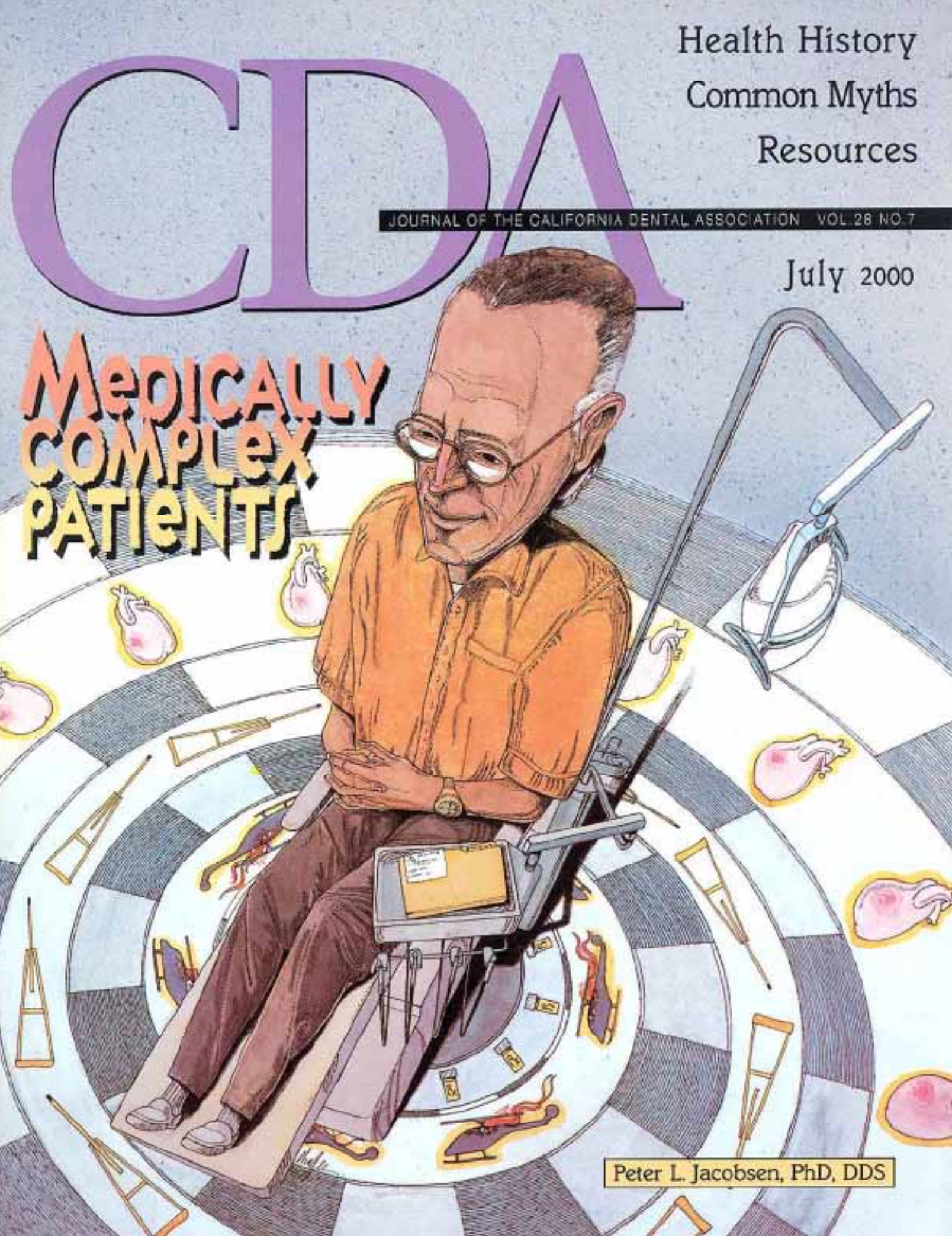
CDA

Health History
Common Myths
Resources

JOURNAL OF THE CALIFORNIA DENTAL ASSOCIATION VOL.28 NO.7

July 2000

MEDICALLY COMPLEX PATIENTS



Peter L. Jacobsen, PhD, DDS

OF THE CALIFORNIA DENTAL ASSOCIATION
Journal
CDA Journal
Volume 28, Number 7
JULY 2000

DEPARTMENTS

FEATURES

CDA JOURNAL, VOL 28, Nº7

A Vision of the Future

JACK F. CONLEY, DDS

Members of the California Dental Association who regularly read the CDA Update, participate in leadership activities in their local component societies, or serve on CDA committees have been well-exposed to many discussions and reports relating to the strategic planning efforts under way at the association. However, until the results of these planning efforts conclude and leadership approves the course of future activity, very little can be predicted with confidence about the direction that current and future programs will take and what kinds of issues the association will value as inherent to its purpose.

In mid-April of this year, Executive Director Tim Comstock addressed a group made up primarily of current and former association leaders. While Tim has been communicating regularly with membership via the pages of the Update, we found his remarks on this occasion to provide an insightful perspective on some of the directions the association will probably embark upon once the planning and approval processes are complete. What we found particularly valuable about Tim's remarks is that he has developed a firm understanding of some important trends that have been influencing the association and the future of the dental profession. They are trends that must be dealt with; and Tim, the person at our helm, showed that he understands the Big Picture -- his vision of the future CDA is coming into focus. He understands some of the major changes in course that must be considered if the association is to achieve the mission and purpose established for it in the current strategic planning process.

I felt that Mr. Comstock's presentation should be shared with all members with access to these pages. It should be helpful to long-time members in understanding some of the reasons for change. Newer and younger members, who have been concerned that membership in organized dentistry has not been fully addressing their professional needs, can take comfort that their concerns will be addressed with new energy in the future.

***

CDA -- 2000 and Beyond

Tim Comstock, executive director

It is a rubric of life, of war, of corporate America, and even of nonprofit professional associations, that structure follows strategy. It cannot be the other way around if you want to build and maintain anything that is responsive, creative, competitive, and capable of change.

I spent 20 years in the world of higher education. Despite its generally left-leaning politics and the individual dynamism and cutting edge pose of many of its members -- there is nothing in the world so conservative as a faculty governance group. Their structural model is specifically designed to ensure that only an act of God or a political cataclysm has even a slight possibility of nudging the enterprise in a different direction.

CDA JOURNAL, VOL 28, Nº7

The CDA/ADA system isn't quite as entrenched as is that of academe, but we've only been at it a bit over a century -- academia has a thousand years on us.

So, why do we need to change? We're strong, and our programs and advocacy serve our members and the profession with skill and honor. We have somehow wound ourselves into the ownership of two class A office buildings within a hundred yards of the State Capitol, and we own an insurance company with reserves in excess of $100 million. So, what's the rush to change?

Here are a few reasons:

- Because we are still operating on a model and with a system that is finely tuned for professional and cultural realities that existed in the 1950s and '60s.
- Because we've piled programs upon services, upon unfunded mandates, onto a staff that is exhausted trying to do it all. We've done this over the years without assigning any relative priorities. Therefore, we do a thousand things -- we are, or try to be, all things to all dentists -- and we aren't really sure that we're doing any of these things really well.
- Because the world has changed and is changing dramatically. John Welch, the innovative leader of General Electric, observed, "When the rate of change outside exceeds the rate of change inside, the end is in sight."
- Because 40 percent of our membership is over the age of 50. Within 10 to 12 years, they won't be paying dues; and we're not capturing sufficient numbers of new, younger members through our present mechanisms to even stay even.
- Because the ranks of new dental grads look and act so very differently from our present membership. What do they care about the advertising canons that compelled us to lay out hundreds of thousands of dollars in a fight with the Federal Trade Commission? How turned on do they get over our never-ending battle to bring fluoridation to California's water supply?

These things are, at most, abstract holograms to new dental grads and young practitioners. We're like the kindly old grandpa to these people. They acknowledge us -- they might even enjoy having us around. But, are we worth $1,500 or more a year out of their pockets -- just to keep around?

I don't think so. If we're going to engage them in our enterprise, we have to show them how and why we are relevant to them and how their being with us is a benefit to them.

Art Dugoni, past CDA and ADA president, and present pre-eminent dental dean, is one of the most respected voices in the country whenever the dental biz is discussed. And Art Dugoni is worried, deeply, about the future of dental education and, therefore, about the future of the profession of dentistry, organized and otherwise, in California and throughout the rest of the country.

Those who are now matriculating from school to the practice are different. They are not boosters and joiners like we were. The pressures on them are far greater and different. The debt they face at an early age, as they take their first steps into practice, is staggering. The rallying cry, our rallying cry, of fee for service only is but another abstraction to them. They've got to live, to eat, to pay off massive loans. Few among them have the capital to buy a practice or start up an office from scratch.

What can we do for them that is relevant and will inspire their trust and enthusiasm? What can we do to address the fact that our dental schools have hundreds of faculty positions unfilled and not fillable because they can't pay enough to attract qualified teachers?

I don't have all the answers -- none of us has all the answers. But, CDA has embarked upon a major program of self-examination in an effort to discover the answers and to become a relevant leader in addressing a multitude of problems that weren't even anticipated 20, 30, or 40 years ago.

Right now, we are at least looking to the future, although we're firmly set in the past and present. To get to the future, we have to be able and willing to drop the baggage from the past. Picture yourself running for a departing train while carrying a 200-pound suitcase. Your odds aren't good. I'm not saying there aren't some things in our suitcase that are valuable -- there are. But we need to judge with great care exactly what have to take with us on the trip we must take.

I see us four or five years down the road -- still solid and sound. I hope we'll be a bit larger and a whole lot more varied in our makeup. We'll likely have fewer programs and maybe fewer total staff in Sacramento. But, we'll do a better job on the programs we'll have. We will be a better partner to our component societies and to the ADA in what I hope is a better-defined and more cohesive tripartite than the rather loosely drawn confederation we see today. I see us, too, as a better teammate to the dental academic enterprise. Finally, I see a leaner, more responsive, and more proactive governance and council structure that will propel CDA into a leadership role in public health forums up and down the state of California.

The road from here to there won't all be smooth. Change upsets a lot of us; that's only human. But we must move on. And, there's a chance that we all won't be there at the end, or at least we all might not arrive there at the same time. I do know that it's a trip we have to take if we are to be a viable, key player in the affairs of our profession five, 10, and 20 years down the road.

As Dan Quayle once said, "If we don't succeed, then we run the risk of failure." Maybe John Kennedy said it a little better when he observed 40 years ago, "The United States has to move very fast even to stand still." His comment rings true for CDA. Now we stand ready to fashion our accommodation with the future.

CDA JOURNAL, VOL 28, Nº7

Research Shows No Fluoride-Bone Fracture Link

By David G. Jones

For more than a half century, fluoridation of public water supplies has been a flash point for voluminous debate. Fluoride opponents have continued their passionate harangues even as scientific evidence has piled up in support of the chemical's safety and efficacy.

Studies continue to be completed showing the hollow nature of shouted claims that fluoride causes all manner of harm. What those studies generally indicate is that fluoride helps ensure improved dental health and that it is especially important for children who do not have the benefit of access to dental care.

An argument currently in vogue with fluoride opponents is that people who ingest fluoridated drinking water are more susceptible to bone fractures as they age. And while they are pointing to studies as their evidence, those "studies" are not standing up to the standards of true science.

The most frequent finding of legitimate studies focusing on the relationship between ingestion of fluoridated water and bone fractures is that there is no connection.

For example, a case-control study's results in England were reported comparing about 500 men and women aged 50 and older with hip fractures to a similar number without the fractures. The results, published in the Jan. 22 issue of The Lancet, showed the chance of fracturing a hip was the same for those who drank water containing fluoride at concentrations of about 1 part per million as for those whose water contained less fluoride.

In another study, 7,500 Chinese in 60 villages were followed for six years to determine the relationship between bone fracture prevalence and long-term natural fluoride exposure in a range of concentrations bracketing 1 part per million.

Barry P. Katz, PhD, director of Indiana University's Division of Biostatistics in the Department of Medicine says he got involved in the middle of the study in 1997 to determine fluoride's effect on bone fracture.

"The basic data show a public health benefit of the lowest fracture rate at between 1 and 1.06 parts per million, which coincides with the level of water fluoridation in the United States," Katz says. "We also looked specifically at hip fractures. We had fewer hip fractures than we had hoped for statistical analysis, but the results seem to support the other data in our study. The results of both parts of the study seem to support the study in England."

Katz says that the study in China was significant because it centered on elderly people who had lived their entire lives in rural farming towns, so the study involved lifelong fluoride exposure.

In a third recent study, more than 7,000 women in the United States were followed for seven years to compare bone fractures in those exposed to water fluoridation for the past 20 years compared to women with no water fluoridation exposure.

Kathy Phipps, who holds a doctorate in public health, was the principal investigator. She is an associate professor at Oregon Health Sciences University in Portland, and has been doing research on the impact of fluoride on osteoporosis and fracture rates for about 10 years.

"We found that there was no increase in hip or vertebral fractures in women exposed to fluoridated water," Phipps says. "In fact, fluoridation in this study had a slightly protective effect."

Phipps said her study was the largest on community water fluoridation and fractures to date. Her research paper was recently accepted for publication in the British Medical Journal.

Robert Selwitz, DDS, MPH, senior dental epidemiologist at the National Institute of Dental and Craniofacial Research

More Evidence Supports Perio-Preterm Birth Link

The more of the mouth affected with periodontal disease, the more likely a woman is to deliver a premature baby, according to an ongoing study of more than 2,000 pregnant women.

The results offer further indications that periodontal disease may be a significant risk factor for preterm births.

Past studies have shown that women with periodontal disease may be up to seven times more likely to deliver a preterm low-birth-weight baby. Preliminary research was recently presented at the American Academy of Periodontology's Specialty Conference on Periodontal Medicine suggesting that the risk for women who have generalized periodontal disease (affecting at least 30 percent of their mouth) is even higher.

"We were amazed by these findings," said Marjorie Jeffcoat, DMD, the study's lead researcher and chair of periodontics at the University of Alabama at Birmingham School of Dentistry. However, women with only a couple of sites in their mouths affected by periodontal disease aren't in the clear. "While there is a definite dose response, the trend for preterm birth was observed in women with as few as two sites with attachment loss," Jeffcoat said.

The women in the study had no other major risk factors for preterm birth.

Office of Science Policy and Analysis, says some studies that seem to support a link between fluoridation and bone fractures must be regarded skeptically.

"Many studies indicating a relationship between water fluoridation and bone fracture are referring to ecological studies that have inherent weaknesses, since fluoride histories for individuals involved were never obtained," Selwitz says.

Selwitz says there are also many confounders unaccounted for by ecological studies, such as individuals' estrogen levels or use of estrogen and vitamin D supplements, smoking, occupational exposure, and dietary patterns that might influence differences in bone disease rates in fluoridated and nonfluoridated areas.

Phipps says she controlled for all of the confounding factors.

"And over the last 10 years, we've moved from ecologic studies to looking at more individual level studies, which allow for greater control," she says. "Based on our and others' current high-quality research, community water fluoridation does not increase an individual's risk of hip fractures."

Selwitz says there is little real evidence linking fluoride and broken bones.

"The sum of the evidence to date appears to indicate that water fluoridation does not increase the risk for bone fracture," he says. "But more research is needed."

Phipps says she believes that the three recent studies allow the scientific community to lay the issue to rest.

"The only population group we haven't looked at in our country is elderly men, but there's no reason to assume there would be a different effect on them compared with women," she says.

Protecting the State of Your Estate

By Marios P. Gregoriou

You've spent years growing your wealth and building your estate, so it is just good sense to plan to protect your assets and pass them on to your beneficiaries according to your wishes. Here's how:

- Write a will. If you do not have a will when you die, the law of your state may determine what happens to your estate, your assets, and any minor children. In addition, the state process, usually governed by probate court, is often slow, sometimes expensive, and open to the public.
- Fund a living trust. Follow through if you set up a living trust. Until you transfer ownership of property or assets to it, the trust is not worth any more to you or your beneficiaries than the paper it's printed on. Unfortunately, many revocable living trusts are set up but remain unfunded.
- Re-title joint-tenancy property. Joint-tenancy-with-right-of-survivorship titling of assets may result in estate planning headaches. Although probate is avoided at the first joint owner's death, it is not avoided at the death of the survivor, thus only delaying estate taxes. Re-titling assets to a credit shelter trust can help avoid probate and provide estate tax savings.
- Use both spouses' applicable exclusion amount. Leaving all property and assets to a spouse may avoid estate taxed at the death of the first spouse, but this approach wastes the gift and estate tax credit of the first to die. A credit shelter trust can maximize each spouse's credit, thus sheltering more assets from estate tax liabilities.
- Re-title ownership of life insurance policies. Most life insurance policies are owned by the insured, causing the policy's face amount to be included in that person's estate at his or her death. Policy owners may consider giving policies directly to the beneficiary or transferring the policies to an irrevocable life insurance trust. Either strategy could help reduce estate taxes.
- Choose an appropriate executor. Naming an inexperienced family member as executor could complicate the demanding task of settling your estate. This is especially true at a difficult and emotional time following a death. Look into the benefit of naming a professional organization to follow through with the duties of an executor.
- Organize your paperwork and files. If you do not provide your executors and beneficiaries with all the paperwork or files pertaining to your property, assets, and wishes, improper distribution and management of your estate may result.
- Update your estate plan. Updating your estate plan from time to time is important so that it is implemented exactly according to your wishes. You will want to update your estate plan when there are changes in your family (births, marriages, divorces, deaths, for example); when the value of your estate significantly increases or decreases; when tax laws change; if you move to another state; or if your profession or career changes.

