the studies, plaque and gingivitis levels were compared after use of a manual and an electric toothbrush of a counter-rotational design with the care being delivered by a dental hygienist or dental assistant.[43] The results after the use of the powered toothbrush were significantly improved over the manual toothbrush. A second study compared plaque removal and gingival inflammation in a group of nursing home patients after using an electric rotary brush with a single tuft of brushes and a manual toothbrush. The care was delivered by dental students.[44] The electric toothbrush again effected a statistically significant improvement in the two parameters measured.

Even though electric toothbrushes seem to be indicated for use in a mentally handicapped population, Bratel and coworkers[45] were unable to demonstrate clearly the superiority of electric over conventional toothbrushes whether used independently or aided. It may be that electric toothbrushes are beneficial for this population, because patients and caregivers find them easier and more pleasant to use.[46]

It remains to be seen whether features such as smaller brush heads, sonic cleaning power, reciprocating brush heads, or a counter-rotational design may have unique effectiveness for this population (Figure 20-13).

In selecting which toothbrush is best for a particular patient, one should consider alignment of teeth in the arch, constriction of the arches, and whether an exaggerated gag reflex is present.

One additional note of caution should be considered before recommending an electric toothbrush for a compromised patient. An overzealously used electric toothbrush can cause considerable damage to the hard and soft tissues in a short time.

Floss-Holding Devices

Dental flossing is not recommended for all compromised patients. Unless the task of toothbrushing can be learned, it is useless to superimpose the more complex task of flossing. To do so can be so discouraging that all attempts at oral hygiene are abandoned. This is true whether the patient or the attendant is performing the program. Flossing, therefore, should be introduced on a selective basis for those patients or attendants who have mastered toothbrushing and consistently show low plaque levels on tooth surfaces.

An adequate assessment of the patient's dexterity and ability to understand the technique must be determined before flossing is introduced.

For some compromised patients, flossing can be performed regularly if a floss-holding device is used. Eight such devices were evaluated by people with upper-extremity limitations.[21] This group rated one device significantly higher for its handle dimensions, ease of threading, and ability to keep the floss taut.[r] Although some compromised patients have learned adaptive techniques allowing them to thread floss-holders themselves, the majority of compromised patients have great difficulty in accomplishing this procedure. One patient with very limited use of his hands described how his wife kept five floss-holding devices threaded on the kitchen counter for use as he needed them. If one became unthreaded during a flossing routine, he simply obtained another. An alternative to multiple floss-holders is to create a plaster of Paris base for the floss-holding device, so that it can be stabilized by one compromised hand while the other completes the threading. The holder can then be used with or without the base, depending on the patient's strengths and desires. There are currently on the market several brands of floss holders claiming to be self-threading. There have been no comparative studies to determine if, in fact, they offer advantages to a compromised population. Therefore, manipulating the floss with or without a floss holder continues to be a barrier for this population.

[r]Floss-Aid Co., Santa Clara, CA.

Interproximal Brushing

In older patients, gingival recession is a common experience. Often the recession is so pronounced that the use of regular dental floss is not effective in cleaning the long expanse of exposed root structure. In this situation some recommend Super Floss,[s] as it is considerably thicker at one end. If the gingival recession has occurred to the extent that the papilla no longer fills the interdental space, an interproximal brush may be beneficial.[47] Individuals who have never used floss or who have difficulty manipulating the dental floss or threading a floss holder seem to adapt more readily to the interproximal brush.

Interproximal brushing may be introduced near the beginning of the preventive program. Because handles of interproximal brushes are long and sturdy, they can

easily be modified in the same manner as the toothbrush. Many interproximal brushes require the assembly of the proper brush head to the handle. This is an intricate task and requires fine-motor skills. Additionally, some patients have severely constricted arches requiring unusual access and angulation of the brush head into the interproximal space. Therefore, the newer preassembled interproximal brushes, those with snap on brush heads and those where the angle of the brush to the handle can be changed from 90° to 180° are recommended for compromised patients (Figure 20-14). Demonstrations of assembly and use are definite requirements.

[s]Oral-B Laboratories, Inc., Belmont, CA.

Prosthesis Hygiene

Compromised patients who wear full or removable partial dentures may need assistance with maintaining proper hygiene of the appliances, which must be removed for thorough cleaning of the oral soft tissues and any remaining natural teeth. The appliances also must be cleaned appropriately and should be left out of the mouth for 6 to 8 hours per day. Modifications to denture-cleaning devices as well as modifications to the dentures may aid in helping compromised patients provide their own denture hygiene.[48] Oral hygiene care by nurses' aides in institutional settings should include removal of all full or partial dentures and scrubbing and soaking of these appliances, as well as the care of the soft tissue and teeth. Dentures are often lost in institutions by the staff, as well as by the patients themselves. As a result, residents may experience digestive complaints, inadequate nutrition, and speech difficulties, all of which can contribute to a poor self-image. Therefore, it is important for the dental consultant to set up denture-identification programs to mark prostheses with the patient's name, Social Security number, or other means of identification. Then any misplaced appliances can be readily returned to their owner.

Other Types of Oral Hygiene Aids

From time to time other oral hygiene aids are promoted for use with patients who are in some manner compromised (Figure 20-15). Frequently, these devices have not undergone any testing prior to their marketing, but are promoted on the basis of potential worth. When or if such testing is accomplished, claims are not always upheld. An example of such a product is the disposable "foam on a stick" device. In a study by Addems and colleagues,[49] able-bodied subjects showed marked increases in plaque and gingival index scores during the week when the foam sticks were used (compared with a week when conventional brushing was performed).

Another study[50] found some equality in removing plaque with the foam sticks in comparison to a regular toothbrush, but it was clear that the toothbrush was more effective in retarding plaque accumulation. Cotton swabs[x] are frequently used for oral hygiene in institutionalized settings. If swabs are used that contain citric acid, significant damage to the dentition can occur in the form of irreversible erosion of the enamel.[51]

In another study by Kambhu and Levy,[52] four devices were compared for efficacy when used on a simulated dependent care population by a nonprofessional caregiver. An unusual toothbrush with curved bristles,[y] as well as an electric toothbrush with ten

different rotating tufts of bristles,[z] were more effective at removing plaque than a conventional toothbrush. A foam stick device came in a distant fourth in the study. The subjects rated the curved-bristle toothbrush as the most comfortable, and the caregiver rated it as the easiest to use.

Another device incorporates three different sets of brush tufts angled around an arc into one toothbrush head.[53] This allows the facial, occlusal, and lingual surfaces of each tooth to be brushed at the same time. Although no difference was found in plaque and bleeding indices when this brush was used in comparison to a regular manual toothbrush, it seemed to be easier to teach its use to mentally retarded individuals. However, this brush configuration does not work in cases of severe gingival recession.

[x]Moi-Stir®, Kingswood Laboratories, Inc., Carmel, IN.

[y]Collis-Curve™ Toothbrush, Collis-Curve, Brownsville, TX.

[z]INTERPLAK®, Bausch & Lomb Oral Care Division, Inc., Tucker, GA.

Disclosing Techniques

Whatever the patient's age, disclosing products should be suggested to visualize plaque when a patient has difficulty in plaque removal. Disclosing solutions are readily available over-the-counter in multidose bottles. Recently, single-dose packaging of disclosing solution with its own cotton-swab applicator has become available and may prove practical for weekly plaque removal effectiveness checks in institutional settings (Figure 20-16). Should the price of disclosing solution serve as a deterrent the cost factor can be minimized by purchasing commercial food coloring, usually available in the bakery section of any grocery store. The food coloring can then be used in place of the disclosing solution to stain dental plaque. The color should be chosen on the basis of which is easiest to see in the mouth. For example, yellow is difficult to detect on teeth because the color is too close to that of natural tooth color. Blue and green, although suitable for teaching plaque control to children, are more difficult for the aging eye to see. Red food coloring is the easiest to visualize for all age groups. A popular color, it can be found packaged in a number of different containers, including individualized plastic bottles that are much easier to use. Two drops of food coloring should be placed on the tongue and the patient advised to use the tongue to wipe the food coloring around all the surfaces of the teeth prior to brushing. An alternative technique when the patient is unable to follow these directions is to have the caregiver apply the food coloring to a cotton swab and gently dab it on the teeth. The plaque is well stained with either of these methods, and, as the volume of liquid used is minimal, little drooling or subsequent staining of the individual's clothes occurs.

Figure 20-12 A pressure-activated toothbrush (Water-Pik) being used by a severely disabled individual.

Figure 20-13 Electric toothbrushes. Top: multiple reciprocating brush head.[o] Center: sonic cleaning brush.[p] Bottom: counter-rotational design.[q]

[o]INTERPLAK®, Bausch & Lomb Oral Care Division, Tucker, GA.

[p]Sonicare, Philips Oral Healthcare, Snoqualmic, WA.

[q]ORALGIENE™ USA, Inc., Culver City, CA.

Figure 20-14 Interproximal brush handle that can accommodate brush at right (90°) or straight (180°) angle.[t]

[t]Proxident holder. Athena Nordic, Fulan, Sweden.

Figure 20-15 Oral hygiene aids. From top: swab on a stick,[u] foam on sticks of varying size (minifoam stick,[v] foam sticks with longer handles.[w])

[u]Moi-Stir®, Kingswood Laboratories, Inc., Carmel, IN.

[v]TOOTHETTE®, Halbrand, Inc., Willoughby, OH.

[w]MEDI-CLENZ®, Specialized Care Co., Hampton, NH.

Figure 20-16 Single dose disclosing solution with cotton swab applicator.[aa]

[aa]DISCLOSE® Beutlich Pharmaceuticals LP, Waukegan, IL.

Preventive Therapies

Dietary Considerations and Alternative Reward Systems

For many compromised patients, foods high in sugar are distributed throughout the day as a reward for having been compliant. Such a reward system encourages between-meal snacking and increases the consumption of highly cariogenic foods.[18] With patients who have decreased neuromuscular coordination or decreased salivary flow, it may be difficult to adequately clear the mouth.[20] Food may remain in the buccal vestibule and between the teeth until the next brushing. To reduce the cariogenic potential, it is necessary 1) to restrict between-meal snacking and 2) to limit the use of highly cariogenic foods.[20] If sweets are to be consumed at all, they should be presented at mealtime and the teeth brushed immediately after eating. Bedtime snacks should be discouraged.

An alternative to a reward system based on sugary treats[20] is to present tokens for later redemption for prizes, such as toys, noncarious food, or outings.

Sealants and Fluorides

In spite of the normalization of handicapped individuals into the mainstream of society, it appears that the non-institutionalized handicapped do not have as high a level of oral health as the rest of the population. The F (filled) value for the DMF (decayed, missing, or filled) scores is often lower in the compromised population, whereas the D and M values are higher than in the general population.[54, 55] Although becoming more common, preventive strategies that would really benefit this population group are often not available on a regular basis. The use of sealants and fluorides should be considered important preventive techniques to assist in caries control for compromised patients.[55]

Sealant application may be more difficult in compromised patients, because it may be more difficult to control moisture contamination. Salivary pooling is often seen in cerebral palsy and muscular dystrophy patients, because they have swallowing difficulties. For the short time needed to apply sealants, antisialogogue medications are usually not indicated. Instead, the sealant may be applied in the conventional manner using the techniques to control saliva flow indicated in Chapter 10. To aid in moisture control the patient should be seated upright rather than in a reclining position.

In a 30-month study of a preadolescent population with Down syndrome living in a hostel-like group setting, the application of dental sealants was 100% effective with a sealant retention rate of 97% in preventing caries over the term of the study.[56]

Regular topical fluoride applications by the dental staff are highly important for the compromised dental patient. A new fluoride-containing varnish developed for dentinal hypersensitivity is now available as a unit-dose application and can be used as a fluoride supplement. It is supplied in two doses: 25 mL for primary dentition and 40 mL for mixed dentition (Figure 20-17). The two may be combined to form a 65 mL dose for the permanent dentition. Fluoride varnish can be quickly painted on and is effective even in a moist field, a particularly important characteristic for some of the developmentally disabled and mentally retarded population who have a disordered swallowing mechanism and are therefore unable to effectively clear their mouths of saliva. For the younger patient, water fluoridation or tablets are essential.[57] Equally important for this population is a home self-applied fluoride program. Several effective techniques are now available for home fluoride application, ranging from mouth rinses to fluoride gels applied with custom-made trays. Rinses are contraindicated for compromised patients who cannot effectively swish the solution around their mouths. Some individuals with muscular dystrophy and some post-stroke patients have an incompetent or hypotonic lip seal and cannot keep solutions in the mouth for the required period. Our experience with office-applied fluoride treatment delivered in a tray requiring the patient to keep the tray in place for a minimum of 4 minutes has demonstrated how difficult it is for many compromised patients to cooperate that long, particularly if there is an active gag reflex. A gel-filled tray also stimulates the flow of saliva, which is often difficult to confine. Neither the patient nor the caregiver likes the drooling that occurs. Therefore, home-fluoride treatments utilizing a tray-delivery method will probably not be successful. An alternative home-fluoride delivery method uses a foam applicator. In a nursing home population, Saunders and colleagues[58] demonstrated that the level of fluoride in saliva was higher 3 hours following delivery by an intra-oral applicator when compared to the fluoride levels in saliva after residents rinsed with a fluoride mouthrinse. More independent populations may find brush-on fluoride gels easier to use, because their application takes advantage of an already learned toothbrushing behavior. Fluorides have been shown to reduce demineralization and enhance remineralization.[59] Therefore, brush-on gel fluorides should be considered for use by elderly compromised patients, particularly those with gingival recession. Fluorides should not, however, be indiscriminately given to patients for unsupervised use if some question exists as to the patient's ability to understand and follow instructions. Although Chan and O'Donnell[60] found little risk of toxicity when a fluoridated tooth- paste was used independently by a population of mentally handicapped children, one must still

exercise caution whenever recommending fluorides.

Chemical Plaque Control

It has been recognized that treatments need to be developed to manage plaque control that are less dependent on the manual dexterity of the patient.[61] The efficacy of applying chlorhexidine (CHX) by swabbing for people with disabilities has been established. In one study[62] CHX was applied by a caregiver once daily, five times per week, for ten weeks using foam sticks.[cc] When compared with applying a placebo by swabbing, the CHX group showed consistent and significant improvement in lower plaque levels, gingivitis, and pocket depths. A subsequent study[63] demonstrated that CHX swabbing was effective at a reduced frequency (twice per week as opposed to five times per week) and was well tolerated with prolonged use (42 weeks).

Application of sustained-release varnishes of CHX and arginine also produced reductions in plaque, calculus, and pocket depths in a mentally retarded population.[64] The effectiveness of a very low concentration (0.06%) of CHX spray delivered by caregivers was evaluated in developmentally disabled patients,[65] and resulted in significant improvement in plaque scores. Thus, for severely disabled or mentally retarded patients, a caregiver can provide CHX applications by various means and improve the periodontal condition. However, it may not be possible for these patients independently to achieve these positive results as demonstrated in a study comparing CHX and an essential oil mouth rinse.[66]

[cc]TOOTHETTE™, Halbrand, Inc., Willoughby, OH.

Implant Care for Compromised Patients

Patients who have become incapacitated subsequent to having dental implants placed are at significant risk for oral hygiene problems.[67] Once the patient or the family or the institutional staff has demonstrated the level of oral hygiene that is attainable, efforts should be made by the practitioner to modify the implant complex to ensure cleansibility. This should not be done until after a rehabilitation program, if warranted, is completed and it is clear that the level of ability has plateaued. During the interim period more dependence should be placed on chemotherapeutic efforts to maintain good oral health than would be appropriate to do over the patient's remaining life span.

Other compromised patients may be well served by implants if the implant design allows for easy cleaning. In a case report of a person with cerebral palsy, the use of magnetic keepers provided a highly cleansable surface.[68]

Figure 20-17 Unit-dose applicators of fluoride-containing varnish.[bb]

[bb]CavityShield™ OMNII Oral Pharmaceuticals, West Palm Beach, FL.

Periodic Preventive Maintenance

Many compromised individuals have a higher incidence of caries and periodontal problems than noncompromised patients and, therefore, they should be seen more frequently.[69] The timing of preventive maintenance appointments should be

individualized and should reflect the patient's or caregiver's ability to perform oral hygiene procedures. Often, compromised patients are either on fixed incomes or have limited resources available to finance their dental care. Others who are enrolled in government or private insurance plans may have more flexibility in procuring dental care on a regular basis. Documentation by the dentist of the patient's disability and the subsequent oral problems often assists the patient in obtaining a more generous interpretation of the services covered by the third-party provider. This is particularly true for government plans. For some, the cost of dental care is assumed by the patient's family, who realize the importance of preventive oral care and are eager to see the patient benefit from such treatment. In general, the compromised patient has limited resources to expend on dental care. For these patients, the dental clinician may wish to consider some innovative financial arrangements to pay for preventive procedures. For example, it might be desirable if a contract could be established whereby the patient is brought in on a regular quarterly schedule for prophylaxis. Each appointment after the first one is performed for a reduced fee if the patient completes the entire series of scheduled visits. Concurrent treatment contracts should also be negotiated for restorative care.

Provider Availability

Although compromised children are usually welcomed in most pediatric dentistry practices, it is often difficult for the similarly afflicted adult patient to find dental personnel with the training, empathy, and patience needed to deal with the patient's disabilities. In recognition of this problem, many dental schools are now providing training in special patient care to current students as well as to practicing dentists in continuing-education courses.[70] These actions should increase the number of dental clinicians with the expertise and willingness to render special care.

Dental Care in an Institutional Setting

Many institutionalized persons have poor oral health.[71] It is often conjectured that this is because residents of institutions are likely to have more severe disabilities than those who are disabled but live in the community, or that the oral care of institutionalized populations is of poorer quality than those not institutionalized. A recent study examined the oral-hygiene habits, gingival bleeding, food diaries, and oral microorganisms of moderately or severely mentally retarded adults before and up to 21 months after relocating into the community from an institutional setting.[72] Of the oral-health parameters measured, none worsened and some improved, demonstrating that the institutional environment does place the compromised patient at greater risk for poor oral health.

The most common role for the dental provider in an institutional setting is that of consultant. In this capacity, the provider advises the administration about the dental needs of the residents and recommends the type and frequency of oral hygiene care to be delivered.[71, 73] The dental clinician should expect to provide in-service prevention-oriented educational training programs for the nursing staff. The administration and the staff must be kept aware of the importance of routine oral-health care.[74] The administrator of a facility may agree to a routine dental-care program, provided that the dentist or dental hygienist trains the staff. This requires an ongoing training program because of frequent turnover of nurses' aides in such facilities. Training aids

may include videotape recordings of the important aspects of preventive care. The dentist should participate in staff meetings when needed. Periodic evaluation of the residents' oral hygiene using an established oral hygiene index helps determine if additional in-service training is needed. A more informed staff relative to the importance of oral hygiene has been shown to result in better oral health care for the residents.[74]

When appropriate, the residents of the various institutions should be encouraged to participate in their own oral hygiene efforts. Instruction in oral-hygiene methods, followed by staff supervision and encouragement, can result in improvements in various periodontal indices.[75]

Even the totally disabled or comatose patient who is no longer taking food by mouth but is being nourished via a gastric tube or intravenous line is subject to intra-oral plaque and calculus accumulation and should have daily oral-hygiene procedures performed. Ironically, it has been shown that, although plaque accumulates at about the same rate in tube-fed and normally fed patients, calculus accumulates faster in tube-fed patients.[76] The objectives for oral hygiene procedures for these patients are basically the same as for all patients except that more care must be taken, including such steps as lubricating the lips of the patient prior to the hygiene treatment. Petroleum jelly is an excellent, inexpensive lubricant that keeps desiccated lips from being injured by mouth props.

The teeth of comatose patients should be brushed in the conventional manner with a soft-bristled toothbrush. Edentulous areas should be wiped gently with gauze or a disposable foam sponge on a stick, both of which can be lightly moistened. If a mobile or central aspirating system is available, a toothbrush can be used that has been manufactured with an aspirating tube[dd] as a part of the brush head.[26] Such a device is an aid to controlling the salivary secretions in the debilitated or comatose patient.

[dd]Plak-Vac, Trademark Medical, Fenton, MO.

Question 4

Which of the following statements, if any, are correct?

A. Often, the intake of cariogenic foods can be better controlled by a guardian through judicious cooking than by a compromised patient.

B. Dry-field operation and patient cooperation are the two salient requirements for sealant placement.

C. Fluoride dentifrices should be utilized by all compromised patients.

D. Preventive care, even though more economic, usually has a lower priority than treatment.

E. The nurses' aides in institutions for compromised patients are usually well trained to take care of oral-health needs.

Summary

Individuals with physical, medical, mental, or emotional problems often have a greater need for dental care than their healthy counterparts. This may be because the disability itself has oral manifestations, but more commonly it is because of (1) the limited capabilities of the individual or the family members to understand and to perform important oral hygiene tasks, (2) a lack of understanding of the importance of preventive dental care, and (3) an inability to finance dental care. When a compromised patient does present to a dental office, the clinician should develop a treatment plan that emphasizes prevention. Assessments should be made of the patient's sensory, cognitive, and functional abilities and be used to customize a preventive plan. When the patient is unable to provide his or her own care, the family or an attendant needs to be taught the appropriate techniques.

Specialized equipment and easy-to-accomplish modifications of conventional oral hygiene devices may be employed to provide oral hygiene care. Strategies such as substituting a noncariogenic reward system to decrease caries incidence are often successful. Dental preventive procedures, such as sealants, fluorides, and chemical plaque control, should be considered for each patient as part of any treatment plan.

The rapport of the compromised patient and his or her family with the dental health provider and the entire office staff is critical to the comfort and compliance of the patient. All members of the office staff need to convey a warm, receptive attitude to these special patients.

Most institutionalized individuals have great oral health needs. The dentist can play a significant role in assessing those needs by communicating recommendations for a daily oral care program to the institutional administrator. Dentists and dental hygienists can offer training to the nurses' aides who provide that day-to-day care.

For many compromised individuals, the retention of teeth in a healthy mouth improves mastication and digestion, as well as helps maintain an adequate nutritional status. The pleasing aesthetics afforded by good oral health help people with disabilities to be more welcomed by others. Good preventive care enhances one's self-esteem. For some patients who are severely compromised, specially adapted appliances may be required to maintain oral health. Many individuals, because of neuromuscular problems, have difficulty functioning with any type of oral prosthesis. Because the natural dentition assumes such an important role in the total living environment of the compromised patient, it is of utmost importance that the patient, caregivers, and the dental team work together to achieve an effective preventive oral-hygiene program for such an individual.

Answers and Explanations

1. A, C, and D—correct.

B—incorrect. The patient should be able to see the dentist's face to better understand and to note the dentist's body language.

2. B and E—correct.

A—incorrect. Remember, do not generalize on what a patient might do because of a handicapping condition; there are always exceptions to the rule.

C—incorrect. Directions given to the handicapped, especially mentally handicapped, should be as simple as possible to get the job done.

D—incorrect. A handshake can determine hand strength but does not assess dexterity or cognition necessary to perform oral hygiene.

3. B, C, and D—correct.

A—incorrect. It is the brush bristles that disturb the plaque—not the dentifrice.

E—incorrect. Very hot water is sufficient to soften a toothbrush handle prior to modification.

4. A, B, and D—correct.

C—incorrect. Fluoride dentifrices are desirable for those who have control of their oral musculature; otherwise, undesirable drooling or swallowing of the dentifrice occurs. Other methods of application of fluoride, however, should always be considered.

E—incorrect. The high turnover of nurses' aides does not permit the development of a good teaching program.

Self-evaluation Questions

1. A definition of a compromised individual is _____. Before initiating any preventive program, it is necessary to evaluate a range of functional, _____. intellectual, and _____ capabilities.

2. A patient may have a simple decrease in visual acuity, which can be noted when the patient begins to _____. If a human guide or guide dog accompanies the patient, they (should) (should not) be allowed in the treatment room. Other visual problems are _____ and _____.

3. Three precautions that should be taken to ensure that instructions are presented with maximum effectiveness to a person with loss of hearing are _____, _____, and _____. When a high-speed handpiece is turned on next to a hearing aid, it is uncomfortable for the patient because _____.

4. Patients with a history of previous _____ often have difficulty in speaking. In Parkinson's disease, multiple sclerosis, and ALS, there is often an impairment of speech, called _____. A severe impairment in the word sequence in speaking is termed _____. When speech impairment and body paralysis occur, communication can sometimes be accomplished by use of _____ (device).

5. The best way to determine the IQ of a patient is to _____. If a homebound patient cannot complete a task, the _____ (person) should be given the responsibility of helping.

6. A simple test to determine hand muscle strength is _____. To determine whether a patient has the cognitive and psychomotor ability to use a toothbrush, the easiest method is to _____.

7. One position that a caregiver might take in brushing the teeth of a compromised individual is _____. Two disadvantages of using toothpaste are _____ and _____.

8. Two mouth props are the _____ and the _____. Of these, the _____ needs to be secured with a piece of dental floss to prevent its being swallowed, while the second, the _____ prop, can cause an overopening of the mouth.

9. At least three modifications of a toothbrush are _____, _____, and _____. Electric toothbrushes can be used by severely weakened patients by _____. One problem that might be experienced after compromised patients begin using an electric brush is _____. The _____ brush is often convenient for cleaning the interproximal embrasures.

10. A good substitute for commercially available disclosants is _____.

11. Patients with trouble in walking need either another person as an _____ to help or a wheelchair.

12. Reward systems should *not* include _____. In placing sealants, the two key factors to success are _____ and _____. In a nursing home, it is the _____ or the _____ who normally conducts in-service training for nurses' aides. To avoid the loss of dentures in a nursing home, it is desirable to _____ (action).

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Chapter 21. Geriatric Dental Care - *Janet A. Yellowitz Michael S. Strayer*

Objectives

At the end of this chapter, it will be possible to:

1. Describe the demographic changes associated with the U.S. population 65 years and older since 1900, and how this segment of the population will appear in 2050.

2. Compare key age-related physiologic changes commonly found in older adults with disease related changes.

3. Identify the most prevalent chronic diseases found in the elderly population.

4. Describe the cognitive impairments generally associated with the older adult population.

5. Compare how patterns of oral-health status have changed since 1970, and how oral-health status will change among older adults by 2030.

6. Identify the three key oral disease processes commonly found in older adults.

Introduction

During the latter half of the 20th century, the age composition of the population changed dramatically, with more people living to older ages and the older population getting older. This demographic change will have a major impact on the delivery of general and oral-health care, as well as on the providers of these services. Although some older adults have physical and/or psychological conditions that require special attention in the dental office setting, one should not assume that all older people share

these conditions. Yet, the greatest challenges in geriatric care focus on the oldest, sickest, frailest, as well as those with multiple medical and/or psychological problems.[1] In order to be best prepared for the future practice of dentistry, oral-health professionals need to be knowledgeable about the general and oral-health status of older adults, the physical changes associated with aging, and how best to optimally address these issues.