Marios Gregoriou is associate vice president financial adviser with Morgan Stanley Dean Witter in Sacramento. He can be reached by calling (800) 755-8041. This article is published for information purposes and is not an offer or solicitation to sell or buy any securities or commodities. Any particular investment should be analyzed based on its terms and risks as they relate to your circumstances and objectives.

CDA JOURNAL, VOL 28, Nº7

Doxycycline Enhances Effects of Root Planing on Gum Disease

A recent clinical trial found that subantimicrobial dose doxycycline significantly improved the results of scaling and root planing in the treatment of adult periodontitis.

The study was published in the May 2000 issue of the Journal of Periodontology.

"Data from this study suggest that [subantimicrobial dose doxycycline] may have an important role in the successful long-term management of adult periodontitis," says lead investigator Jack G. Caton, DDS, MS, professor of periodontology at the University of Rochester's Eastman Dental Center. "These results demonstrate that effective nonsurgical treatment may require a two-pronged approach aimed at both reducing bacterial load through the use of [scaling and root planing] and suppressing the destructive enzymes that are produced in response to bacteria, utilizing [doxycycline]."

Caton and colleagues report that twice-daily adjunctive treatment with 20 mg capsules of doxycycline hyclate yielded a substantial improvement in clinical attachment levels and probing depths compared with scaling and root planing and a placebo capsule.

The multicenter, double-blind clinical trial was conducted at five dental schools: the State University of New York at Buffalo, West Virginia University Medical Center, the University of Florida at Gainesville, the University of Pennsylvania, and the Eastman Dental Center at the University of Rochester.

Study Cites Varied Reasons Children Cry in the Dental Office

A crying child in a dental office can make treatment next to impossible if the child is not calmed down and doesn't cooperate with the treatment, but the child may not be crying out of fear or pain, write Dan Zadik, DMD, and Benjamin Peretz, DMD, in the January-February 2000 Journal of Dentistry for Children.

While it is easy for children to utilize fear in the dental office as a catchall reason for crying, there may be other explanations for the child's behavior.

Since children cry for a variety of reasons, dentists should have a wide range of behavior management methods to meet the needs of individual children. The authors refer to H.G. Elsbach's theory that crying is a mode of expression and should be used as a diagnostic tool. Elsbach classified four types of crying: fear-motivated crying accompanied by hysterical behavior, pain-motivated crying, compensatory-motivated crying, and steady-state siren-like crying.

The authors cite a study involving 104 parents who accompanied their children, ages 2 to 13, for dental treatment at the Department of Pediatric Dentistry at the Hebrew University-Hadassah Faculty of Dental Medicine in Jerusalem. Each child had at least one session for an operative procedure after an initial evaluation. All parents were present in the operatory during treatment.

About 48 percent of the children fully cooperated in previous dental procedures, while 24 percent exhibited limited cooperation and 28 percent were totally uncooperative. The mother accompanied most children (61 percent), while 36 percent came with the father. Four children came with a grandparent.

Findings showed that when a child cried with no evidence of pain, most parents preferred stopping treatment and calming the child before resuming treatment. Most parents also felt they had to assist the operator in calming the child.

One aspect of the study showed that at least some of the children were not fearful or even excited, but either cried to avoid the treatment or had a general tendency to cry. Stopping treatment for these children may serve as a reinforcement to continue crying, and for these children a "matter of fact" approach is the method of choice, the authors write.

Zadik and Peretz stress that dentists should acquire a repertoire of behavioral and cognitive skills to regulate patients who cry. With effective guidance by the dentist, a child can learn to collaborate during the dental experience, they note. The successful completion of treatment, they say, is viewed as a partnership between the dentist and parent. It is, however, important that the dentist inform the parent about the method to be used and receive the parent's consent.

Disabled Dentists Sought for Study

The Oregon Health Sciences University is conducting a qualitative investigation on the education and practice of dentists with disabilities. OHSU invites such dentists to share the aspects of their education and profession that have been positive and/or negative in relation to their disability.

To participate, contact Jared Schultz, project coordinator, at (503) 232-9154, Ext. 128.

Family History Leads to Gene Disorder Discovery

The study of a family's unusual history of missing teeth dating back to 1645 has led researchers at the University of Texas-Houston to identify the responsible gene, reports Lisa Garvin of the University of Texas-Houston Health Science Center Department of Public Affairs

According to an article in the January/February 2000 American Association of Orthodontists Bulletin, a mutation of the gene known as PAX9 is the cause of a rare form of oligodontia in a Houston family.

Study co-author Dr. Monica Goldenberg, an orthodontic resident, noticed a 13-year-old patient was missing 14 permanent teeth. After questioning the mother, Goldenberg learned the patient's father and two brothers had the same condition. In 43 family members, 21 were found to have missing molars.

DNA samples from affected family members revealed an extra nucleic acid on the PAX9 gene, which disrupted the normal protein sequence and caused the defect. The mutation is dominant, meaning offspring of an affected parent have a 50 percent chance of inheriting the missing-tooth trait, and it appears in every generation.

The study marks the first time a human disorder has been linked to a mutation on PAX9, which is one of a family of nine master genes that regulate organ development.

Others involved in the study and gene analysis were Drs. Rena D'Souza, Marion Messersmith, Parimal Das, David Stockton, and Pragna Patel.

Details of the study are in the Jan. 1, 2000, issue of Nature Genetics magazine, and more details on the description of the family will be published in the Journal of Dental Research.

D'Souza is interested in identifying other families with multiple congenitally missing teeth and can be contacted at (713) 500-4218 or by e-mail at rdsouza@mail.db.uth.tmc.edu .

Honors

The American Association of Dental Schools has named Arthur A. Dugoni, DDS, as the third recipient of the AADS Distinguished Service Award. Dugoni is dean of the University of the Pacific School of Dentistry.

CDA JOURNAL, VOL 28, Nº7

First, Do No Harm

PETER L. JACOBSEN, PHD, DDS

AUTHOR

Peter L. Jacobsen, PhD, DDS, is the director of oral medicine at the University of the Pacific School of Dentistry.

First, do no harm. This directive is central to providing health care and is a part of the Hippocratic oath. This issue of the *Journal of the California Dental Association* is dedicated to the management of the types of patients to whom, inadvertently, it may be possible to do more harm than good. This group of patients is commonly referred to as medically compromised or, more appropriately, medically complex.

The obvious and essential first step in ascertaining whether a patient is medically complex is to take a good medical history and follow it up with an interview. The opening article by my colleagues and I provides a revised, updated, and complete health history. We also provide an interview sheet that is formatted to ensure that the most important health history questions are properly asked and that the patient fully understands the health history questionnaire. The questionnaire has also been translated into 10 languages, which make up 95 percent of the languages, other than English, spoken by Californians. The use of a translated questionnaire ensures that the questions are properly understood. As importantly, it documents that dental practitioners and the California Dental Association are committed to the highest quality of dental care for the state's diverse population.

This issue also contains excellent articles on specific aspects of medical complexities. Michael J. Wahl, DDS, in his article, "Demystifying Medical Complexities," looks at some of the dental mythology regarding medical management. Richard L. Wynn, PhD, has written a review of medications used for cardiovascular disease and their affect on dentistry. He provides some excellent protocols on how to safely manage patients on anti-coagulants.

Finally, because medically complex patients are, well, complex, and there is always more to be known, Lewis R. Eversole, DDS, MSD, MA, and William M. Carpenter, DDS, MS, have provided a list of resources, including the American Dental Association and National Institutes of Health, from which additional information can be obtained about specific medical problems or treatment protocols. Certainly there are some worthwhile Internet resources to bookmark for quick access.

Next month's Journal will contain four more articles to help complete your library of information on the frequently encountered challenge of doing no harm.

CDA JOURNAL, VOL 28, Nº7

An Updated Multiple Language Health History for Dental Practice

Peter L. Jacobsen, PhD, DDS; Richard Fredekind, DMD, MA; Alan W. Budenz, MS, DDS; William M. Carpenter, DDS, MS

ABSTRACT To properly assess a patient, a variety of questions are necessary to evaluate signs and symptoms of medical problems and to find out about diagnosed medical problems and specific medical treatments, including the use of drugs or medications. All of these factors can have a bearing on dental management. Because of the diversity of the population, a variety of languages are spoken in California and the United States. So that dentists can communicate with those diverse groups, a health history form developed at the University of the Pacific School of Dentistry has been translated into 10 languages. The translated forms and the corresponding English form are presented along with an explanation and rationale for their use.

AUTHORS

Peter L. Jacobsen, PhD, DDS, is the director of oral medicine at the University of the Pacific School of Dentistry.

Richard Fredekind, DMD, MA, is an associate professor in the Department of Diagnosis and Management and a group practice administrator at UOP School of Dentistry.

Alan W. Budenz, MS, DDS, is an assistant professor of anatomy and chair of the Department of Diagnosis and Management at the UOP School of Dentistry.

William M. Carpenter, DDS, MS, is professor and chairman in the Department of Pathology and Medicine at the UOP School of Dentistry.

First, do no harm. That is a basic tenet in the delivery of health care. A complete medical history is the required centerpiece of every patient evaluation, ensuring that we do no harm. In dentistry, the medical history has always been considered an integral part of the initial interview process and the ongoing care of a patient. It is designed to identify medical problems or conditions that would put the patient (or the practitioner, in the case of infectious diseases) at an increased risk during dental procedures.

Most dental practices will have a patient complete a health history questionnaire, which makes the process more efficient and ensures that no questions are missed. The validity of the health history questionnaire is well-documented.[1]

This questionnaire must be followed up with a verbal interview to ensure that the patient properly understood the questions and to give the clinician an opportunity to ask about any positive responses.

The health history has become increasingly necessary and appropriate because a growing number of patients have ongoing medical problems. An extensive survey of more than 29,000 dental patients in the Netherlands revealed that 1 out of 5 answered "yes" to having a medical problem.[2] As patients grow older, they have more medical problems, the problems are more severe, and the potential for complications during dental therapy increases.

All patients, even young and apparently healthy ones, require a thorough health history review. The incidence of disease

CDA JOURNAL, VOL 28, Nº7

increases with age, however; and statistics clearly indicate that the population as a whole is aging. Medications are allowing medically compromised people to live longer and remain mobile. Patients are also keeping their teeth longer and, therefore, need and desire more dental procedures. All of these factors create a dental, medical, and legal environment that requires a current and thorough medical history.

The Department of Pathology and Medicine and the Department of Diagnosis and Management at the University of the Pacific School of Dentistry have developed a thorough health history questionnaire to evaluate all patients presenting to the school. This questionnaire is up-to-date and conforms to all the legal requirements for a health history (see Health History form). The accompanying health history interview sheet completes the medicolegal requirements for a thorough evaluation and assists the practitioner in ensuring that no medical problems have been overlooked (See Health History Interview Sheet form).

Based on the diverse populations seen at the dental schools in California, as well as in the rest of the United States, the University of the Pacific health history has been translated into several languages. The numbering and sequence of all the questions on all the health histories is exactly the same. This allows the correlation of the health history questions. The questions on one health history form exactly match the same numbered question on the other health history forms. An English practitioner can effectively evaluate a Vietnamese-speaking patient's medical problems by using the appropriate health history translation and then correlating it to the English version. For that matter, a dentist who speaks primarily Spanish or any of the other included foreign languages, can effectively evaluate an English-speaking patient by correlating the health history forms.

The 1990 Census3 revealed that although English is the official U.S. language, a significant number of people in California primarily speak another language at home. In California, Spanish is the second most common language spoken at home, although other languages are also relatively common (**Table 1**). The languages selected for interpretation range from those spoken commonly in California (Spanish, Cantonese) to the relatively obscure (Hmong). Although the Hmong speakers do not account for large numbers, relative to the population of California, they make up a significant population group in certain locales and in some individual dental practices. Being capable of communicating with diverse populations is crucial to those patients and those dentists.

TABLE 1.

Language Spoken at Home

(1990 Census, California; Total surveyed 27,383,547)

Language	Number of people	% of population
English	18,764,213	68
Spanish	5,478,712	20
Chinese	575,447	2
Tagalog	464,644	2
Vietnamese	233,074	1
Korean	215,845	1
Japanese	147,451	0.5
Russian	44,978	0.1

The series of dental-related health histories that follow make up the largest collection of correlated health history translations for dental offices in the world. The health history itself and the number of languages into which it has been translated have been updated since its prior publication in the *Journal of the California Dental Association* in 1993.4

A sensitivity to and an appreciation for the diversity found in California and in specific practices ensures a high standard of personalized professional dental care. This respect is an important first step in establishing appropriate patient rapport and optimal working relationships.

Health History Form

The health history form is divided into specific segments designed to elicit different types of information from the patient.

General Questions

Section I is designed to elicit general information about the patient's health and whether they have seen a physician recently, are currently in pain, or have had any problems with prior dental treatment.

Signs and Symptoms

Section II focuses on various signs and symptoms that are indicative of medical problems. Signs are indications of disease that can be observed by the practitioner, e.g., swollen ankles. Symptoms are problems that, though often indicative of disease, can be experienced only by the patient, e.g., the pain from a toothache.

As can be noted in this section and others, no time frame is given for the problem or complaint. Therefore, the patient may answer "yes" for a sinus problem that had occurred many years ago. This is intentional because judging the relevance of the time frame is the responsibility of the clinician. Pertinent information may be missed if the questions are restricted to problems that have occurred in the past year. A "yes"

CDA JOURNAL, VOL 28, Nº7

response may require a medical consult for a definite diagnosis.

Specific Diseases

Section III concentrates on specific diseases that have been previously diagnosed. All of the diseases, signs and symptoms, and other questions on the health history have dental relevance. Again a medical consult may be required depending on the current situation.

Three changes have been made to this section in this revised version of the health history. Asthma has been added to item #35 because of its increased frequency in the population and the potential of a flare-up during dental treatment. Due to the increasing frequency of latex allergies and the prevalence of latex-containing supplies in dentistry, item #38 now asks about latex allergies specifically. Finally, ARC (AIDS-related complex) has been omitted from item #40 because it is a term and concept no longer used in describing HIV-disease.

Treatments

Section IV discusses medical treatments and prosthetic devices, which can have a bearing on dental management. Decisions regarding dental management depend on the patient's specific situation and the extent of the treatment and/or resultant outcome.

Medications and Drugs

Section V elicits important information on prescription and over-the-counter medications, natural remedies, and any other drugs the patient might be taking. This new health history has incorporated a prompter about natural remedies because the use of these compounds is so widespread. One study found that up to 42 percent of the population has utilized "alternative medicine" therapies at least once in the previous year.[5] The current medication history is extremely pertinent and documents the extent of any problems identified on other parts of the health history (and at times the existence of problems not identified by the patient). This information can have a bearing on management as well as the potential for adverse drug reactions. The tobacco and alcohol use and history should also be addressed by the dental professional.

Women Only

Section VI elicits specific information relative to women such as pregnancy, nursing, and use of birth control pills.

All Patients

Section VII consists of a catchall question designed to elicit information the patient believes is appropriate to provide but which has not been otherwise queried.

It is then appropriate for the patient to sign and date the health history form. The patient should review the health history at selected intervals and re-sign it after each review thereby documenting that there have been no changes. The frequency of review depends on the practitioner's preference and the volatility of the patient's medical status. Most commonly, offices have patients review and update their health histories approximately every six months to one year, usually at their hygiene recall visit. If there have been any health changes, it is appropriate for the clinician to review the pertinent information and re-sign the interview sheet to document the review.

Health History Interview Sheet

The health history interview sheet ensures that any questions answered positively are followed up appropriately and documented. It also provides a location for any significant findings and a description of any dental management considerations. Medicolegally, this also allows a separate area for the dentist's edits, comments, and notes. The dentist should not alter the patient questionnaire or add notes on that sheet. If the dentist believes an edit on the questionnaire is crucial for clarification, any edit should be dated and initiated by the patient. The most important set of reminders on the interview sheet are the six questions on the right-hand side, which should be asked of the patient even if the patient has already answered them in the negative on the questionnaire. These six areas are extremely important in dentistry, and it is appropriate to ensure that the patient properly understood the intent of the questions. These crucial questions are as follows.

Cardiovascular

Cardiovascular problems make up the bulk of the medical problems that require dental management considerations. One study estimates that approximately 51 percent of those patients with medical complexities have cardiovascular problems, and the incidence of cardiac problems increases rapidly as patients age.[2] Most specifically, heart problems or heart murmurs should be investigated. Patients may or may not understand the term "cardiovascular," but if they have had a heart problem or murmur, they will recognize that terminology and answer appropriately. Frequently, a patient will answer "no" to "heart murmur" on the questionnaire but acknowledge the diagnosis of such during the interview.