The "elderly" segment of the population is diverse and has been subdivided into the following categories:

1. People aged 65 to 74 years are the *new* or *young elderly* who tend to be relatively healthy and active;

2. People aged 75 to 84 years are the *old* or *mid-old,* who vary from those being healthy and active to those managing an array of chronic diseases;

3. People 85 years and older are the *oldest-old,* who tend to be physically more frail. This last group is the fastest-growing segment of the older adult population.

Although the elderly have traditionally been defined by as those over age 65 years, age 75 may be a more appropriate age to consider, allowing for some flexibility to account for the many variations found in this population.

Demographic Trends

The 20th century has been experiencing an unprecedented "graying of America" (Table 21-1).[2,3] Today, 35+ million Americans or one in eight are age 65 or older, compared to only 3.1 million people at the turn of the century. Since 1990, the population 65+ years increased 10.6%, compared to an increase of 9.1% for the under-65 population.[2] The older population is also getting older. Compared to the population in 1900, in 1999 the 65 to 74 age group (18.2 million) was 8 times larger, the 75 to 84 group (12.1 million) was 16 times larger and the 85+ group (4.2 million) was 34 times larger, thus making the 85+ population the fastest-growing cohort of both older adults and the population as a whole.

By 2030, there will be about 70 million older persons, more than twice their number in 1999. Representing 13% of the population in 2000, the 65+ population is expected to grow to be 20% in 2030. In addition, the number of centenarians—people at least 100 years old—almost doubled in the past decade.

Although this growth pattern of the aging population slowed down during the 1990s, because of the decrease in the birth rate during the 1930s' Depression, a rapid increase is expected between the years 2010 and 2030, when the "baby boomers" will reach age 65. By 2030, the 65+ population will rise to close to 20% of the population.[2]

Ethnic and minority populations are also projected to increase during this century, with the number of Hispanic-American and Asian-American populations to increase substantially by 2050. Minority populations are projected to represent 25.4% of the elderly population in 2030, an increase from 16.1% in 1999. Between 1999 and 2030, the white population is projected to increase by 81% compared to 219% for older

minorities, including Hispanic-Americans (328%), African-Americans (131%), American Indians, Eskimos and Aleuts (147%), and Asians and Pacific Islanders (285%). These dramatic population changes will continue to alter the availability and delivery of general and/or oral health care.

Life Span/Life Expectancy

Life span is generally defined as the maximal length of life potentially possible—the age beyond which no one can expect to live. Human beings have a life span of approximately 120 years. Life expectancy is the average number of years a group of individuals born during the same time period or cohort is expected to live. Between 1900 and 1997, life expectancy at birth increased from 46.3 years to 73.6 years for males, and from 48.3 to 79.4 for females.[4] (See Table 21-2A and 21-2B.) Life expectancy also increased for the 65-year and older cohort, from 11.9 years in 1900 to 17.7 years in 1997 (Table 21-2A and 21-2B).[4] These increases in life expectancy are primarily caused by advances in medical technology, and environmental and public-health measures. The increase in the population 75 and 85 years and older is of particular concern to health-care providers, since this age group tends to present with the highest frequency of physical and cognitive disorders.

As the population ages, the distribution of older adults varies greatly by gender. At age 55, there are approximately 100 females for every 100 males; at age 65 there are approximately 122 females for every 100 males; whereas at age 85, there are 259 females for every 100 males. Thus, as the size and proportion of elderly increases in the future, females will continue to outnumber males in the older age groups.

Marital Status

Marital conditions and living arrangements of older persons vary tremendously by gender. Most men spend their later years married and in family settings, whereas most older women spend their later years as widows outside of family settings. This situation is primarily because most women marry older men, women have a longer life expectancy, and thus outlive their spouses. In addition, widowed or divorced men generally remarry rather than continue to live alone. Older widowed men have a remarriage rate that is over eight times higher than older widowed women.[5]

Living Arrangements

Although approximately one-third of the elderly live alone, the majority of older non-institutionalized adults live in a family setting. However, these figures vary by gender and advancing years. Most men 75 years and older live with their spouses or other family member, compared to less than 50% of the women in this age group. Although only a small percentage (4.5%) of the 65+ population lived in a nursing home in 1997, the percentage increases dramatically with age, ranging from 1.1% for persons 65-74 years to 4.5% for persons 75-84 years and 19% for persons 85+ years of age.[6]

Question 1

Which of the following statements, if any, are correct?

A. The new-elderly today are older than the new-elderly of yesteryear.

B. The oldest-old are dying off too fast to be a significant economic and health problem for the taxpayers.

C. The percentage increase in white population over the next thee decades will outnumber each of the following ethnic and cultural groups: Asian and Pacific Islanders, African-American, Hispanic-American, African-American and American Indian, Eskimo, and Alaskan Aleut.

D. At age 85, there are more single men than single women.

E. The majority of individuals over 85 live in nursing homes.

Education

The educational level of the older population is increasing. Between 1970 and 1999, the percentage of older adults having completed a high school education rose from 28% to 68%. This group varied considerably by race and ethnic origin, with 73% of whites, 68% of Asians and Pacific Islanders, 45% of African Americans and 32% of Hispanics.[2]

In summary, elderly of the future will be older, better educated, have better control of their finances, include more females, and have more minorities than ever before. With these changes, the elderly of the future will be significantly different than today's elderly population.

Health Status

The study of aging includes not only diseases that cause morbidity and mortality but also the conditions that cause disability and decline in independent functioning. Table 21-3[7] lists the most common causes of death among elderly people in the United States, and Table 21-4[7] lists the most common chronic conditions effecting adults in different age groups.[7] The three leading causes of death in the elderly are: *diseases of the heart, malignant neoplasm* (cancer) and *cerebrovascular disease* (stroke). Eliminating deaths caused by heart disease would add an average of 5 years to life expectancy at age 65 and would lead to a marked increase in the proportion of older persons in the population.[8] Yet, if cancer as a cause of death were eliminated, the average life span would be extended by less than 2 years.

The most common chronic conditions are arthritis, hearing impairment, hypertension and heart disease. The majority of health conditions and diseases are the result of the accumulation of ones' lifestyle, genetic factors and environmental conditions.

For many older Americans, chronic disease is a fact of life; however, most older adults perceive themselves in a positive manner. Approximately 80% of the elderly have at least one chronic medical condition. Yet, almost 71% of non-institutionalized older adults describe their general health to be excellent, very good or good, compared with others their age.[4]

Physiologic Changes Associated with Aging

Physiologic changes have a cumulative effect as they relate to the continuum of biologic, psychologic, social, and environmental processes of aging. Many of the deficits traditionally thought to be associated with aging are actually signs of pathologic processes. Changes occur for all people, tissues, and organs, however these changes occur with differing rates and individual variability. Variations occur at every age and in every part of the body. Since many internal changes can mimic disease manifestations, and normal changes can mask signs of disease processes, it is very important for those who care for the aged to be knowledgeable about the changes that do occur with aging, rather than identifying changes either as pathologic or associated with the aging process.[9] The four characteristics of physiologic aging are: *universal, progressive, decremental,* and *intrinsic.*

The determinants of aging are complex, and include environmental exposures, genetics, lifestyle, and physiologic and psychological factors. Physiologic age-related changes are not mutually exclusive but rather synergistic and impact each other. In general, as the body advances in years, it tends to become less adaptive to stress. Physiologic changes associated with aging can modify every system in the body, and impact the style and manner in which dental care is delivered.

The major results of the aging process are: a) a reduced physiologic reserve of many body functions (i.e., heart, lungs, kidney); b) an impaired homeostasis mechanism by which bodily activities are kept adjusted (i.e., fluid balance, temperature control and blood pressure control); c) an impaired immunonologic system, as well as related increased incidence of neoplastic and age-related autoimmune conditions.[10]

The loss of elders' ability to function to capacity includes a decline in respiratory function and the inability to accommodate to temperature changes. It is important for the dental team to be aware of these changes; in particular when older adults are challenged by trauma, acute illness, or external temperature extremes. In each of the incidences, older adults tend to be less able to maintain a stable, internal physiologic state. As dental practitioners tend to maintain their office at a cool temperature, the decline in an older persons' baroreceptor function may cause the person to feel cold, which can impact their postural reflexes, causing the patient to be susceptible to orthostatic hypotension.[10] In addition, it is helpful to keep a blanket in the office to keep patients comfortable.

The cardiovascular system of older adults tends to be more likely to develop ischemia, arrthymias, and heart failure, especially when concurrent illness is present. With increased exercise and/or stress, there is an increase in cardiac output. For older adults, the work of the heart is increased as blood is pumped through a less compliant arterial system.

Slight increases in systolic blood pressure are not unusual for those in older age groups, however, one must ensure the pressure stays within acceptable values (<160/95 mm Hg) Systolic hypertension is a strong risk factor for stroke and heart failure, and warrants treatment if it remains consistently elevated over 160 mm Hg, regardless of age. Diastolic blood pressure is not known to change with older age. Although both blood pressure lability and the prevalence of "white coat hypertension"

are increased in the elderly, the clinical significance of these phenomena is controversial.[11, 12]

Nutrition deficiencies are common in the elderly patient. In community-dwelling elderly, anorexia and micronutrient deficiencies are common.[13] Anorexia is multifactorial, affected by changes in taste and smell, lifestyle, physiologic, and psychological changes. Multivitamin supplementation can often improve nutritional status and immune function in this population of older adults.[14]

Loss of subcutaneous fat and decrease in elastic tissue in the dermis render the skin of older adults more susceptible to tear-type injuries. In addition, wound healing is impaired in older adults.[15] Injuries from minor trauma, loss of the skin's effectiveness as a barrier, and a decrease in immune function renders the older person skin prone to infections.[16]

Age-related eye changes are common in older adults. The majority of older adults experience presbyopia, or age-related changes in the lens and iris of the eye. Persons with *presbyopia* have difficulty focusing on *near* objects, often requiring the use of reading glasses. In addition, those with presbyopia experience a greater loss of dynamic visual acuity (viewing objects in motion) than in static acuity. The ability to adapt to sudden changes of light and darkness also diminishes with age. An example of this change is experienced when moving from a brightly lit area to a darkened one, as when entering a movie theatre from the snack area. Yellowing of the lens and accumulation of insoluble protein in the center of the lens fibers eventually develop into *cataracts*, which not only reduce visual acuity, but also cause increases in light scattering, rendering the older adult with poor vision and a sensitivity to glare.[17]

Hearing impairment is common over the age of 60; with a prevalence of 25 to 30% among community-dwelling elders and close to 70% in residents of long-term care facilities. *Presbycusis* is the most common type of hearing loss in older adults and is caused by both pathology and, in some cases, auditory processing.[18] Presbycusis causes gradual, progressive *bilateral hearing loss*, predominantly in the higher frequencies, as well as decline in speech discrimination. Both atherosclerosis and cumulative noise exposure may contribute to presbycusis. Communication with an individual affected by presbycusis is enhanced by slow, distinct vocalization at a low pitch. Shouting can actually be painful to the patient and does not improve the ability to understand what is being said.

Question 2

Which of the following statements, if any, are correct?

A. The elimination of heart disease as a cause of death would result in a greater increase in life expectancy for the total population than if cancer were the first to be eliminated.

B. Each, and all of the following are major signs of the aging process: (a) reduced physiologic reserve of many body functions; (b) impaired homeostasis; (c) impaired immunologic system; (d) increased number of neoplastic conditions; (e) heart conditions, and (f) stroke.

C. Wound healing of the elderly following oral surgery would be expected to be slower than among the young.

D. Presbyopia and presbycusis are both common problems among elderly patients entering the dental office.

E. In counseling an older individual, it is advisable to evaluate if he/she can fully communicate and understand, as well as being able to consent and participate in a proposed treatment regimen.

Bone remains metabolically active throughout life. Age-related bone loss is extremely common, reflecting an imbalance between bone resorption by osteoclasts and bone formation by osteoblasts. *Osteoporosis*, a common problem in the elderly, is an age-related disorder characterized by a decrease in bone mass and by an increased susceptibility to fractures. Losses in bone mass with advancing age are multifactorial, including inactivity, estrogen deficiency, nutritional deficiencies and age-related changes. Clinically, advanced osteoporosis can present with chronic back pain, from mechanical strain caused by kyphosis or vertebral compression fractures. Recent studies indicate that changes in alveolar bone as a result of osteoporosis may contribute to the progression of periodontal disease.[19] Also, a significant decrease in bone mass of the mandible may lead to fragility and increased resorption, risk of fracture, and failure of osseointegration of implants. Prevention, rather than treatment, is the key to the management of osteoporosis. Exercise, vitamins, a balanced diet, dietary calcium, and estrogen play a role in the treatment and prevention of osteoporosis.

Despite extensive neuronal loss, cognitive function in the absence of pathology is well preserved for the most part. Certain neuropsychologic abilities do show decrement with age.[20] Difficulty with *word-finding* is a common complaint of healthy older adults, and a common symptom of cognitive disease. However, as most older adults do not experience cognitive diseases, with sufficient time older persons do find the desired word as successfully as those in younger age groups. *Processing speed* is also slower in older adults. Thus, complex tasks that require quick responses, especially in the context of distracting stimuli, can be hard for the elderly to perform. The central nervous system undergoes significant changes during the course of aging. Decreased response time is often seen in the elderly population, but there is a wide variation between individuals.

The immune system becomes less competent with age. However, the degree of deficiency is not severe enough that opportunistic infections occur commonly in the elderly population.

It is the responsibility of the dental team to be aware and to address the commonly seen age-related changes of aging. Both modifications of office design and patient management techniques are best incorporated in the dental practice addressing the "graying of America."

Cognitive function in those ages 80 years and older is influenced by the high prevalence of dementing illnesses.[21] See section on Cognitive Function.

Functional Status

Functional status is a critical indicator of health and well-being in the older person, and is one of the most challenging issues in health care of older adults. Functional status is often a better descriptor of an individual than the presence of specific diseases, as impairments in physical and cognitive functioning predict mortality, institutionalization, and the type and amount of health-care services needed. Identifying one's functional status requires a comprehensive *health assessment,* including an assessment of the individual's *functional abilities, health status, physical, psychological,* and *oral-health status.*

A functional assessment evaluates one's ability and limitations to complete basic tasks of daily life.[7] Functional status is defined in terms of Activities of Daily Living *(ADLs)* and Instrumental Activities of Daily Living *(IADLs).* Activities of Daily Living are those abilities that are *fundamental to independent living,* such as bathing, dressing, toileting, transferring from bed or chair, feeding and continence. Instrumental Activities of Daily Living (IADLs) are more complex *daily activities* such as using the *telephone, preparing meals* and *managing money.* The individual's ability to complete ADLs and IADLs will affect the person's ability to access and maintain their oral health care regimen.

Functional limitations serve as key indicators of an older person's ability to remain independent in the community, their quality of life and active life expectancy. Approximately 72% of the population aged 65+ years reported having no difficulty with ADLs and IADLs, while about 10% had difficulty with 3 or more ADLs.[22] As age increases, the percentage of the population having no difficulty with ADL's or IADLs decreases. The two most common IADLs identified by the elderly are *difficulty walking* and *getting outside.*[23] These common conditions may require dental-health professionals to modify the individuals' treatment plan, and to consider schedule times that are optimal for the patient.

Cognitive Changes Associated with Aging

Inaccurate assumptions and false beliefs about mental health and the cognitive changes of aging have resulted in an overemphasis on decrements often associated with older adults. Recent studies of the aging brain show that major cognitive declines do not occur in the absence of disease, trauma, or stress. Developmental transitions, life events and environmental changes may interfere with older adults' ability to concentrate and to think clearly. Research has ascertained that one's intellect does not decline as an outcome of aging but rather, as a result of many conditions including poor nutrition, disease and hormonal changes.[24] An older person usually takes longer to learn the same information as a younger adult, but when given sufficient time, the end result is similar for both individuals. In general, more time is needed for an older person to encode, that is, to retrieve or to recall the information. In later life, mental health is measured by the capacity to cope effectively with relationships and environment and by the satisfaction experienced in doing so.

Because of the multiplicity of factors that relate to the treatment of the elderly, it is important to evaluate the ability of the patient to communicate and to understand,

consent to and participate in the treatment. The practitioner must determine, either through an interview or through professional services, the capacity of the individual to respond to treatment.

The *most common type of dementia* in the elderly is senile dementia of the *Alzheimer's type (SDAT),* accounting for approximately 80% of all dementia's seen in the elderly. The second most common cause of dementia in the elderly is multi-infarct dementia or *vascular dementia,* accounting for 15 to 25% of cases.[25]

Alzheimer's disease is a progressive, degenerative, dementing illness that attacks the brain and leads to the loss of memory, intellectual capacity, thinking, and behavioral changes. SDAT is characterized by the accumulation of *neuro-fibrillary tangles* and *senile plaques* within the cerebral cortex.

Alzheimer's disease has an insidious onset that manifests as loss of recent memory, impairment of judgment and personality changes. Those with SDAT may experience confusion, personality and behavior changes, impaired judgment, difficulty finding words, finishing thoughts or following directions. When experiencing the early stages of the disease, the individual generally maintains good social skills and is often able to "disguise" the presence of the disease. In its early stage, the disease is often very difficult to assess, and is generally denied by family members.

The cause of SDAT is not known and currently there is no cure for it. Although SDAT has been found in younger age groups, it afflicts approximately 10% of those over the age of 65 and *47% of those over age 85.* The disease progresses from 2 to 20 years, and presents as a complex picture of overlapping symptoms, reflecting a continuous decline in memory, thinking, and behavior. Cognitive skills and competency in life skills decline. There is loss of memory, language, intellectual prowess, concentration, emotionality, and altered spatial motor performance. Both verbal and nonverbal communication is affected.

There are numerous reversible causes of these symptoms in older adults including infection, dehydration and vitamin deficiencies. Attentive health-care professionals, in particular oral health-care professionals may be one of the first caregivers to detect some of the early, subtle changes associated with Alzheimer's. It is incumbent upon the health-care professional to assist the individual to obtain a comprehensive assessment to correct the reversible causes whenever possible and to assist the individual and family in the opportunity to address the care and management of the individual.

Oral-Disease Patterns

During the past 50 years, one of the major changes in oral-disease patterns in the United States has been a steady *decrease in the rate of edentulism.* It is likely that for the first time in recorded history there are now more older adults with natural teeth than without teeth. In 1986, almost 30% of those 65 to 74 years were edentulous, whereas in 2024 it is predicted that only 10% of this group will be edentulous.[26] This decline in edentulism appears to be the result of water fluoridation, increased public awareness of preventive approaches, improved access to services, and a decrease in early tooth loss.[27]

Although the prevalence of edentulism increases in the non-institutionalized older age groups (10% of 45 to 54 years, 28.4% of 65 to 74, and 52.5% of 85+ years), these rates have steadily decreased over time.[26] This decline in tooth loss results in more natural teeth at risk for caries (coronal, recurrent, and root) and periodontal diseases. As these trends continue more restorative and preventive services will be needed in future dental practices.

Recent reports have found the prevalence of coronal caries is decreasing for children and young adults of middle to high socioeconomic status. Although dental caries has not traditionally been perceived as a problem for the elderly, decay rates have been found higher in some adult groups than in children. As long as teeth are present, individuals remain at risk of dental caries.[28] Unfortunately, many older adults do not place a priority on oral health care, and view seeing a dentist only to relieve pain and discomfort. For those not receiving routine dental care, and as a result of increased deposits of secondary dentin and a reduced sensory ability, many older adults tend to seek care only when their decay is in a late stage. A survey of Iowa's' population found 30% of dentate elderly had untreated coronal decay, with 77% having either a new coronal or root lesion in the last 3 years.[29]

Root caries is common and frequently occurs in this age group. Root caries has been found in 65% of the males and 53% of the females in the 1985-86 NIDR study.[30] With the use of new preventive approaches and restorative materials, the dilemma associated with restoring root-carious lesions is expected to diminish in the future.

Contrary to many long-held views, periodontal disease is *not* an age-related disease. Although the prevalence of periodontal disease appears to increase with age, this is likely due to the long-standing cumulative nature of the disease, with its onset earlier in adulthood. In addition, much of the available data reflects cross-sectional studies that do not present a generalizable view of the population. Longitudinal survey data is needed to document the progression of the disease. It is estimated that *90% of adults 65+ years* need periodontal treatment, with 15% needing complex treatment.[31] With the rate of periodontal disease progression partly related to the mass and composition of the oral microbiota and the host's ability to respond to this microbial population, research has focused on new diagnostic and treatment modalities, such as DNA diagnostic probes, enzymatic assays and bacterial analyses, the use of lasers, new pharmaceutical preparations and subtraction radiography. With new diagnostic methods complementing traditional clinical techniques, earlier identification of periodontal disease and risk factors will be possible, as well as early treatment to help reduce disease progression and its subsequent loss of teeth.

Question 3

Which of the following statements, if any, are correct?

A. Determining the ability of an individual to perform the Activities of Daily Living (ADL) and the Instrumental Activities of Daily Living (IADL) is a better means to evaluate a person's functional age than using age alone.

B. Periodontal disease is associated with the older individual; however, periodontal

disease is not caused by aging—it is caused by oral neglect while aging.

C. The four general key admonitions to retaining teeth for a lifetime are: (1) have frequent recalls and dental examinations, and (2) have early diagnoses for disease and disease risk, followed by appropriate (3) early preventive and/or (4) restorative treatment.

D. Approximately 47% of individuals over 85 are afflicted with dementia of the Alzheimer's type.

E. Presbycusis, but not presbyopia, are physical problems of many seniors who will visit a dental office.

Like most cancers, oral cancer occurs primarily in the older age segments of the population, with the majority of cases diagnosed after age 65 and more than 95% occurring after age 40.[32] The key issue related to oral cancer problem is the need for *early and effective diagnosis.* Although the primary risk factors for the development of oral squamous cell carcinoma have traditionally included alcohol abuse and use of tobacco products, these risk factors do not have to be present for a lesion to develop. Thus, it is incumbent upon the oral health care professional to provide oral cancer examinations to all patients on a regular (at least annual) basis. As the multifactorial etiology of oral cancer becomes evident, other factors including alterations in cellular oncogenes, as well as microbial and viral infections may be found to play a role in the pathogenesis of premalignant lesions and oral squamous cell carcinoma.[33] Given that early diagnosis of oral cancer greatly improves the prognosis of the disease, and that many factors influence the timing at which oral cancers are diagnosed, i.e., lack of access to care and patient delay in seeking treatment,[34] oral-health professionals must provide when appropriate, routine comprehensive intra- and extraoral examinations of their patient populations.

It is essential to recognize that no broad, generalized decremental changes in oral health occur simply with age. Healthy older people can expect to keep their teeth, throughout their lifetime. However, in the presence of one or more medical conditions and/or their treatments, oral functions may be altered which can then impact upon the patients' general and oral health status.

Through frequent recall visits and regular professional examinations, adults will be better able to maintain their dentition throughout their life. Prevention of oral disease is the critical component for oral-health maintenance. In addition to promoting and monitoring the basic oral-hygiene practices, the practitioner needs to be aware of the changing physical, psychologic, socioeconomic, and medication status of their older adult patients. In addition, the practitioner needs to be ready and willing to intervene and make necessary modifications to treatment as well as referrals to community resources. Older adults and their caregivers need to be educated and have their education reinforced so to enhance their knowledge of oral care protocols.

Long-Term Care

Long-term care refers to health, social and residential services provided to chronically disabled persons over an extended time.[35] Longitudinal studies in the United States

suggest that persons 65 years and older have a 40% chance of spending some time in a long-term care facility before they die. Of those who enter nursing homes, 55% will spend at least one year there, and over 20% will spend more than five years there. Two of the most common symptoms that lead to nursing home placement are incontinence and behavioral problems such as wandering or disruptive actions often associated with dementia and Alzheimer's disease.[36]

The majority of the 65+ population lives in the community, while only 4.5% are in nursing homes.[21] Of those institutionalized in 1990, 1% were 65 to 74 years, 6% were 75 to 84 years, 19% were 85 to 89 years, 33% were 90 to 94 years, and 47% were 95 and older. Given the growth of the 85+ population, a substantial increase in the future use of nursing homes appears inevitable.[37]

The delivery of oral-health care to residents in long-term care facilities or for those who are homebound presents special challenges for the professional. In addition to the mode of care delivery, this patient population tends to be frail, functionally dependent and often lacking any level of self-interest in their oral health. Cognitive declines, lack of motivation, physical impairment, and chronic medical problems all contribute to a decrease in self-care ability and increase risk of oral disease. This population has been characterized as having high levels of edentulism, coronal and root caries, poor oral hygiene, periodontal diseases and soft tissue lesions.[38, 39]

For the first time, the provision of oral-health care is mandated in long-term care institutions. The Omnibus Budget Reconciliation Act (OBRA) of 1987 (Public Law 100-203) requires all nursing facilities "must employ comprehensive assessments to determine specific health care needs of those residents who participate in Medicaid or Medicare Part B programs. The assessment must be conducted not later than 14 days after admission and can be amended up to 30 days after admission. Nursing facilities must review the resident's condition for any changes in health status every 3 months and perform the comprehensive assessment annually. The assessments are conducted by a registered nurse, with input from a physician or other health care practitioner(s) as necessary." While these assessments are not required to be completed by a dental professional, dentists and/or dental hygienists need to be identified as part of the health-care team of the long-term care facility. Given a rapidly growing population of dentally aware older adults, future population growth patterns and technological advances in portable and mobile dental equipment, oral-health care programs in long-term care facilities will become feasible and common locations for providing dental care.

One major influence on dental utilization is having dental insurance. Since dental insurance is generally provided as an employee benefit, the elderly typically do not have this benefit. According to the 1989 NHIS[26], 50% of the 35 to 54 year, 37% of the 55 to 64 years and 15% of the 65+ population had private dental insurance. Dental benefits are not included in Medicare, and very few states provide dental service to adults through the Medicaid program. In 1990, nearly 75% of edentulous persons 35+ years did not have private dental insurance compared to about half of the dentate population.[40] Since edentulous persons are less likely to have visited a dentist for the past 5 years, routine examinations for and early treatment of oral soft-tissue diseases are precluded. Thus, the elderly edentulous population may be identified as one of the major underserved populations of this country.

Question 4

Which of the following statements, if any, are correct?

A. The two most common causes of cancer are excessive consumption of alcohol and smoking; the two most important ways to insure recovery from cancer are early diagnosis and early effective treatment.

B. Two out of every five (40%) of Americans over 65 will spend time in a nursing home before death.

C. The majority of institutionalized individuals usually have private dental insurance.

D. The two most common causes for nursing home attendance are incontinence and osteoporosis.

E. Dental care is authorized for the elderly under Medicare.

Summary

With more older adults and more teeth per older adult, the composition of services rendered to this population will change dramatically in the next decade. The oral health professional of today and of the future will be called to treat an ever-increasing number of older adults. The future elderly will be different than the current cohort seen today. The future elderly will have more teeth, visit the dentist more often, have a higher level of education, better finances and a dramatically different perspective of needs. They are and will continue to be a heterogeneous mix of individuals with various levels of functional, socioeconomic and oral health status. Advances in materials and technology in combination with the changing patterns of oral diseases will continue to have dramatic effects on the practice of dentistry.

The role of the oral health-care professional will focus more on the diagnosis and treatment of oral diseases and disorders, using new aids and devices such as lasers, CAD/CAM, and molecular probes. As diseases of the hard tissues are resolved, more emphasis will be placed on the diagnosis and treatment of soft tissue lesions. With new and improved diagnostic skills, the older adult, the group identified as having the highest risk of oral cancer may no longer require the extensive and often disfiguring surgical remedies currently in place. Preventive oral-health approaches need to be maintained throughout the lifespan.

In order to provide optimal care to the aging population, one must remain current on oral medicine, pharmacotherapeutics and changing technologies. Oral health-care professionals must address how this aging population will manage in the dental facility, and, at a minimum, have accessible offices, large-sized type medical history forms available, as well as easy-to-read signs, health literature, and appointment cards.

Answers and Explanations

1. A—correct.

B—incorrect. The elderly are living longer as a group, hence being the oldest, the sickest and the frailest, they constitute a major health (and political) challenge.

C—incorrect. The truth is that between 1999 and 2030, the white population is projected to increase by a tremendous 81%; however this will be drawfed by the estimated 328% by Hispanic-Americans, 285% by Asian and Pacific Islanders, 147% by American Indians, Eskimos and Aleuts, and 131% by African-Americans.

D—incorrect. There are more older widows because women marry earlier than men, hence often outlive their spouse.

E—Incorrect. Majority means over 50%. The correct percentage of individuals over 80 living in nursing homes approximates 19%.

2. A, B, C, D, E—correct.

3. A, B, C, D—correct.

E—Incorrect. BOTH presbycusis (hard of hearing) and presbyopia (far sightedness) are problems for the dental office; patient mobility and patient communication are jeopardized by these two impairments.

4. A, B—Correct.

C—incorrect. The answer is that many nursing home residents do not have dental insurance. Part of this problem is due to the fact that many have dentures and do not believe they need insurance. Others do not have the money for insurance that buys access to professional care, nor do they have the motivation or physical ability to desire to maintain self-image.

D—incorrect. Incontenence is one of the two major problems. Osteoporosis is not; (it can be coped with in home environments). The second major reason for institutionalizing individuals is the dementia characteristic of Alzheimer's disease.

E—incorrect. Medicare does not subsidize routine dental care.

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Chapter 22. Preventive Dentistry in a Hospital Setting

- *Norman O. Harris*
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Objectives

At the end of this chapter it will be possible to

1. Discuss the scope of dental services available at community, federal, and large metropolitan hospitals.

2. List at least eight categories of patients seen on a hospital service who benefit from a hospital dental service.

3. Name or describe the personal oral-hygiene items that, at a minimum, each hospitalized patient should possess at time of admittance, and that should also be stocked in the hospital store.

4. Cite several adverse side effects of chemotherapy and radiation therapy for cancer.

5. Outline the responsibilities of the oral surgeon, the speech therapist, the pediatrician and the pediatric dentist in the effort to make life as near-normal as possible for a cleft-palate individual.

6. Orient students about the administrative functions of a hospital and examples of a hospital dental and medical service in action.

7. Compare peritoneal dialysis with hemodialysis.

8. Explain why dentists favor pregnant women having folate during the first four

months of pregnancy.

9. Tell another student what precautions you are going to take with this afternoon's high-risk cardiac patient.

Introduction

The idea of providing for the delivery of dental services in a hospital setting is not new.[1] The first dentist to practice in an American hospital was Richard Courtland Skinner, who immigrated to Philadelphia in 1788.[2] Later, he was the first dentist to ask for and receive an official appointment to a medical institution. In the 1790s, Skinner created the first hospital dental clinic in the Dispensary of the City of New York to treat the indigent. It was not until the middle of the 19th century that Garretson and Hullihan laid the foundation for the practice of hospital dentistry.[3] Approximately 200 years after Skinner, in 1987, it was reported that about 40,000 dentists had hospital privileges.[4] In 1901, the Philadelphia General Hospital developed the first dental-intern training program. In spite of the fact that about 60% of all hospitals have dental programs, not all hospitals extend staff privileges to dentists. Presently, the *admission* of dentists to a hospital staff and the extent of dental *privileges* is a determination *made by each individual hospital* and is reflected in the hospital by-laws.

The following partial job description illustrates the typical daily tasks of a hospital dentist: "The hospital dentist focuses on serving those who cannot receive dental care through the traditional delivery systems. Patients who are medically and/or mentally compromised (e.g., with cancer, heart disease, HIV/AIDS, Alzheimer's Disease), often suffer from a debilitating anxiety towards dental treatment. In addition, the service provides care for victims of emergency and trauma to the head and neck regions, offers consultation services for other hospital services, and furnishes dental care to patients residing in the facility. Hospital dentistry renders the full range of surgical, restorative, consultative, maintenance and preventive outpatient procedures offered in private practice settings. The hospital practice also offers services for restorative and surgical procedures completed under general anesthesia."[5]

The majority of the dental care rendered in many hospitals is focused on the diagnostic and treatment care necessary to *support the recovery* of medically compromised patients or patients admitted for serious head and neck diseases, infection, or trauma. The U.S. health-care system recognizes that the training needed by dentists and dental hygienists for hospital practice can be incorporated into a teaching hospital's mission. To support this need for training dental personnel, teaching hospitals support training programs for *graduate students,* and rotate them through a dental department and other appropriate departments such as emergency medicine, internal medicine, and anesthesiology. Every year more than 1000 American dentists receive training in hospitals. At the same time, dental hygiene students and dental assistants have provided dental hygiene services for ambulatory and nonambulatory patients. The scope of the dental services within a hospital varies greatly, being dependent on such factors as bed capacity, economic support, level of specialty expertise, and proximity to medical and research centers.

As a result of the influence of these needs and limitations, dental services of hospitals

can arbitrarily be placed in one of three categories:

1. Smaller nonfederal hospitals (private and community) that provide care to *only support recovery* of the patient's primary complaint from the time of admission. These hospitals usually have only a *short-term* commitment to the patient for a period during which care is being rendered for a primary complaint. Accordingly, the dental service available is generally concerned with delivering maxillofacial care resulting from accidents. The oral surgeon may be on-call for cases arriving at the emergency room—or the problem may simply be handled by an attending surgeon. In these smaller hospitals, it is assumed that any patient requiring dental care, other than that pertaining to the primary complaint, will visit a private dentist following the period of hospitalization.

2. Military and other federal hospitals that provide total oral-health needs for their active duty or eligible personnel. These services include diagnosis, treatment planning, preventive and treatment services, counseling and programmed recalls. Similarly, Veterans Administration and U.S. Public Health Service Hospitals provide complete care to eligible in-and-outpatients.

During war years, military hospitals are often at the forefront in innovative oral and general surgery.[6] The expertise gained in conflict is then brought back to the peacetime world. For instance, in World War I with its trench warfare, coupled with violent charges across battlefields blanketed with high explosives and a continual hail of high velocity small arms fire, produced devastating wounds of the face and body at a rate never known before.

It is of interest to know that in 1914, there was no general concept for reconstructing a destroyed mandible. It was between 1914 to 1918 that the transplantation of bone emerged as the best approach to restoring the lower jaw. Even then, there was a puzzle as to why one transplant would be a success and a failure in another. It was a Dr. Carl Partsch, a German Army Surgeon, who is credited for pointing out that the success of fracture healing and bone transplantation depended on the *tight contact* between undamaged surfaces, or between stumps and transplantation pieces.[7]

3. Large metropolitan and university teaching hospitals have expanded their scope of dental services to include treatment for patients with serious systemic diseases associated with a *high risk for life-threatening emergencies*—such as heart disease. For these patients, when crises occur in the hospital, life-saving drugs and/or emergency teams are immediately available. Also, many patients are accepted who have a high risk for *transmitting of disease,* such as AIDS. Finally, to support advanced-degree programs, dental clinics are available to the public. Graduates from these degree programs often remain in hospital dentistry; others enter academia, while others go *well prepared* into private practice.

Dental Needs of Hospitalized Patients

Dentists as well as other health professionals realize that oral health cannot be divorced from the general health of the hospitalized patient. Many oral conditions are intimately related to systemic diseases. Optimally, total health care requires the *combined* efforts of the medical and dental professions.

Several reports indicated that long-term hospitalized or chronically ill patients had many significant dental needs. To verify these reports, the American Dental Association's Council on Hospital Dental Services' surveyed 1,634 individuals. It was estimated that about 80% of all patients admitted to a hospital had some form of oral pathosis that *required* treatment. The majority of these patients was unaware of dental problems and typically did not have a family dentist. The six greatest dental treatment needs were the same dental needs as of the general population—dental caries, periodontal disease, plaque or calculus deposits, nonrestorable teeth, partial or complete edentulism. They also discovered that adequate, functional, fixed, and removable prosthetic devices were nearly nonexistent and that the level of dental care previously provided was very low. Poor oral hygiene, gingival inflammation, and papillary hyperplasia were the most prevalent periodontal problems. Almost all of the dentures examined were inadequate, with gross amounts of materia alba present on the denture and in the buccal vestibule. About 57% of patients with acute dental pain had not received palliative care.

There are several categories of hospital patients, each with special needs for dental services. Among others, such patients include: (1) head and neck cancer patients; (2) cleft-palate cases, (3) AIDS patients; (4) renal, liver, and heart-failure patients; (5) patients in need of prophylactic antibiotics; (6) comatose patients; (7) paraplegics, quadriplegics, and amputees; (8) postsurgical patients; (9) diabetic patients; (10) psychiatric patients; (11) obstetric patients; and (12) organ-transplant candidates and recipients. Each individual in each of these groups requires specialized care and counseling. No treatment plan fits all!

Administrative Requirements

If a dentist or a dental hygienist is to be used in a hospital preventive dentistry program, the hospital administrator *must be receptive* to the idea. While dental hygienists may be employed by a hospital through normal employment channels, dentists must be *admitted* to the hospital staff and be *privileged to provide specific* services. Hospitals have well-defined regulations for the admitting and privileging processes. The existing medical staff and nurses must support any additional ward dental activity that has to be meshed with the other professional time requirements associated with patient care. Unfortunately, the subject of dental care is not emphasized in the formal training of the majority of nondental health professionals. Some medical professionals who have been trained in hospitals that sponsored post-doctoral dental-education programs may be more knowledgeable about hospital dental care. However, this education *usually* must come through continuing personal relationships between dental and medical staff personnel and through continuing education programs.

The Hospital Dental Department

Accreditation and Regulations

A hospital is administered by (1) a *governing body* entrusted with the responsibility for the overall organization and conduct of the institution. This body may be a Board of Directors, managers, or trustees and this body will establish policies that are in

accordance with documents written by the Founders, the hospital administration and by the laws of the state and nation in which the hospital is located. Hospitals will appoint a (2) *chief operating officer* who has the day-to-day responsibility of maintaining administrative structure, decorum, quality of hospital care, planning and fiscal policies. The (3) *medical-dental staff* of a hospital is the third component of the hospital administrative structure and is composed of its appointed Chairpersons and elected representatives.

Hospitals, in order to be permitted by their respective states to deliver care and to receive reimbursement from federal payors, must participate in a formal accrediting process. Most, if not all hospitals in the United States are accredited by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO). The website of the JCAHO lists as its mission: "To continuously improve the *safety and quality* of care provided to the public through the provision of health care accreditation and related services that support *performance improvement* in health care organizations." In addition, teaching hospitals in the United States and Canada that sponsor postdoctoral dental education programs are *periodically assessed and accredited* by their respective *national dental associations.* In the United States, the accrediting agency is the Commission on Dental Accreditation of the *American Dental Association.* Goals and objectives for both U.S. and Canadian postdoctoral dental programs are similar. Postdoctoral teaching programs evaluate a variety of proficiencies—professional skills and practice management, restorative dentistry, prosthetics, endodontics, orthodontics and pediatric care, oral pathology, oral surgery, periodontics, pharmacology and hospital functioning. To add to this list may be emergency care, sedation anesthesia, and some aspects of public health.[8]

Dental Department Administration and Sections

The Dental Department is the administrative and clinical hub for *all* dental operations in the hospital. The Department Chairperson is selected by the Hospital Board based on her/his demonstrated leadership and professional competence, and has equal status and responsibilities as the Chairs of other departments. Examples of these responsibilities include a require- ment for interdepartmental cooperation and consultation, major and minor oral surgery, operation of a public clinic, clinical and basic research, Ward dental care of dental in-patients, recruitment of department manpower, and for budget preparation to meet expected department needs and for future institution expansion. To accomplish these tasks, many of the duties are delegated to the Section Chiefs—surgery, prosthetics, periodontics, general practice, pediatric dentistry, and research.

The multichair dental clinic where the majority of routine dental care occurs, usually accepts an outpatient patient load as part of the postdoctoral training program. Many times, this patient load consists of the indigent, with or without government or insurance support. This general practice supervised teaching program is essentially a private-practice environment, and it is a splendid training background before entering practice. It also helps provide the dental manpower to meet the hospital's requirement for a fully functioning dental service.

The Dental Department's maxillofacial team is an essential component to a hospitals mission. In a large metropolitan hospital where emergency surgery is a daily routine,

the operating room duties for major craniofacial surgery are usually shared by teams of *Board-Certified* medical and dental maxillofacial surgeons. Oral surgeons treat victims with major soft and hard tissue damage of the face and head. These injuries arise from many sources—automobile accidents, knife and gunshot wounds,[9] and societal and domestic violence.[10] The oral surgeons also provide orthopedic and plastic surgery to correct facial skeletal disharmonies and *developmental defects* such as cleft lip and palate.[11] Some oral surgeons specialize in more extensive *plastic* surgery, while oral *cancer surgery* is another major task for a well-trained oral surgeon.

The successful outcome of any minor or major surgery is determined by whether the patient feels as though he/she can socialize without impediment, whether there is acceptable speech, and whether dental appearance and oral function is satisfactory.[12]

The dental department's maxillofacial *prosthetic section* is an essential component of the maxillofacial team. The absence or loss of soft and/or hard tissue resulting from developmental defects, or following trauma or surgery, often is *too great for corrective surgery* alone. With training beyond the usual prosthodontics graduate programs, some prosthodontists and laboratory technicians become proficient in sculpting customized dental or cosmetic prostheses.[13] Obturators are needed at times to bridge palatal clefts.[14] Some hospitals have established prosthetic centers specializing in the fabrication of eyes or facial parts lost by trauma or disease;[15] others have specialized in constructing ears lost because of trauma or congenital absence.[16]

In the past, these external and intra-oral prostheses usually *were* held in place with adhesives.[17] Now, whenever possible, they are magnetically connected to implants that are *integrated into bone* for more permanent fixation.[18] Once inserted, a *make-up artist* teaches the patient how to apply different shades of creams and powders to camouflage the prosthesis.

Intra- and Extra-hospital Cooperation

Many times there is a need for cooperation between the physicians of other services and the personnel of the dental department. For example, there have been needs for endoscopic tooth extractions from the nasal cavity requiring the skills of a nose and throat professional.[19]

Physician intervention is often indicated to cope with seemingly routine tasks of a dental service that turn into emergencies. A few examples are: near-fatal bleeding following over self-medication with quinine,[20] treating of periodontal disease to help control diabetes mellitus (and possibly vice versa), reducing the possibility of a bacteremia of oral origin becoming a problem of cardiovascular pathology, or in receiving help in removing a denture that has lodged in a throat.[21] Some of these problems can be anticipated from a patient's history record, many cannot.

Daily there are requests for clinic consultations and examination of newly admitted patients. There are also many requests from the private sector for extractions under anesthesia, or for dental care of high-risk persons. Some are referred because of patient fear and anticipated difficult extractions, while still others are referred because of mental handicaps, moderate-to-severe behavioral problems or a history of

seizures.[22]

Consultations between appropriate hospital services and private referring practitioners is encouraged, both as a form of courtesy to the referring professionals and to ensure that patient management is appropriate.

Preventive Dentistry

In most smaller hospitals, there are no dental departments, hence no provisions for an active primary preventive program. In one study of short hospital stays, (at least one week) all 33 respondents to a questionnaire indicated they were unable to carry out their self-care as well as they could at home.[23] This finding would probably have been different if the hospital had had a policy of asking each self-sufficient patient admitted, to bring a small kit with a toothbrush and floss to carry out daily self-care procedures. In addition, the hospital store should stock high-demand dental products including fluoride dentifrices, floss threaders, disclosants, fluoride mouthwashes, and denture cleaners. The pharmacies should be in a position to dispense prescriptions for chlorhexidine.

In these hospitals, nurses or nurses' aides must provide essential post-operative dental procedures involving the oral structures.[24] An example might be the daily fluoride spray-cleaning of the mouths of mandibular fracture patients (where the interdental wiring makes mechanical plaque control difficult).

For military hospitals, the task is much easier. All essential dental items are stocked in the hospital exchange for patient purchase. A preventive dental officer is designated by the Chair to insure that the needs of Ward and clinic patients are served. Usually a visit to the clinic is scheduled for an examination as *soon as is appropriate* following admission. At this time, a dental examination is accomplished to determine preventive and treatment needs. Since, by policy, a military patient is held for recuperation before being returned to full-duty, there is often slack time for accomplishing needed dental services before being returned to his/her unit. During this time, counseling should be directed to the need and methods of self-care—mechanical plaque control (brush, floss, and irrigation), and chemical plaque control (use of fluoride mouth rinses for achieving limited reminerlization), and chlorhexidine (for temporary suppression of caries and gingivitis microbiota). Also, during this time, more intensive remineralization procedures can be instituted for interproximal radiographic lucencies that are without signs of overt cavitation. Since adults *do* develop occlusal caries, sealants should be placed in any deep pits and fissures.

For the clientele of the clinics of *large and university hospitals*, preventive dentistry takes on a more urgent and ominous tone. Many of these patients are high-risk patients who have serious chronic diseases that have been treated at the institution or elsewhere. For these patients, preventive dentistry means preventing the occurrence of life-threatening emergencies as well as protecting the oral structures from disease. The same procedures suggested in other chapters apply for these patients, except that (1) all the preventive actions and risk assessments are monitored at shorter intervals; and (2) all emergency precautions for each condition must be in place before treatment begins.

In total-care hospitals, a preventive dentistry cart should be available to the dental officer, dental hygienist or designated person making the ward rounds. Such a cart should be stocked with items such as disposable plastic mouth mirrors, a flashlight, a hand mirror for patient viewing, sterile tongue blades, cotton applicator sticks, mouth props, and seizure sticks (double tongue blades wrapped in gauze and taped securely). Additional aids can include a spray unit and high-volume, low-vacuum suction unit, aspirator tips, gauze squares, and disclosing tablets. Mouthrinses can be made up in quantity by the pharmacy for Ward use.

Question 1

Which of the following statements, if any, are correct?

A. The first hospital dental service in America was established in New York only a few years after the Revolutionary War.

B. To become a professional member of a hospital staff, a candidate must be privileged.

C. A hospital dental service must be accredited by medical and dental rules and regulations established by the political and professional entities legally claiming jurisdiction.

D. The Chairman of the various departments of a hospital only vote on propositions pertaining to their respective services.

E. Pit-and-fissure sealants should be considered for adults with deep occlusal fissures.

To this point, hospital administration has been emphasized; this permits the business and professional aspects of a hospital to function harmoniously. From this point on, *examples* of the professional responsibilities and patient problems of a hospital population will be discussed. The diseases selected for brief discussion—cancer, cleft palate, HIV/AIDS, cardiovascular disease, renal disease, and diabetes mellitus are all pathologies that will be encountered by dentists and dental hygienists over a professional lifetime. These encounters will occur regardless of whether the practice is a dental office, military facilities, academia, public health, or hospital clinic. The main focus should be in *recognizing* impending or actual emergencies and immediately *initiating a programmed and already rehearsed response.*

Xerostomia

Many xerostomia ("dry mouth") cases that are seen in a hospital clinic are a combined physician-dentist concern. This hypo-secretion of saliva is seen in several medical and dental conditions—Sjogren's syndrome, intake of xerogenic drugs, dryness of psychogenic or idiopathic origin, diabetes, candidosis, and excessive alcohol consumption.[25]

Xerostomia is especially troublesome when it results from radiation therapy used in the treatment for cancer when the glandular tissue of the major and minor salivary glands are usually destroyed. A drastic *decrease* of saliva flow occurs, and an inverse

increase in salivary viscosity soon follows. The xerostomia following radiation is generally *irreversible*, although some researchers have reported modest recoveries in some patients (Figure 22-1). Xerostomia diminishes the saliva's buffering capacity resulting in a *major increase of cariogenic organisms* such as mutans streptococci and lactobacilli.[26-28] Cariogenic activity becomes rampant and accelerated under these "dry mouth" conditions. A radiation-induced caries, termed *radiation caries*, is especially destructive, displaying a cervical and incisal predilection on the teeth for, first plaque and then caries that can rapidly amputate the crown. (Figure 22-2). It should be emphasized that the caries is not caused by the radiation, but is a result of the xerostomia that allowed unrestricted growth of the cariogenic plaque bacteria.

Because the salivary changes following radiation are *permanent*, the threat of radiation caries exists for all teeth *throughout the reminder of the patient's life.* Preventive therapy involves meticulous attention to daily self-care consisting of oral hygiene, a low-carbohydrate diet, frequent dental checkups (at least every 3 months), topical fluoride gel, and chlorhexidine rinsing. Conversely, the xerostomia that occurs following the intake of xerogenic drugs and chemotherapy usually *regresses to normal* following discontinuance of the offending cause.

Salivary stimulants and saliva substitutes are options to provide some relief to the dry mouth. Generally, saliva substitutes consist of a wetting agent (carboxymethylcellulose or sorbital), electrolytes, fluoride, and flavoring. However, saliva substitutes are poorly accepted by patients because of their short duration of action (10 to 15 minutes). As such, the majority of xerostomic patients prefer frequent sips of water to the saliva substitutes.[29] Sugarless lemon drops and chewing gum, are recommended as methods for increasing salivary output.[26,30] A prescription for pilocarpine, a saliva stimulant, has also successfully increased salivary output in studies involving a variety of xerostomic populations.[31]

Figure 22-1 Mean flow rates of stimulated whole saliva in 42 patients with cancer before, during, and after radiotherapy. (From Dreizen S, Brown LR, Daly TE, et al. Prevention of xerostomia-related dental caries in irradiated cancer patients. *J Dent Res.* 1977; 56:99-104.)

Figure 22-2 Severe radiation caries in the maxillary and mandibular anterior region of an adult treated for head and neck cancer. (Courtesy of the late Dr. Simon Katz, Indianapolis, IN.)

Head and Neck Cancer

Cancer treatment is usually centered at major medical centers having both the facilities and personnel to continually seek new and better approaches to cancer therapy. Examples are the MD Anderson Hospital in Houston, Texas, and the Memorial Sloan-Kettering Cancer Center in New York City. For the year 2001 the American Cancer Society has estimated that there will be approximately 20,000 new cases of *oral cancer* in the United States. This same projection indicates that approximately 7,800 will die of the disease.[32] Tobacco is the cause of an estimated 390,000 premature deaths in the United States annually. Despite the fact that the *incidence of cancer is increasing*, early diagnosis and significant advances in therapy have resulted in a *much longer and more productive life* for the cancer patient.[33]

Most head and neck cancers are *not* diagnosed early. In examining this problem with

dentists in Maryland, where the mortality rate for oral and pharyngeal cancer is seventh- highest in the nation, focus groups found need for continuing education to correct: (1) inaccurate knowledge about oral cancer, (2) inconsistency in oral examinations, (3) lack of confidence in when and how to palpate for abnor- malities, and (4) lack of time to routinely conduct oral cancer examination. Most cancers of the mouth are *easy* to see, while those of the floor of the mouth can be *easily* palpated.[34] The typical early intraoral lesion is usually an *indurated ulcer.* Any ulcer that persists for *3 weeks should be biopsied.*[34] The early detection, and early referral leading to a more favorable outcome of therapy, *is* a crucial responsibility of the *general dentist!*

Cancer Prevention

A tragedy associated with many head and neck cancer deaths is that in *most cases* the disease *could have been prevented.* The great majority of head and neck cancers arise from *three* sources: (1) *smoking* and the use of smokeless tobacco, (2) exposure of the lips and face to the *sun,* and (3) excessive consumption of *alcohol.* Tobacco products account for *three-fourths* of all intraoral malignant (squamous cell) cancer cases (Figure 22-3) in the United States.[35] *Basal cell* carcinomas of the face are often caused by an excess exposure to the natural *sun* or to "*tanning devices.*" *Excess alcohol consumption* is also a major contributory factor to the development of oral and pharyngeal cancer[36,37] with alcohol appearing to synergize with tobacco as a risk factor for all the upper aerodigestive tract cancers.[38,39]

Even for those ignorant of, or who ignore the admonitions by the American Cancer Society concerning smoking, sun exposure and alcohol, there is frequently a second chance. Usually, there are early nonhealing, *precancerous* changes in the oral cavity or on the face that should alert an individual to immediately seek professional care. Cessation of the use of tobacco, avoiding excessive exposure to the sun, and consuming alcohol in moderation *will help* return the tissues to normal. Finally, if the cancer is diagnosed in the early stages, *immediate treatment* is usually followed by a "clinical cure," provided there is not a return to the original habits.

The Profile of the Oral-Cancer Patient

The average oral cancer patient is over 40 years of age, with men afflicted more often than women. The most common intraoral cancer is a *squamous cell* cancer that might have been preceded by *leukoplakia.*[34] After a patient has been confronted with an often terrifying diagnosis of head or neck cancer, the scene becomes one of crisis. The patient will quickly undergo a series of examinations and tests by the various hospital services that will be involved in the treatment phase, including dentistry.[40] Following these examinations, the seriousness of the cancer is designated by a *TNM* classification system (staging) with scoring assigned in three areas of clinical assessment: *(T)*umor size, involvement of the lymph *(N)*odes draining the area and presence/extent of *(M)*etastasis. The greater the scoring numbers from 0 to 4 in each of the categories, the *more serious* the prognosis.