Infectious Diseases

This is designed primarily to define any active infectious problem. Hepatitis is the most common infection with dental implications or complications. Another important infectious disease is advanced HIV infection or AIDS because of concurrent oral problem and systemic changes. Both hepatitis and HIV disease can lead to hematologic changes (e.g., increased bleeding) and problems with healing. Since all patients are treated as though they are infectious (universal precautions), this section is not designed to elicit information that would alter infection control protocols. Infection control protocols should be the same for all patients, except for active tuberculosis patients. This information on infectious diseases may also be important in the management of a parental exposure incident, if one occurs.

Allergy to Medicines

Allergies are always a concern in dentistry because of the variety of medications used. Penicillin and aspirin have relatively high allergic rates; and the responses can be very severe, including life-threatening anaphylactic reactions. A patient should be asked about allergies in general and if they have allergies to antibiotics, pain medications (including aspirins), local anesthetics, or narcotics specifically. (Allergies to medications used in dentistry are extremely rare and often based on a previous anxiety-based adverse response). A patient's reaction to drugs still needs to be identified even though they may be nonallergic reactions such as side effects, idiosyncratic reactions, and responses to toxicities.

Hematologic

Positive answers relative to prolonged bleeding or bruising may identify a bleeding problem. This problem can occur as a result of many conditions, including the use of medications such as aspirin.

Medications

As on the written health history, the medications a patient is taking reveal extremely important information about his or her medical condition. It documents that the nature of the medical problem is enough to warrant medical treatment. It also provides information for awareness relative to particular side effects, problems with certain medications, and potential for certain drug interactions. Information about over-the-counter, natural, and herbal medications should also be elicited and recorded.

Other Medical Problems Not Asked

In a comfortable one-on-one confidential setting, this catchall question may elicit information about medical problems that may have a bearing on dental therapy. This question may also uncover a variety of anxieties or concerns a patient may have but will not write down. A patient may be more open to discussing such concerns once he or she has established rapport with the practitioner.

Summary

The incidence of medical problems increases as the population ages and more medically compromised people are ambulatory and present for dental care. A patient-generated health history and the doctor-conducted health history interview are the standard of care, a medicolegal requirement, and a crucial aspect of proper dental patient management.

To properly assess a patient, a variety of questions are necessary to evaluate signs and symptoms of medical problems and to find out about diagnosed medical problems and specific medical treatments, including the use of drugs or medications. All of these factors can have a bearing on dental management. Because of the diversity of the population, a variety of languages are spoken in California and the United States. To communicate with those diverse groups, the UOP health history has been translated into 10 languages.

The utilization of a fully interpreted health history ensures that adequate information is elicited from the patient and illustrates the respect and concern a practitioner has for each patient.

Since the questions on the various translated questionnaires all correspond numerically with each other and with the questions in English, a positive response in a language not spoken by the clinician can be correctly interpreted and appropriate interview questions completed. Of course, if adequate follow-up cannot be conducted because of a language barrier, an interpreter can be requested to ensure that no medical problems arise during dental therapy because of confusion about the medical history.

Acknowledgments

The authors would like to acknowledge the contributions of the faculty the Department of Pathology and Medicine and the Department of Oral Diagnosis and Patient Management at the University of the Pacific Dental School. They have put many hours into developing the health history and perfecting it over the past several years. Their academic input has been crucial to the quality of the health history.

The authors would also like to acknowledge the following people for their efforts toward translating the English version of the health history into their native languages. Their contributions, time, and effort were invaluable and are much appreciated.

- Chinese – Dr. Hai Loo, Dr. Stephen Yao, Dr. Polly Chan
- Farsi – Dr. Katayoun Alaei, Dr. Soheil Goel, Dr. Faroud Hakim
- Hmong – Mr. Hai Her, Ms. Mao Her (dental student)
- Korean – Dr. Myoung Lee, Ms. SuJin Mayeda
- Laotian – Mr. Nai Her
- Russian – Dr. Leonid Tolstunov, Ms. Yelena Ostrovsky (dental student)
- Spanish – Dr. Guillermo Canjura, Garrett Guess (dental student), Dr. Noelle Fannuci, Dr. Oriol Llena
- Tagalog – Dr. Bert Masangkay
- Thai – Dr. Noellette Falkow, Dr. Warasiri Pitakanonda
- Vietnamese – Dr. Lich Khoung, Ms. Chan Long Nguyen (dental student)

Transcend of Davis, Calif., typeset and certified the translations.

References

1. De Jong KJM, Borgmeijer-Hoelen A, and Abraham-Inpijn L, Validity of a risk related patient administered medical questionnaire for dental patients. Oral Surg, Oral Med, Oral Path 72:527-33, 1991.
2. Smeets EC, De Jong KJM, and Abraham-Inpijn L, Detecting the medically compromised patient in dentistry by means of the medical risk related history. Prevent Med 27:530-5, 1998.
3. Census of Population and Housing, 1990, Summary Tape File 3, CA. Population: Language Spoken at Home/prepared by the Bureau of the Census. The Bureau State Census Data Center, Washington, 1991.
4. Jacobsen PL and Fredekind R, A multiple language health history for dental practice. J Cal Dent Assoc 21(5):25-7, 1993.
5. Eisenberg DM, Davis RB, et al, Trends in alternative medicine use in the United States, 1990-1997. J Am Med Assoc 280:1569-75, 1998.

To request a printed copy of this article, please contact/ Peter L. Jacobsen, PhD, DDS, UOP School of Dentistry, 2155 Webster St., San Francisco, CA 94115 or at pjacobse@uop.edu.

CDA JOURNAL, VOL 28, Nº7

Demystifying medical complexities

Michael J. Wahl, DDS

ABSTRACT Although dentists often fear treating medically complex patients, in many cases this fear may be based not on scientific facts but rather on a "mythology" of the dangers of dentistry. Dentistry is a remarkably safe profession, even for most medically complex patients. The myths of endocarditis, artificial joint infections, local anesthetics and vasoconstrictors, dental surgery in anticoagulated patients and patients on antiplatelet drugs, and antibiotic interference with oral contraceptives are discussed. Although dental treatment is not usually a risk factor for endocarditis, practitioners should consult the 1997 American Heart Association statement for recommendations for endocarditis prevention. Most artificial joint patients should not receive antibiotic prophylaxis. Local anesthetics and vasoconstrictors can be used safely in most medically complex patients. Neither continuous anticoagulation nor antiplatelet medications should be withdrawn for dental surgery. Scientific studies have failed to document an interaction between antibiotics used in dentistry and oral contraceptives.

AUTHOR

Michael J. Wahl, DDS, is an assistant attending dentist at Christiana Care Health Services in Wilmington, Del., and a clinical assistant professor of surgery at Allegheny University Hospitals. He is also in private practice in Wilmington, Del.

Many dentists, from those just out of school to experienced practitioners, approach dental treatment of medically complex patients with a mixture of two emotions: fear and paranoia. Fortunately, dentistry is not really as dangerous as many dentists are taught. Dental treatment has a remarkable safety record that is the envy of other health care professions.

Many things in dentistry that are done because they have always bee done. Many dentists may recommend certain types of materials, techniques, or treatments in patients because that was the way they were taught. Tradition is not necessarily a good way to practice dentistry.

Dentists are frequently taught that physician consultation can provide easy answers to the confusing questions of how to treat certain patients while also insulating the consulting dentist from liability in malpractice suits in the event of an unfavorable outcome. Actually, the dentist (not the physician) is ultimately responsible for the dental treatment.[1-3] Physician consultation, rather than insulating the dentist from liability in malpractice suits, can actually be a contributing factor in a malpractice suit, especially if the dentist relied on medical advice that the dentist knew or reasonably should have known to be erroneous.[4]

For example, a physician may recommend (unnecessary) antibiotic prophylaxis to prevent endocarditis for a patient undergoing a dental extraction because the patient had undergone

heart bypass surgery (even though the American Heart Association does not recommend antibiotic prophylaxis in such a situation). If the dentist follows this advice, and the patient suffers a serious reaction to the antibiotic, then it is likely that both the dentist and the physician will be named in a subsequent malpractice action. "I did what the physician told me to do," is unlikely to be a valid legal defense. Although physician consultation can and should be a valuable adjunct to dental treatment decisions, it is no substitute for good clinical judgment based on current scientific studies and available national medical and dental group recommendations.

A dental license is not a license to defer dental treatment decisions to nondentists, even if the nondentists are physicians. Physicians are extremely well-trained in medicine, but usually not at all trained in dentistry. It is therefore not surprising, for example, that many physicians believe that root canal therapy carries a higher risk of hemorrhage than professional cleanings.[5] Generally, if there is enough information from the patient's medical history, then established scientific and national medical or dental group guidelines are sufficient to treat the patient. If there is information unknown to both the patient and the dentist, it may be appropriate to contact the physician for such information.

Well-meaning dentists and physicians often make dental treatment on medically complex patients more complex than it should be. Areas for which this can be the case include endocarditis, artificial joint infections, local anesthetics and vasoconstrictors, dental surgery in anticoagulated patients and patients on antiplatelet drugs, and antibiotic interference with oral contraceptives. Much of the thinking in these areas is not based on serious scientific thought but, rather, on tradition. In each area, a common myth will be presented, then the facts will be discussed. In each area, dentistry will be shown to be much safer than is commonly believed. Finally, the question of physician consultation and/or legal issues will be addressed in each area.

Myth 1

Dental procedures are a common cause of and antibiotics are an effective and harmless preventive of infective endocarditis.

Endocarditis is a rare heart infection that is caused by bacteremia, a bacterial invasion of the bloodstream. Although the bloodstream is usually sterile, most people have occasional bacteremias throughout the day. Usually, these bacteria quickly and harmlessly pass through the system; but, in rare cases, the bacteria can colonize on the heart valve, causing a heart infection known as infective endocarditis.[6] Although endocarditis can occur in any patient, the principal risk factor is pre-existing heart valve abnormalities.[7,8]

Roughly half of all cases of endocarditis are caused by the viridans streptococci,[9] a group of microorganisms found abundantly in the mouth and gingival sulcus but also found on the skin, genitourinary tract, and eye. The initial flulike symptoms of streptococcal endocarditis usually occur within two weeks after the bacteremic incident.10 It is therefore difficult to determine definitively which bacteremic incident actually caused the subsequent infection, but it is not difficult to rule out any incidents that occurred more than two weeks before the initial symptoms.

Since some dental procedures can cause bacteremia by oral organisms, many cases of endocarditis have been blamed on the dentist. But several studies have shown that dental procedures cause very few, if any, cases of endocarditis of oral origin.[11-13] Most cases of endocarditis of oral origin are caused by frequent bacteremias from poor dental health and hygiene (bleeding gums and longstanding dental infections).[12] This is not surprising since in a typical month ending with an extraction, a patient with poor home care would have 5,000 more minutes of oral bactermia from "physiologic" sources (like brushing, chewing, and random bacteremias) than from the dental extraction.[12]

Still, it is possible that a dental procedure can cause a bacteremia that leads to endocarditis, and patients with pre-exisiting heart valve abnormalities are at greater risk of developing endocarditis than those without such abnormalities.[14] The American Heart Association has issued statements on the prevention of endocarditis, most recently in 1997.[15] This 1997 statement has been endorsed by the American Dental Association.[16] Although it accurately points out that the most important preventive for endocarditis is good oral health and home care, the AHA also recommends that some patients with certain heart problems should receive antibiotic prophylaxis before certain dental procedures that cause significant bleeding.

Although it is widely agreed upon that antibiotics can be a valuable adjunct in treating infections, there is still controversy as to whether antibiotics are effective in preventing infections.[7,17] Although antibiotic prophylaxis has been shown to prevent experimental endocarditis in animals, there have been "apparent failures" of antibiotic prophylaxis to prevent endocarditis in humans.18 Case/control studies have shown that antibiotic prophylaxis is effective less than half the time.[19,20] Risk/benefit studies have shown that in some scenarios, the small risk of serious reactions (e.g., gastrointestinal upset, hearing loss, allergy, anaphylaxis, and even death) to antibiotics actually can be greater than the even smaller risk that a patient would develop endocarditis.[21,22] Durack has stated it may be time to scale back on antibiotic prophylaxis and eliminate it altogether except for the highest risk patients having the highest risk procedures (e.g., patients with artificial heart valves or previous history of endocarditis having extractions or

gingival surgery).[23]

Practitioners usually have neither the time nor the interest to become expert in the area of endocarditis prevention. The 1997 AHA statement, written by a prominent group of experts in endocarditis, is a simple and useful guide for the practicing dentist. It provides a list of patients and dental procedures for which antibiotic prophylaxis is recommended and those for which it is not recommended. Although it is not meant to be a standard of care in all cases, it is a virtual certitude that in a malpractice case involving endocarditis, the AHA statement will be consulted. Since it will not be reprinted here, readers are urged to consult a copy of the statement and related articles.[24,25]

Physician Consultation

Physician consultation is often an excellent tool for information on a patient's heart condition. Consultation is generally not recommended for deciding whether or not to administer antibiotic prophylaxis for a specific dental procedure or the choice, dosage, or timing of the antibiotic. Instead, the latest (1997) AHA statement should be consulted.

Myth 2

Dental treatment is a common cause of and antibiotics are a harmless and effective preventive of artificial joint infections.

As the population ages, there are more joint replacement surgeries being performed, including hips, knees, and shoulders. These artificial joints can become infected from bacteremias; and such infections can be very serious, leading to further surgeries, disfigurement, and even death. In the past, dentists were urged to prescribe antibiotic prophylaxis for these patients to prevent such infections. It is now clear that most dental patients should not receive antibiotic prophylaxis to prevent artificial joint infections.[26-28]

Although there have been many documented artificial joint infections, only a few of these have been blamed on dental treatment. But after examining these few cases carefully, several authorities came to the same conclusion: in none of these cases was the dental treatment the probable cause of the subsequent joint infection.[29-31]

The American Dental Association and the American Academy of Orthopaedic Surgeons issued a statement in 1997 concluding that antibiotic prophylaxis was not indicated for most patients with artificial joints. The only patients for whom antibiotic prophylaxis should even be considered is a small number of high risk artificial joint patients (e.g., patients with previous artificial joint infections, insulin-dependent diabetes, and radiation-induced immunosuppression).[2] Even among these increased-risk patients, it is still within the standard of care not to recommend antibiotic prophylaxis to prevent artificial joint infections: "Practitioners must exercise their own clinical judgment in determining whether or not antibiotic prophylaxis is appropriate."[2] Other groups have issued similar statements. The American Academy of Oral Medicine has stated that "there is insufficient scientific evidence to support routine antibiotic prophylaxis for patients with prosthetic joints who are receiving dental care."[32] The British Society for Antimicrobial Chemotherapy recommends against any antibiotic prophylaxis before dental treatment in these patients.[33]

Some authorities have noted that because there is good evidence that artificial heart valve patients are at risk of endocarditis from dental treatment, so artificial joint patients must be at risk of artificial joint infections from dental treatment.[34] But while the single leading cause of prosthetic valve endocarditis is streptococcus, found abundantly in the mouth, the single leading cause of prosthetic joint infections is staphylococcus.[7,26] Staphylococci are ubiquitous on the skin, but are not commonly found in the blood after dental procedures.[35,36] Also, unlike cases of endocarditis, with an incubation period of a few days to two weeks, late artificial joint infections have been associated with much longer incubation periods, from four months to almost nine years.[30,37-39]

After placing artificial knee joints in 23 rabbits, Blomgren injected staphylococci into each rabbit. Four of the 10 surviving rabbits had artificial joint infections.[40,41] This experiment has been called the best evidence supporting antibiotic prophylaxis to prevent prosthetic joint infection after dental treatment.[42] But there were serious flaws in this small study, and it has little relevance to dental treatment. The organism used was a staphylococcus, which is rarely, if ever, found in the blood after dental treatment. In addition, the inocula were more like septicemias than transient bacteremias after dental treatment: More than half of the rabbits died from these massive inocula. Animal experimentation has therefore failed to show that dental treatment causes artificial joint infections.

Risk/benefit and cost/benefit analyses have shown that the risks and costs of antibiotic prophylaxis outweigh the potential benefits:[43-47] Because of the risks of serious reactions to certain antibiotics, when all prosthetic joint patients receive antibiotic prophylaxis for dental treatment, there may actually be a net loss of life as compared to no use of prophylaxis.

Physician Consultation

It may be prudent to consult the patient's physician if there is a discrepancy between what the physician recommends and what the American Academy of Orthopaedic Surgeons/ American Dental Association 1997 statement recommends to find out if the physician has information about the patient's medical history that is unknown to the dentist.

If the patient or the patient's physician insists on antibiotic prophylaxis, then

it is still acceptable to treat the patient. Jacobsen summed it up well: "Let the physician prescribe the medicine; the dentist can then do the dentistry."[48] This way the dentist may not be faced with a legal problem from potential harm from an unnecessarily prescribed drug.