As a result of this whirl of activity, the patient is often left completely bewildered as to why teeth have suddenly become so important. Many of these individuals are in their fifth or sixth decade of life—some with a history of excellent oral hygiene, and some with a history of dental neglect. In addition to the impact of the diagnosis of

cancer or the specter of possible facial mutilation and suffering from the cancer treatment, the patient is confronted with a possible full-mouth extraction. The result is additional stress to the patient's self-esteem and morale. Then, too, there is the fear of a family economic disaster as a result of medical expenses and loss of income, as well as fear of death. At this time, it is important to be cognizant of the *psychological and physical strain* placed on patients by their diagnosis and projected treatment. As such, patients may not be receptive to care and their pain reaction threshold may be depressed. Education, patience, and a compassionate demeanor will help establish rapport and facilitate care for most patients. However, in some cases the fear, economic, and social pressures are too great to cope with; in these cases pharmacosedation should be considered. [26,41,42]

Treatment of Oral Cancer

Before treatment begins, all concerned departments comprising the Hospital Tumor Board convene to contribute of their expertise. Singh at the Eastman Dental Institute has pointed out that when the oncology team includes a dentist, the risk of development of serious complications, such as osteoradionecrosis (to be discussed later), are significantly reduced in the cancer patient. Other personnel such as family services workers and a chaplain may be included to contribute to issues that are not directly treatment oriented, yet important to a patient and family. In these meetings, the projected treatment details are discussed—chemotherapeutic agents to be used, the degree of anticipated immunosuppression, the total radiation dose, and the tissue fields (or ports) to be irradiated. Throughout the therapy, the progress of each patient will be evaluated—all with the object of achieving a better end result. Out of these meetings comes a consensus as to the best pretreatment, treatment and post-treatment regimens to be used for each patient.

The treatment for cancer of the oral cavity involves a choice of *surgery, radiation, chemotherapy,* or combinations thereof. Surgery is used to e*xcise* smaller cancerous lesions or to *debulk* large tumors (remove as much as possible of large volume cancers). As a co-therapeutic modality, *chemotherapy or radiotherapy* can be an option *following* surgery. The addition of radiotherapy adds two very disconcerting problems to the treatment regimen—*mucositis*[43] and *osteoradionecrosis (ORN).* Both are due to damage to the end arteries supplying the mucous membrane of the mouth or to the bone.

Chemotherapy and radiation produce their own complications; chemotherapy involves the use of plant alkaloids, and tumor antibiotics to *kill cells undergoing mitosis,* of which cancer cells are the most rapidly dividing. It is during mitosis that even a normal cell is most sensitive to chemotherapy.

The objective of using radiation has the same objective as chemotherapy—i.e., to *kill* rapidly growing cells. Unavoidably, in both cases other *normal* dividing host cells are also destroyed, such as *hemopoietic cells* (blood-forming cells) of the bone marrow, *epithelial* cells of the oral mucosa and gastrointestinal tract, and *endothelial* cells of terminal arteries are also killed. Host damage by radiation is usually *localized* to the field of irradiation, while the side effects of chemotherapy are *systemic.*

Figure 22-3 Intraoral squamous cell carcinoma. The patient was seen in a

Hospital Dentistry Department for a complaint of oral pain. His social history revealed tobacco and excessive alcohol use for many years. A biopsy by the general dentist revealed squamous cell carcinoma. Despite appropriate referral for treatment, the patient died within 6 months. (Courtesy of Dr. Jeffery L. Hicks, University of Texas Dental School, San Antonio, TX.)

Question 2

Which of the following statements, if any, are correct?

A. The xerostomia that often follows radiation therapy is permanent; that following the use of xerostomic drugs is usually confined to the period of drug use.

B. An ulcer that does not heal in two months should be carefully inspected and possibly biopsied by a professional.

C. The causes for most oral and orofacial cancers are self-inflicted—smoking, drinking, and tanning.

D. A large cancerous growth may be debulked by surgery prior to radiation therapy.

E. The damage from radiation therapy is local; the damage from chemotherapy is systemic.

Mucositis

The mucositis resulting from damage to the mucous membrane is characterized by a mouth that has a raw, ulcerated, and painful mucous membrane that often makes eating and swallowing *very* painful.[44] There is often a reluctance of a patient to eat following the onset of mucositis. Patients will selectively eat cool, soft, high-carbohydrate foods that promote caries development. Patients must be encouraged to take vitamin supplements and eat balanced and creative diets that minimize mucocitic pain, yet promote the desire to eat. Having the patient *consult a dietician* is salient to successfully manage this side effect.[45]

Since the effects of *chemotherapy* are systemic, there are several *systemic* complications. Aside from mucositis the complications include immune suppression (neutropenia—a low-neutrophile count), hemorrhage, and infection secondary to other foci of infection such as dental abscesses and periodontal infection. The use of chemotherapy, *does* suppress mitosis. However, it does *not* result in a marked *decrease in saliva* (xerostomia) production or in a prolonged mucositis such as seen following radiation therapy. Both the mucositis and any xerostomia tend to peak and to *regress within one or two weeks after treatment.*[46,47] Chemotherapy does *not* cause osteoradionecrosis (to be discussed later). However, chemotherapy is a significant source of morbidity and mortality. *Systemic infections resulting from the immunosuppression are responsible for 70% of deaths following chemotherapy.* Fortunately, mucositis occurs in only about 40% of the patients.

Osteoradionecrosis (ORN)

Many serious complications result from radiation therapy. These include a permanent xerostomia with an accompanying accelerated caries and periodontal disease development, altered smell, dysphonia (speech), malnutrition, trismus, and osteoradionecrosis. But by far, the most serious complication is the potentially disfiguring occurrence and treatment of *osteoradionecrosis.*

Osteoradionecrosis is caused by a destruction of the end arteries *to the bone that were in the radiation beam.* There is a severe *reduction* or *cessation* of osteocyte and osteoclast activity.[48] A rough guideline defines ORN as "an area of exposed necrotic bone that has persisted *three months without signs of healing*" (Figure 22-4). A more optimistic viewpoint of osteoradionecrosis is that only 10% of all cases treated with radiation develop the condition; a more pessimistic outlook is that the risk of ORN is perpetual and continues to increase with the passage of time because the blood supply to the affected area *never* improves. As the total absorbed dose of radiation to bone *increases,* so does the likelihood of ORN developing. Any subsequent surgical trauma to the area, i.e., dental extractions, increases that risk. Because of the density and reduced blood supply to the mandible, one study found that the mandible was affected more than the maxilla by a 95 to 5% margin.[49]

Since the consequences of surgical treatment of osteoradionecrosis are so debilitating, *prevention is the crucial therapy.* The *conservative* treatment consists of analgesics, antibiotics, irrigation and debridement (removal of necrotic tissue) and occasionally, hyperbaric oxygen therapy.[50-52] Hyperbaric oxygen therapy (HBO) increases tissue oxygenation of the tissues by administering 100% oxygen to the patient while in a pressurized chamber (2.4 atmospheres). Under these conditions, tissue oxygenation is increased, which in turn promotes the healing process by encouraging the formation of a connective tissue matrix and *capillary budding.* Unfortunately, HBO is not effective as a singular therapy—and it is time-consuming and expensive.

When the necrotic bone of osteoradionecrosis is extensive, aggressive surgical *excision* of the necrotic areas often becomes necessary—a surgery that can be physically *disfiguring and disabling.*

The initial signs and symptoms of ORN include constant, throbbing pain, along with a soft tissue breakdown over the necrotic bone. Later signs and symptoms include suppuration, a fetid oral odor, possible pathologic fractures and orocutaneous fistulae. Attention to subtle oral changes is required for an early diagnosis.

Dental Intervention, Cancer

If possible, all needed dental surgery for the newly diagnosed cancer patient should be accomplished *prior* to radiotherapy. All teeth with a questionable prognosis should be extracted, such as those with moderate-to-severe periodontal disease, extensive caries, impacted third molars and irreversible pulpitis. In making a decision to extract or retain teeth, consideration should be given to the past evidence of the patient having maintained a fastidious level of plaque control, overall prognosis, attitudes and expected compliance with written and verbal preventive dentistry instructions.

After exodontia there should be a 21-day waiting interval *before* radiation since the risk of developing ORN increases with a shorter interval elapsing between surgery

and radiation therapy.

Following radiation, all dental care should be *conservative,* emphasizing endodontic and other tooth retention measures instead of extraction. Unfortunately, regardless of the adequacy of self-care of the preserved teeth, the risk of ORN is *perpetual,* even though the risk exists only for the osseous tissue that was within the field of radiation. Recognizing that patient compliance with oral care instructions following radiotherapy is reported to be less than 50%, frequent recalls are essential.[53] Research to date has clearly shown that routine self-care hygiene measures combined with use of fluoride therapy plus regular dental monitoring, significantly reduces the incidence of postradiation caries and the progression of periodontal disease.[54]

Figure 22-4 Massive mandibular osteoradionecrosis secondary to heavy x-ray irradiation for epidermoid carcinoma in the jaw. (Courtesy of Dr. Robert P. Johnson, San Antonio, TX.)

Cleft Palate

Cleft palate is one of the most common of all birth defects, but very little is known of its cause.[55] Anatomically, it may involve either the right or left, or both the left and right sides of the embryonic incisal bone and lip, as well as the hard and soft palates. At the very minimum there might be evidence of only a slightly bifid uvula. Genetics has been believed to be an important factor in the development of cleft lip/cleft palate for the past 60 years, but no genes have been yet isolated.[56] Several environmental factors have been studied—cigarette smoking, alcohol consumption, organic solvents, and anticonvulsant drugs—with no conclusions.[57]

There is considerable interest that use of vitamin supplements during the first 4 months of pregnancy might have a protective effect.[58] Another study suggested that a folic acid deficiency may be responsible for different malformations through a common mechanism that interferes with embryonic development.[59]

In Europe, 1 out of *every 700 births* a cleft palate is present.[59] In the United States the Centers for Disease Control and Prevention have listed it as approximately 1 in 1,000 births with cleft palate alone, and 1 in 2,500 with cleft lip.[60] As soon as possible after the birth of the child with the cleft, the parents, the oral surgeon and a selected pediatrician must become involved in immediate decisions relating to the projected long series of primary, secondary and tertiary treatments that will be needed to provide the child with a life as near-normal as possible. Counseling should include a briefing with the parents about the future needs for surgery and rehabilitation procedures.

Surgical cleft-lip closure usually commences as early as age 3 *months,* and cleft palate repair at *1 year.* When speech begins, the advice and corrective actions of a qualified speech therapist is essential to modify the hypernasal speech of a cleft palate individual as well as to insure the intelligibility of the child's speech. The next major surgery is at approximately *9 to 10 years* of age, when bone grafting may be employed to restore the maxillary anterior alveolar ridge. After the healing of the anterior alveolar ridge surgery, there should be *orthodontic* corrections of the maligned teeth and jaws. *Prosthetic* appointments are needed to replace those teeth that are still missing, or to bridge the cleft for better eating, breathing, or speech.

During this first decade of continual monitoring, it is the responsibility of the pediatrician to assure that the child remains well nourished and healthy. For example, ear infections are a persistent problem for the child with cleft palate. On the other hand, the pediatric dentist is similarly charged with continuously monitoring risk for oral diseases, and taking the necessary preventive actions to abort onset of the plaque diseases.[61]

The cost of care of a cleft palate case is considerable and requires outside help.[59] In Pisa, Italy, for the 800 children born with clefting, the cost is 80 million Euros (1 Euros = +/- 1 dollar), or $100,000 per child. This sum is not exhorbitant, when it is considered that the following members make up the lifetime Cleft Plate Team responsible for the care of each cleft palate birth:

- An audiologist (to assess hearing)
- A surgeon, usually a plastic surgeon, or an oral/maxillary surgeon, or a head-and-neck surgeon, or a craniofacial surgeon, or a neurosurgeon, (to perform corrective surgery)
- A pediatric or general dentist, (to prevent and treat dental problems)
- A dental hygienist, (to provide professional dental preventive care)
- A prosthodontist, (to provide specialized prostheses)
- An orthodontist (to align the remaining teeth prior to prosthetics)
- A geneticist, (to screen patients for craniofacial syndromes and consult parents about the risk of having additional children with clefts)
- An otolaryngologist, (to treat ear, nose, and throat problems)
- A pediatrician, (to monitor overall health and development)
- A psychologist or other mental health specialist, (to support the family and assess any adjustment problems)
- A speech-language pathologist (to assess and correct speech and breathing when swallowing)
- A school counselor (to aid in child's integration into school and educational programs.)
- A social services expert (to aid the family in securing financial help from government, services and fraternal/charitable organizations)

Question 3

Which of the following statements, if any, are correct?

A. A child born with a cleft of the lip and palate should have both surgically corrected about 3 months after birth.

B. There are more clefts of the lip than of the hard palate.

C. Xerostomia is one of the symptoms of radiation damage to the salivary glands.

D. The identification of a pre- or early cancerous lesion is mainly the task for general practitioners.

E. Two major problem sequellae of radiation therapy are osteoradionecrosis and

mucositis.

Aids

Acquired immune deficiency syndrome (AIDS) is a symptomatic infectious disease in which lymphocytes; specifically *T helper lymphocytes (CD4 cells*) are invaded and impaired by a retrovirus—the human immunodeficiency virus (HIV). The subsequent *gradual decline* in the number of protective CD4-lymphocyte cell population results in an *immunosuppression* (loss of effectiveness of the body immune defenses) that places the host at risk for *opportunistic infections and cancers.* Another commonly referred to lymphocyte is the *T-suppressor lymphocyte (CD8).* The two lymphocyte cell populations *may* act in suppressing HIV infection *until they can no longer contain the virus.*

The AIDS virus is transmitted by blood and sexual contacts and the principal contributing behaviors are male-to-male sexual activity and parenteral drug abuse. Initially, the disease was confined mainly to those participating in these activities; however, the disease incidence is now increasing in the heterosexual population engaging in unprotected sex with females at greater risk of infection than males.[62] HIV/AIDS was projected to infect between 5 and 6 million persons worldwide by the year 2000.[62] Patients infected with the virus are termed HIV-positive (HIV^+). Following initial infection, HIV patients may remain asymptomatic for years during viral dormancy. Nearly all patients *eventually* develop symptoms that classically consist of opportunistic infections such as pneumocystitis carinii pneumonia,[a] Candida esophagitis, toxoplasmosis, mycobacterial infections and cytomegalovirus.[63,64] Patients are considered to have progressed from HIV^+ (Human Immunodeficiency Syndrome) to AIDS when the CD4 lymphocyte cell count drops below 500 cells per milliliter of blood.[b] Crossing this threshold results in patients being at increased risk of opportunistic infections.

AIDS patients are at risk for developing certain cancers and the development of these cancers signal a relatively *poor prognosis for survival.* The most common cancer-like disease developed by patients as a result of HIV infection is *Kaposi's sarcoma* (Figure 22-5).[64] Patient mortality attributable to AIDS is *nearly 100%* and is most often the result of an *opportunistic infection.*[63] Longevity has been increased by the use of *Highly Active Anti-Retroviral Therapy,* abbreviated HAART. HAART is a treatment where a combination of anti-HIV drugs—usually three—are administered even before the symptoms of AIDS develop.

Some of the opportunistic infections and cancers affecting AIDS patients are manifested orally, allowing *dental practitioners* to play a major role in diagnosis, patient monitoring, and management. The most common oral infection is *candidiasis—a yeast infection,* which afflicts 75% of all AIDS patients at least once in their disease course. The appearance of *hairy leukoplakia,* is another manifestation of AIDS. It is a corrugated, white, raised lesion located on the tongue.[65] The leukoplakia *cannot* be wiped off. The presence of this lesion appears to be an accurate *predictor* of progression from being HIV^+ to AIDS. A*pproximately 83%* of patients who develop hairy leukoplakia are diagnosed with AIDS *within 30 months.* This benign lesion does not require treatment and can usually be diagnosed based on clinical appearance without the need for biopsy.[65] *Aphthous ulcers* are also common

in AIDS patients ranging from small, discrete lesions to large denuded areas. Kaposi's sarcoma appears as a flat or raised, purple to brown lesion and occurs orally in *50%* of all AIDS patients. The lesions are most commonly seen on the *hard palate* and when indicated, biopsy is suggested for definitive diagnosis. Treatment of these lesions is needed only when they interfere with patient function.[64] Oral Non-Hodgkin's Lymphoma (NHL) is another cancer which is increasingly seen in patients with AIDS. NHL occurs as a soft tissue mass, commonly on the palate or gingiva and may exhibit ulcerations. The occurrence of NHL signals a relatively *poor prognosis*. HIV^+ dental patients who develop oral candidiasis, hairy leukoplakia, Kaposi's sarcoma, or NHL should be instructed to notify their physician. Development of these complications may indicate a need for a change of medical treatment.

Acute periodontal infections are common in AIDS patients. HIV gingivitis appears as an erythematous band of inflammation on the marginal gingiva that will *not* improve with oral-hygiene measures. HIV periodontitis is a rapidly progressive infection characterized by soft tissue ulceration and necrosis, and loss of periodontal attachment. Clinically, the disease mimics *acute necrotizing ulcerative gingivitis* (ANUG). Present treatment recommendations include aggressive scaling and root planing combined with chlorhexidine therapy and antibiotics. *Early* treatment of the HIV gingivitis may prevent the development of HIV periodontitis.[66]

The immuno-compromised status of these patients requires the same aggressive *preventive* approach to caries and periodontal disease suggested for cancer patients. Potential sources of bacteremia should be definitively treated to prevent the development of an oral infection that threatens the patient's systemic health.[67] Xerostomia is a common finding in HIV^+ patients. The decreased saliva production may be caused by HIV-associated gland disease or as a side effect of HIV medications.

[a]There is no need to memorize the names of these diseases. That will come in Pathology. Just remember that an HIV patient becomes more susceptible to opportunistic diseases as the disease progresses from HIV to AIDS.

[b]The normal CD4 count ranges from about 600 to 1600.

Perspectives, Responsibilities, Patient's and Dentist's Rights

The AIDS epidemic that came to the forefront in the 1980's has been a very serious disease characterized by a very high death rate. In the field of dentistry, all diagnostic or treatment procedures for HIV/AIDS individuals *is fraught with some degree of danger for the dental team.*[68]

The report of several years ago that a Florida dentist had transmitted the AIDS virus to six persons elevated the infection-control procedures of dentistry to public debate.[69,70] Much of the controversy was probably due to media reporting that favored sensation over rational discourse.[71] However, the debate brought to the forefront the *moral* and *legal* responsibilities of the *dentist to the patient*, and *vice versa*, the responsibilities of the HIV/AIDS patient to the dental team. Anti-discrimination laws restricted the ability of the practitioner to control admittance of new patients, while the Occupational Safety and Health Administration (OSHA) has forced an upgrade in

the infection-control requirements of the dental office.[72]

There has been worldwide concern of the public that going to the dentist is beset with the possibility of transmission of the virus caused by incomplete sterilization of instruments. This concern has been exaggerated, but is not entirely baseless. In Hong Kong, teachers and secondary-school students were polled to determine risk perceptions. Approximately one-half of each group was concerned about contracting HIV infection during dental visits, while 65% of the students and 57% of the teachers believed that a dentist did not have sufficient knowledge to identify AIDS patients.[73]

A mail questionnaire study was completed in Denmark to determine the time, steam and temperature settings used by Danish dentists to sterilize instruments. At the end of the study, it was concluded that 3.4% of the autoclaves had not operated properly.[74]

With the outbreak of a worldwide epidemic, dentists began to use additional disease barriers to protect themselves and their patients—operating uniforms, glasses, masks, and gloves. Yet there appears to be differences among the specialties in use of protective devices. For instance, general dentists outperform orthodontists in use of gloves (92 to 85%), masks (75 to 38%), eyewear (84 to 60%), gloves (92 to 85%) and heat sterilization of handpieces (84 to 57%). Additional precautions were increased in both groups when the patients were *known* to have HIV/AIDS.[75]

It is not known how many patients disclose their HIV status in the patient history form—one study set the figure at 70%. With this 30% uncertainty, dentists must provide maximum recommended barrier techniques and sterilization procedures on the assumption that any patient could be HIV infected.[76]

One of the omnipresent hazards of dentistry is living with the possibility of *needle-stick* and/or *cut* injuries. The actual injury pales in concern to the "terror factor," real or imagined, of contracting a potentially lethal injury.[77] This psychological effect is important considering that 60% of the dentists in a Denmark study reported having at least one needle-stick or cutting injury during the previous year.[74]

There is a *bias* by many dentists for caring for HIV/AIDS patients.[78] At times the bias has been caused by the concern of losing non-HIV^+ patients who discover that HIV/AIDS patients are being treated in a practice. At other times it is caused by sociocultural biases. In a study in Japan, 71% of the dentists surveyed felt as though they had a *moral responsibility* to treat HIV/AIDS patients, but only 16% were *willing* to treat HIV/AIDS patients.[79] In several studies it was believed that additional education is needed to improve knowledge and attitudes relative to the treatment of HIV/AIDS patients.[80] In a survey of dentists in Lothian, Ireland where care is mandated, the dentists considered that their professional background allowed them to cope with disease transmission problems in treating AIDS patients.[81]

As a final note for those worried about contracting the disease from dental professionals, |it is worthwhile to quote from an abstract from a paper by Robinson and de Blienk: "The risk of acquiring human immunodeficiency virus (HIV) infection from a health-care worker is 2,000 times less than that of dying from a car accident. It is 700 times less probable than perishing from being struck by lightning or suffering a fatal fall. Despite the rarity of the risk of health-care-worker-to-patient, the

transmission of the disease in the workplace has been the focus of investigation by congressional, federal, state, and local agencies. If all HIV transmission from health care workers to patients were prevented using current guidelines and legislation, the epidemic of AIDS would be reduced by 0.0006%."[82]

Figure 22-5 This HIV^+ patient is exhibiting a Kaposi's Sarcoma on the hard palate. This lesion was treated with radiation to prevent its enlargement. (Courtesy of Dr. Jeffery L. Hicks, University of Texas Dental School, San Antonio, TX.)

The Patient with Cardiovascular Disease

Cardiovascular disease (CVD) is a term that embraces a *varied* array of cardiac pathoses. Generally, CVD patients can be subdivided into (1) ischemic heart disease (IHD—poor blood supply to heart muscle), (2) myocardial infarction (MI—scarred heart muscle from previous heart attack), (3) hypertension, (4) valvular and congenital heart disease, (5) dysrhythmias, (irregular and erratic pulse) and (6) congestive heart failure (CHF-failing heart muscle).

The following three sections on cardiovascular disease, renal disease, and diabetes mellitus is *not* meant to provide a detailed account of the drugs and detailed treatment modalities used in their treatment. Instead, it is another series of snapshots of what happens in different departments of a hospital—and are applicable for use in a private practice.

Each year, more than 350,000 adult Americans die each year of sudden cardiac arrest. The fatal event is unpredictable and can occur in patients with no history of cardiac disease or cardiac symptoms.[83] CVD is the *leading cause* of death in the United States and is responsible for twice as many deaths as cancer. Irrespective of the underlying cardiovascular problem, *all dental practices* should be prepared in case that a fatal events threatens or occurs in *your office, and to one of your patients.* Waters and others have laid out a path that should help prepare for such a catastrophe. The preliminary precautions begin on the initial contact with the patient. At this time an assessment is made of the *stress tolerance* of the patient. Such a tentative assessment is made following an interview that expands on the information contained in the patient's medical and dental history. Such details include the prospective patient's narration of health status, drugs being taken, any past heart attacks etc., followed by a possible consultation with the patient's cardiologist. For dental legal record purposes, an informed consent form should be completed outlining the expected treatment, and the expected compliance by the patient.[84]

Preparing for an Office Emergency

The dentist should maintain a drug cabinet stocked with current fresh drugs. All office personnel should be included in an emergency plan—one that establishes who is to call EMS if necessary and who would secure the emergency drug(s). It is assumed that the dentist would accomplish the initial cardiopulmonary rescusitation, if necessary. A maneuver involving the office team should be rehersed at intervals to ensure an efficient operation if it is ever necessary to initiate an actual rescue plan.

The Typical Appoinment

All appointments should be *short*—less than an hour if possible—and preferably in the morning.

The patient's chart is reviewed to refresh the memory about details of the patient's medical and dental background.

The treatment phase should be preceded with the taking of the patient's blood pressure. If it exceeds the systolic and diastolic limits suggested by the cardiologist, the appointment should be terminated until the blood pressure is within the established limits. If the treatment is of emergency nature (extraction), consultation with the cardiologist is recommended.

Effective stress management may include preoperative sedation with the decision being dependent upon the *expected stress tolerance* inherent in the proposed operation. *Stress tolerance is a gauge of the heart's ability to sustain additional stress.* Patients with poor stress tolerance (and high risk) include those with severe hypertension, uncontrolled cardiac heart failure, severe dysrhythmias (tachycardia, abnormally fast heart beat and bradycardia, abnormally slow heart beat), or hemodynamic instability (erratic blood pressure), unstable angina (new onset of chest pains, pain at rest, pain that is poorly controlled with medication, or pain that has recently changed character). Severe hypertension is characterized by a diastolic pressure greater than 115 mm of mercury, and a systolic blood pressure greater than 200 mm. There are an estimated 58 million cases of hypertension in the United States, with fewer than 5% having a curable cause. Many do not know they have high blood pressure, or its consequences, (usually heart attack or stroke) hence the term, "*the silent killer*."

At the end of the appointment, if the patient has been sedated, she/he should not be allowed to depart the office unless accompanied by a care giver who can help the patient in activities such as driving or crossing streets.

On the Day of the Emergency

Everything is going along fine, until suddenly the patient complains of a chest pain. The dentist immediately and correctly ceases the dental treatment. It is now up to him/her to immediately as*sess* the problem and *react* to the emergency. What is to be done? Not all chest pains are a serious heart attack.[85] Not all hyperventilation heralds a heart attack. The drugs in the emergency cabinet may be sufficient; however, if the dentist's diagnosis is that the pain is considered more serious, the EMS should be immediately contacted by the office personnel. This is a time that the dentist stands alone and must demonstrate decisive leadership as planned in past maneuvers. In a worst case scenario where the heart has stopped, (cardiac failure with unconsciousness) cardiopulmonary resuscitation (CPR) is absolutely necessary. It must be initiated immediately, with the stark reality that if the heart is not restarted in about 4 minutes, cerebral hypoxia jeopardizes life continuance.[86] Adding to the crisis is the fact that the chance of success towards getting the heart restarted is reduced by 10% for every minute that passes. There is not sufficient time to wait for the arrival of the EMS to apply advance resuscitation possibilities, such as the use of the automated external defibrillator. With its use, an electrical shock to the heart muscle offers a

much better chance to restart heart action. Although dental offices do not usually have a need for a defibrillator over the lifetime of the practice, its early use instead of CPR, can mean the difference between life-or-death. Passenger planes carry defibrillators for passenger emergencies. They are usually available in large gathering areas such as sports arenas and industrial clinics.[83]

Critique

There are several questions that can be asked. The most critical would question whether the dentist has the current level of training needed to correctly assess the severity of the case. A second question would be, "Should all dental offices stock defibrilators despite the fact that very few dentists experience a life-or-death situation during their entire professional careers." The third question would be, "If in the estimation of the cardiologist the victim is considered to be a very high risk patient, why was she/he not referred to a hospital with a dental clinic?" In such a facility, monitoring during the treatment is routine, all emergency drugs are available, and a crisis team is available within minutes of being summoned for any emergency.[87]

Education Needs of the Dentist

In the United Kingdom, in 1999, senior officers in oral and maxillofacial surgery expressed dissatisfaction in their training in resuscitation.[88] These were professionals with graduate training, working with daily high risk operations. Yet, our above dentist was confronted with the same kind of critical decisions learned in a lecture hall some ten- or- fifteen years ago—or more—in dental school. He/she had the leadership, but not the academic background or experience to make the expeditious and informed decisions that were necessary. In view of the rarety of a life-or-death event in any dental office, there is a need for an intensive refresher course, much as given by the airlines to selected crew members to provide defibrilator resuscitation of airline passengers suffering a possible heart attack. Included in such a course should be a refresher session on CPR, mainly because "All healthcare professionals are expected to be competent in cardiopulmonary rescusitation."[88]

A Final Topic—Antibiotic Prophylaxis

Once there is an invasive procedure involving blood and possible blood borne bacteria, there is the basis for a debate as to whether antibiotics should be administered to at risk patients (prophylactic antibiotic coverage). This category of patients includes immunocompromised patients and end-stage organ disease (kidney, liver), patients with certain cardiovascular manifestations (heart murmur, prosthetic valves) and patients with prosthetic joint replacements. The purpose of the antibiotic coverage is meritorious i.e., to get the antibiotic into the blood stream to eliminate the microorganisms before they reach the target organ; and secondly to get the antibotic into the target organ to eliminate any invading organisms. Acting on this common sense belief, antibiotics were used before all invasive procedures to prevent infection. However, with the extensive use of antibiotics, it was soon noted that several strains of bacteria began to appear that were resistant to antibiotics. It was not long before the American Heart Association drew up some guidelines (Table 22-1 and 22-2) listing specific conditions for which antibiotics should definitely be used, with other conditions left to the judgement of the clinician. There are minor disagreements with

these guidelines, but from a practical viewpoint (the medicolegal viewpoint) there appears to be a consensus to provide prophylaxic antibiotic coverage to at risk patients undergoing *oral surgery, periodontal treatment, and implant placement.*

Question 4

Which of the following statements if any, are correct?

A. Patients are considered to have deteriorated from HIV to AIDS status when the laboratory count of the CD4 lymphocyte has decreased to less than 500 cells per milliliter.

B. As a safety precaution, barrier techniques (masks, glasses, rubber gloves, etc.) should be used while treating all patients on the common sense assumption that all patients are HIV/AIDS infected.

C. One of the best indicators of patient risk, is the patient's medical history.

D. Antibiotic prophylaxis is required for all cardiovascular patients.

E. A defibrillator is more effective than CPR in restarting the heart after heart failure.

Renal Disease

Renal disease affects three percent of the United States population, of which 122,000 individuals require routine renal dialysis and approximately 40,000 have been recipients of kidney transplants. Renal disease is divided into *acute* (ARF) and *chronic renal failure* (CRF), the causes of which could include hypertension, drug reactions, renal obstruction and diabetes. Because ARF is an acute medical emergency, patients with this problem are *not* commonly encountered by dental practitioners and dental hygienists in a private practice setting. Chronic renal failure—also called *end-stage renal failure (ESRD),* is usually the result of a progressive loss of renal function that *gradually* destroys the nephrons, (which accomplish the vital glomerular filtration) and eventually causes irreversible kidney damage. Few symptoms are manifest until up to 75 percent of glomerular filtration capability is lost. This loss of efficiency of blood filtration and increased build-up of waste in the vascular system affects *every organ* in the body.[89]

The two treatments for CRF are *dialysis* and *kidney transplantation.* One or the other is required or death ensues. The removal of wastes can be accomplished by *hemodialysis* and *periotoneal dialysis.* Dialysis is a palliative therapy that maintains life over a long and often demoralizing time as a patient waits till he/she reaches the top of a list to receive a donor kidney.

*Hemo*dialysis involves a requirement to surgically create a *shunt* which serves as a permanent access to the arterial and venous vascular systems. The usual sites of placement of the arterio/venous shunt is in the *forearm or upper arm.* By connecting the hemodialysis machine to this access, the patient's blood can be circulated through a dialyzer unit that acts as an artificial kidney and extracts the wastes from the patient's blood (Figure 22-6). *Nine to 12 hours* of hemodialysis is required *each week,*

usually divided into three sessions on alternating days (M-W-F or T-Th-S).

Continuous ambulatory peritoneal dialysis is a viable option for patients with poor vascular accesses for hemodialysis, or who prefer this method of dialysis. In continuous ambulatory peritoneal dialysis, dialysate is placed into the abdomen through a surgically placed permanent catheter and then drained after 4 to 6 hours. New dialysate is introduced after the old is drained, so that dialysis can be continued—if desired, throughout the day and night—hence the term *continuous.* It also permits the patient to be *ambulatory* during the dialysis period that can be accomplished at home with less risk of infection.[90] It is estimated that approximately one third of the Canadian chronic dialysis patients are on peritoneal dialysis.[91]

In contrast to dialysis, *kidney transplants* are the only means to normalize kidney function. They also provide a much better opportunity for survival than long term hemodialysis.[92] This emphasizes the need for early transplantation once the diagnosis of ESRD is made.[93] The problem is that the number of individuals needing dialysis is growing by 10% to 15% per year—and there just are not enough donors.[94]

The donor source for renal transplants can be either from an immediately deceased corpse (cadaveric transplant), or from a living donor (allogenic transplant). Allogenic transplants are much more successful. Once a successful kidney transplant is in place, a *life-long* immunosuppression drug regimen is *required* to minimize the possibility of organ rejection.

Figure 22-6 Patient and Hemodialysis Machine. An End Stage Renal Disease Patient is receiving dialysis treatment while connected to a dialysis machine. The patient is connected to the machine via tubing placed into a shunt in the patient's right arm. (Courtesy of Dr. Jeffery L. Hicks, University of Texas Dental School, San Antonio, TX.)

Salient Systemic Signs and Symptoms of ESRD Anemia

Dorland's medical dictionary defines anemia as "A reduction below normal in the number of erythrocytes per cubic mm., the quantity of hemoglobin, or the volumn of the packed cells per 100 ml. of blood." This definition sounds innocuous; however, one research group suggests a more ominous connotation applicable to the anemia of a dialysis patients, viz., "We suggest there is a triangular relationship, a vicious circle between chronic heart failure, chronic kidney insufficiency, and anemia where each of these three can both cause and be caused by the other."[95]

There are several important contributing factors to the development of an anemia of a hemodialysis patient: (1) Many *red cells are destroyed* by the hemodialysis unit; (2) there is a deficiency of *erythropoietin* (a protein that stimulates red cell production) and results in a *reduced output* of erythrocytes); and, (3) a lack of erythopoietin needed for normal formation of *hemoglobin.* A chronic or severe hemoglobin deficiency is highlighted by the results of a study of a group of hemodialysis patients, where "The relative risk of death and hospitaliza- tion are *inversely* associated with hemoglobin levels."[96]

The loss of erythrocytes is increased by *bleeding.* This is a platelet problem since they are essential in the clotting process. Platelets can be destroyed during hemodialysis;

their function to aggregate (clot) can be negated by the high blood urea content, or clotting can be prevented by the residual presence of heparin that is used during dialysis.

Renal Osteodystrophy

Renal osteodystrophy[c] is a disorder of bone seen in the endstage of renal disease. Vitamin D is normally manufactured in the kidney, a source that is lacking in advanced kidney disease. In its absence there is a deficiency of calcium absorbed from the gut for essential body functions. To compensate, the parathyroid glands secrete a hormone that causes a withdrawal of calcium from bone. However, this secondry hyerparathyroidism results in any remodeled bone being dystrophic and often subject to spontaneous fracture. Renal osteodystrophy disease begins relatively early in the development of chronic renal failure.[97]

It is a challenge to the nephrologist to adjust the medications to simultaneously control the anemia, the deficient mineral metabolism of the ESRD patient, and other emergency medical needs as they arise. The *lack* of success is highlighted by the experienced mortality rates in patients with ESRD that are 10-100 times those without ESRD.[98]

[c]Dystrophy = not normal; osteodystropy = dystrophy of bone; renal osteodystrophy = bone dystrophy of renal origin.

Hypertension

High blood pressure is considered a significant risk factor for the development of ESRD. Also, high blood pressure has been looked at as a possible predictor of mortality for hemodialysis patients.[99] Closely parallelling this risk factor is another of interest from a dental viewpoint, namely that uric acid might have a pathogenic role in the development of hypertension, vascular disease, and renal disease.[100] A few decades ago, American dentistry joined in the campaign against high blood pressure by taking the blood pressure of all patients before providing dental care. Unfortunately, only an estimated 5% of those who are informed of their personal high blood pressure take active steps to reduce it as an *important* health hazard.[101]

Infection

Infection is a constant companion of the ESRD patient. The shunt that is absolutely necessary for hemodialysis is vulnerable to infection from both within (blood borne bacteria) and from without (bacteria around the shunt). As much as 50% of hospitalization costs for ESRD is related to access problems.[102] The infections that occur are not a matter of just morbidity, but also of possible mortality. The solution is to use antibiotics to cope with blood borne pathogens, and exquisite cleanliness and hygiene around the shunt area. A clinician should make every effort to avoid traumatizing or infecting this vital arterio/venous access structure. For instance, the blood pressure sphygnomometer cuff should *never* be placed on this arm.

Infection of dialysis patients with hepatitis B and C viruses ranges from 20-30%. The probable source of the infection is probably from contaminated equipment and

nosocomial transmission.[103] Because of the risk of transmission of the viruses, a consultation with the patient's primary care physician is recommended to determine the patient's infectious status before beginning dental care. However, *with appropriate vaccines and universal infection control measures, the risk to health care workers is minimal.*

Antibiotic Prophylaxis

Antibiotic prophylaxis prior to any dental treatment for a hemodialysis patient that causes bleeding (calculus removal, scaling, and planing) *may be* recommended to avoid infection of the shunt by pathogenic blood borne bacteria. The decision concerning antibiotic prophylaxis should be *jointly made* between the dental practitioner and the patient's nephrologist. However, antibiotic prophylaxis to prevent endocarditis in the dialysis patient is no longer *specifically recommended* by the American Heart Association, thus leaving the decision up to the judgment of the practitioner and/or the nephrologist.

Oral Manifestations of ESRD

There are several signs and symptoms of ESRD that are both of interest and important to the dentist and the dental hygienist. Most are due to the *high level of urea in the saliva.* (Under normal circumstances, the urea is filtered out in the urine by the healthy kidney). The changes in homeostasis that are attributable to urea are: (1) taste changes and breath malodor (halitosis) due to the urea in the saliva breaking down to ammonia; (2) a higher saliva pH and buffering capacity, also due to the high alkaline urea. Additional signs and symptoms of ESRD include: a lower flow of stimulated and unstimulated saliva, due to the excess loss of fluid through the diseased kidney; bone dystrophies seen in x-rays, such as a missing lamina dura around the roots of the teeth and abnormal trabeculae (the framework of bone) that can be traced to the secondary hyperparathyroidism; pallor of the oral mucous membrane, with ecchymosis and petechia[d] that are all caused by the anemia.

[d]Ecchymosis = Area bleeding beneath the mucous membrane or skin; Petechia = Pinpoint bleeding beneath the mucous membrane or skin.

Chronic Renal Failure and the Plaque Diseases

There is a consensus that there is a lower prevalence of caries in a ESRD population. Nunn et al found the prevalence of caries to be low in the ESRD patient.[104] It is believed that the lower caries prevalence that is characteristic of patients with ESRD, is due to the higher concentration of salivary (alkaline) urea nitrogen. Obry found the urea level in salivary analysis to be 513 +/- 210 mg/100ml *prior to* patient dialysis. *Following* dialysis, the saliva level dropped to 241 +/- 82 mg/100 ml. These figures are in contrast to 110 +/- 48 mg./100 ml. for the control group.[105]

It should be noted that in one study after the plaque was exposed to carbohydrate the configuration of the Stephan curve for both ESRD patients and the control group paralleled each other, but because the pH of the ESRD group being initially elevated, the drop did not as often reach the critical level required for demineralization.[106]

Miscellaneous Factors Relating to the Care of ESRD Patient

Any routine dental appointment for a chronic renal failure patient should be scheduled for the day before, or the day after hemodialysis. This policy avoids the possibility of the patient coming to the dental office from one stressful appointment, to another equally or more stressful experience.

If *onset* of the renal failure begins *before* the development of the teeth is completed, there is the possibility of enamel *hypoplasia* and *intrinsic staining* caused by the high blood levels to urea and relative calcium deficiency.[104,107] These abnormalities probably date from the time of the onset of the ESRD.

The *intrinsic stain* of any teeth caused by uremia cannot be removed with scaling or prophylaxis (the same as for intrinsic stains of fluoride and tetracycline). This is because during the formation of the teeth, the stain completely permeates throughout the enamel.

If antibiotic coverage is *desired* in order to prevent infection of the shunt or cause further kidney damage, then the American Heart Association' recommendations (Table 22-1) should be followed. A simple way of achieving antibiotic coverage is to have the nephrologist administer intravenous vancomycin (an antibiotic) at the time of dialysis. For dialysis patients, the duration of coverage of vancomycin is approximately 5-to-7 days, giving the dental practitioner ample time to perform any needed dentistry.[108]

Post Transplantation Dental Concerns

A successful transplant helps normalize daily activity. However, the patient is exchanging one problem for another. Infections are emerging as causes of morbidity and mortality because of the lifelong requirement for daily immunosuppressive drug therapy to prevent rejection of the kidney transplants.[109] Following the kidney transplant, *all* dental treatment expected to *cause bleeding* should be preceded by antibiotic prophylaxis (according to the American Heart Association guidelines).

Another problem generated by the immunosuppressant drugs is *hyperplasia* of the gingiva (Figure 22-7). There is a 30 percent incidence of gingival hypertrophy for patients taking the suppressive drug, cyclosporin A. This side effect can be minimized with scrupulous oral hygiene; however, if necessary, a gingivectomy or gingival flap is often needed.[110,111]

Of interest, in one study the data suggested that following a successful transplant, with the restoration of normal kidney function, there was again an increased risk of caries.[106]

There are very few studies that point out any specific periodontal problems in transplant patients not encountered by normal individuals. Once the diagnosis of ESRD is made, it should be the responsibility of the nephrologist to coordinate the patient's hemodialysis schedule and medical care. This schedule should also include provisions for an appropriate evidenced-based dental recall schedule to maintain as healthy periodontium as possible. (Chapter 23).

This need is well pointed out by Naugle et al. where 100% of ESRD patients on dialysis had some form of periodontal disease. Sixty four percent displayed a severe gingivitis (28%) or early periodontitis (36%). Naugle also pointed out that these two periodontal conditions represent a bacterial foci that can contribute to a blood infection that can increase a patient's risk to morbidity or mortality.[112]

As a final note, not all successful transplants end happily. There are reports of transplants bringing with them problems that were part of the donor's legacy. This possibility is covered in a self-explanatory article entitled, "Risk for tumor and other disease transmission by transplantation: a population-based study of unrecognized malignancies and other diseases in organ donors."[113]

Figure 22-7 Several immunosuppressive drugs taken to prevent rejection of an organ transplant, and some taken to suppress seizures, produce the same gingival overgrowth. Surgery is indicted to remove the excess tissue, and patients must exercise excellent oral hygiene to prevent or delay regrowth. (Courtesy of Dr. Jeffery L. Hicks, University of Texas Dental School, San Antonio, TX.)

Diabetes Mellitus

Diabetes mellitus (DM) is a *common endocrine* disorder. It is estimated that approximately *16 million* Americans are afflicted with the disease—a number that is projected to *double* by 2010. Twenty percent of U.S. citizens over 60 have DM.[114] An estimated 5.4 million of the 16 million do not realize they have the disease. This is unfortunate, since early and continued treatment helps prevent some of the disastrous consequences of DM. These consequences can range from blindness, to amputations of limbs, periodontal disease, renal failure, hypertension, neuropathy, cardiovascular disease and a *great* reduction in the quality of life. African Americans, Hispanics and American Indians are especially susceptible to diabetes.[115]

Diabetes mellitus is caused by an insufficient supply of insulin because of either a lack of production by the *islet cells of the pancreas,* a deficit of insulin receptors, or an error in insulin metabolism (insulin resistance). Insulin is the key that allows the blood glucose to *enter* the body cells to *provide for energy needs.* Without it, the body cells are literally starving for the energy-giving glucose, while just outside in the blood supply the needed glucose continues to build up to toxic levels in the blood and spill over into the urine.

There are two main sub classifications of DM, Type 1, and Type 2 (both with Arabic numerals, not Roman numerals). Type 1 is often the result of genetic omission, or as a result of an autoimmune destruction of the pancreatic beta cells *early in life.* By the time the disease is identified up to 80% of the beta cells have usually been destroyed. Approximately 5-10% of the cases of diabetes are in this category.

For glycemic control of Type 1, *exogenous insulin is absolutely necessary to maintain life.* This insulin is self-injected by the patient throughout life, or is automatically dispensed by a strapped-on insulin pump (Figure 22-8). The pump automatically senses a changing level of glucose in the blood, and adjusts the dose accordingly.

Type 2 usually develops *later in life* and is often associated with *overweight* and relatively *inactive individuals.* Approximately 90-95% of the cases fall into this category. Often Type 2 DM can be controlled by a combination of exercise, diet and/or oral hypoglycemic agents.[116] Both types can be recognized by the presence of three clinical signs: *polyuria* (frequent urination), *polydipsia* (frequent urge to drink), and *polyphagia* (frequent urge to eat). A positive diagnosis of DM usually follows a physician's clinical examination that is verified in routine blood tests as an excess of glucose, or in urine tests with the presence of glucose. Type 1 is increasing *slowly* in numbers. On the other hand, the number of Type 2 diabetics is escalating *rapidly* and is expected to *double* before 2010.[114] Many in the news media and public health call it an *epidemic.*

The Relationship of Diabetes Mellitus to the CPITN

If there is a relationship between the level of blood serum glucose and periodontitis, then a high glucose level for an individual should have a significant parallel high CPITN score. There are two studies that verify this assumption.

A large-scale study involving 10,590 subjects in Israel charted abnormal blood glucose (levels over 120 ml/dl) with elevated CPITN scores of above 4.5.[117] In the second smaller study of 40 subjects—20 with diabetes, and 20 control subjects. It was found that there was a *steady increase* in blood serum glucose (142-173 mg/dl) that paralleled that of an increasing CPITN score (13.5-19.1).[118]

Periodontal Disease and Diabetes Mellitus—Bi-Directional Diseases

In reviewing the literature, there is a consensus that periodontal disease has an adverse effect on the severity of DM, and vice versa that the severity of DM has an adverse effect on the severity of periodontal disease—a *bi-directional* relationship (Figure 22-9). The bi-directional etiology signals the need for cooperation between the medical and dental professions, as echoed in the following statements.[119]

Statement: *Poorly controlled diabetics* have a *greater incidence* of severe periodontal disease compared with those patients who are well controlled or have no diabetes mellitus.[120]

Statement: Periodontitis is a common problem in patients with diabetes. The relationsip between these 2 maladies appears bi-directional—insofar that the presence of one condition tends to promote the other, and that meticulous management of either may assists treatment of the other.[121]

Statement: New evidence suggests that advanced *periodontal disease may interfere with diabetes mellitus control* and the physician should be made aware of the patient's periodontal status.[122]

Statement: Not only does diabetes affect the periodontium, but periodontal infection can adversely impact glycemic control in diabetics.[123]

Statement: Equally important is the fact that there are *no* studies of acceptable design that refute this bi-directional relationship between periodontal disease and DM.[124]

In 1999 and again in 2000, the American Academy of Periodontology issued position papers about the relationship of diabetes and periodontal diseases.[125,126] These two position papers point out that there *is* a relationship between the two diseases and that all patients should be informed of that relationship, especially where periodontal disease might increase the risk of DM complications and vice versa.

Caries

Theoretically, there is a basis for *either* a decreased or increased caries prevalence for Type 1 diabetics. Normally, if there is a good self-care (with an effective mechanical and chemical plaque control regimen), there is a lower count of cariogenic organisms and a lower DMFS. If there is a diet with minimum carbohydrate there is a lower caries incidence. Another moderator is the flow of saliva, with an inverse relationship between flow rate and caries development. A look at several studies is necessary to determine what factor(s) are *most determinant* for the caries.

Twetman et al. after a three year study with adolescents of 8-15 years of age, concluded that the *main most influencial determinants for high caries development over the period of the study were metabolic control,* poor oral hygiene, previous caries experience and high levels of lactobacilli. *Also there was a higher glucose in the resting saliva.*[127]

Other researchers have either confirmed or added to these findings. For instance:

Statement: *Poor control of diabetes* was found to be associated with caries.[128]

Moore et al. confined their studies to the flow of saliva, and reported that dry mouth (xerostomia) was more prevalent in diabetics than in the control subjects, with more complaints from diabetics with *poor metabolic control.*[129]

Xerostomia in diabetic patients is usually secondary to the dehydrational effects of the disease process itself. Greater amounts of fluids are eliminated via the urine (polyuria) and less is available for the saliva. Thus, the appropriate therapy for xerostomia of DM origin is to *restore insulin balance.*[130]

In another study, home care practices were similar, and all subjects had received similar regular dental treatment. In conclusion, *it was poor metabolic control of diabetes that was found to be associated with caries.*[131] Taking all previous statements into account, it is poor glycemic control that accounts for the excess prevalence and severity of the plaque diseses.

First Appointment

Every dental office will treat diabetes patients.[122] Upon admittance, the patient will be asked to fill out a personal medical and dental history for the use of the dentist and the dental hygienist

The first appointment should have two major objectives. The first should be to establish rapport with the new patient, and the second is to learn more about the

diabetic background of the patient. As the interview unfolds, any critical information should be added to the medical and dental history record. This would include information on dosage, time schedules, method of administration, previous adverse experiences with insulin control, number of hospitalizations, and physician recommendations.

At this time, the dentist should carefully explain the relationship between DM and periodontal disease. According to an article by Sundberg, 83% of the DM patients were *not* aware of the linkage between the systemic and the oral disease. It is necessary that the patient should know in the beginning that there will be many visits to the dentist for prophylaxes, monitoring and possible periodontal treatment, that in turn will help maintain metabolic balance in the medical treatment.[132]

Another salient educational item is the subject of smoking. If the patient smokes or uses smokeless tobacco, cessation is required if periodontal health is to be maintained. In comparing the effects of smoking on diabetic and non-diabetic men, it was found that the parameters usually assessed for periodontal disease—plaque index, gingival index, bleeding score, probing depth, loss of attachment, and missing teeth were all greater for the diabetic men.[133] It is counterproductive and ridiculous for a periodontist to treat a disease, if at the same time the subject is practicing a habit that is *blocking* the effect of the treatment. (See Chapter 13 for a full discussion of the adverse effect smoking has on the periodontium.) Another reality is that if smoking continues, there is an increased risk for cardiovascular disease that is one of the serious consequences of diabetes mellitus. With the above education and counseling complete, routine treatment appointments can be made.

Routine Appointments

When the known DM patient arrives for any appointments it is wise to determine whether she/he has had their prescribed insulin dosage and when the next dosage is due. Before treatment begins, the blood pressure needs to be recorded. If any surgery is contemplated, consultation with the patient's physician is desirable since the possibility of infection is omnipresent. There is always an *increased susceptibility to infection* of soft tissue as well as *a delayed healing of wound sites.* This increased hazard is probably due to the high content of glucose in the soft tissues that is ideal for bacterial growth. It is because of this increased risk of blood borne bacteria that all oral surgery (including periodontal treatment) should be accomplished with antibiotic prophylaxis.[122,134]

The First DM Emergency—the Hypoglycemic Episode

The most common emergency is the *Hypoglycemic Episode*—an unexpected *decline* in the blood glucose level. This is manifest by the patient feeling weak, exhibiting mood changes, incoherence, sweating, and tachycardia. All operative procedures should cease, and the patient *immediately given a fast acting oral carbohydrate from the emergency cabinet*—glucose tablets or gel, candy, juice followed by a determination of the glucose level.[e] This can be determined easily with a relatively economic electronic glucose monitor, which is quite accurate. If there is no apparent progress after 10 minues, repeat ingestion of the fast acting carbohydrate. *If there is recovery,* the patient should eat a snack or a meal to prevent a rebound to the

hypoglycemic state. If there is no apparent progress, the 911 phone call should be made, since with further delay, the symptoms begin to become ominous—unconsciousness, low blood pressure, hypothermia, seizures, coma and death.[135,136]

[e]Diabetic mellitus patients often carry a supply of fast acting carbohydrate agents of their choice. A soft drink (*not a dietetic* drink will often suffice.)

A Day in the Life of a Diabetic

Like for any other disease diagnosis, compliance with an attending physician's advice can be expected to range from full to minimal cooperation. For the diabetic, glycemic control and his/her general health status directly depend on this compliance. And what does this compliance entail????