Myth 3

Dental patients on continuous anticoagulant or antiplatelet drugs should have these drugs withdrawn before dental surgery.

Anticoagulant medications such as warfarin sodium (coumadin) are often administered continuously (daily) to patients to prevent complications such as stroke and thromboembolisms.[49,50] Although these medications are often lifesaving, they can also increase the risk of hemorrhage after medical or dental procedures. Anticoagulation is measured by the International Normalized Ratio or Prothrombin Time Ratio. The American Heart Association51 and the American College of Chest Physicians[52] recommend a therapeutic level of anticoagulation of no higher than INR 3.5 or about PTR 2.0 (although some authorities[53] have stated no higher than INR 4.0, or about PTR 2.2). Although 73 percent of physicians surveyed in 1996 recommend withdrawing coumadin before some or all dental procedures, more physicians recommended withdrawing coumadin for root canal therapy than for professional cleanings, even though root canal therapy carries little or no risk of hemorrhage.[5]

In extensive reviews of the literature, it was shown that in more than 950 patients on continuous anticoagulant therapy (many at higher than currently recommended therapeutic levels) receiving more than 2,400 surgical dental procedures, only 12 patients (<1.3 percent) needed more than local measures (e.g., biting on gauze or tea bags, suturing, or application of cellulose sponges) to control hemorrhage.[54,55] Only three of these patients (<0.31 percent) were anticoagulated within or below currently recommended therapeutic levels, but these were not well-documented. In other words, there has never been a well-documented case of hemorrhage uncontrolled by local measures after dental surgery in a continuously anticoagulated patient. On the other hand, of 526 patients having 575 interruptions of continuous anticoagulation, five (0.95 percent) suffered serious emobolic complications; four of these patients died. There is no difference in blood loss between dental extraction patients continuing anticoagulation and those in whom anticoagulation is withdrawn.[56]

Continuous (daily) antiplatelet medications (aspirin) are often used to prevent a heart attack. Many physicians recommend withdrawing aspirin before dental surgery.5 Although there have been case reports of postoperative hemorrhage after dental surgery,[57-59] controlled studies have not indicated a risk in patients taking low-dose (100 mg/day) aspirin. In an interesting study of patients taking aspirin undergoing unexpected (emergency) major operations (including appendectomy, cholecystectomy, intestinal resection, or mastectomy), there was no difference in perioperative blood loss between the study group and a control group of patients not taking aspirin undergoing the same procedures.[60] The aspirin group also had no postoperative bleeding problems. In a similar study of patients undergoing dental extractions, the aspirin group was no different than the placebo (acetaminophen) group.61 A recent study came to the same conclusion: There is no need to stop aspirin before oral surgery.[62]

Anticoagulants and antiplatelet drugs are often lifesaving; dental surgery is almost never life-threatening. When used at currently recommended therapeutic levels, these medications should not be withdrawn for dental surgery.

Physician Consultation

Physician consultation or consultation with the patient's medical laboratory is often helpful for the anticoagulated patient's latest level of anticoagulation (INR). Consultation is generally not recommended on the question of whether or not to withdraw anticoagulation once the dentist knows that the patient is at therapeutic levels of anticoagulation. For patients on continuous antiplatelet medications such as aspirin, physician consultation is usually not necessary unless there are other areas of concern in the medical history, especially those that affect bleeding time.

Myth 4

Local anesthetics and vasoconstrictors should be used with special caution in most medically compromised patients.

Although the safety of local anesthetics and vasoconstrictors has often been questioned, the reality is that they should be used the same in most medically compromised patients as they are in otherwise healthy patients.[63] For example, in spite of drug manufacturer warnings that there may be a severe hypertensive or hypotensive reaction between MAO inhibitors and vasoconstrictors in anesthetics,[64] animal,[65] and human[66,67] studies indicate there is no such interaction.

Although drug manufacturers still state that amide local anesthetics may cause severe hyperthermic reactions in patients susceptible to malignant hyperthermia,[64] animal and human studies show amide local anesthetics do not pose a special risk in such patients.[68-71] This is fortunate because the only injectable anesthetics in single-use dental cartridges manufactured in the United States for dental use (and virtually the only injectable anesthetics used by dentists) are amides such as lidocaine, mepivacaine, prilocaine, etidocaine, and bupivacaine. The ester anesthetic procaine (Novocain) is no longer manufactured in dental cartridges in the United States.[72]

Although the anesthetic preservative

methylparaben has been banned by the Food and Drug Administration in the United States for single-use dental cartridges,[73] there are small quantities of sulfite antioxidant preservatives for vasoconstrictors added to anesthetics with vasoconstrictors. Sulfites are also used in beer, wine, and much restaurant food. Restaurant meals typically contain about 25 to 200 mg of sulfites, which is at least 27 times the amount of sulfite in a single cartridge of lidocaine with 1:100,000 epinephrine (0.9 mg).[74] Again, in spite of drug manufacturers' warnings to the contrary, the vast majority of asthmatic patients can safely receive anesthetics with sulfite-containing vasoconstrictors.[75,76] Although a few steroid-dependent asthmatic patients may be sensitive to low doses of sulfites, a general rule is that if a patient can drink beer or wine or eat a restaurant meal, then the patient can safely have sulfite-containing local anesthetics.[74,77]

Allergy to amide local anesthetics is extremely rare, and there are few if any well-documented cases of true allergy to amide local anesthetics.[72] Some patients claim they are allergic to Novocain, but further questioning often reveals their symptoms more consistent with a psychosomatic reaction or an intravascular injection of vasoconstrictor-containing anesthetic.

To prevent lip mutilation after dental procedures, some dentists administer anesthetics without vasoconstrictors, thinking that the lip anesthesia will not last as long when using anesthetics with vasoconstrictors. Scientific studies, however, have shown that although pulpal anesthesia may last longer when vasoconstrictors are added to local anesthetics, the duration of lip anesthesia is the same with or without vasoconstrictors.[78,79] But anesthetics with vasoconstrictors have less anesthetic per cartridge. Mepivacaine 3 percent plain has 50 percent more anesthetic per cartridge (54 mg) than lidocaine 2 percent with epinephrine (36 mg). Lidocaine has a significantly higher maximum recommended dose (13.9 cartridges in a 150-pound adult; 4.4 cartridges in a 50-pound child) than does mepivacaine plain (7.4 cartridges in a 150-pound adult; 2.8 cartridges in a 50-pound child). There have been several unfortunate case reports of overdoses of mepivacaine plain during dental appointments, leading to the death of small children.[80,81] Inclusion of a vasoconstrictor might have slowed the anesthetic absorption rate and reduced its toxicity.

When a vasoconstrictor is necessary, epinephrine is often preferred over levonordefrin in medically compromised patients.82 Because epinephrine is a more potent vasoconstrictor than levonordefrin, mepivacaine with 1:20,000 levonordefrin has five times as much vasoconstrictor (90 mcg levonordefrin per cartridge) as does lidocaine with 1:100,000 epinephrine (18 mcg epinephrine per cartridge). The effects of epinephrine may be potentiated by tricyclic antidepressants. Anesthetics with epinephrine should therefore be used cautiously in patients taking tricyclic antidepressants, but levonordefrin-containing anesthetics should not be used at all in such patients because its effects are potentiated even more.[66,83]

Physician Consultation

Generally a physician consultation is not necessary unless more information is needed about the patient's medical history.

Myth 5

Antibiotics used in dentistry interfere with oral contraceptives, thereby contributing to unwanted pregnancies.

Dentists have long been indoctrinated with the fear of wrongful death lawsuits for negligence in prescribing medications. Now, there are even warnings about "wrongful birth" lawsuits. A wrongful birth or unwanted pregnancy, it is said, can be caused by a dentist failing to warn a woman of a possible interaction between her birth control pills and an antibiotic that the dentist prescribes. The dentist could then legally be liable for damages, including child support payments. Since many women of childbearing age are taking oral contraceptives and since dentists frequently prescribe antibiotics, this issue can be of great concern to practicing dentists. Some have advised that women who are prescribed antibiotics should be warned of a potential interaction and advised to use an additional birth control method.[84-86] At least one dermatologist has gone so far as to withhold needed antibiotics from any women on birth control pills.[87]

This concern came to a climax in the lay media: A tabloid newspaper addressed the issue under the headline "A dental visit can make you pregnant!" Fortunately, after reviewing the scientific and medical literature, it appears that the fears of antibiotic interference with oral contraceptives are not based on scientific evidence but instead on irrational myths.

There have been case reports of unwanted pregnancy after administration of antibiotics, including many of the antibiotics used in dentistry. These case reports have led many to conclude that the antibiotics caused the oral contraceptive to fail. This type of reasoning has been called "post hoc, ergo propter hoc" (after this, therefore on account of this). But simply because a pregnancy occurred after antibiotic ingestion does not prove that the antibiotic helped cause the pregnancy. From 1968 to 1984 there were 63 pregnancies reported in England in women on oral contraceptives who were concurrently receiving antibiotics (not including rifampin, which has been scientifically shown to decrease oral contraceptive efficacy).[88] From 1973 to 1984, there were more than 300 million prescriptions for antibiotics (not including rifampin) in England. In a landmark article questioning the

interaction between antibiotics and oral contraceptives for the first time in the dental literature, Becker pointed out that such statistics introduce the likelihood of mere chance correlation between unwanted pregnancies and antibiotics.[89]

Many of the case reports of unwanted pregnancy blaming antibiotics occurred in women also taking other medications known to decrease contraceptive efficacy. In addition, some of these reports may be related to the known 1 percent failure rate of oral contraceptives. Finally, many women may skip doses of the contraceptive, which may lead to unwanted pregnancy. For example, it is estimated that up to 50 percent of teenage girls on oral contraceptives miss doses. The average teenager misses three pills during a 28-day cycle, and the failure rate among teenagers is eight times (8 percent) the failure rate among all women (1 percent).[90]

The American Dental Association reported on possible antibiotic interference with oral contraceptives in 1991.[91] Although it is now doubtful that any antibiotics used in dentistry are truly responsible for oral contraceptive failure, those listed by the American Dental Association were only the penicillins, tetracyclines, sulfonamides, erythromycin, metronidazole, griseofulvin, and cephalosporin. Clindamycin was not implicated in oral contraceptive failure.

Rifampin was first implicated in decreased contraceptive efficacy in 1971.[92] Since then, other antibiotics have been blamed, including the penicillins (amoxicillin, ampicillin, and penicillin), metronidazole, the macrolide antibiotics (including erythromycin and roxithromycin), cotrimoxazole, and the tetracyclines. Rifampin, a potent enzyme-inducing antituberculosis drug generally not used in dentistry, has been scientifically shown to decrease the levels of oral contraceptives.[93,94] Similar studies on ampicillin,[95,96] metronidazole,[97] tetracycline,[98] roxithromycin,[99] cotrimoxazole,[100] temafloxacin,[101] and doxycycline[102] have consistently failed to show decreased contraceptive levels in women taking these antibiotics concurrently. One antibiotic, cotrimoxazole, has been shown to increase levels of estrogen and progesterone.[99] Unlike rifampin, none of these antibiotics are enzyme inducers.

To prevent wrongful birth lawsuits from unwanted pregnancies, dentists prescribing antibiotics are trained to recommend additional forms of contraception to women taking oral contraceptives. But giving such advice can unnecessarily upset women and could theoretically lead to lawsuits for emotional distress and/or loss of consortium. Also, because of receiving this advice, many women may refuse to take antibiotics at all, which may lead to greater infection, another potential lawsuit. In addition to exhaustively reviewing the literature, Hersh[103] analyzed a California legal proceeding in which a woman and her husband sued her gynecologist for the "wrongful life" of their child allegedly caused by an interaction between her oral contraceptive medication and the penicillin V that the dentist prescribed.[104] The plaintiffs lost the case because they were unable to show scientific studies that documented a significant interaction or that she became pregnant while taking the penicillin. In addition, under California law, practitioners are not required to discuss risks of very low incidences. Hersh pointed out that the failure rate would have to exceed double the expected failure rate for a practitioner to be required to discuss the potential risk.

Antibiotics used in dentistry have not been shown to decrease oral contraceptive efficacy. Of all antibiotics studied, only rifampin has been scientifically shown to decrease oral contraceptive efficacy. On the other hand, diarrhea or breakthrough bleeding from any cause (including antibiotics) can be a sign of decreased absorption of oral contraceptives, and patients in these situations should use additional birth control methods.[105] Since oral contraceptives are not 100 percent effective, many patients may wish to use additional methods of contraception anyway.

Conclusion

Dental treatment on many medically complex patients may not be so complex. Although dental treatment generally does not cause endocarditis, it can. Dentists should consult the 1997 American Heart Association statement for recommendations on antibiotic prophylaxis in patients at risk for endocarditis. Most patients with artificial joints should not have antibiotic prophylaxis -- the only ones for whom antibiotic prophylaxis should even be considered are patients at high risk. Neither continuous anticoagulation nor continuous antiplatelet medications should be withdrawn for dental surgery unless patients are at higher than therapeutic levels or they have other medical considerations. In spite of many admonishments to the contrary, local anesthetics and vasoconstrictors can be used without special reservation in virtually all medically complex patients. Local anesthetics with vasoconstrictors should be preferred over plain anesthetics in children. Scientific studies have failed to document an interaction between antibiotics used in dentistry and oral contraceptives. Physician consultation can be a valuable adjunct to dental treatment but should not be a substitute for the dentist's clinical judgment based on current scientific literature and available national medical and dental group guidelines.

References
1. Todd KM, A legal perspective on antibiotic prophylaxis. *J Am Dent Assoc* 128:1007, 1997.
2. American Dental Association, American Academy of Orthopaedic Surgeons, Antibiotic prophylaxis for dental patients with total joint replacements. *J Am Dent Assoc* 128:1004-7, 1997.
3. Jacobson JJ, Prosthetic joint patients [letter]. *J Am Dent Assoc* 125:1432-4, 1994.
4. Wahl MJ, Prosthetic joint patients [letter]. *J Am Dent Assoc*

125:664-6, 1994.
5. Wahl MJ, Howell J, Altering anticoagulation therapy: a survey of physicians. *J Am Dent Assoc* 127:625-38, 1996.
6. Pallasch TJ, A critical appraisal of antibiotic prophylaxis. Int Dent J 39:183-96, 1989.
7. Pallasch TJ, Slots J, Antibiotic prophylaxis and the medically compromised patient. Periodontol 2000 10:107-38, 1996.
8. Little JW, Antibiotic prophylaxis for prevention of bacterial endocarditis and infectious major joint prostheses. Oral Maxillof Surg Infec 2(3):93-101, 1992.
9. Yagiela JA, Prophylactic antibiotics: cardiac and prosthetic considerations. *J Calif Dent Assoc* 23(10):29-40, 1995.
10. Starkebaum M, Durack D, Beeson P, The "incubation period" of subacute bacterial endocarditis. Yale J Biol Med 50:49-58, 1977.
11. Bayliss R, Clarke C, et al, The teeth and infective endocarditis. Br Heart J 50:506-12, 1983.
12. Guntheroth WG, How important are dental procedures as a cause of infective endocarditis? Am J Cardiol 54:797-801, 1984.
13. Strom BL, Abrutyn E, et al, Dental and cardiac risk factors for infective endocarditis. A population-based, case-control study. Ann Intern Med 129:761-9, 1998.
14. Steckelberg JM, Wilson WR, Risk factors for infective endocarditis. Infect Dis Clin North Amer 7(1):9-19, 1993.
15. Dajani AS, Taubert KA, et al, Prevention of bacterial endocarditis: recommendations by the American Heart Association. J Am Med Assoc 277:1794-1801, 1997.
16. Dajani AS, Taubert KA, et al, Prevention of bacterial endocarditis: recommendations by the American Heart Association. *J Am Dent Assoc* 128:1142-51, 1997.
17. Wahl MJ, Myths of dental-induced endocarditis. Arch Intern Med 154:137-44, 1994.
18. Durack DT, Kaplan EL, Bisno AL, Apparent failures of endocarditis prophylaxis: analysis of 52 cases submitted to a national registry. J Am Med Assoc 250:2318-22, 1983.
19. van der Meer JTM, van Wijk W, et al, Efficacy of antibiotic prophylaxis for prevention of native-valve endocarditis. Lancet 339:135-9, 1992.
20. Lacassin F, Hoen B, et al, Procedures associated with infective endocarditis in adults: a case control study. Eur Heart J 16:1968-74, 1995.
21. Bor DH, Himmelstein DU, Endocarditis prophylaxis for patients with mitral valve prolapse: a quantitative analysis. Am J Med 76:711-17, 1984.
22. Clemens JD, Ransohoff DF, A quantitative assessment of pre-dental antibiotic prophylaxis for patients with mitral-valve prolapse. J Chron Dis 37:531-44, 1984.
23. Durack DT, Antibiotics for prevention of endocarditis during dentistry: time to scale back? Ann Intern Med 129:829-31, 1998.
24. Pallasch TJ, Gage TW, Taubert KA, The 1997 prevention of bacterial endocarditis recommendations by the American Heart Association: questions and answers. *J Calif Dent Assoc* 27(5):393-9, 1999.
25. Pallasch TJ, Antibiotic prophylaxis: significance of its recent evolution. *J Calif Dent Assoc* 25(9):619-32, 1997.
26. Wahl MJ, Myths of dental-induced prosthetic joint infections. Clin Infect Dis 20:1420-5, 1995.
27. Wahl MJ, Clinical issues in the prevention of dental-induced endocarditis and prosthetic joint infection. Pract Perio Aesth Dent 6(6):25-32, 1994.
28. Little JW, Managing dental patients with joint prostheses. *J Am Dent Assoc* 125:1374-8, 1994.
29. Council on Dental Therapeutics, American Dental Association. Management of dental patients with prosthetic joints. *J Am Dent Assoc* 121:537-38, 1990.
30. McGowan DA, Hendrey ML, Is antibiotic prophylaxis required for dental patients with joint replacements? Br Dent J 158:336-8, 1985.
31. Thyne GM, Ferguson JW, Antibiotic prophylaxis during dental treatment in patients with prosthetic joints. J Bone Joint Surg [Br] 73B:191-4, 1991.
32. Eskinazi D, Rathbun W, Is systematic antimicrobial prophylaxis justified in dental patients with prosthetic joints? Oral Surg Oral Med Oral Pathol 66:430-1, 1988.
33. Simmons NA, Ball AP, et al, Case against antibiotic prophylaxis for dental treatment of patients with joint prostheses. Lancet 339:301, 1992.
34. Rubin R, Salvati EA, Lewis R, Infected total hip replacement after dental procedures. Oral Surg 41:18-23, 1976.
35. Hall G, Hedström SÅ, et al, Prophylactic administration of penicillins for endocarditis does not reduce the incidence of postextraction bacteremia. Clin Infect Dis 17:188-94, 1993.
36. Heimdahl A, Hall G, et al, Detection and quantitation by lysis-filtration of bacteremia after different oral surgical procedures. J Clin Microbiol 28:2205-9, 1990.
37. Maniloff G, Greenwald R, et al, Delayed postbacteremic prosthetic joint infection. Clin Orthop Rel Res 223:194-7, 1987.
38. Poss R, Thornhill TS, et al, Factors influencing the incidence and outcome of infection following total joint arthroplasty. Clin Orthop 182:117-26, 1984.
39. McCarthy J, Stingemore N, Clostridium difficile infection of a prosthetic joint presenting 12 months after antibiotic-associated diarrhoea. J Infect 39:94-6, 1999.
40. Blomgren G, Hematogenous infection of total joint replacement: an experimental study in the rabbit. Acta Orthop Scand 52(suppl 187):1-64, 1981.
41. Blomgren G, Lindgren U, Late hematogenous infection in total joint replacement: studies of gentamicin and bone cement in the rabbit. Clin Orthop 155:244-8, 1981.
42. Cioffi GA, Terezhalmy GT, Taybos GM, Total joint replacement: a consideration for antimicrobial prophylaxis. Oral Surg Oral Med Oral Pathol 66:124-9, 1988.
43. Jacobson JJ, Schweitzer SO, Kowalski CJ, Chemoprophylaxis of prosthetic joint patients during dental treatment: a decision-utility analysis. Oral Surg Oral Med Oral Pathol 72:167-77, 1991.
44. Little JW, The need for antibiotic coverage for dental treatment of patients with joint replacements. Oral Surg Oral Med Oral Pathol 55:20-3, 1983.
45. Tsevat J, Durand-Zaleski I, Pauker SG, Cost-effectiveness of antibiotic prophylaxis for dental procedures in patients with artificial joints. Am J Public Health 79:739-43, 1989.
46. Jacobson JJ, Schweitzer S, et al, Chemoprophylaxis of dental patients with prosthetic joints: a simulation model. *J Dent* Educ 52:599-604, 1988.
47. Jacobson JJ, Schweitzer S, et al, Antibiotic prophylaxis for dental patients with joint prostheses? A decision analysis. Inter J Tech Assess Health Care 6:569-87, 1990.
48. Jacobsen PL. Guidelines stressed [letter]. *J Am Dent Assoc* 125:798, 1994.
49. Herman WW, Konzelman JL, Sutley SH, Current perspectives on dental patients receiving coumarin anticoagulant therapy. *J Am Dent Assoc* 128:327-35, 1997.
50. Beirne OR, Koehler JR, Surgical management of patients on warfarin sodium. J Oral Maxillofac Surg 54:1115-8, 1996.
51. Hirsh J, Fuster V, Guide to anticoagulant therapy. Part 2: oral anticoagulants. Circulation 89:1469-80, 1994.
52. Hirsh J, Dalen JE, et al, Oral anticoagulants: mechanism of action, clinical effectiveness, and optimal therapeutic range. Chest 102 (suppl):312S-26S, 1992.
53. Cannegieter SC, Rosendaal FR, et al. Optimal oral anticoagulant therapy in patients with mechanical heart valves. N Engl J Med 333:11-7, 1995.
54. Wahl MJ, Dental surgery in anticoagulated patients. Arch Intern Med 158:1610-16, 1998.
55. Wahl MJ, Myths of dental surgery in patients receiving anticoagulant therapy. *J Am Dent Assoc* 131:77-81, 2000.
56. Campbell JH, Alvarado F, Murray RA, Anticoagulation and minor oral surgery: Should the anticoagulation regimen be altered? J Oral Maxillofac Surg 58:131-5, 2000.
57. Thomason JM, Seymour RA, et al, Aspirin-induced post-gingivectomy haemorrhage: a timely reminder. J Clin Periodontol 24:136-8, 1997.
58. Davis MJ, Aspirin-induced prolonged bleeding: report of case. *J Dent* Child 43:350, 1976.
59. McGaul T, Postoperative bleeding caused by aspirin. *J Dent* 6:207-9, 1978.
60. Ferraris VA, Swanson E, Aspirin usage and perioperative blood loss in patients undergoing unexpected operations. Surg Gynecol Obstet 156:439-42, 1983.
61. Pawalk DF, Itkin AB, et al, Clinical effects of aspirin and acetaminophen on hemostasis after exodontics. J Oral Surg 36:944-7, 1978.
62. Ardekian L, Gaspar R, et al, Does low-dose aspirin therapy complicate oral surgical procedures? *J Am Dent Assoc* 131:331-5, 2000.
63. Wahl MJ, Local anesthetics and vasoconstrictors: myths and facts. Pract Periodont Aesth Dent 9:649-52, 1997.
64. 1997 Physicians' Desk Reference. Medical Economics, Montvale, NJ, p 563.
65. Yagiela JA, Duffin SR, Hunt LM, Drug interactions and vasoconstrictors used in local anesthetic solutions. Oral Surg Oral Med Oral Pathol 59:565-71, 1985.
66. Boakes AJ, Laurence DR, et al, Interactions between sympathomimetic amines and antidepressant agents in man. Br Med J 1:311-15, 1973.
67. Elis J, Laurence DR, et al, Modification by monoamine oxidase inhibitors of the effect of some sympathomimetics on blood pressure. Br Med J 2:75-8, 1967.
68. Wingard DW, Bobko S, Failure of lidocaine to trigger porcine malignant hyperthermia. Anesth Analg 58:99-103, 1979.
69. Harrison GG, Morrell DF, Response of MHS swine to IV infusion of lignocaine and bupivacaine. Br J Anaesth 52:385-7, 1980.
70. Steelman R, Holmes D, Outpatient dental treatment of pediatric patients with malignant hyperthermia: report of three cases. *J Dent* Child 59:62-5, 1992.
71. Gielen M, Viering W, 3-in-1 lumbar plexus block for muscle biopsy in malignant hyperthermia patients. Amide local anaesthetics may be used safely. Acta Anaesthesiol Scand 30:581-3, 1986.
72. Clinical action of specific agents. In, Malamed SF, Handbook of Local Anesthesia, 4th ed. Mosby, St. Louis, 1997, pp 49-73.
73. The cartridge. In, Malamed SF, Handbook of Local Anesthesia, 4th ed. Mosby, St. Louis,1997, pp 91-9.
74. Simon RA, Sulfite sensitivity. Ann Allerg 56:281-8, 1986.
75. Bush RK, Taylor SL, et al, Prevalence of sensitivity to sulfiting agents in asthmatic patients. Am J Med 81:816-20, 1986.
76. Goldfarb G, Simon R, Provocation of sulfite sensitive asthma. J Allergy Clin Immunol 73:135 [Abstr 107], 1984.
77. Pérusse R, Goulet J-P, Turcotte J-Y, Contraindications to vasoconstrictors in dentistry, Part II. Oral Surg Oral Med Oral Pathol 74:687-91, 1992.
78. Hersh EV, Hermann DG, et al. Assessing the duration of mandibular soft tissue anesthesia. *J Am Dent Assoc* 126:1531-6, 1995.
79. Yagiela JA, Local anesthetics: a century of progress. Anesth Progr 32:47-56, 1985.
80. Moore PA, Preventing local anesthesia toxicity. *J Am Dent Assoc* 123(9):60-4, 1992.
81. Hersh EV, Helpin ML, Evans OB, Local anesthetic mortality: report of case. *J Dent* Child 58:489-91, 1991.
82. Robertson VJ, Taylor SE, Gage TW, Quantitative and

qualitative analysis of the pressor effects of levonordefrin. J Cardiovasc Pharm 6:929-35, 1984.
83. Patient evaluation. In, Jastak JT, Yagiela JA, Donaldson D, Local Anesthesia of the Oral Cavity. WB Saunders, Philadelphia, 127-44, 1995.
84. Donley TG, Smith RF, Reduced oral contraceptive effectiveness with concurrent antibiotic use: a protocol for prescribing antibiotics to women of childbearing age. Compend Contin Educ Dent 11:392-5, 1990.
85. Gibson J, McGowan DA, Oral contraceptives and antibiotics: important considerations for dental practice. Br Dent J 177:419-22, 1994.
86. Bainton R, Interaction between antibiotic therapy and contraceptive medication. Oral Surg Oral Med Oral Pathol 453-5, 1986.
87. Coskey RJ, Tetracycline and oral contraceptives [letter]. J Am Acad Dermatol 5:222, 1981.
88. Back DJ, Grimmer SFM, et al, Evaluation of Committee on Safety of Medicines yellow card reports on oral contraceptive-drug interactions with anticonvulsants and antibiotics. Br J Clin Pharmac 25:527-32, 1988.
89. Becker DE, Drug interactions in dental practice: a summary of facts and controversies. Compend Contin Educ Dent 15:1228-42, 1994.
90. Sondheimer SJ, Update on oral contraceptive pills and postcoital contraception. Curr Opin Obstet Gynecol 4:502-5, 1992.
91. ADA Health Foundation Research Institute, Department of Toxicology. Antibiotic interference with oral contraceptives. *J Am Dent Assoc* 122(12):79, 1991.
92. Orme ML'E, Back DJ, Breckenridge AM, Clinical pharmacokinetics of oral contraceptive steroids. Clin Pharmacokin 8:95-136, 1983.
93. Back DJ, Breckenridge AM, et al, The effect of rifampicin on norethisterone pharmacokinetics. Eur J Clin Pharmacol 15:193-7, 1979.
94. Back DJ, Breckenridge AM, et al, The effect of rifampicin on the pharmacokinetics of ethinylestradiol in women. Contraception 21:135-43, 1980.
95. Back DJ, Breckenridge AM, et al, The effects of ampicillin on oral contraceptive steroids in women. Br J Clin Pharmac 14:43-8, 1982.
96. Friedman CI, Huneke AL, et al, The effect of ampicillin on oral contraceptive effectiveness. Obstetr Gynecol 55:33-7, 1980.
97. Joshi JV, Joshi UM, et al, A study of interaction of low-dose combination oral contraceptive with ampicillin and metronidazole. Contraception 22:643-52, 1980.
98. Murphy AA, Zacur HA, et al, The effect of tetracycline on levels of oral contraceptives. Am J Obstet Gynecol 164:28-33, 1991.
99. Meyer B, Müller F, et al, A model to detect interactions between roxithromycin and oral contraceptives. Clin Pharmacol Ther 47:671-4, 1990.
100. Grimmer SFM, Allen WL, et al, The effect of cotrimoxazole on oral contraceptive steroids in women. Contraception 28:53-9, 1983.
101. Back DJ, Tjia J, et al, The lack of interaction between temafloxacin and combined oral contraceptive steroids. Contraception 43:317-23, 1991.
102. Neely JL, Abate M, et al, The effect of doxycycline on serum levels of ethinyl estradiol, norethindrone, and endogenous progesterone. Obstet Gynecol 77:416-20, 1991.
103. Hersh EV, Adverse drug interactions in dental practice: interactions involving antibiotics, Part II of a series. *J Am Dent Assoc* 130:236-51, 1999.
104. LaCasa C, California court denies wrongful birth claim. J Law Med Ethics 24(3):273-4, 1996.
105. Miller DM, Helms SE, Brodell RT, A practical approach to antibiotic treatment in women taking oral contraceptives. J Am Acad Dermatol 30:1008-11, 1994.

To request a printed copy of this article, please contact: Michael J. Wahl, DDS, 1601 Concord Pike, Wilmington, DE 19803 or at WahlMichaelJ@aol.com.

CDA JOURNAL, VOL 28, Nº7

Cardiovascular Drugs and Dental Considerations

Richard L. Wynn, PhD

ABSTRACT This paper provides current information on the pharmacologic management of cardiovascular diseases. It also describes the drugs used to treat five common cardiovascular disorders -- heart failure, coronary artery disease, atrial fibrillation, hypertension and unstable angina -- and lists their dental implications. This information can be used to monitor patients for potential adverse drug reactions and drug interactions and to provide an information base for medical consultation.

AUTHOR

Richard L. Wynn, PhD, is a professor at the University of Maryland Dental School

This paper describes the drugs used to treat five common cardiovascular disorders: heart failure, coronary artery disease, atrial fibrillation, hypertension, and unstable angina. For each condition, a discussion of the drugs used in the management of that condition is provided. The drugs are also listed in Tables 1 through 5. Some of the listings are redundant because the drugs are used to treat more than one cardiovascular disorder. As a convenience to the reader, each table has been constructed as a stand-alone listing of drugs for the given disorder. The dental implications of these drugs are listed in Tables 6 and 7. Each of these two tables is a consolidation of the drugs from Tables 1 through 5. Table 6 describes the more frequent cardiovascular, respiratory, and central nervous system adverse reactions that may be seen in the dental patient. Table 7 describes the effects on dental treatment reported for these drugs. It is suggested that the reader use Tables 6 and 7 to check for potential effects that could occur in medicated cardiovascular dental patients. It is the purpose of this paper to provide the most current information on the pharmacologic management of cardiovascular diseases. This information can then be used to monitor patients for potential adverse drug reactions and drug interactions and to provide an information base for medical consultation.

Drugs to Treat Heart Failure

Heart failure is a condition in which the heart is unable to pump sufficient

blood to meet the needs of the body. It is caused by impaired ability of the cardiac muscle to contract or by an increased workload imposed upon the heart. The most significant underlying cause of heart failure is coronary artery disease. Other contributory causes are hypertension, diabetes, idiopathic dilated cardiomyopathy, and valvular heart disease. It is estimated that heart failure affects approximately 5 million Americans.1 The New York Heart Association functional classification is regarded as the standard measure to describe the severity of a patient's symptom.2 Class I is characterized by having no limitation of physical activity. There is no dyspnea, fatigue, palpitations or angina with ordinary physical activity. There is no objective evidence of cardiovascular dysfunction. Class II includes those patients having slight limitation of physical activity. These patients have fatigue, palpitations, dyspnea, or angina with ordinary physical activity but are comfortable at rest. There is evidence of minimal cardiovascular dysfunction. Class III is characterized with marked limitation of activity. Less than ordinary physical activity causes fatigue, palpitations, dyspnea, or angina; but patients are comfortable at rest. There is objective evidence of moderately severe cardiovascular function. Class IV is characterized by the inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest, and any physical activity undertaken increases discomfort. There is objective evidence of severe cardiovascular dysfunction.

Drug Classes and Agents for Treatment

Drug classes and the specific agents used to treat heart failure are listed in **Table 1**. Angiotensin converting enzyme inhibitors reduce left ventricular volume and filling pressure and decrease total peripheral resistance. They induce cardiac output modestly and induce natriuresis. These ACE inhibitors are usually used in all patients with heart failure if no contraindication or intolerance exists. This group of drugs is considered the cornerstone of treatment, and they are used routinely and early if pharmacologic treatment is indicated.

Diuretics increase sodium chloride and water excretion resulting in reduction of preload, the degree of tension on the cardiac muscle when it begins to contract. By reducing preload, the diuretics relieve symptoms of pulmonary congestion associated with heart failure. They may also reduce myocardial oxygen demand. The thiazides, the loop diuretics, and the potassium-sparing agents are all useful in reducing preload by way of their diuretic actions. See **Table 1** for a list of the specific diuretics.