Every morning, it will involve a *finger stick* to secure a drop of blood to determine the blood glucose level, using an electronic glucose monitor. If the blood glucose level is low, a decreased quantity of insulin is necessary. If breakfast is to include considerable more carbohydrate than normal, the amount of insulin must be increased. From 4 to 8 finger sticks a day is required to determine if glucose levels are remaining within pre established levels. A well controlled diabetic attempts to maintain a blood glucose level of 75-125 mg/dl. Additional finger sticks are necessary before events such as driving a car (safety) or before appointments (dental) to maintain a reasonably normal glucose blood level over the time to be involved. For some "brittle" diabetics where the insulin levels are erratic and unpredictable, night finger sticks may be necessary. Finger sticks and hypodermic self-administration of insulin become a monotonous way of life. However, they are *absolutely necessary day-in-and-day-out over the remaining years of life.* When the enormity of this daily task is fully realized, the unceasing support of a spouse, of the family, other diabetics, and the attendant medical community helps to *bridge the discouragement of the unending tasks necessary to preserve life.*

For those diabetics who have an insulin pump, the dosage is pre-programmed by the endocrinologist to inject insulin several times a day into the *abdominal cavity*. There is sufficient insulin in the pump reservoir for at least three days. The wearer must release additional insulin for any increased intake of carbohydrates or emergency. In addition, every third day, the patient must refill the pump reservoir, accomplish skin hygiene of the entry point, and replace the small catheter connecting the pump to the abdominal cavity.

For any diabetic, the holy grail is a cure that will eliminate the need for the repetitious finger sticks and self- or pump-administered insulin. World-wide research programs to achieve such a cure abound. As reported in an excellent article in the New Yorker magazine, pancreatic cells have been implanted into humans that produce the needed insulin.[137] However, to offset this outstanding achievement, has been the fact that the availability of donor islet cells (from pancreases) is miniscule in relation to the tremendous need.[138-140] Yet, it is a very promising beginning, even though immunosuppressive drugs are still required.

The most promising preventive program that is immediately available as well as

affordable for all ages is a personal crusade of *healthy eating*, *daily physical exercise*, and *maintaining a normal weight*.

Figure 22-8 Insulin Pump. Worn beneath the clothes, this insulin pump is driven by an internal computer that can be programmed to inject specific amounts of insulin at specific times. More accurate glucose control is possible with the pump than with the typical 2 or 3 manual injections per day. (Courtesy of Dr. Jeffery L. Hicks, University of Texas Dental School, San Antonio, TX.)

Figure 22-9 Periodontal disease as part of the Diabetes Mellitus problem. Examination and blood glucose testing of this patient with Diabetes Mellitus, Type 2, revealed severe periodontitis and hyperglycemia. The patient admitted to observing poor glucose control and to being chronically hyperglycemic. (Courtesy of Dr. Jeffery L. Hicks, University of Texas Dental School, San Antonio, TX.)

Question 5

Which of the following statements, if any, are correct?

A. The saliva urea content of the average ESRD patient before hemodialysis is about twice as high as a ESRD patient undergoing hemodialysis; in turn, the saliva content of the patient undergoing hemodialysis is roughly twice as high as a non-diabetic control.

B. Dental foci of infection can damage either the shunt used for hemodialysis, or the transplant that replaces the need for hemodialysis.

C. Diabetes mellitus is charcterized by an insufficient amount of glucose within the cell membranes, and an excess of glucose in the blood supply outside the cell membranes.

D. Without a relatively continuous glycemic control in DM, an uncontrolled periodontitis can be anticipated.

E. The successful transplantation of islet cells still requires immunosuppressive drugs.

Summary

The first part of this chapter provided an overview of the objectives and the administration of a hospital. The second half related to some of the diseases that are being treated daily in a hospital. Most frustrating, in many cases prevention practiced before admission could have aborted the onset of the primary complaint. To cite a few examples: (1) in the case of facial cancer (basal cell) the etiology probably was excessive exposure to the sun in earlier days (and still continues with a generation that better understands the consequences). (2) For many, the more deadly intra oral cancer (squamous cell) could probably have been avoided by rejecting the earlier use of tobacco products (smoking and chewing tobacco). (3) Another failure in cancer prevention occurred when the individual failed to seek professional advice for that small mucosal ulcer that would not heal, but only enlarged in size; or, (4) the lesion could have been missed by the dentist in the last dental examination (if the person had access to a dentist). Another example of the need for close cooperation by

Departments in the hospital is illustrated by the need for glycemic control by the diabetic patient. This control requires the combined effort by kidney specialists, endocrine experts, the periodontist. and other specialists to care for the complications, if and when they are diagnosed. The end result of this cooperation, is better health, and a better life for the diabetes mellitus patient. DM is truly an example of a bi-or-multi directional disease!

In dealing with diseased, traumatized, and mental and physically handicapped patients, there is always the concern for the unexpected. All hospital services have plans to react to most emergencies, ranging from use of emergency drugs to crisis teams that respond immediately when advanced resuscitation procedures are required. In all of these responses, someones life is often in balance. Even under the best of circumstances, the outcome is never guaranteed. In private practice, it is only a rare event such as the hypoglycemic episode that arises. Occasionally for some dentist in the United States a more serious emergency occurs. He/she is responsible for diagnosing and reacting to the emergency. The reaction must be immediate and correct. Like for the crisis team, it is an awesome responsibility, and the consequences can also be catastrophic, even though the response might have been correct.

For prevention of the plaque diseases in systemic disease, the guidelines outlined later in Chapter 23 apply. For self-care for most caries or periodontal patients, it is the usual need for mechanical plaque control with the "brush, floss, and flush" routine, coupled with chemical plaque control—fluoride mouthrinses and appropriate use of chlorhexidine (or other antimicrobial agents) to reduce the possibility of caries and gingivitis. It is not difficult to figure what is to be done, but instead, how to motivate individuals to do them. The main variable experienced in continuing care of chonically ill patients, is the time interval between recalls for prophylaxes and monitoring to avoid the need for secondary and tertiary prevention procedures. A second challenge is the question of how to best deliver preventive care to individuals who need help with self-care—such as the arm amputees, the unconscious, facial fractures, where the mandible and maxilla are wired together, and the mentally handicapped.

It is not long before a dentist in a hospital environment learns that dentistry does not end at the third molar. This chapter has pointed out how systemic disease can modulate oral disease, and vice versa. In the case of renal disease, the accompanying uremia can even benefit caries control!

Hospital dental practice is now becoming an alternative dental career to the present options of private practice, academia, the military and public health. It is an exciting, challenging professional career of continual learning from colleagues and personal experiences. It is also a major contribution of the profession to insure better dental care for the disabled and diseased as well as for the healthy.

Finally, as a staff member of the hospital, there are four areas where there is a need for your participation in prevention: Your patient needs to be protected from body harm (Hippocratic Oath); you need to protect yourself and your co-workers from the transmission of diseases of patients; you need to protect yourself and your institution from malpractice suits; and if you are in a non-hospital environment (private practice) when an emergency occurs, you need to remember the numbers 911.

Answers and Explanations

1. A, B, C, E—correct.

D—incorrect. The Department Chairpersons function as an Executive Board and act together for the good of the institution.

2. A, C, D, E—correct.

B—incorrect. A non-healing ulcerous lesion should be carefully examined and possibly biopied after a period of about 3 weeks, not three months.

3. C, D, E—correct

A—incorrect. At 3 months of age, only the surgical correction of the lip should be considered. The correction of the anterior alveolar ridge should await until the patient is about 10 years of age.

B—incorrect. There are more clefts involving the hard palate than involving the lips and hard palate. During the embryonic period there is a cleft between the maxillary bones that fuses from the lip back towards the uvula. At times the fusion will proceed normally and for unknown reasons, leave a normal lip. This partial fusion accounts for the lips sometimes being spared and the palate being involved.

4. A, B, C and E—correct.

D—incorrect. Since a cardiac emergency is probably the most serious occurrence that can happen in a dental office, probably the safest professional and safest legal position is to consult with the patient's cardiologist as well as review the current recommendations of the American Heart Association. If still in doubt, *empirical propylaxis* (prophylaxis coverage because the operator wants to be on the safe side) is a logical response.

5. A, B, C, D, and E—correct.

Self-evaluation Questions

1. A dentist (and other hospital professional staff personnel) must be _____ to indicate competency to practice in a hospital dental service.

2. The accrediting agency for dental hospital services in the United States is the _____ (organization).

3. The prosthetic device used to bridge a palatal cleft is known as an _____.

4. The more scientific term for "dry mouth" is _____.

5. Bone necrosis that follows cancer radiation treatment is known as _____.

6. An HIV case becomes an AIDS case when the CD4 lymphocyte drops below _____ lymphocytes per milliliter of blood.

7. Relieving a patient's _____ is one of the best means to assure an uneventful appointment with a high risk cardiac patient.

8. A bi-directional disease is one where both can help influence the outcome or the severity of the other.

9. A systemic disease that reduces the incidence of caries is _____.

10. If you had a patient have a cardiac arrest in your office, would you have a better chance of resuscitation with (CPR) (a defibrillator). Circle your selection.

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Chapter 23. Rationale, Guidelines, and Procedures for Prevention of the Plaque Diseases - *Norman O. Harris Marsha A. Cunningham-Ford*

Objectives

At the end of this chapter, it will be possible to

1. Describe the two reversible stages that occur between histological normalcy and development of overt lesions for each of the plaque diseases, i.e., caries and periodontal disease.

2. Explain why the initial/annual dental examination is so important to the present and future dental health of a patient.

3. Name seven caries-activity indicators (CAIs), and four periodontal-activity indicators (PAIs) and explain why they should be included in the initial/annual examination.

4. Explain how the CAIs and PAIs that are included in the initial/annual dental examination can be used as an aid in preparing the patient's education, treatment, prevention, and maintenance plans.

5. Discuss two diagnostic scenarios in which the use (or misuse) of an explorer for caries diagnosis can result in the insertion of many unneeded occlusal and smooth surface restorations.

6. Propose a flexible recall schedule based on a patient's level of treatment urgency (risk), and explain how risk determination can be used to channel patients into a more closely monitored caries and/or periodontal maintenance program.

7. Critique the advantages and disadvantages for the development of national guidelines for preventive dental care.

8. State five (out of the six listed) clinical environments in which a zero-or-near-zero

dental plaque disease prevention program can be fully implemented without major changes of present clinic facilities and manning personnel.

9. Contrast what you think *can be done to prevent the plaque diseases with what is being practiced.*

Introduction

In the quest for a zero or near-zero incidence of the plaque diseases, i.e., caries, and periodontal disease(s), the critical requirement is that the signs and symptoms of impending disease be identified at a time when progression toward overt cavitation and/or periodontitis can be *prevented, arrested, or reversed.* Three salient factors make this preventive objective feasible: (1) both dental caries and periodontitis are the result of a prolonged presence of pathogenic plaques affecting the enamel, cementum, and/or contiguous gingiva; (2) in most cases, both diseases can be controlled by mechanical and chemical plaque control regimens; and, (3) both of the plaque diseases must go through a continuum of two *reversible* interim stages from histological normalcy to clinical pathology.

The earliest stage of the plaque diseases is *in situ* involvement. For caries, this stage is marked by the *microscopic demineralization* of the crystalline structure of the enamel rods.[1] For periodontal disease, it is the early *infiltration of inflammatory cells* beneath the sulcular epithelium.[2] Neither the early demineralization of caries nor the early cellular infiltration of gingivitis can be directly seen. However, these microscopic beginnings of impending plaque disease can be *suspected* on the basis of noninvasive caries and periodontal risk assessment tests and indices that are *now* available to dental and dental hygiene professionals.

An *in situ* involvement, unless arrested or reversed, merges into the next stage of progression of the caries process—*the incipient lesion.* For caries it is manifest by the clinical appearance of a "*white spot*" on the enamel that is due to a more extensive subsurface rod demineralization.[3] Incipient lesions can occur on any surface as a *precaries lesion.*[a] They may occur (1) interproximally, *apical* to the contact point, (2) as *cervical white spots,* (3) on the *walls* of the deep occlusal fissures, and (4) on *buccal* and *lingual surfaces*—wherever there is plaque stagnation. These precaries lesions can be easily seen on dry, well-lighted buccal, lingual, and gingival enamel surfaces.[4] They are more difficult to detect on the occlusal surface where "sticky" pits and fissures should always be highly suspect as having incipient or even early undetected carious lesions.[5] The presence of "white spots" on the smooth interproximal surfaces are usually first identified in radiographs.[6,7] In periodontal disease, the incipient lesion is an inflammation of the gingiva, i.e., *gingivitis* with gingival bleeding being one of the first noticeable manifestations.[8] The incipient lesions of *both* caries and gingivitis can be reversed to histological normality—which by definition, represents a cure.

The third and final stage of the plaque diseases is the *overt* lesion. For caries, this stage is heralded by *cavitation* with bacterial infiltration. For periodontal disease, it is characterized by *nonreversible* changes in the periodontium such as an apical migration of the epithelial attachment and bone loss (periodontitis). At the overt lesion stage of the plaque diseases, treatment is usually indicated. There are two

possible exceptions where noninvasive preventive regimens may possibly be used to reverse overt caries, namely the use of antibacterial agents and/or remineralization therapy to arrest root decay, and the use of sealants to arrest early pit-and-fissure caries.[9,10]

Not all *in situ* lesions progress to the incipient stage, nor do all the incipient lesions progress to the overt stage of caries and/or periodontitis.[11] However, it is extremely important to note that no overt plaque disease lesion occurs at any site without first beginning as an *in situ* manifestation, and then progressing to an incipient lesion before becoming overt. *Thus, any prevention program must focus on identifying and reversing the in situ and incipient stages of the plaque diseases with the same, or greater diligence than is now given to searching for and treating overt disease.* It is the purpose of this chapter to summarize how the dentist, the dental hygienist, and the other members of the office team can realistically accomplish this goal. Such an achievement will allow the profession to move from a traditional emphasis on secondary and tertiary preventive dentistry, *to a primary preventive focus and commitment.*

[a]Precaries lesion; Since an incipient caries lesion can, in many cases, be remineralized, it should not be considered in the same category as an overt lesion where a restoration is usually indicated.

The Initial and/or Annual Dental Examination

The *initial/annual dental examination* is a *most* important event in the entire oral health program of a patient. At this time, a person seeking dental care has the opportunity and *expectation* to have his or her current oral status carefully assessed by a professional, a treatment plan prepared, restorative care accomplished, and *a comprehensive preventive dentistry program initiated* that will help forestall future plaque disease. This baseline examination is one against which all future examinations should be compared to evaluate time-function deviations from baseline oral health.

The first phase of the initial/annual examination begins in the reception area of the dental office. Here the patient is asked to complete the usual paperwork that includes the patient's medical and dental histories. Included in these histories should be a carbohydrate-intake questionnaire and questions relating to systemic factors and behavior patterns that might affect past, present or future oral disease development. Before the clinical examination, the dentist and hygienist should carefully scrutinize the patient's history, both for evidence of transmittable disease, as well as for conditions indicating a relationship of systemic conditions to oral disease. For example, a patient taking anti-hypertensive medications, tranquilizers, or many other drugs, often has an accompanying xerostomia and an increased caries risk. The medical history also helps identify conditions such as Crohn's disease[12] or diabetes mellitus[13] that are background systemic factors that raise the risk for caries and periodontal disease, respectively.

The initial/annual examination should be divided into two stages: (1) a *clinical* and a *roentgenographic* phase to *locate, diagnose,* and to *record* sites of incipient and overt plaque disease; and, (2) *laboratory tests and indices* to help identify the *risk* of in *situ*

and/or incipient plaque disease. These are ultimately the responsibilities of the *dentist*[14,15] but the laboratory phases can be performed by the dental hygienist. The examination should include as a minimum:

- A visual examination of all the intraoral and craniofacial tissues for diseases, other than the plaque diseases (cancer for instance)
- A set of bitewing and periapical radiographs as part of the examination for interproximal enamel radiolucencies and loss of alveolar bone
- A *visual* and mouth mirror examination of all the teeth for incipient and overt coronal and root caries, and an explorer examination for suspected fractured restorations, and secondary (i.e., recurrent) caries
- A record made as to which occlusal surfaces need sealants
- A record made as to which "white spots," (interproximal radiolucencies *without* evidence of cavitation), and root surface caries that require remineralization therapy and,
- A periodontal probe examination of all gingival sulci

The *final phase of the initial/annual* examination can be accomplished by a dental hygienist as part of the first prophylaxis appointment. This phase should include: a plaque index, a calculus site recording, a saliva flow rate notation, and appropriate laboratory tests to establish caries risk. All can be easily accomplished in the dental office to furnish valuable baseline information on the background of the patient's plaque diseases. The indices also furnish valuable information needed to help establish the level of preventive treatment required.

A plaque index is both an indicator of caries *and/or* periodontal disease activity. However, it does not discriminate as to which plaque disease is involved. As the plaque score (Chapter 13) increases over 10% there is an increasing probability that the plaque bacteria are causing damage to the teeth or to the periodontium. The plaque index also provides a means of evaluating, at sequential appointments, whether previously recommended plaque control measures have been implemented. The sites of calculus accumulation can be recorded at the same time, thus providing a further means of identifying areas of saliva stagnation where plaque control methods such as tooth brushing, flossing and irrigation have not been adequate.

Microbiologic caries activity tests provide a good assessment of caries risk, especially if the results are compared with previous baseline results. Dip-slide[b] kits are commercially available for evaluating the salivary levels of mutans streptococci and lactobacilli, respectively (Figure 23-1A and 1B). Any increase in the number of cariogenic bacteria between annual examinations should be viewed as indicating an increased risk for disease; therefore, it is prudent to take action to reduce the bacterial count. Microbiologic test scores when considered along with the carbohydrate intake score, serve as tools for assessing patient compliance with previous dietary and oral hygiene counseling intended to reduce the bacterial challenge.

Stimulated saliva should be collected in a calibrated tube in order to determine flow rate per minute. This latter datum—especially if the flow rate is below one milliliter per minute (xerostomia)—is often important in helping to identify the cause of an individual's caries. There is little difference between the data collected for treatment planning and the data needed for prevention programs. Perhaps the greatest contrast

between the two is in the decision-making process of what to do with the data. The dentist making the initial/annual examination should have the knowledge and experience to *identify* both incipient and overt lesions in the clinical and radiographic exam- inations, and the *wisdom* to differentiate between the two in selecting appropriate treatment options. A treatment-oriented decision of caries usually results in an invasive treatment procedure resulting in damage to the tooth, while a preventive-oriented decision usually leaves the tooth intact.[16]

A second major difference between the treatment and preventive aspects of the examination lies with office-time priorities. In the usual treatment plan, time commitments emphasize disease eradication. In an ideal preventive program, optimal time is allowed for *both* primary and secondary prevention. Another critical difference is that the treatment plan ends when the recorded pathology is successfully treated. In the preventive plan, the information gained in the initial/annual examination can be arrayed in a manner that permits the development of reasoned patient behavior modification and monitoring strategies that can be used to prevent future plaque disease development.

[b]Dip-slide kits for office counts of mutans streptoccus and lactobacillus are available from Ivoclar Vicadent, Amherst, NY, 14228.

The Computer Age

The percentage of dental practices using computers at the beginning of 1999 has been estimated at 89%.[17] Articles are beginning to appear in the literature on the advantages of hitech dental offices.[18-21] The initial use of computers was for administrative purposes—billing, recall appointments, electronic insurance claims, payroll and inventory control. However, with the passage of time, software became available that directly supported clinical needs. For instance, there are cosmetic imaging programs, interactive patient education CD-ROMs, self-administered patient histories, diet-analysis programs, software to determine the indications, contraindications, and incompatibilities of available drugs, on-line professional information and even remote consultations over the internet.[22] Even more benefits are coming.[23] However, three of the most current and promising areas for computer support of the dental clinical examination are: (1) *computerized charting,* (2) *the use of the intraoral videocamera (IVC),* and (3) *(filmless) digitalized roentgenography (DXR).*

Computerized charting programs are now commercially available for recording the presence of overt or incipient plaque disease lesions (Figure 23-2). Other parts of the computer software often permit *recording the presence and severity of selected caries and periodontal activity indicators* that are the basis of risk assessment *at the time of testing.* Commonly used evidence-based caries-activity indicators (CAIs) are the (1) plaque index, (2) quantification of cariogenic organisms, (3) saliva flow rate, (4) frequency of intake of refined carbohydrate (sugar). Equally important are the (5) number of "sticky" occlusal pits and fissures, (6) coronal and root caries, (7) incipient buccal and lingual smooth surface lesions, and the (8) number of interproximal lucencies (incipient lesions) *without cavitation.* For periodontal disease, the periodontal-activity indicators (PAIs) are the (1) plaque index, (2) calculus, and (3) gingival bleeding indices, and the (4) Periodontal Screening and Recording system

(PSR) for pocket depth.[24,25] These plaque disease indicators can be entered into the computer at chairside by use of a keyboard, mouse, touch screen, or voice activation. Note that the plaque index is useful as a risk indicator for *both* caries and periodontal involvement. Other indicators for either of the plaque diseases can be added or substituted in a computer format. For instance, the flow rate of gingival crevicular fluid might eventually serve as a PAI for dental offices having the necessary equipment.[26,27] However, the above four PAIs are now well known, easy to accomplish, and presently used by dentists and dental hygienists in caries and periodontal control programs.

Figure 23-1 A After 48 hours, mutans streptococci become visible on test strip. The two left slides have less than 100,000 colony forming units (CFU) per milliliter of saliva and constitute a relatively low risk for caries. The two samples to the right indicate a high risk with more than 100,000 CFU/ml.

Figure 23-1 B A small convenient office incubator for processing Caries Risk Test slides. Both illustrations courtesy of Ivoclar Vivadent, Amherst, NY 14228.

Figure 23-2 Periodontal Computer Assisted Record Form. A periodontal computer assisted record form that, along with other options available with the accompanying software, permits easy comparison with the details of previous periodontal examinations. When the probing is accomplished with the Florida Probe illustrated in Figure 13-6, entries can be automatically made on the record form. Color coding [not possible on this black and white photo] allows easy detection of differences in gingival sulci probing depths of 1-4 mm (black), greater than 5 mm (red). Note the other parameters that are part of a complete periodontal examination, such as PSR, plaque index, mobility, furcations, and recession. Courtesy of the Florida Probe Corporation, Gainesville, FL.

Question 1

Which of the following statements, if any, are correct?

A. Incipient "white spot" lesions are found only on the facial and lingual surfaces of teeth.

B. "White spots" in the enamel indicate that the carious process has passed the incipient stage.

C. The *in situ* stage of gingivitis is characterized by sulcular bleeding upon gentle probing.

D. The presence of dental plaque is an important evidence-based indicator for both caries and periodontal disease.

E. The CAIs and PAIs are more accurate in indicating *present vulnerability,* than of *predicting* future caries and/or inflammatory periodontal disease.

The Intraoral Videocamera (IVC) and the Digitalized X-Ray (DXR)

The recent introduction of the intraoral videocamera (IVC) and the digitized dental x-ray (DXR) provide two powerful high-tech instruments, that when coupled with a

computer, video recorder or color printer, facilitate a more accurate identification, charting, and understanding of the sites of both incipient and overt plaque disease.[28]

The IVC, which is a miniature camcorder, provides a uniform lighting at all sites being examined (Figure 23-3). It permits magnification of questionable areas to *improve* the possibility of locating incipient and overt lesions, as well as sites of plaque accumulation, calculus deposition, defective restorations, and periodontal problems. The IVC has the advantage of allowing an area to be freeze-framed to study a questionable site better, or if desired, to make high-quality color photographs by outputting the image to a high-resolution color printer. Cracked or fractured amalgams are easy to identify. A later replay of the examination tape on the high-resolution television screen is an outstanding means of educating and motivating patients.[29] Administratively, the modem of the computer can be used to transmit the necessary images for immediate insurance verification of anticipated treatment and preventive costs, or to send treatment information to remote sites.

The digitalized x-ray (DXR) utilizes intraoral film-size *electronic sensors,* which are connected to the computer (Figure 23-4). The existing office x-ray unit provides the source of energy. No liquid processing is necessary as the image is processed electronically within the computer.[30] The digitalized image is more uniform in contrast than is now possible with film. By use of computer control, it is possible to zoom in on small details, and to use enhancement techniques to lighten or darken areas of the screen for better diagnosis.[31] An important advantage of DXR is that it only requires *50% or less* ionizing radiation than is necessary in conventional dental radiography.[32] This improved safety factor results in a better balancing of the individualized need for more frequent radiographic monitoring against the ethical responsibility to minimize patient exposure to ionizing radiation.[33] Like the IVC, the DXR images can be downloaded for patient education and study, insurance verification, or for use in treatment or preventive programs. There is no deterioration of the stored digitalized images with time. Finally, a most important point is that the diagnostic value of the image is considered equal to traditional bitewing radiography when downloaded to a *high-resolution* monitor or printer.[34]

Figure 23-3 A high-tech merging of electronics and optics to make an intraoral video camera. Note the small camera lens at the end of the camera shaft. The images obtained are seen on the operatory monitor during the examination as well as being transmitted over the office hardwiring to the business center where it can be stored or printed, or if necessary sent out as an e-mail for insurance purposes.

Figure 23-4 Three different intraoral film-size sensors—bitewing, anterior, and pediatric—used for DXR. Variations in energy received by the sensor are transmitted by the attached wire to the computer for electronic processing. The resulting image can be presented on a high resolution monitor, stored, printed or treated as e-mail if needed.

Caries and Primary Prevention

Explorer Misdiagnosis and Misuse

The hi-tech advancements to dental diagnostic science discussed above, have arrived

at a time when the traditional explorer is coming into question as often being an *accessory to iatrogenic dentistry.* With the millions of explorer diagnoses of caries-or-no caries being made every day in dental offices throughout the world, it is essential to look at any evidence that might indicate potential or actual patient harm, as well as possible solutions.