Digitalis glycosides have been used in the treatment of heart failure for more than 200 years. Digitalis drugs increase cardiac output by a direct positive inotropic action on the myocardium. Inotropic action is defined as the contractile force of the cardiac muscle. This increased cardiac output results in decreased venous pressure, reduced heart size, and diminished compensatory tachycardia.

Beta-adrenergic receptor blocking drugs (beta blockers) are used in the treatment of heart failure because they reduce the number of deaths. A meta-analysis of randomized clinical trials showed that the beta blockers significantly reduced all causes of mortality, with carvedilol (Coreg) showing the greatest efficacy.[3] The overall risk of death was reduced by more than 30 percent. The beta blockers used to treat heart failure are listed in **Table 1**.

Other drugs used in the treatment of heart failure are referred to as supplemental agents. The direct-acting vasodilators reduce excessive vasoconstriction and the workload of the failing heart. The catecholamines and phosphodiesterase inhibitors are alternative agents with positive inotropic effects. The catecholamines and phosphodiesterase inhibitors are effective for short-term therapy and have not been demonstrated to prolong life during long-term therapy. The names of these supplemental drugs are found in **Table 1**.

Drugs to Treat Coronary Artery Disease

Coronary artery disease is the cause of about half of all deaths in the United States.[4] CAD has been shown to correlate to the levels of plasma cholesterol and/or triacylglycerol-containing lipoprotein particles. Secondary prevention of CAD focuses on therapies to reduce morbidity and mortality in patients clinically documented to have the disease. In contrast, primary prevention focuses on averting the development of CAD. Lipid-lowering and cardioprotective drugs provide significant risk-reducing benefits in the secondary prevention of CAD. Reducing total and low-density cholesterol levels primarily through the inhibition of hydroxymethylglutaryl coenzyme A (HMG-CoA) reductase by the statin drugs significantly improves survival. Cardioprotective drug therapy includes antiplatelet/anticoagulant agents to inhibit platelet adhesion, aggregation and blood coagulation; beta blockers to lower heart rate, contractility and blood pressure; and the ACE inhibitors to lower peripheral resistance and workload. See **Table 2** for a listing of these drugs.

Drugs to Treat Atrial Fibrillation

Atrial fibrillation is an arrhythmia characterized by multiple electrical activations in the atria resulting in scattered and disorganized depolarization and repolarization of the myocardium. Atrial contraction can lead to an irregular and rapid rate of ventricular contraction. The prevalence of atrial fibrillation within the U.S. population ranges from 1 percent to 4 percent,[5,6] with the incidence of the disease increasing with age. It is often associated with rheumatic valvular disease and nonvalvular conditions including coronary artery disease and hypertension. Coronary artery disease is present in about one-half of the patients with this form of arrhythmia. Atrial fibrillation is a major risk factor for systemic and cerebral

embolism. It is thought that thrombi develop as a result of stasis in the dilated left atrium and are dislodged by sudden changes in cardiac rhythm.[7] About 10 percent of all strokes in patients older than 60 are caused by atrial fibrillation.7

The cornerstones of drug therapy for atrial fibrillation are the restoration and maintenance of normal sinus rhythm through the use of anti-arrhythmic drugs; ventricular rate control through the use of beta blockers, digitalis drugs or calcium channel blockers; and stroke prevention through the use of anticoagulants.[8] Therapy for atrial fibrillation also includes the management of arrhythmias by cardiac pacemaker and ablation therapy.

Antiarrhythmic Drugs

Cardiac rhythm is conducted through the sinoatrial and atrioventricular nodes, bundle branches, and Purkinje fibers. Electrical impulses are transmitted within this system by opening and closing of sodium and potassium channels. Antiarrhythmic drugs are classified by which channel they act upon, a classification known as Vaughn Williams after the author of the paper describing them.[9] The so-called Class I agents act primarily on sodium channels, and the Class III agents act on the potassium channels. In addition, there are subclassifications within the Class I agents according to effects of the drug on conduction and refractoriness within the Purkinje and ventricular tissues. Class IA agents show moderate depression of conduction and prolongation of repolarization; Class IB agents show modest depression of conduction and shortening of repolarization; Class IC agents show marked depression of conduction and mild or no effect on repolarization. Class IA and IC agents are effective in the treatment of atrial fibrillation. Class IB agents are not used to treat atrial fibrillation but are effective in treating ventricular arrhythmias. This class includes lidocaine and phenytoin. **Table 1** lists the drugs and the categories used to treat atrial fibrillation. Class II drugs are the beta-adrenergic blocking drugs and Class IV are the calcium channel blockers.

Restoring and Maintaining Normal Sinus Rhythm

Cardioversion induced by drugs can usually restore sinus rhythm in patients with atrial fibrillation. Class IA drugs (disopyramide, procaineamide, quinidine), Class IC drugs (flecainide, propafenone), and Class III antiarrhythmics (amiodarone) are all effective in restoring normal sinus rhythm. Success rates may vary greatly and are complicated by the high rate of spontaneous conversion.[10,11] The drugs used for pharmacologic conversion are also used to maintain sinus rhythm.

Ventricular Rate Control

It is accepted practice to treat patients with medication when the resting ventricular rate exceeds 110 beats per minute. Digoxin, calcium channel blockers, and beta-adrenergic blockers are used in the regulation of the ventricular rate. Digoxin increases the vagal tone to the atrioventricular node, calcium channel blockers slow the atrioventricular nodal conduction, and the beta-adrenergic blocking drugs decrease the sympathetic activation of the atrioventricular nodal conduction.

Stroke Prevention

Reports from stroke prevention trials indicate that patients with atrial fibrillation incurred a 4 percent annual risk of stroke if not treated.[12] Also, patients with heart failure or coronary heart diseases were three times more likely to have a stroke than those without risk factors. Anticoagulation therapy with warfarin reduced the stroke risk by 64 percent.[12] Warfarin was found to be more effective than aspirin in all age groups in the Stroke Prevention in Atrial Fibrillation II Trial.[13,14] To achieve optimal levels of anticoagulation with the lowest incidence of bleeding, the International Normalized Ratio is usually maintained between 2 and 3.[15-17] For patients younger than 60 with atrial fibrillation and no other risk factors, no anticoagulant therapy is needed. Aspirin in the amount of 325 mg daily is recommended for those older than 60.[7]

Drugs to Control Hypertension

In the United States, almost 50 million adults 25 to 74 years of age have hypertension. Hypertension is defined as systolic blood pressure ≥ 140 mm Hg, and/or diastolic pressure of > 90 mm Hg.[18] People having blood pressure above normal are considered at increased risk of damage to the heart, kidney, brain, and eye that results in premature morbidity and mortality.[19] Individuals with high normal blood pressure (systolic blood pressure of 130 to 139 mm Hg and diastolic blood pressure of 85 to 89 mm Hg) should be monitored and encouraged to reduce blood pressure by nondrug measures, including weight control, restriction of sodium and alcohol, and participation in an exercise program.[18] Stage 1 hypertension is blood pressure of 140 to 159/90-99 mm Hg. If the desired blood pressure is not achieved with nondrug measures within six months in patients with Stage 1 hypertension without target organ disease and/or clinical cardiovascular disease, pharmacologic therapy is suggested.[18]

The suggested initial goals of drug therapy are the maintenance of an arterial pressure of ≤140/90 mm Hg with concurrent control of other modifiable cardiovascular risk factors.[18] Further reduction to 130/85 mm Hg should be pursued if cardiovascular and cerebrovascular function is not compromised. The Hypertension Optimal Treatment randomized trial using patients 50 to 80 years of age found that the lowest incidence of major cardiovascular events and the lowest risk of cardiovascular mortality occurred at a mean achieved diastolic blood pressure of 82.6 and 86.5 mm Hg respectively.[20] The classes of medications used for the treatment of hypertension include:

- Diuretics;
- Beta-adrenergic receptor blocking agents (beta blockers);
- Alpha 1-adrenergic receptor blocking agents (alpha 1 blockers);
- Agents that have both alpha and beta adrenergic blocking properties (alpha/beta blockers);
- ACE inhibitors;
- Angiotensin II receptor blockers;
- Calcium channel blocking agents; and
- Supplemental agents such as central-acting alpha 2 - adrenergic receptor agonists and direct acting peripheral vasodilators.

Table 4 lists the drug categories and representative agents used to treat hypertension.

Current Thinking Regarding Antihypertensive Drug Selection

Medications in the first eight categories in **Table 4** were held to be equally effective in two large scale studies reported in the New England Journal of Medicine and the Journal of the American Medical Association,[21,22] and it was determined that any of the medications could be used initially for monotherapy. According to the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure,[18] diuretics or beta blockers are recommended as initial therapy for uncomplicated hypertension. If a diuretic is selected as initial therapy, a thiazide diuretic is preferred in patients with normal renal function. If necessary, potassium replacement or concurrent treatment with a potassium-sparing agent may prevent hypokalemia. Loop diuretics are used in patients with impaired renal function or who cannot tolerate thiazides. Diuretics are well-tolerated and inexpensive. They are considered the drugs of first choice for treating isolated systolic hypertension in the elderly.[18]

Beta blockers are the agents of choice in patients with coronary artery disease or supraventricular arrhythmia and in young patients with hyperdynamic circulation.

Beta blockers are alternatives for initial therapy and are more effective in white patients than in black patients. Beta blockers are not considered first choice drugs in elderly patients with uncomplicated hypertension.

The beta blocking drug carvedilol also selectively blocks alpha 1 receptors and has been shown to reduce mortality in hypertensive patients.

Alpha 1 adrenergic blocking agents can be used as initial therapy. The alpha 1 blocking agent prazosin and related drugs have an added advantage in treating hypertensive patients with coexisting hyperlipidemia since they seem to have beneficial effects on lipid levels. Selective blockade of the post synaptic alpha 1 receptors by prazosin and related agents reduces peripheral vascular resistance and systemic blood pressure. In addition, all alpha 1 adrenergic blocking agents relieve symptoms of benign prostatic hyperplasia.

ACE inhibitors are the preferred drugs for patients with coexisting heart failure. They are useful in initial therapy in hypertensive patients with kidney damage or diabetes mellitus with proteinuria, and in white patients.[18] No clinically relevant differences have been found among the available ACE inhibitors.[23] They are well-tolerated by young, physically active patients and the elderly. The most common adverse effect of the ACE inhibitors is dry cough. Angiotensin II receptor blockers produce hemodynamic effects similar to ACE inhibitors while avoiding dry cough.[24] These agents are similar to the ACE inhibitors in potency and are useful for initial therapy.

The calcium channel blocking agents are effective as initial therapy in both black and white patients and are well-tolerated by the elderly. These agents inhibit entry of calcium ion into cardiac cells and smooth muscle cells of the coronary and systemic vasculature. Nifedipine (Procardia) and amlodipine (Norvasc) are more potent as peripheral vasodilators than diltiazem (Cardizem). Long-acting formulations of the calcium channel blockers have been shown to be very safe despite some earlier reports that short acting calcium channel blockers were associated with a 60 percent increase in heart attacks among hypertensive patients given a short-acting calcium antagonist.[25]

Supplemental antihypertensive agents include the centrally acting alpha-2 agonists and direct-acting vasodilators. These agents are less commonly prescribed for initial therapy because of the impressive effectiveness of the other drug groups. Clonidine (Catapres) lowers blood pressure by activating inhibitory alpha 2 receptors in the central nervous system, thus reducing sympathetic outflow. It lowers both supine and standing blood pressure by reducing total peripheral resistance. Hydralazine (Apresoline) reduces blood pressure by directly relaxing arteriolar smooth muscle. Hydralazine is given orally for the management of chronic hypertension, usually with a diuretic and a beta blocker.

Drugs to Treat Unstable Angina Pectoris

Numerous physiologic triggers can initiate the rupture of plaque in coronary blood vessels. Rupture leads to the activation, adhesion, and aggregation of platelets and the activation of the clotting cascade, resulting in the formation of occlusive thrombus. If this process leads to complete occlusion of the artery, then acute myocardial infarction with ST-segment elevation occurs. Alternatively, if the process leads to severe stenosis and the artery remains patent, then unstable angina occurs. Triggers that induce unstable angina include physical exertion, mechanical stress due to an increase in cardiac contractility, pulse rate, blood pressure, and vasoconstriction.

Unstable angina accounts for more than 1 million hospital admissions each year.26 In 1989, Braunwald devised a classification system for angina according to the severity of the clinical manifestations.[27] These manifestations are defined as acute angina while at rest

(within the 48 hours before presentation), subacute angina while at rest (within the previous month but not within the 48 hours before presentation), or new onset of accelerated (progressively more severe) angina. The system also classifies angina according to the clinical circumstances in which unstable angina develops, defined as either angina in the presence or absence of other conditions such as fever, hypoxia, tachycardia, thyrotoxicosis, and whether or not ECG abnormalities are present. Recently, the term "acute coronary syndromes" has been used to describe the range of conditions that includes unstable angina, non-Q-wave myocardial infarction, and Q-wave myocardial infarction.

Pharmacologic therapy to treat the syndrome of unstable angina includes antiplatelet drugs, antithrombin therapy and conventional antanginal therapy with beta blockers, nitrates, and calcium channel blockers. These drug groups and selected agents are listed in **Table 5**.

Antiplatelet Drugs

Aspirin reduces platelet aggregation by blocking platelet cyclooxygenase through irreversible acetylation. This action prevents the formation of thromboxane A2. A number of studies have confirmed that aspirin reduces the risk of death from cardiac causes and fatal and nonfatal myocardial infarction by approximately 50 percent to 70 percent in patients presenting with unstable angina.[28] Ticlopidine is a second-line alternative to aspirin in the treatment of unstable angina and is also used as adjunctive therapy with aspirin to prevent thrombosis after placement of intracoronary stents. Ticlopidine blocks ADP-mediated platelet aggregation. Clopidogrel inhibits platelet aggregation by affecting the ADP-dependent activation of the glycoprotein IIb/IIIa complex. Clopidogrel is chemically related to ticlopidine but has fewer side effects than ticlopidine.

Platelet glycoprotein IIb/IIIa receptor antagonists. Antagonists of glycoprotein IIb/IIIa, a receptor on the platelet for adhesive proteins, inhibit the final common pathway involved in adhesion, activation, and aggregation. There are three classes of inhibitors. One class is murine-human chimeric antibodies of which abciximab is the prototype. The other two classes are the synthetic peptide forms (e.g., eptifibatide) and the synthetic nonpeptide forms (e.g., tirofiban). These agents have been used to treat unstable angina in combination with heparin and aspirin to significantly reduce the incidence of death or myocardial infarction.

Antithrombin Drugs

Unfractionated heparin in combination with aspirin is used to treat unstable angina. Unfractionated heparin consists of polysaccharide chains that bind to antithrombin III and cause conformational change that accelerates the inhibition of thrombin and factor Xa by antithrombin III. Unfractionated heparin is, therefore, an indirect thrombin inhibitor. Unfractionated heparin can only be administered intravenously. Low-molecular-weight heparins have a more predictable pharmacokinetic profile than the unfractionated heparin and can be administered subcutaneously. These heparins have a mechanism of action and use similar to unfractionated heparin.

The direct antithrombins decrease thrombin activity in a manner independent of any actions on antithrombin III. One such direct antithrombin is lepirudin, also known as recombinant hirudin. This agent is a highly specific direct thrombin inhibitor with each molecule capable of binding to one molecule of thrombin and inhibiting its thrombogenic activity. Direct antithrombins are used for the prevention or reduction of ischemic complications associated with unstable angina.

Warfarin (Coumadin) elicits its anticoagulant effect by interfering with the hepatic synthesis of vitamin K-dependent coagulation factors II, VII, IX, and X. Although warfarin appears to be somewhat effective after myocardial infarction in preventing death or recurrent myocardial infarction, its effectiveness in the treatment of acute coronary syndrome is questionable. Combination therapy with aspirin and heparin followed by warfarin has resulted in reduction of recurrent angina, myocardial infarction, death or all three at 14 days as compared to aspirin alone.[29] Another study, however, failed to show any additional benefit from a combination of aspirin and warfarin compared to aspirin alone in the treatment of acute coronary syndrome.[30]

Conventional Antianginal Therapy (Beta Blockers, Nitrates, Calcium Channel Blockers)

Current thinking is that there is a definite link between unstable angina and acute myocardial infarction. In this regard, beta blockers are currently recommended as first-line agents in all acute coronary syndromes.[28] A meta analysis of studies involving 4,700 patients with unstable angina demonstrated a 13 percent reduction in the risk of myocardial infarction among patients treated with beta blockers. The various preparations of beta blockers appear to all have equal efficacy.[28] The effects of beta blockers are thought to be due to their ability to decrease myocardial oxygen demand.