The use of the explorer to search for carious lesions is being questioned in Europe[35] and the United States.[36] This concern is generated by the fact that explorer examinations can possibly cause irreparable damage to the surface of immature enamel;[37-39] carry cariogenic bacteria from one infection site to other vulnerable deep pits and fissures;[40] and *most important, explorer catches have not proved to be valid indicators of the presence, or absence, of questionable occlusal caries.*[41,42] The diagnosis of pit-and-fissure caries with convoluted fissures is precarious by either visual or explorer techniques. The diagnostic value of an explorer examination of the occlusal surfaces *decreases* as the "stickiness" of the fissure increases—this at a time when *the need for validity increases.* For instance, the sensitivity of an explorer examination can decrease from 80% for wide fissures to 52% for those that are narrow.[42] Penning and coworkers employed an explorer to examine the occlusal surface of 100 extracted teeth that had *no visible cavitation,* and which were later x-rayed and sectioned. Only 24% of the caries lesions that were eventually found were discovered by use of the explorer.[43] Lussi conducted a study involving 34 dentists who serially examined 61 extracted teeth. A histological study of the teeth was accomplished to provide the "correct" diagnoses. The results demonstrated that the dentists were more likely *not to treat decayed teeth than to "restore" sound teeth.* Forty two percent of the teeth were correctly diagnosed, only one tooth was correctly diagnosed by all, and two were never correctly diagnosed.[42] As a result of the lack of validity of the explorer-based examination of occlusal surfaces, *several clinical researchers have suggested that the explorer is no more effective for locating pit-and-fissure caries than is a visual examination coupled with professional judgment.*[44,45] This conclusion argues for the use of an *explorerless* mouth-mirror-only (or preferably the use of the IVC) in dental examinations, (Figure 23-5) but relegates the explorer to such tasks as locating calculus, checking the marginal integrity of newly placed restorations and sealants, and in searching for secondary caries. Even this latter use has been subject to question.[46]

An example of overtreatment caused by *misuse* of the explorer is the case of the incipient caries lesion. In a caries examination, "white spots" on the buccal and lingual surfaces can be easily seen; whereas, interproximal enamel lucencies ("white spots") are usually first detected by radiography. *Once incipient lesions are identified and recorded, there is no need to probe this precarious site.* To do so will often convert the incipient lesion into an overt lesion requiring a restoration. Lesions over 0.1 to 2.0 mm. can be created in extracted teeth by such misuse of the explorer.[37] Without the probing *penetration* of the surface zone that allows a subsequent bacterial invasion, remineralization therapy of the "white spots" can very often be successfully implemented with no eventual damage to the tooth.

As a general summarizing concept of *diagnostic errors,* Downer[47] and Bader[48] have both pointed out that in routine caries examinations, whether as a result of the x-ray diagnosis or from the use of an explorer, there is a high probability of *false-positive* and *false-negative* diagnoses. As that line of delineation between disease-and-no

disease is approached, it becomes even more difficult, in fact impossible, to positively identify a tooth as carious-or- noncarious. In this gray area the toss of a coin can be as accurate as the explorer. These diagnostic errors are the result of perceptual and tactile variations between examiners, different criteria for treatment decisions, lack of precision of diagnostic technology, and differences in diligence to search for pathology and *impending* disease. This fallibility has been recognized for years, and several methods have been, or are being tested to provide more validity to caries diagnosis. Some of the more promising methods used are bite wing and digitalized roentgenography (the latter with its capability of image enhancement); fiber-optic transillumination (FOTI), visual examination especially when using a video intraoral camera, disclosing dyes, electrical conduction (or resistance) and high intensity focused light. In the latter cases, the reflectance from the tooth surfaces of the ultraviolet, fluorescent, laser, or infra red light are being measured by a light meter.[49] Until a better solution is developed, penetration of the surface zones of incipient lesions or false-positive diagnoses for caries can be as damaging to the teeth as is bacterial-induced cavitation, since in a clinical situation the end result is the insertion of unneeded restorations

Experience and studies also indicate that restorations are not-for-a-lifetime.[50-53] The high failure of amalgams from marginal defects, overhangs and recurrent caries has been noted for many years.[54,55] More contemporary studies show an improvement in longevity of restorations, but still the cycle of placement and replacement is strongly evident. Mjor et al., in a recent study of 6,761 restorations, found that the average longevity for an amalgam restoration placed in adults (> or = 19 years of age) was 11 years. Resin-based composites averaged 8 years, glass ionomers 4 years, and resin modified glass ionomer, 2 years.[56] Each successive replacement involves a greater loss of tooth structure and a greater danger of eventual tooth loss. *Thus, preventing one new cavity results in a multiplier effect of preventing several recurrent lesions over a lifetime.* For this reason, the present treatment of overt, and arrest, or reversal of incipient lesions, *must be accorded an urgent priority of preventive care*.

Probably the two most effective and economical dental-office preventive procedures available to rapidly reduce caries incidence, and often the least used, would be the early placement of sealants in all deep convoluted ("sticky") occlusal fissures, and the timely use of remineralization procedures for all noncavitated incipient smooth surface lesions. To epitomize the potential of the combined use of sealants and remineralization strategies, Ripa concluded that caries could practically be eliminated in U.S. schoolchildren by the combined use of sealants to protect the occlusal surfaces, and fluoride rinses to protect the smooth surfaces.[57]

Figure 23-5 With a patient watching the monitor during the examination, much patient education can be accomplished.

Question 2

Which of the following statements, if any, are correct?

A. The IVC camcorder documentation of the dental examination can be watched on a monitor screen by both the patient and the dentist.

B. The main disadvantage of DXR is the fact that it requires more x-ray energy than

bitewing films.

C. Several clinical research studies have demonstrated that explorer catches are not valid indicators of the presence or absence of occlusal caries.

D. According to several clinical researchers, an explorer examination of "sticky" occlusal surfaces results in many false-positive and false-negative diagnoses.

E. A false-positive recording of caries status for a tooth usually results in the placement of a restoration in a non carious tooth.

Sealants

The placement, replacement, and retention of a sealant in "sticky" caries-prone pits and fissures can *prevent* and/or *arrest incipient occlusal lesions* (Chapter 10).[58-62] Sealants provide a perfect *solution* to the dilemma of coping with the false-positive diagnoses of occlusal caries. Their placement in deep convoluted fissures of occlusal surfaces negates the possibility of wrongfully restoring the many teeth that would have received inadvertent false diagnoses for caries. The sealing of a fissure also sequesters the cariogenic organisms to prevent the seeding of other areas in the mouth. Sealing of the fissures can be accomplished easily, quickly, painlessly and economically.

The sealant serves to interdict the nutrient supply to the fissure microorganisms, leading to their extinction, and the hardening of the underneath dentin.[63] The placement of an occlusal sealant is probably the most conservative solution to preventing or arresting the early caries process (if present), thereby avoiding the probability of future primary or recurrent lesions and their sequelae.[61] Sealant placement should be followed by a topical application of fluoride applied at the dental office, as well as a daily home exposure to a fluoride dentifrice and/or mouthrinse to help protect the whole tooth. Traditionally, the majority of sealants were placed on the occlusal surfaces of children's teeth. However, the *anatomy* of the occlusal fissure and the *risk assessment of the patient,* not the age of the individual should be the guide to any decision to place or not to place a sealant. *Lest we forget,* (1) *for every occlusal restoration seen at any age, a sealant was indicated previous to the development of the overt lesion and* (2) *for every smooth surface lesion seen at any age, remineralization was probably indicated previous to the development of cavitation!*

Tooth, Heal Thyself—Remineralization

The microscopic loss (demineralization) and regaining of mineral (remineralization) on the *surface* of the teeth occurs *continuously* in a human environment.[64] Remineralization can be a *short-term* defense response to the daily microscopic loss of mineral from food abrasion and ingestion of acid foods and condiments such as oranges, pineapple, cola drinks and vinegar. Of equal importance is the body's ability, under *longer-term* favorable conditions, to repair radiographic lucencies that are seen in radiographs extending from the enamel surface into as far as the outer 1/3 to 1/2 of the dentin. The rehardened subsurface lesions are usually microbiologically inactive with a hardness that approaches or exceeds that of the original enamel or dentin.[65]

The equilibrium between de- and remineralization can be greatly biased in favor of oral health by:

1. Reducing the population of cariogenic bacteria in the oral environment by mechanical and chemical plaque control measures. This is accomplished by frequent patient use of tooth brushing, flossing and irrigation ("brush, floss and flush"); it is also aided by the periodic use of therapeutic mouthrinses such as chlorhexidine and fluoride.

2. A drastic reduction of refined carbohydrate (sugar) intake.

3. Initiatiation of remineralization and protective strategies that enhance and supplement the crucial saliva-driven tooth remineralization.[66,67] Examples include multiple exposures to fluoride found in community-water supplies; of *professional* applications of fluoride varnish/gels, hygienist use of fluoride prophylactic pastes; and the *home use* of fluoride products such as fluoride dentifrices and mouthrinses.

4. Stimulating the flow of saliva with its mineralizing constituents. This can be accomplished by chewing non-sugar containing chewing gum, especially a gum flavored with xylitol, a non-acidogenic and non-cariogenic polyol[68] (Chapter 6.)

Remineralization of teeth is not a new concept. In 1884, one of Dr. J. D. White's "tips" in *Dental Cosmos* pointed out that "many teeth coming through the gums had an exceed- ingly defective look". For these teeth he suggested that the finger should be used to rub chalk on the teeth twice a day. As much of the chalk as possible should be allowed to remain. Limewater was to be used to rinse the mouth instead of regular water.[69]

During the period of 1910 to 1920s, Head, a physician presented convincing experimental evidence that teeth could be hardened.[70] In one paper he wrote that he and several other dentists had seen *white spots* disappear, after which he asked, "If the spot disappears, would not this prove that the enamel is not a dead, inert substance that we are supposed to consider it?"[71]

By the 1920s, there was considerable agreement that recalcification (remineralization) could occur, but still considerable debate as to whether "calcification and recalcification" was physiological via the pulp, or a chemical phenomenon from addition of constituents from the saliva.[72,73]

It was not until the 1950s that there was more positive proof of caries arrestment. Muhler in his extensive research of a stannous fluoride dentifrice (Crest) found that the end-of-the-year examinations, many of the original charted lesions no longer existed. These he termed reversals.[74]

Following the initiation of water fluoridation, it was believed that the caries decrement that followed was caused by the formation of a more resistant fluorapatite crystal on the enamel surface, which to some extent was true. However, it was not till Silverstone described the carious lesion as comprised of a surface zone, the body of the lesion, and the dark and translucent zones, that it was demonstrated that with appropriate fluoride therapy, the subsurface carious lesion could be remineralized.[11]

Today it is recognized that fluoride acts to *both* prevent demineralization and enhance remineralization. (Chapters 3 and 11.)

Remineralization therapy to arrest and reverse incipient smooth surface lesions is often mentioned in *research* journals, less mentioned in *practice-oriented* publications, and rarely employed *routinely* in clinical practice. Yet, the remineralization of incipient lesions with *no* evidence of cavitation is an essential *noninvasive* preventive option to maintain a lifetime caries-free dentition. As early as 1970, von der Fehr and coworkers in a study involving multiple daily sugar mouthrinses, *visually* observed the development of "white spots" within 23 days. Remineralization of these "white spots" was successfully accomplished by use of dental hygiene and fluoride mouth rinses.[3] It was about 2 years later that a similar study was reported, only in this case, the students also used a 0.2% chlorhexidine mouthrinse twice-a-day to suppress the cariogenic organisms. There were no signs of early caries. *The chlorhexidine had made a difference.*

"White spots" are *not* rare occurrences. Mejare and colleagues visually inspected 598 surfaces of premolars extracted for orthodontic reasons as well as the adjacent surfaces of neighboring teeth and found that 51% had incipient lesions, of which only 5% demonstrated cavitation.[75] Mejare and Malmgren in another study found that at age 16, the mean number of incipient lesions per individual was 3.0[76] In a Scottish study, 2,917 incipient lesions were found visually on the buccal and lingual surfaces of the teeth of 2,854, 13-year-old school children. At the end of 2 years, approximately *three quarters* of the lesions had *remained static* or *regressed.* Furthermore, where incipient lesions were originally identified, there was a greater amount of plaque, as well as a greater possibility of locating other incipient lesions.[4] *The identification of one incipient lesion can be a warning sign of a more generalized caries status requiring attention.*

The *extreme importance* of considering all radiolucencies for remineralization is illustrated by a study in Denmark. The interproximal lucencies of 1,080 *preoperative* radiographs were examined *following* placement of restorations. Most lucencies found were those demonstrating demineralization but *not* cavitation. Only 10% demonstrated cavitations, meaning that *90% should have been considered for remineralization in lieu of restoration.* As a result, it was concluded that the original diagnosing dentists needed to better recognize lesions amenable to remineralization.[77] To further emphasize this problem of inappropriate over-treatment, Thylstrup and coworkers, in a roentgenographic study of interproximal caries, estimated the *only one out of four patients receiving restorations, needed restorations.*[78] Unfortunately, many believe that operative intervention is necessary to restore minimal interproximal lucencies despite a plethora of information that remineralization is a preferable *noninvasive* option for tooth longevity. For example, in a questionnaire study in Scotland, it was found that 44.2% of Scottish dentists would fill an approximal lesion confined to the enamel of a hypothetical 12-year-old *prior* to cavitation.[79]

Another factor to be considered when an interproximal surface of a tooth is prematurely restored is the *iatrogenic damage* caused to the proximal surface of the adjacent sound tooth. In one study, more than 50% of surfaces adjacent to approximal restorations were damaged, often presenting with radiographic changes.[80,81]

Every interproximal radiolucency *not showing cavitation,* should be considered for remineralization therapy and longitudinal monitoring; *only* when a lesion demonstrates cavitation should it be considered irreversible. Pitts and Rimmer, in examining 1,588 teeth, found that *100%* of the teeth with only "white spots" had sound enamel surfaces with no cavitation; even 89.5% of the enamel lucencies extending to the *dentinoenamel junction* had no cavitation. However, all lucencies extending into the proximity of the pulp *did have* cavitation.[82]

The New Zealand School Dental Service successfully uses fluoride varnish (Durophat) to enhance remineralization of the teeth of children having an interproximal lucency that is *not more than halfway through the enamel and no cavitation.*[83] Others use the dentinoenamel junction as the threshold.[84] For instance, Elderton believes that a radiolucency should extend *into* the dentin or *show cavitation* before operative intervention.[85] Dodds has suggested that even dentinal lucencies *without apparent cavitation* should be considered for possible remineralization therapy since further caries progression is often blocked by sclerosing or secondary dentin.[86]

Clinicians in Europe and globally are beginning to think conservatively.[87,88] The following 1999 paper from Scandinavia illustrates this trend. The study by Majare et al. is entitled "Caries assessment and restorative thresholds reported by Swedish dentists." The study included the responses of 651 (out of 923) dentists who indicated that for an adolescent with low caries activity and good oral hygiene, 90% would *not* automatically restore an approximal lucency if its radiographic appearance did not show obvious progression *to the outer 1/3 to 1/2 of the dentin.* Moreover, 67% would only consider immediate restorative treatment of an occlusal surface if *obvious cavitation* and/or if radiographic signs of dental caries could be observed. Other interesting findings were that the younger, more often than older dentists would postpone restorative treatment, and that dentists in private practice would restore approximal caries at an earlier stage of progression than dentists in the Public Health Dental Service.[89]

There are no U.S. reports available for comparison.

The Remineralizable Root Caries Lesion

With an aging population retaining their teeth longer, there is an increasing prevalence of root caries.[90,91] Approximately 28% of the population seeking treatment who are under 60 years of age have root caries;[92] over 60 years of age, the estimate increases to approximately 40 to 63%.[93] Fifty-three percent of the root lesions are on the *facial* surface, followed in order by the distal, lingual and mesial surfaces.[94]

Like coronal caries, root surface caries occur in distinct and important stages—incipient, arrested, overt, and restored. The incipient stage that precedes frank (overt) cavitation is described as a "well-defined softened area, yellowish or light brown in color, but *without* cavitation upon initial inspection, that is, undisturbed *before* probing.[95] There is no "white spot" or subsurface lesion as found in coronal caries. This softened mass can often be remineralized by daily plaque removal with a toothbrush and a fluoride dentifrice, plus appropriate spaced professional applications of fluoride.[96-98] As a part of surface rehardening, there is also a redistribution of

mineral within the lesion.[99] Thus, like incipient coronal caries, the easily visible root caries site should *not* be subject to explorer manipulation if contemplating remineralization procedures. The influence of fluoride on root surface caries is the same as for coronal caries, i.e., reducing demineralization and enhancing remineralization.

Several chemotherapeutic approaches have been used to arrest root caries lesions. Billings and associates,[100] and Markitzui and colleagues used *topical fluorides.*[9] Nyvad and Fejerskove found that where root lesions were accessible (mainly *facial* surfaces) to brushing with a *fluoride dentifrice,* it was possible to convert 54% of the active root caries to an inactive status. Only 8% of the approximal lesions reverted to inactive status.[100,101] Schaecken et al. found that the use of chlorhexidine and fluoride caused a *hardening* of root caries as well as markedly suppressing the mutans streptococci over several months.[102]

Following a histopathology examination of extracted teeth with root caries, Schupack and associates concluded that remineralization depended on (1) the degree of active sclerosis of dentinal tubules underlying the lesion, (2) the degree of the bacterial infection of the dentin, (3) the degree of progression of the lesions, and (4) the location of the lesions at the various root surfaces[103]

The alternative to *preventing* active root surface caries is the insertion of restorations that soon become the sites of secondary caries. Fejerskov expressed this thought by stating, "The more fillings inserted, the more likely is the risk of developing more caries lesions."[104] Remineralization therapy of root caries, especially of the facial surface, appears to have an equal or better probability of success in restoring an incipient or early overt lesion to inactive status, than does the insertion of a restoration.

Fluorides and Remineralization

Regimens to maximize the potential for remineralization as well as to minimize demineralization consist of the use of home and office chemical and mechanical plaque control techniques, multiple fluoride therapies, and a severely limited intake of sugar—exactly the same techniques as used to prevent the development of the incipient lesions in the first place. The multiple fluoride therapies include fluoride dentifrices and mouth rinses, office applications of topical fluoride liquids, gels, or use of a fluoride varnish or remineralizing paste at specific sites, and even the possible prescription of fluoride tablets in fluoride-deficient areas, regardless of age.[105] Once remineralization has been successfully accomplished in the presence of fluoride, the remineralized areas often are *more resistant to future acid attack than previous to demineralization.*

The two times when a tooth is especially vulnerable to demineralization and caries are (1) immediately after eruption and before maturation of the enamel, and (2) at any time during life following prolonged bacterial acid-induced demineralization. To meet the first of these two challenges, several *professionally applied fluoride* applications should be made from the time the first tip of the erupting cusp appears until the tooth is in occlusion. Even after full eruption is attained, fluoride should be applied several times professionally during the first year of intraoral maturation.

In the second instance, the many daily cycles of de- and remineralization at different periods throughout life are usually subclinical and are not possible to detect.[11] One of the best ways to ensure enamel protection from caries progression during such periodic negative mineral balances, is by the daily exposure of the teeth to fluoride, such as fluoridated water and by use of over-the-counter products such as fluoride dentifrices and fluoride rinses. (Figure 23-4).

The Potential

The successful sealing of all deep pits and fissures and the remineralization of all incipient lesions would by definition, result in preventing all carious lesions (except for those caused by diagnostic error and explorer misuse) from progressing to the overt stage requiring restorations. Any concern of possible unnoticed caries progress during remineralization therapy or beneath a sealant can be allayed by shorter individualized recall schedules to permit longitudinal monitoring.[106,107] To further allay concern, the slow advance of the pre-caries front is seen in a report by Majare et al., where 50% of the lucencies at the dentinoenamel junction required an average of 3.1 years to progress into the dentin.[108] This provides ample opportunity to later substitute invasive operative treatment, if necessary.[109] To reinforce these previous facts, Pitts, in a 3-year study of regular adolescent attendees, found that 80% of the original radiolucencies apparently regressed or arrested spontaneously.[110] Thus, noninvasive approaches of sealant placement and remineralization therapy are viable and a preferred approach to maintaining intact teeth. *Dental caries is a preventable disease where even established precaries lesions can be arrested or reversed—with appropriate patient cooperation.*[111]

Question 3

Which of the following statements, if any, are correct?

A. Sealants become more cost-effective as the fissures on the occlusal surface deepens and become more convoluted ("sticky").

B. Sealants should be placed over all "sticky" fissures where there is the possibility of a false positive or a false-negative diagnosis.

C. "White spots" result in a permanent change in color and surface topography of a tooth.

D. Usually, more "white spots" regress or remain static than those that progress to overt lesions.

E. Caries of both coronal and root surfaces can only occur when demineralization exceeds remineralization over time.

Periodontal Disease and Primary Prevention

A Rational Approach to Preventing Gingivitis

This short section will briefly review a few facts appropriate to this chapter that were learned from Chapters 4 and 13.

1. A high plaque-index score and/or the presence of calculus usually portends a gingivitis.

2. Gingivitis usually can be reversed by office-instituted and home self-care hygiene measures that remove or severely disturb the dental plaque. Since the periodontal tissues return to histological normalcy, this home and professional care represents a *cure.*

3. Once the gingivitis is successfully treated, the next recall interval should be based on the present treatment urgency, that is, the higher the apparent *risk,* the shorter the time interval should be before the next appointment.

Periodontal-activity indicators (PAIs) and guidelines for appropriate *therapy, counseling, and recall schedules* can be developed in the same way as previously discussed for caries-activity indicators (CAIs). Again, the four evidence-developed indicators are the plaque index, calculus severity, number of bleeding sites on probing, and the Periodontal Screening and Recording system (PSR) for pocket depth (see Chapter 13). The PSR was introduced in 1993 by the American Dental Association and the American Academy of Periodontology as a method to screen all adult patients 18 years of age and older for evidence of periodontal disease.[24]

Only 5 minutes is needed to accomplish the PSR screening (probing). The zero-to-four coding system used for each of the sextants of the patient's mouth provides a numerical basis to suggest the periodontal treatment urgency as well as patient management guidelines commensurate with the patient's periodontal status. The PSR should be recorded at all initial/annual examinations, and at all recalls, especially where the previous periodontal treatment urgency was of concern. A color-coded, beaded probe tip is used to measure sulcus depth enabling the PSR to be quickly accomplished.

To increase the accuracy of determining the depth of the periodontal pockets, as well as to facilitate the ease of recording pocket depth, a computer-coupled periodontal probe was developed to automatically signal a computer to record the pocket depth of a tooth after encountering a given resistance at the base of the gingival sulcus.

Unlike the CAI laboratory tests in which caries risk is related mainly to mutans streptococci and lactobacilli levels, no parallel laboratory "*screening tests*" are available for periodontopathogens. A number of *diagnostic* laboratory tests for *Actinobacillus actinomycetemcomitans, Porphyromonos gingivalis,* and *Prevotelle intermedia* and other suspect periodontopathogens are used to aid in making treatment decisions, but their cost-benefit as routine microbiologic screening PAIs has not been adequately assessed. However, much research effort is now being devoted to finding some genetic marker for identifying present and future risk for periodontal disease.

Plaque and Plaque Control

Dental plaque is *always* present at the interface between the saliva and the tooth

surface. Any site with more than a minimal amount of plaque should be considered an *infected area* in need of constant and effective daily disruption or removal by use of a toothbrush, dental floss, and irrigation; and/or by the suppression of pathogenic bacteria with antimicrobial agents. Initially, a marginal gingivitis of local origin occurs as a result of bacterial metabolic end products in the supragingival plaque. This early periodontal involvement (gingivitis) is usually the first sign apparent to the patient in the form of "pink toothbrush" caused by blood on the toothbrush. Starting at an early age, it is essential that a person should learn to use plaque-control measures that remove or severely disturb plaque at least once a day. The tooth- brushing, flossing, and irrigation that are accomplished by the individual while the periodontium is healthy is intended to *prevent the onset* of gingivitis. Any eventual gingivitis may or may not progress to periodontitis. If it does, it was the supragingival plaque that probably served as the initial seeding loci of the microorganisms found in the subgingival plaque. If unsuccessful in maintaining gingival health, the preventive effort should immediately be directed to professional intervention to arrest the disease process before an entrenched subgingival plaque causes additional damage to the periodontium.

Periodontal maintenance care can reduce *both* the number of root caries and the number of pockets.[112]

Chlorhexidine Gluconate

In August 1986, the U.S. Food and Drug Administration (FDA) approved the *prescription* sale of Procter & Gamble's Peridex, a 0.12% chlorhexidine gluconate solution as a mouth-rinse. This action was soon followed by the American Dental Association (ADA) also approving the product as useful in the control of gingivitis. Since that time, it has been found that chlorhexidine not only provides a potent adjunctive agent for the treatment of periodontal disease(s), but also is highly effective for suppressing cariogenic plaque bacteria.[112-116]

Chorhexidine is considered the "gold standard" for effectiveness as an oral antimicrobial agent. With the exception of fluoride, it is considered the most effective drug available in the war against mutans streptococci.[117] The effectiveness of chlorhexidine stems from its substantivity; that is, (1) it *adsorbs* to the infected tissue in the target area, (2) it is *released slowly,* and (3) it is *released in an active form.*

The dental uses of chlorhexidine are impressive.[118] For soft-tissue therapy it is used to treat gingivitis, periodontitis, stomatitis, herpes simplex, ulcers, and before and after oral surgery.[119] For caries control, it has been employed with success in mouthrinses, gels and varnishes. It has been combined with fluorides in varnishes to suppress Streptococcus mutans, and with fluoride to accelerate remineralization.[120,121] One three-year study of 12 to 14 year old high-risk students with $>10^6$ *Streptococcus mutans* that used 1% chlorhexidine gel experienced an 81% reduction in caries incidence.[122] There have been relatively few complaints, and these have have mainly targeted tooth stain (77% of the complaints), transient bitter taste (12%), excess calculus, and dry mouth (6%).[119] The small amount of tooth stain that occurs following continued use of chlorhexidine rinses can usually be easily removed at home by some of the rotary powered toothbrushes, or certainly by a prophylaxis. Some studies now being conducted indicate that when chlorhexidine is combined with

peroxyborate, an antioxidant, the results are claimed to be superior to chlorhexidine alone, yet without the problems of taste and staining.[123]

In controlled mouthrinse programs, chlorhexidine has both *prevented* a buildup of plaque and *reduced* plaque accumulations, if used along with brushing.[124] Once gingivitis is diagnosed, chlorhexidine rinses plus other self-care oral hygiene measures are usually adequate to return the inflamed gingival to normal. Once the subgingival plaque becomes involved, subgingival irrigation with chlorhexidine becomes an option.[125] The irrigation instrument can be a powered pulsating jet stream, or as simple a device as a plastic canula attached to a plastic squeeze bottle.[83]

To briefly summarize its great utility in plaque disease control, chlorhexidine has been used with toddlers,[126] with high-risk schoolchildren,[127] with high-risk elders,[128] with the handicapped,[129] and with the mentally handicapped.[130] It has found use in orthodontics,[131] in periodontics,[132,133] in caries-control for overdenture patients,[134] and with post-radiation patients.[135]

"Scorecards" and Guidelines

Professional *ethics* and the *law* mandate that health-care providers present patients with a disclosure of *full and accurate information* (informed consent)[136] about all contemplated invasive or noninvasive treatment procedures, as well as the likely costs and benefits of each.[137,138] Often *missing* from these discussions are science-based preventive regimens that offer options to both the patient and dental professional for the non invasive control of the plaque diseases.[139,140] In June 1995, *The Journal of the American Dental Association* supplement, entitled "Caries Diagnosis and Risk Assessment," aptly defined in *narrative style* the current needs for oral-preventive strategies and management.[141] Earlier, Anderson et al. concisely pointed out the same steps were required for a comprehensive preventive dentistry plan.[142] Needed is a way to bring all the narrative information into a workable paradigm at the time of the initial/annual examination.