Nitrates, such as nitroglycerin, are widely used in the management of unstable angina. Nitrates elicit a number of effects, including a reduction in oxygen demand, arteriolar vasodilation, augmentation of collateral coronary blood flow, and reduction in the frequency of coronary vasospasm. Intravenous nitroglycerin is one of the first line therapies for unstable angina because of the ease of dose titration and the rapid resolution of effects. Continuous nitrate therapy with oral and transdermal patch preparations has resulted in tolerance to the beneficial effects of nitrates. A six- to eight-hour daily nitrate-free interval will minimize the tolerance phenomenon. Also, supplemental use of vitamin C appears to prevent nitrate tolerance.[31] The different nitrate preparations used to treat unstable

angina are listed in **Table 5**.

Calcium channel blockers such as nifedipine, verapamil, and diltiazem cause coronary vasodilation and reduced blood pressure. Because of these actions, the calcium channel blockers were thought to be a drug group that could be effective in the treatment of unstable angina. However, a meta analysis of studies in which patients with unstable angina were treated with calcium channel blockers found no effect of the drugs on the incidence of death or myocardial infarction.[28] More recently, it has been shown that treatment with diltiazem and verapamil may result in increased survival and reduced rates of reinfarction in patients with acute coronary syndrome. Current thinking suggests that calcium channel blockers should be used in patients in whom beta blockers are contraindicated or those with refractory symptoms after treatments with aspirin, nitrates or beta blockers. The different calcium channel blockers used to treat unstable angina are listed in **Table 5**.

References

1. American Heart Association, Heart and stroke facts: 1996 statistical supplement. National Center of the American Heart Association, Dallas, 1996, p 15.
2. The Criteria Committee of the American Heart Association, New York City Affiliate, 1994 revisions to classification of functional capacity and objective assessment of patients with diseases of the heart. Circulation 90:644-5, 1994.
3. Heidenreich PA, Lee TT, Massie BM, Effect of beta-blockade on mortality in patients with heart failure: a meta-analysis of randomized clinical trails. J Am Coll Cardiol 30: 27-34, 1997.
4. Libby P, Molecular basis of the acute coronary syndromes. Circulation 91:2844-50, 1995.
5. Albers GW, Atwood JE, et al. Stroke prevention in nonvalvular atrial fibrillation. Ann Intern Med 115: 727-36, 1991.
6. Cairns JA, Connolly SJ, Nonrheumatic atrial fibrillation. Risk of stroke and role of antithrombotic therapy. Circulation 84:469-81, 1991.
7. Morley J, Marinchak R, et al, Atrial fibrillation, anticoagulation and stroke. Am J Cardiol 77:38A-44A, 1996.
8. Pritchett EL, Management of atrial fibrillation. N Eng J Med 326:1264-71, 1992.
9.Vaughn Williams EM, A classification of antiarrhythmic actions reassessed after a decade of new drugs. J Clin Pharmacol 24:129-47, 1984.
10. Sopher SM, Camm AJ, Atrial fibrillation: maintenance and sinus rhythm versus rate control. Am J Cardiol 77:24A-37A, 1996.
11. Gilligan DM, Ellenbogen KA, Epstein AE, The management of atrial fibrillation. Am J Med 101:413-21, 1996.
12. Golzari H, Cebul RD, Bahler RC, Atrial fibrillation: restoration and maintenance of sinus rhythm and indications for anticoagulation therapy. Ann Intern Med 125:311-23, 1996.
13. Atrial Fibrillation Investigators, Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation: analysis of pooled data from five randomized controlled trials. Arch Intern Med 154:1449-57, 1994.
14. Stroke prevention in atrial fibrillation investigators, Warfarin versus aspirin for prevention of thromboembolism in atrial fibrillation. Stroke prevention in atrial fibrillation II study. Lancet 343:687-91, 1994.
15. Hylek EM, Skates SJ, et al. An analysis of the lowest effective intensity of prophylactic coagulation for patients with nonrheumatic atrial fibrillation. N Engl J Med 335:540-6, 1996.
16. Stroke prevention in atrial fibrillation investigators, Adjusted-dose warfarin versus low-intensity, fixed-dose warfarin plus aspirin for high-risk patients with atrial fibrillation: stroke prevention in atrial fibrillation III randomized clinical trial. Lancet 348:633-8, 1996.
17. The European Atrial Fibrillation Trial Study Group, Optimal oral anticoagulant therapy in patients with nonrheumatic atrial fibrillation and recent cerebral ischemia. N Engl J Med 333:5-10, 1995.
18. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI). Ann Intern Med 157:2413-46, 1997.
19. Kannel WB, Blood pressure as a cardiovascular risk factor. J Am Med Assoc 275:1571- 6, 1996.
20. Hansson L, Zanchetti A, et al, Effects of intensive blood pressure lowering and low-dose aspirin in patients with hypertension: principal results of the Hypertension Optimal Treatment (HOT) randomised trial. HOT Study Group. Lancet 351:1755-62, 1998.
21. Materson BJ, Reda DJ, et al, Single-drug therapy for hypertension in men. A comparison of six antihypertensive agents with placebo. N Eng J Med 328:914-21, 1993.
22. Neaton JD, Grimm RH Jr, et al, Treatment of Mild Hypertension Study. Final results. Treatment of mild hypertension Study Research Group. J Am Med Assoc 270:713-24, 1993.
23. Leonetti G, Cuspidi C, Choosing the right ACE inhibitor, a guide to selection. Drugs 49: 516-35, 1995.
24. Kaplan NM, Perspectives on the new JNC VI guidelines for the treatment of hypertension. Formulary 32:1224-31, 1997.
25. Furberg CD, Psaty BM, Meyer JV, Nifedipine: Dose-related increase in mortality in patients with coronary heart disease. Circulation 92:1326-31, 1995.
26. Graves E, National Hospital Discharge Survey. Annual survey 1996. Series 13, no. 4. National Center for Health Statistics, Washington, DC, 1998.
27. Braunwald E, Unstable angina: a classification. Circulation 80:410-4, 1989.
28. Yeghiazarians Y, Braunstein JB, et al, Unstable angina pectoris. New Eng J Med 342:101-14, 2000.
29. Cohen MC, Adams PC, et al, Combination antithrombotic therapy in unstable rest angina and non-Q-wave infarction in nonprior aspirin users: primary end points analysis from the ATACS trial. Circulation 89:81-8, 1994.
30. Coumadin Aspirin Reinfarction Study (CARS) Investigators, Randomised double-blind trail of fixed low-dose warfarin with aspirin after myocardial infarction. Lancet 350:389-96, 1997.
31. Bassenge E, Fink N, et al, Dietary supplement with vitamin C prevents nitrate tolerance. Am J Cardiol 77:24C-30C, 1996.
32. United States Pharmacopeial Dispensing Information, Therapeutic monograph, atrial fibrillation. In, USPDI volumes I and II update. The United States Pharmacopeial Convention Inc, Rockville, MD, December 1998:2118-40.
33. United States Pharmacopeial Dispensing Information, Therapeutic monograph, atrial fibrillation. In: USPDI volumes I and II update. The United States Pharmacopeial Convention Inc, Rockville, MD, April 1998: 630-45.
34. United States Pharmacopeial Dispensing Information, Therapeutic monograph, atrial fibrillation. In, USPDI volumes I and II update. The United States Pharmacopeial Convention Inc, Rockville, MD, November 1998, 1954-82..
35. United States Pharmacopeial Dispensing Information, Drug information for the health care professional, volume I, ed. 19. The United States Pharmacopeial Convention Inc, Rockville, MD, 1999.

To request a printed copy of this article, please contact/Richard L. Wynn, PhD, Dental Department of Pharmacology, University of Maryland, 666 W. Baltimore St., Baltimore, MD 21201 or e-mail at rlw001@dental.umaryland.edu

Table 1.

Drugs Used to Treat Heart Failure

Angiotensin converting enzyme (ACE) inhibitors*

Benazepril (Lotensin)
Captopril (Capoten)
Enalapril (Vasotec)
Fosinopril (Monopril)
Lisinopril (Prinivil)
Perindopril (Aceon)
Quinapril (Accupril)
Ramipril (Altace)
Trandolapril (Mavik)

Diuretics

Thiazides
- Hydrochlorothiazide (Hydrodiuril)

Loop diuretics
- Furosemide (Lasix)

Potassium-sparing agents
- Amiloride and hydrochlorothiazide (Moduretic)
- Spironolactone (Aldactone)
- Spironolactone and hydrochlorothiazide (Aldactizide)
- Triamterene and hydrochlorothiazde (Dyazide)

Digitalis glycosides

Digoxin (Lanoxin)
Digitoxin

Beta-adrenergic receptor blocking drugs

Bisoprolol (Zebeta)
Carvedilol (Coreg)
Labetolol (Normodyne, Trandate)
Metoprolol (Lopressor)

Supplemental agents

Direct-acting vasodilators
- Hydralazine (Apresoline)
- Nitroglycerin
- Sodium nitroprusside (Nitropress)

Catecholamines
- Dobutramine (Dobutrex)
- Dopamine

Phosphodiesterase inhibitors
- Amrinone (Inocor)
- Milrinone (Primacor)

*Regarded as the cornerstone of treatment of heart failure and should be used routinely and early in all patients.

From USPDI volumes I and II update, December 1998 [32]

Table 2.

Drugs Used in the Treatment of Coronary Artery Disease

Reduction of total and low-density cholesterol levels

HMG-CoA reductase inhibitors
- Fluvastatin
- Lovastatin
- Pravastatin
- Simvastatin
- Atorvastatin
- Cerivastatin

Fibrate group
- Gemfibrozil
- Clofibrate

Bile acid resins
- Cholestyramine
- Colestipol

Nicotinic acid

Cardioprotective drug therapy

Antiplatelet/anticoagulant agents
- Aspirin
- Ticlopidine
- Clopidogrel
- Warfarin

Beta -adrenergic receptor blockers
- Metoprolol
- Atenolol
- Propranolol

Angiotensin Converting Enzyme (ACE) inhibitors
- Captopril
- Enalapril
- Ramipril
- Lisinorpil
- Fosinopril

Table 3.

Drugs Indicated for the Treatment of Atrial Fibrillation *

Class I Antiarrhythmic Agents

Disopyramide (Norpace) (I A)
Flecainide (Tambocor) (I C)
Procainamide (Pronestyl) (I A)
Propafenone (Rythmol) (I C)
Quinidine (I A)

Class II Antiarrhythmic Agents (Beta-adrenergic Blocking Drugs)

Cardioselective (beta 1 – receptor block only)
- Acebutolol (Sectral)
- Atenolol (Tenormin)
- Betaxolol (Kerlone)
- Metoprolol (Lopressor)

Noncardioselective (beta 1 and beta 2 – receptor block)
- Nadolol (Corgard)
- Penbutolol (Levatol)
- Pindolol (Visken)
- Propranolol (Inderal)
- Timolol (Blocadren)

Class III Antiarrhythmic Agents

Amiodarone (Cordarone)

Class IV Antiarrhythmic Agents (Calcium Channel Blocking Agents)
- Diltiazem (Cardizem)
- Verapamil (Calan)

Miscellaneous Agents

Digitalis
Anticoagulants
- Warfarin (Coumadin)
- Aspirin

From USPDI volumes I and II update, April 1998 [33]

Table 4.

Drug Categories and Representative Agents for the Treatment of Hypertension *

Diuretics
Thiazide-type
Hydrochlorothiazide (HCTZ)
Loops
Furosemide (Lasix)
Potassium-sparing
Triamterene (Dyrenium)
Potassium-sparing combinations
triamterene/HCTZ (Dyazide)

Beta blockers
Cardioselective beta blockers
Atenolol (Tenormin)
Noncardioselective beta blockers
propranolol (Inderal)

Alpha, blocker
Prazosin (Minipress)

Alpha/Beta blocker
Carvedilol (Coreg)

Angiotensin-converting enzyme (ACE) inhibitors
Lisinopril (Prinivil)
enalapril (Vasotec)

Angiotensin-converting enzyme inhibitors/ diuretic combination
Captopril and HCTZ (Capozide)

Angiotensin II receptor blockers
Candesartan (Atacand)
Losartan (Cozaar)

Calcium channel blocking agents
Amlodipine (Norvasc)
Nifedipine (Procardia)
Diltiazem (Cardizem)

Supplemental agents
Central-acting alpha 2-agonist
Clonidine (Catapres)
Direct-acting peripheral vasodilator
Hydralazine (Apresoline)

From USPDI volumes I and II update, November 1998 [34]

Table 5.

Drugs Used to Manage Unstable Angina

Antiplatelet drugs
Aspirin
Ticlopidine (Ticlid)
Clopidogrel (Plavix)
Glycoprotein IIb/IIIa receptor antagonists
Abciximab (ReoPro)
Eptifibatide (Integrilin)
Tirofiban (Aggrastat)

Antithrombin drugs
Indirect thrombin inhibitors
Unfractionated heparin (Heparin)
Low molecular-weight heparin (Enoxaparin)
Direct thrombin inhibitors
Lepirudin (Hirudin)
Dicumarols
Warfarin (Coumadin)

Conventional antianginal drugs
Beta blockers
Atenolol (Tenormin)
Bisoprolol (Zebeta)
Carteolol (Cartrol)
Vadolol (Corgard)
Propranolol (Inderal)
Nitrates
Erythrityl tetranitrate (Cardilate)
Isosorbide tetranitrate (Diltrate – SR, Isordil)
Isosorbide mononitrate (Imdur)
Nitroglycerin (sublingual tablets, oral tablets, Sublingual spray, transdermal patch)
Pentaerythritol tetranitrate (Duotrate, Peritrate)
Calcium channel blockers
Diltiazem (Cardizem)
Nifedipine (Procardia, Adalate)
Verapamil (Calan)

Table 6.

Cardiovascular/Respiratory/Nervous System Effects Caused by Drugs Used for Cardiovascular Disorders

Alpha 1 blocker

prazosin (Minipress): More frequent: orthostatic hypotension, dizziness; Less frequent: heart palpitations; Rare: angina.

Alpha/Beta blocker

carvedilol (Coreg): More frequent: bradycardia, postural hypotension, dizziness; Rare: A-V block, hypertension, hypotension, palpitations, vertigo, nervousness, asthma.

Angiotensin-converting enzyme (ACE) inhibitors

benazepril (Lotensin): Less frequent: dizziness, insomnia, headache; Rare: hypotension, bronchitis.
captopril (Capoten): Less frequent: tachycardia, insomnia, transient cough, dizziness, headache; Rare: hypotension
enalapril (Vasotec): Less frequent: chest pain, palpitations, tachycardia, syncope, dizziness, dyspnea; Rare: angina pectoris, asthma
fosinopril (Monopril): Less frequent: orthostatic hypotension, dizziness, cough headache; Rare: syncope, insomnia.
lisinopril (Prinivil): Less frequent: hypotension, dizziness; Rare: angina pectoris, orthostatic hypotension, rhythm disturbances, tachycardia,
perindopril (Aceon): Less frequent: headache, dizziness, cough (incidence > women 3:1); Rare: hypotension.
quinapril (Accupril): Less frequent: hypotension, dizziness, headache, cough; Rare: orthostatic hypotension, angina, insomnia.
ramipril (Altace): Less frequent: tachycardia, dizziness, headache, cough; Rare: hypotension.
trandolapril (Mavik): Less frequent: tachycardia, headache, dizziness, cough (more frequent in women); Rare: hypotension.

Angiotensin-converting enzyme inhibitors/diuretic combination

captopril and HCTZ (Capozide): Less frequent: tachycardia, palpitations, chest pain, dizziness; Rare: hypotension

Angiotensin II receptor blockers

Candesartan (Atacand): Less frequent: chest pain, flushing; Rare: myocardial infarction, tachycardia, angina, palpitations, dyspnea.
Losartan (Cozaar): Less frequent: hypotension without reflex tachycardia, dizziness; Rare: orthostatic hypotension, angina, A-V block (second degree), CVA, palpitations, tachycardia, sinus bradycardia, flushing, dyspnea.

Antiplatelet/anticoagulant agents

Aspirin, ticlopidine, clopidogrel, warfarin: no cardiorespiratory or nervous system effects reported.
Abciximab (ReoPro): More frequent: hypotension, pain; Less frequent: bradycardia
Eptifibatide (Integrilin): More frequent: hypotension, bleeding.
Tirofiban (Aggrastat): More frequent: bleeding; Less frequent: bradycardia, dizziness, headache.