Guidelines, or standards target specific diseases, help establish a patient's level of risk and suggest approaches to diagnosis, treatment and maintenance to meet that level of risk. The guidelines are intended to help in decision making, as well as achieving a level of consistency of treatment throughout a population.[143] They should also increase the number of options to patient care, reduce patient costs, and improve the predictability of achieving desired outcomes.[144] *They should also leave room for professional judgment.* To accomplish these objectives, there are now efforts to develop nationwide professional guidelines of practice for both medical procedures[143-145] and for dentistry.[146-148]

As new research advances evolve for controlling or conquering the plaque diseases, any upgrading of standards should represent a consensus of the general practitioners, specialists, educators in dental and dental hygiene schools, research units, insurance servers and consumer groups. All guidelines should be subject to *continual review and change* as accomplished by the periodic updated recommendations by the American Heart Association,[149] the American Cancer Society and the American Academy of Periodontology.[150] As these guidelines continually evolve, parallel efforts are needed to educate the public and the profession about the new benefits

resulting from use of new procedures and technologies.

Putting it all Together—Examination, Treatment, Prevention

In the first edition of this text (1981), Harris introduced the concept of caries-activity indicators (CAIs) to develop a patient's oral-health profile ("scorecard") at the time of the initial/annual dental examination, and to link it with the treatment, prevention, patient education plans, and recall schedule.[151] This was followed in the second edition (1987) by Harris and Scheirton[152] suggesting that the same approach could be used for periodontal disease. These patient plaque disease profiles ("scorecards") can be used to match the severity of the evidence-based disease indicators with primary preventive dentistry responses commensurate with the severity of the plaque disease(s). This approach acknowledges that all individuals have different dental disease proclivities. Those at higher risk require more aggressive and closely monitored primary preventive therapies. Evidence-based indicators such as the plaque index, the bleeding index and semi quantitative microbiological testing should be employed to help establish whether a patient is at high, moderate, or low risk—and to adjust treatment and monitoring schedules accordingly.[153]

As an explanatory note, the CAIs and PAIs are *not being used as predictors* of impending plaque disease in terms of sensitivity and specificity;[154-156] instead they are being used *to estimate the patient's present vulnerability* and to take the necessary actions to return any indicator scores to as near zero as possible before the next appointment. A suggested approach to a guideline that uses the "scorecard" of the initial/annual examination to aid in selecting individualized treatment options is shown in Tables 23-1 and 23-2.

The first step following the entire examination is to secure a printout of the "scorecard" listing the oral health profile of the patient and the treatment urgency of each of the CAIs and PAIs. Tables 23-1 and 23-2 list the selected CAI or PAI clinical findings, laboratory tests, or indices considered important in the development of the plaque diseases and indicate the relative treatment urgency (risk) of the several indicators on a scale of 0 to 4. For example, if the finding for any of the caries indicators is negative, the zero in the appropriate column of the zero treatment urgency rows is circled—or entered into the computer. If, however, incipient or overt lesions are identified, circle the "1 or >1" listing found for each caries category in the Treatment Urgency 4 row. [Note: There is no intermediate possibility between Treatment Urgency 0 and 4—either a tooth is at risk due to incipient or actual lesions and should be given priority treatment, or it is at no risk]. In the example of Table 23-1, a Treatment Urgency of 4 is circled because of one incipient buccal lesion ("white spot") and two deep ("sticky") fissures—both without cavitation. The level-4 rating will be retained until the two fissures are covered with sealant, and remineralization therapy completed for the "white spot." If the two entries had been 0, the treatment urgency score would have been 3 because of the plaque index score. In these examples, the indicator values originally entered in the computer as part of the initial/annual dental examination are now highlighted (circled) in the appropriate columns and rows. The overall treatment urgency listing in the last column is based on the most urgent (highest number) entry in any of the columns, in this case, again 4 because of the incipient lesions and "sticky" fissures. With this Treatment Urgency of 4 established, a second printout is made that links the patient's "scorecard" to carefully

considered individualized primary preventive dentistry suggestions (Tables 23-3 and 23-4). Thus, the CAI and PAI "scorecards" provide profiles of each patient's caries and periodontal health, respectively. As a summary listing of priority needs, they are understandable to the patient for discussing informed consent for treatment and economic options. For the practitioner, they are valuable in helping to provide guidelines suggesting the treatment for each of the levels of treatment urgency listed on the "scorecard." Thus, with the integrated caries and periodontal examination "scorecards", the dentist and the dental hygienist can use the original examination data as a patient-motivation instrument by highlighting *specific* patient oral health problems—and solutions.

Once "scorecards" and guidelines have been completed for the office records, a copy of each should be given to the patient as part of the informed consent, education-counseling process, and to emphasize the commitment the dental office has to prevention. Vice versa, it identifies the areas requiring patient cooperation. If desired, a printout can be supplied that lists the progress (or retrogression) of any/or all of the indicator scores recorded over the past several examinations.

Question 4

Which of the following statements, if any, are correct?

A. The Periodontal Screening and Recording system (PSR) aids in diagnosis and suggests treatment options commensurate with the severity of the patients' periodontal status.

B. Chlorhexidine is used to suppress *both* cariogenic bacteria and periodontopathogens of the plaque.

C. Disease guidelines attempt to standardize disease diagnosis and to suggest treatment options based on disease severity, yet allow for professional judgment.

D. A nationally accepted guideline, i.e., one accepted by the American Dental Association, by state dental societies, by research authorities, dental school faculties, and by practitioners, would aid in improving uniformity of care and enhance the predictability of outcome of a disease process.

E. Computer storage of data contained in the initial/annual dental examination record permits convenient downloading of a patient's oral health profile ("scorecard") based on disease severity; with another keystroke, a guideline for suggested treatment, patient education, and preventive dentistry options can be displayed.

The Recall Appointment

The recall appointment provides an opportunity for planned screenings and prophylaxes between annual examinations with the time interval being based on CAI and PAI findings at each successive recall. Philosophically, the recall period of high-risk patients should not exceed the time necessary for a pathogenic plaque to reform and to again allow its microbial population to damage the enamel, cementum, and/or gingiva. Once the pathogenic plaque is removed by prophylaxis, several studies have

documented that it requires at least 3 months for the plaque to regain its disease-causing potential—even though there may be minimal compliance with home care plaque control programs.[157,158] Since *plaque and time* are the key factors in the development of both incipient caries lesions and gingivitis, each prophylaxis, by removing the pathogenic plaque, *essentially resets the time clock of plaque pathogenicity back to near zero.*

There is a need for a flexible recall interval, based *on the level of treatment need,* as suggested in Tables 23-3 and 23-4. Others have indicated that the same periodic oral hygiene procedures and monitoring are effective in reducing the incidence of *both* caries and periodontal disease.[116,159] If possible, the periodontitis and the caries maintenance visits should both be kept in synchrony on the same 3- to 6-month interval recall. In this way, a requirement for a 3-month recall for caries would aid in plaque prevention control for gingivitis, or vice versa, even though the *risk* of the other might be low. For example, fluoride applications for coronal caries would aid prevent root caries where the cementum is exposed. Chlorhexidine varnishes are effective in helping suppress *both the cariogenic and periopathodontic bacteria.*

Finally a high-plaque index can provide a warning sign for *either* caries or periodontitis. The index should ideally be 10% or less. When the plaque score increases markedly, it should cause professional concern. Additional education and counseling, laboratory tests and indices, such as bacterial quantification for caries, and/or the bleeding index for gingivitis is indicated. Thus, the accomplishment of a plaque index at each recall can alert the hygienist about the possible existence of *in situ* and incipient lesions for *either* or *both* of the plaque diseases, while the accompanying prophylaxes can greatly aid in averting the initiation and progression of *in situ* and incipient lesions for *both* diseases. Recall programs can be easily handled by use of computer programs. With very little training, an office manager or dental assistant can enter changes in appointments and generate programmed recall notices that are automatically printed out for regular mailing or e-mailing appointment reminders. Such timesaving and improved administrative practices are essential, even in small dental practices.

Carbohydrate Intake

The carbohydrate intake index provides an easy method to assess a patient's intake of refined sugars, especially if the intake is high. This sugar intake score is based on the patient filling out a carbohydrate intake short form administered by the dental office personnel. As the frequency of intake, and the retentivity of the different sugary foods increase, so does the carbohydrate intake score (Appendix 23-2).

The Dental Hygienist — Primary-Prevention Specialist

As a primary dental prevention specialist in the dental office, the dental hygienist is salient in insuring emphasis on prevention. Academic dental-hygiene education provides a comprehensive curriculum that includes, among other requirements, clinical participation in applying pit-and-fissure sealant use, remineralization therapy, fluoride applications, patient education and oral-health promotion, dietary counseling, prophylaxis, root scaling, root planing and subgingival irrigation—all areas of expertise that are needed for an optimum plaque- disease prevention and control

programs.

Under *a total immersion approach to prevention,* the "tooth-cleaning" appointments required for high-risk patients should *not* be perceived as prophylaxes performed mainly for aesthetic purposes, but rather as part of a caries and/or periodontal disease prevention and maintenance program which will require longer appointment periods. Shallhorn and Snider[160] and Pfeifer and Pfeifer[161] found that it took approximately 53 and 57 minutes, respectively, to complete a recall appointment. The multiple appointments of individuals in these more intensive preventive programs will require additional hygienist time and responsibilities—*and compensation.* Additional hygienists will probably be required as well as an assistant due to the increased patient flow.[162] In turn, to administratively support the increased enrollment of patients in preventive programs, there should probably be one front desk individual who can act as an appointment coordinator to ensure that recall visits are scheduled, and followed up.

If patient compliance with home mechanical and chemical plaque control instructions for caries and periodontal involvement has been adequate between visits, the treatment urgency level for the average patient should drop and remain at a low score. This possibility will permit additional patients to enter the preventive program to maintain a full and expanding schedule for the office dental-hygiene sector.

Patient Education

Traditionally, dentistry has been considered a treatment-oriented profession. The concept of going to the dentist to prevent disease is mainly associated with a twice-a-year prophylaxis and the use of fluorides. Probably the best way to start in the changeover from a treatment to prevention orientation is to conduct a separate appointment for *dental education and promotion devoted to the patient's own problems* as identified at the initial/annual examination (Appendix 23-3).[163] Like the annual medical examination and evaluation, this period should have a fee. It is essential to enlist the patient as a "co therapist" if a home prevention program is to be a success. For patients to assume this role, they need to know *what* is expected, *how* it is to be accomplished, *why* it is necessary and *how much* it will cost. This is the same information as is required as part of informed consent

At the end of the session, the patient should fully realize that he or she is at risk, but that the plaque diseases can often be prevented or reversed as a result of recommended therapy. As a part of medicine's "one third rule," one third of all patients with chronic disease can be expected to comply with instructions, one third to comply erratically, and one third not to comply at all.[164] It is also necessary to inform the patient that all treatment eventually fails without a wholehearted and effective self-care commitment. Only the patient can decide whether he or she wants to pay to prevent disease—or to accept disease and pay for more expensive treatment. If the patient continually fails to comply with instructions, it is expedient to document a warning advisory in the dental record to counter any possible future legal repercussions.[165]

One individual from the dental office should be selected to carry out the main thrust of the education program. The person selected should be a dental hygienist or a health

educator. The main attributes desired are that the person be mature, intelligent, and compassionate; has leadership ability; likes people; is persuasive; has the ability to improve on the daily presentations; and has the flexibility to adapt presentations to meet each patient's needs and attitudes. The entire education session can be facilitated by focusing on the "scorecard" listings for the CAIs and PAIs, especially when the Treatment Urgency level is above 2. At this time, the importance of plaque control can be introduced, using edited portions of the video camera tape to show the location and extent of plaque in the patient's mouth. The carbohydrate-consumption questionnaire previously filled out, becomes much more meaningful when discussed in context of the acid-producing capability of the bacteria in the patient's plaque. The need for laboratory tests to determine the number of acid-producing bacteria in the patient's saliva becomes equally consequential when it is made clear that the risk of decayed teeth increases as the number of acidogenic bacteria increases. As stated by Krasse, "To run a caries preventive program without using microbiological methods is like running a weight control program without a scale."[166] Finally, the relationship of these CAIs to *in situ*, incipient or overt caries lesions can be related to the demineralization that occurs between the times of tooth health to tooth cavitation. This backdrop then provides the basis for introducing and discussing the "guidelines" for customized preventive strategies for the patient's own treatment urgency level.

At this point, probably the two most important subjects related to *caries* prevention and requiring discussion with a patient are: (1) the need and advantages of having *sealants* placed in all "sticky" fissures; and (2) the need to consider all incipient smooth surface lesions (without evidence of cavitation) for *remineralization therapy* and careful monitoring—not restorations. The advantage of these two exceedingly important prevention actions are not generally known and understood by the public, nor actively promoted by the profession.

Plaque-induced gingivitis is the most common form of periodontal disease.[123] All patients should be cognizant of the fact that "pink toothbrush" is an important warning sign of gingivitis that can be self-diagnosed. If not cleared up within a week by more vigorous self-care efforts at home, an immediate visit to the dental office is indicated. A combined professional and self-care program is usually all that is necessary to return the gingival to normal. On the other hand the neglect of this critical early stage of gingivitis creates an environment in the gingival sulcus that favors a more pathogenic flora. Intermittent episodes of gingivitis at a younger age *increase the risk* of a later periodontitis.[167]

The data acquired as part of the initial/annual dental examination as summarized in the "scorecards" and guidelines can form the basis for individualizing the educational presentation. The integration of all these subjects should make the educational phase for each patient much more motivational and longer lasting than an abstract discussion of preventive dentistry.

Education of the Professionals

There is a need for the dental professionals to manage caries to the maximum extent possible by using non-invasive preventive measures. Both smooth-surface and pit-and-fissure lesions are preventable and reversible. Certainly, the restorative approach to caries control has not been successful, especially for those individuals without

funds nor access to dental care—both in the United States and especially in nations without the resources of the United States.

In the past, few dental schools provided courses in *primary* prevention that rival those of secondary and tertiary preventive dentistry in terms of time, money, staff and commitment. In 1989, in an editorial in the *Journal of Dental Research,* Thylstrup identified two major reasons that the benefits of prevention are not universally available. First, dental schools do not yet inculcate in their students the importance of preventing disease. Second, and probably, most important, *no reward* is given for the prevention or reversal of ongoing plaque diseases which have been proved reversible.[78]

Anusavice has added another reason that appears as innocuous as it is important, viz., "*We do not examine for, or monitor incipient plaque disease.*"[88] Until the smooth surface and occlusal sites of incipient caries are identified and *recorded for priority care,* little non-invasive remedial action will occur for these pre-caries lesions. Until CAIs and PAIs as well as summarizing "scorecards" for caries and periodontal disease appear as part of the clinical record, there is no organized format to present each patient with individualized invasive and non-invasive preventive and treatment options.

Two other reasons might be cited. The first is the fact that many of the older dentists find it *difficult to accept the concept of remineralizing pre-caries lesions* seen as radiolucent areas—sites that in former dental school days were considered as diseased tissue that must be restored. Concurrent with this viewpoint is the fact that once a "white spot" develops, there is an *over-estimation* of the velocity of caries progression through the enamel and dentin.[168] Now it is realized that caries advances over a greatly varied time span that can range from months, years or to reversal.[108] Equally important is the fact that the advancing front of the incipient lesion can probably be remineralized until it has, according to Scandinavian studies, advanced to half way through the dentin, i.e., if cavitation has not occurred before reversal takes place.[108]

In evaluating Thylstrup's charge that the preventive care curriculum in dental schools is truncated, the ADA's Survey of Curriculum Clock Hours of Instruction is enlightening. In the 1997-98 school year, U.S. dental schools devoted a mean of 66 hours of curriculum time to prevention. Didactic instruction on *prevention* included mean of 93 hours out of a mean of 5228 hours in U.S. dental schools' curricula—or approximately 2% of the time.[169] In addition, the ADA's Accreditation Standards require that "graduates are competent in providing oral health care within the scope of general dentistry—including health promotion and disease prevention."[170]

In a 1999 questionnaire, Yorty et al. detailed some of the ongoing preventive initiatives being conducted in the dental schools. Forty-two of the fifty-five dental schools (76%) responded to the polling. Not all the queries were answered uniformly. Eighty-one percent reported having formal *risk training programs,* with 38% having criteria for low-, moderate-, and high-risk patients. Sixty-two percent had developed specific recall schedules based on that risk. Two-thirds of the respondents (17/25) consider the option of remineralizing or sealant placement for early primary (incipient) lesions. Thirty-seven of the schools indicated that the level of penetration of the caries front before restoration, was the outer one-third of the dentin.[171] Clearly,

this study portends a trend towards a more conservative (preventive) approach to clinical dentistry.[171]

Economics can be a potent factor in changing health attitudes for both a patient and the professional. There is an acute need for *a reasonable reimbursement to dentists and dental hygienists practicing preventive dental care*, the same as is expected for dental treatment. The idea of purchase of good oral health is alien to Americans who have traditionally accepted restorations, extractions, and prosthetic devices as a means to cope with dental disease. The public *expectations* and *demand* for preventive dental care must change if the profession is to move into an era in which prevention replaces a need for restorations. In these opening years of this new millennium, the profession must prepare the *public* and *itself* for their new roles of dental prevention and full dentulism.

Clinical methods *now* exist to identify, arrest, or reverse the incipient plaque disease lesions that are precursors to overt lesions. It should be so easy to move the focus of dentistry to the practice of dentistry in this direction. The difference between prevention and treatment is simply an intact tooth, a restored tooth, or no tooth.

Implementing Preventive Programs

There are six major treatment environments in which the aforementioned intensive preventive routines can be implemented immediately: (1) private practice environments, (2) military dental services, (3) public-health clinics, (4) public school dental programs, (5) industrial work sites and (6) dental-health maintenance organizations. In each instance, the strategies for reducing the incidence of both of the plaque diseases, involve few changes in clinical physical facilities or in daily operating routine. Only a demonstration of leadership and a commitment to primary prevention is needed to identify and reverse the risk factors of impending plaque disease, rather than limiting the examination to a search for and treatment of pathology.

In the military, the economics of change from treatment to prevention should pose no major problem since appropriated funds are already available for dental care. In other dental settings, additional insurance and personal or public funds are initially required. However, a drastic reduction of later expenditures for the restoration of primary and secondary caries lesions, tooth fractures, periodontal treatment, endodontics, extractions, bridges, and dentures would soon compensate for the increased outlays from any of aforementioned sources. In private practices, it could lead to *contracts to prevent*, rather than treat dental disease

The Immunity Factor

In 1960, Keyes[172] and Fitzgerald and Keys[173] demonstrated that *S. mutans* caused caries in rodents. Once it was established that caries was an infectious disease, it was realized that caries might be controlled by use of vaccines. Several institutions in the United States and England were already accomplishing vaccine research. There was the *prediction* that a vaccine would be available within 3 to 10 years.[174] In 1979 Cohen reported on a 9-year study where two *actively* immunized monkeys had zero and a small lesion respectively, whereas the three surviving control animals exhibited

56,69 and 73 decayed surfaces.[175]

The 3- to 10-year time-table was not realized for one major reason, viz., it was suggested that with active immunity, there might be a cross reaction with heart muscle.[176] With that possibility, (1) even if a vaccine was developed, there was no assurance it would be accepted by the Food and Drug Administration (FDA). Even if approved by the FDA, potential manufacturers were wary of possible huge lawsuits.[177] Besides, caries was on the wane in developed countries and there appeared to be no urgency to risk major problems since caries was not a life-threatening disease. Much of the research stopped or slowed. Anticipation turned to disappointment. However, in Guys Hospital in England, the search was continued to develop a topically applied *passive vaccine* against *Streptococcus mutans.* This would by-pass the major problem of cross-reactions.

Every dentist should read the delightful easy-to-read article by JK-Ma.[178] It is a fascinating account citing some of the problems and successes that were encountered by the Guy's Hospital group over the past 20 years on the way to a prototype dental caries vaccine. The test vaccine has already successfully passed animal and a small-scale human tests. It is now in a Stage II test program with a larger number of subjects. The initial test answered two crucial questions, viz, (1) it is possible with the vaccine to *suppress Streptococcus mutans* (SM) and (2) to *prevent* caries. The vaccine is topically applied to the teeth, a process called *local passive immunization,* thus eliminating the possibility of involving the body's immune system. The antibody that has been developed by genetic engineering has targeted the *adhesins* responsible for the attachment of *Streptococcus mutans* (SM) to the tooth.

The development of the antibodies is a fascinating story in itself. In assembling the desired antibody by genetic engineering, it required four successive critical successes to develop the final antibody with the correct configuration to duplicate that of the human antibody. The amazing part of the genetic assembly process was the fact that each phase of the more-and-more complex antibody, was accomplished using the lowly tobacco plant that cannot ordinarily be induced to produce mammalian antibodies. Once developed, the continual harvesting of the plant *seeds* insured the perpetuation of future crops of tobacco plants, with each generation duplicating the original created antibody configuration. This plant biotechnology supplied an economical and reliable source of antibodies needed for the study.

The preparation of the mouth for application of the vaccine was also ingenious. A two-week mouth rinse program using chlorhexidine was sufficient to clear the mouth of most *Streptococcus mutans.* If no further action was taken at this time, the mouth was quickly repopu-lated with SM. However, if the vaccine was applied to the acquired (salivary) pellicle of the teeth, this repopulation did not occur. It is estimated that this passive immunity would possibly last from three months to a year. To increase the universal value of these antibodies, studies are being conducted as to the feasibility of incorporating them into dentifrices and mouthrinses.[178,179.]

A possible dental caries vaccine for the future? Remember, we are now in the beginning of the 21st Century.

Question 5

Which of the following statements, if any, are correct?

A. The twice-a-year prophylaxis is adequate in helping to prevent, arrest, and reverse the progress of either of the plaque diseases.

B. The plaque is an etiologic factor for both caries and periodontal disease; therefore, a prophylaxis at appropriate intervals and recorded observations for PAIs and CAIs should greatly help to prevent, arrest, or reverse both plaque diseases.

C. Remineralization of overt carious lesions is a common practice in Scandinavia.

D. The cost of primary prevention to prevent one overt plaque-disease lesion can save a much greater cost of later successive secondary- and tertiary- prevention procedures.

E. Mammalian antibodies can be duplicated in plants.

Summary

By definition, if all incipient caries and all incipient periodontal lesions could be prevented, arrested or reversed in the incipient stages, there would be no overt lesions to treat. In order to approach this goal for all people, it will probably require an active public-health immunization program of the immensity and intensity that characterized smallpox elimination. We have not yet reached that point of dental disease suppression; however, the completion of the Human Genome Project does greatly enhance that possibility—not only for dental disease but for all infectious and genetic diseases. At the present time, the ravages of the dental plaque diseases can be greatly minimized for those individuals who have access to a dental facility, and the commitment to comply with a dental prevention and treatment programs established by a dentist. Guidelines would help establish a level of excellence and uniformity for both treatment and prevention intervention. When called upon, it is the responsibility of the dentist to examine, detect and perform—or delegate—the necessary primary and secondary procedures necessary to assure maximum patient dental health in the present and in the future. The initial/annual examination is a most important event in attaining these objectives. Here it is possible to simultaneously identify and record treatment and preventive needs, establish present risk level, specifically focus counseling and education addressing patients' specific needs, as well as making recommendations for home care. Empirically, the home care involves daily oral-hygiene procedures—toothbrush (with fluoride toothpaste), dental floss, irrigation and supplemental mouthrinses recommended or prescribed by the dentist. The two most useful mouth rinses in the dentist's armamentarium are fluoride to increase tooth resistance and enhance remineralization, and chlorhexidine to suppress Streptococcus mutans, help cure gingivitis, and as an adjunct for treating periodontitis. If a short-time passive vaccine does becomes available, it will be a major step towards a lifetime of intact teeth for those who have routine access to dental care.

By the end of the initial treatment cycle, all overt lesions should be restored; all incipient smooth and root surface lesions should be undergoing remineralization therapy; all necessary sealants should be in place; and, all gingivitis should be under maintenance control. Also, by this time, the patient should have the knowledge and

understanding to participate with the oral-health professionals in a team effort of self-care. The outcome of such a comprehensive, integrated, and personalized plaque disease prevention program involves the participation of the entire office team plus the compliance of the patient. As a result of such a controlled prevention and monitoring program, there should be an early dramatic reduction in the incidence of the plaque diseases and their sequelae. This is as it should be—the hallmark of the dental profession should be oral-health maintenance and enhancement, not just disease treatment.

Answers and Explanations

1. D, E—correct.

A—incorrect. A "white spot" can occur on *any* surface; however, it cannot be visually observed on every surface. For instance, it cannot be seen at the bottom of pits and fissures. It is usually first observed as a radiolucency in x-rays of the interproximal surfaces.

B—incorrect. The incipient stage begins as a "white spot" and continues until there is surface cavitation, which by definition is an irreversible overt carious lesion.

C—incorrect. The *in situ* stage of gingivitis is characterized by an infiltration of body defense cells beneath the sulcular epithelium.

2. A, C, D, E—correct.

B—incorrect. The opposite is true. Much less x-ray energy is required for the DXR images than with the intraoral film.

3. A, B, D, E—correct.

C—incorrect. While "white spots" are a translucent white, once remineralized, they usually take on the hue of the enamel.

4. A, B, C, D, E—correct.

5. B, D, E—correct.

A—incorrect. The twice-a-year prophylaxis is adequate for the majority of people; however, there are many who could benefit from a more frequent schedule. In other words, the interval between prophylaxes should be flexible and be based on risk.

C—incorrect. Remineralization can only occur BEFORE the overt caries lesion stage.

Self-evaluation Questions

1. The two stages of caries development, prior to the overt lesion development are _____ and _____; the two stages of periodontal disease prior to periodontitis are _____ and _____.

2. Three advantages of the intraoral videocamera are: _____, _____, and _____.

3. Three advantages of the digital X-ray are: _____, _____ and _____.

4. List five reasons for having the problem of false-negative or false-positive diagnoses for caries: _____, _____, _____, _____ and _____.

5. How does a sealant *prevent* caries on the occlusal surface? _____. How does a sealant *arrest* early caries on the occlusal surface? _____.

6. Define *substantivity:* _____. Give an example of the agent in a mouthrinse with this attribute: _____.

7. Give the reason why patients are not demanding remineralization therapy instead of restorations (YOUR evaluation).

8. Why do you believe, or not believe that a vaccine can be developed for periodontal disease?

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