Beta blockers

Acebutolol (Sectral): Less frequent: chest pain, bradycardia, hypotension, dizziness, dyspepsia, dyspnea; Rare: ventricular arrhythmias
Atenolol (Tenormin): Less frequent: bradycardia, hypotension, chest pain, dizziness, dyspepsia, dyspnea; Rare: ventricular arrhythmias
Betaxolol (Kerlone): Less frequent: bradycardia, palpitations, dizziness; Rare: chest pain
Bisoprolol (Zebeta): More frequent: lethargy; Less frequent: hypotension, chest pain, bradycardia, headache, dizziness, insomnia, cough
Labetolol (Normodyne, Trandate): Less frequent: orthostatic hypotension, dizziness, nasal congestion; Rare: bradycardia, chest pain
Metoprolol (Lopressor): More frequent: dizziness; Less frequent: bradycardia, heartburn, wheezing; Rare: chest pain, confusion.
Nadolol (Corgard): More frequent: bradycardia; Less frequent: dizziness, dyspepsia, wheezing; Rare: congestive heart failure, orthostatic hypotension, confusion, paresthesia.
Penbutolol (Levatol): Less frequent: congestive heart failure, dizziness; Rare: bradycardia, chest pain, hypotension, confusion.
Pindolol (Visken): More frequent: dizziness; Less frequent: congestive heart failure, dyspnea.
Propranolol (Inderal): More frequent: bradycardia; Less frequent: congestive heart failure, dizziness, wheezing; Rare: chest pain, hypotension, bronchospasm.
Timolol (Blocadren): Less frequent: bradycardia, dizziness, dyspnes; Rare: chest pain, congestive heart failure

Table 7. Cont.

Calcium channel blocking agents

Amlodipine (Norvasc): Less frequent: palpitations, dizziness, dyspnea; Rare: hypotension, bradycardia, arrhythmias

Diltiazem (Cardizem): Less frequent: bradycardia, dizziness; Rare: dyspepsia, paresthesia, tremor.

Nifedipine (Procardia): More frequent: flushing, dizziness; Less frequent: palpitations, hypotension, dyspnea; Rare: tachycardia, syncope

Verapamil (Calan): Less frequent: bradycardia, congestive heart failure, hypotension; Rare: chest pain, hypotension.

Class I antiarrhythmics

Disopyramide (Norpace): More frequent: exacerbation of angina pectoris, dizziness; Less frequent: hypotension, hypertension, tachycardia, dyspnea; Rare: syncope, flushing, hyperventilation.

Flecainide (Tambocor): More frequent: dizziness, dyspnea; Less frequent: palpitations, chest pain, tachycardia, tremor; Rare: bradycardia, nervousness, paresthesia.

Procainamide (Pronestyl): Less frequent: tachycardia, dizziness, lightheadedness; Rare: hypotension, confusion, disorientation.

Propafenone (Rythmol): More frequent: dizziness; Less frequent: palpitations, angina, bradycardia, loss of balance, dyspepsia, dyspnea; Rare: paresthesia

Qunidine: Less frequent: hypotension, syncope, lightheadedness, wheezing; Rare: confusion, vertigo, angina, edema.

Class III antiarrhythmics

Amiodarone (Cordarone): More frequent: dizziness, tremor, paresthesia, dyspnea; Less frequent: congestive heart failure, bradycardia, tachycardia; Rare: hypotension.

Digitalis glycosides

digoxin; digitoxin: Rare: atrial tachycardia, sinus bradycardia, ventricular fibrillation, vertigo

Diuretics

Thiazide-type Rare: hypotension

Loops: More frequent: orthostatic hypotension, dizziness

Potassium -sparing: Less frequent: hypotension, bradycardia, dizziness; Rare: flushing

Potassium-sparing combinations: Rare: dizziness

HMG-CoA reducatase inhibitors

Atorvastatin, fluvastatin, lovastatin, pravastatin, simvastatin: Less frequent: headache, dizziness

Nitrates

Nitroglycerin drugs: More frequent: postural hypotension, flushing, headache, dizziness; Rare: reflex tachycardia, bradycardia, arrhythmias.

Supplemental drugs for heart failure

Hydralazine (Apresoline): More frequent: tachycardia, headache; Less frequent: hypotension, nasal congestion; Rare: edema, dizziness

Sodium nitroprusside: Less frequent: palpitations, headache

Dobutamine (Dobutrex): Less frequent: tachycardia, chest pain; Rare: headache, dyspnea.

Amrinone (Inocor): Less frequent: arrhythmia, chest pain.

Milrinone (Primacor) More frequent: arrhythmias; Less frequent: chest pain;

Supplemental antihypertensives

Central-acting alpha 2-agonists; clonidine (Catapres): More frequent: dizziness; Less frequent: orthostatic hypotension, nervousness/agitation; Rare: palpitations, tachycardia, bradycardia, congestive heart failure

Direct-acting; hydralazine (Apresoline): More frequent: tachycardia, headache; Less frequent: hypotension, nasal congestion; Rare: edema, dizziness

From information found in professional package insert for individual agent or the USPDI35

More frequent = > 10 percent incidence; less frequent = 1 percent to 10 percent; rare = < 1 percent incidence.

Table 7.

Cardiovascular Drugs: Dental Drug Interactions and Effects on Dental Treatment *

Any drug known to cause skin allergy is likely to cause a mouth allergy as well.

Alpha 1 blocker

Prazosin (Minipress): Significant orthostatic hypotension a possibility; monitor patient when getting out of dental chair; significant dry mouth in up to 10% of patients.

Alpha/Beta blocker

Carvedilol (Coreg): See nonselective beta blockers.

Angiotensin-converting enzyme (ACE) inhibitors

The NSAID indomethacin reduces the hypotensive effects of ACE inhibitors. Effects of other NSAIDs such as ibuprofen not considered significant.

Angiotensin-converting enzyme inhibitors/diuretic combination

Captopril and HCTZ (Capozide): No effect or complications on dental treatment reported.

Angiotensin II receptor blockers

No effect or complications on dental treatment reported.

Beta blockers

Cardioselective beta blockers; i.e. atenolol (Tenormin): No effect or complications on dental treatment reported. Noncardioselective beta receptor blockers: Any of the noncardioselective beta blockers (i.e. nadolol, penbutolol, pindolol, propranolol, timolol) may enhance the pressor response to vasoconstrictor epinephrine resulting in hypertension and reflex bradycardia. Although not reported, it is assumed that similar effects could be cause with levonordefrin (Neo-Cobefrin). Use either vasoconstrictor with caution in hypertensive patients medicated with noncardioselective beta adrenergic blockers.

Calcium channel blocking agents

Cause gingival hyperplasia in approximately 1 percent of the general population taking these drugs. There have been fewer reports with diltiazem and amlodipine than with other CBs such as nifedipine. The hyperplasia will usually disappear with cessation of drug therapy. Consultation with the physician is suggested.

Antiplatelet/anticoagulant agents

Aspirin may cause a reduction in the serum levels of NSAIDs if they are used to manage post-operative pain; clopidogrel: if a patient is to undergo elective surgery and an antiplatelet effect is not desired, clopidogrel should be discontinued 7 days prior to surgery; warfarin: signs of warfarin overdose may first appear as bleeding from gingival tissue; consultation with prescribing physician is advisable prior to surgery to determine temporary dose reduction or withdrawal of medication; eptifibatide: bleeding may occur while patient is medicated with eptifibatide; platelet function is restored in about 4 hours following discontinuation;

Class I antiarrythmics

Disopyramide: Increased serum levels and toxicity with erythromycin. High incidence of anticholinergic effect manifested as dry mouth and throat. Flecainide: No effects or complications on dental treatment reported. Procainamide: systemic lupus-like syndrome has been reported resulting in joint pain and swelling, pains with breathing, skin rash. Propafenone: >10 % experience significantly reduced salivary flow; taste disturbance, bitter or metallic taste. Qunidine: secondary anticholinergic effects may decrease salivary flow, especially in middle age and elderly patients. Known to contribute to caries, periodontal disease and oral candidiasis.

Class III antiarrythmics

Amiodarone: Bitter or metallic taste has been reported.

Digitalis glycosides

Digitalis: Use vasoconstrictor with caution due to risk of cardiac arrhythmias. Sensitive gag reflex induced by digitalis drugs may cause difficulty in taking dental impressions. Erythromycin and tetracyclines may increase digitalis serum levels.

Diuretics

Thiazide-type: Many diuretics are known to reduce salivary flow. Monitor for xerostomia. Loops: NSAIDs may increase chloride and tubular water reuptake to counter-act loop type diuretics. Many diuretics are known to reduce salivary flow. Monitor for xerostomia. Potassium-sparing: Many diuretics are known to reduce salivary flow. Monitor for xerostomia. Potassium-sparing combinations: Many diuretics are known to reduce salivary flow. Monitor for xerostomia.

HMG-CoA reductase inhibitors

Concurrent use of erythromycin and some of the statin drugs may result in rhabdomyolysis.

Nitrates

No effects or complications on dental treatment reported.

Supplemental drugs for heart failure

Amrinone, milrinone: no effects or complications on dental treatment reported.

Supplemental antihypertensives

Central-acting alpha 2-agonists; clonidine (Catapres): Greater than 10% of patients experience significant dry mouth. Hydralazine (Apresoline): No effect or complications on dental treatment reported.

From Drug Information Handbook for Dentistry, 5th ed. RL Wynn, TF Meiller, HL Crossley, Lexi-Comp, Inc, Hudson (Cleveland), 1999-2000.

CDA JOURNAL, VOL 28, Nº7

Resources for Treating Patients With Medically Complex Problems

LEWIS R. EVERSOLE, DDS, MSD, MA, AND WILLIAM M. CARPENTER, DDS, MS

AUTHORS

Lewis Roy Eversole, DDS, MSD, MA, is a professor of pathology and medicine at the UOP School of Dentistry and a head and neck pathology consultant to Pathology Consultants of New Mexico.

William M. Carpenter, DDS, MS, is professor and chairman in the Department of Pathology and Medicine at the University of the Pacific School of Dentistry.

Patients seeking dental consultation and care may present with signs and symptoms of undiagnosed medical problems or conditions or may have a significant health history and be under the care of a physician. Current and previous medical illness and use of pharmacologic agents will often affect dental care, particularly when surgical procedures are in the general treatment plan. Dentists will be caring for an aging population whose members are enjoying longer life spans and greater quality of life due to improvements in health care and disease prevention. The degenerative diseases of aging will be more frequently encountered in the dental office and may put the dentist in a quandary with regard to patient management. The explosion of knowledge and increasing detail of information surfacing daily concerning medical conditions can place practitioners at a disadvantage because being knowledgeable about all this new information and how it affects dental practice is simply not feasible. However, up-to-date information on specific conditions is available through publications, consultation, Web sites and chat groups.

The purpose of this article is to inform the practitioner of the availability of those resources that will assist in answering clinical questions regarding dental management of medically complex patients. The clinician may not know where to go to look for current information on relevant subjects. Herein is compiled a

list of resources that the dental practitioner can consult when questions arise about managing patients with medical problems.

Dental patients can present with various pathologic conditions that require special consideration. Subjects with atherosclerotic vascular occlusive disease, hypertension, diabetes, chronic liver disease, renal insufficiency, neoplastic disease, neurologic deficits, and infectious diseases are often taking numerous drugs. When contemplating dental treatments, the practitioner will need to review the patient's medical history and arrive at decisions that will protect the patient from complications. In this scenario, time is not of the essence; the dentist can consult available resources and devise a modification in the dental treatment plan for a later appointment. In the case of medical emergencies, time is critical and there will be no opportunity for consultation. In such instances, the practitioner will have to be well-versed in life support, airway control, and cardiopulmonary resuscitation. The primary resource for medical emergencies arising in the dental office is calling emergency services at 9-1-1.

For the patient with a significant medical history, the dentist has a wide array of resources to consult. The sources available to everyone in practice are listed in **Table 1**.

Direct consultation with the patient's physician is always an option when one is concerned about medical problems that affect dental care. Unfortunately, not all physicians are familiar with dental procedures and may not comprehend the significance of the dentist's concerns. However, there are more than 50 dental schools in the United States, and all of these institutions have a department that includes oral medicine, oral pathology or oral diagnostic sciences. The faculty members in these institutions are often available and willing to consult over the telephone. Some dental schools offer Internet consultation on their Web sites. Interested practitioners need only call the school to acquire the appropriate departmental phone numbers and Web site addresses.

Published texts and articles have always served as the primary resources for information on medical problems as they affect dental practice. The American Academy of Oral Medicine has published The Clinician's Guide to Treatment of the Medically Compromised Dental Patient and The HIV Patient. A list of these and other publications is given in **Table 2**. Texts that deal with medical illness and dental practice usually have relatively current recommendations for management. The American Dental Association Council on Access, Prevention, and Interprofessional Relations has published a series of oral health guidelines that are available through ADA catalog sales.

Of course, everything one would ever need or want to know can also be found on the Internet. Web sites with information germane to the dental care of medically complex patients are listed in **Table 3**. The National Library of Medicine Web sites hosts Grateful Med, a medical and dental publication search engine (http://igm.nlm.nih.gov/). This site is simple to use. It is not necessary to know the exact title of an article to use the system. Rather, the format allows for searches according to subject. There are two boxes that will help the user limit the search to the area of interest (**Figure 1**). For example, a dentist might be concerned about a potential bleeding disorder in one of his or her patients who is taking coumadin for thromboembolic disease. To access information from the medical literature on this issue, the dentist would type "bleeding disorder" into the first box and designate the box as "subject" The second box will help narrow the search so that retrieval of articles will focus on dental care. To do this, one would type "dental," "dentistry," or "tooth extraction" in the second box and label it as "subject." Once the categories have been selected, the use hits the "perform search" button. After the results are displayed, the user chooses the articles he or she is interested in and clicks on the box that reads "full citation" to peruse the abstract (**Figure 2**). The user can then download the selected abstracts by clicking the box next to the articles of interest and hitting the "download to disk" button. Choose "download only the records you have selected" and "long records with abstracts" and click on "prepare to download" (**Figure 3**). Then choose the IGM format and click "download now." This will combine all the selected abstracts into one file that can then be saved onto the user's hard drive as text and opened with a word processing program. The practitioner will then have the current literature on this subject he or she is researching.

Other Internet resources include list-serves and bulletin boards. For example, the Bulletin Board of Oral Pathology has a list-serve that anyone can join. By subscribing to the list, participants will receive all posted messages and can post responses. Oral pathologists and oral medicine specialists utilize the bulletin board to discuss current issues.

These are some of the most helpful references to assist the practitioner in caring for the medically complex patient. In the future, schools and other organizations will no doubt expand the resources available for consultation on all aspects of dental care.

To request a printed copy of this article, please contact/ Lewis Roy Eversole, DDS, MSD, MA, UOP School of Dentistry, 2155 Webster St., San Francisco, CA 94115. Or e-mail the author at leversol@uop.edu.

Table 1.
Information Systems for Medically Complex Patients

Direct Consultations
Patient's treating physician
Oral medicine and oral pathology faculty
Published Data
Dental journals
Textbooks
Monographs and guidelines
Information Technology
Medlar Grateful Med Literature Search
Internet bulletin boards, list-serves, and chat groups

Table 2. Published Resources for Medically Complex Patients

Textbooks

Little JW, Falace DA, et al, Dental Management of the Medically Compromised Patient, 5th ed. Mosby-Year Book, St Louis, 1996.

Eversole LR, A Pocket Guide. WB Saunders Co, Philadelphia, 1996.

Silverman S, Oral Cancer. American Cancer Society Inc and BC Decker Publishing, Ontario, 1998.

Silverman S, Eversole, L, Truelove E, Essentials of Oral Medicine. BC Decker Publishing, Ontario, 2000.

Lynch MA, Brightman VJ, Greenberg MS, Burkett's Oral Medicine, 9th ed. Lippincott Williams & Wilkins, Philadelphia, 2000.

Coleman GC, Nelson JF, Principles of Oral Diagnosis. Mosby-Year Book, St Louis, 1993.

Bricker SL, Langlais RP, Miller CS, Oral Diagnosis, Medicine and Treatment Planning, 2nd ed. Lea & Febiger, Philadelphia, 1994.

Sonis ST, Fazio RC, Fang L, Principles and Practice of Oral Medicine, 2nd ed. WB Saunders Co, Philadelphia, 1995.

Neidle EA, Yagiela JA, Pharmacology and Therapeutics for Dentistry, 3rd ed. CV Mosby, St Louis, 1989.

Journals

Journal of the American Dental Association

Journal of the California Dental Association

Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics

Compendium of Education

Monographs

American Academy of Oral Medicine. Contact Dr. Abraham Reiner, executive coordinator, 159 W. 53rd St., New York, NY 10019-6050

- Clinician's Guide to Treatment of HIV-Infected Patients
- Clinician's Guide to Treatment of Medically Compromised Dental Patients

ADA Oral Health Care Booklets, write Catalog Sales, P.O. Box 776, St. Charles, IL 60174

- Cancer Chemotherapy
- Cardiovascular Disease
- Chemically Dependent Patients
- Diabetic Patients
- End Stage Renal Disease
- Head and Neck Cancer
- Hepatic Disease
- Physical and Medical Disorders
- Women's Oral Health Issues

Table 3. Internet Resources

Bulletin Board of Oral Pathology — http://www.sdm.buffalo.edu/bbop/

American Academy of Oral Medicine — http://www.oralmedicine.com

American Academy of Oral and Maxillofacial Pathology http://www.aaomp.org (under development)

Medline search — http://igm.nlm.nih.gov/ — once open, click on "Medline"

CDA JOURNAL, VOL 28, Nº7

The Price of Success

Robert E. Horseman, DDS

How are you? No, really, how do you feel? 130/70, or near? Dentistry is not exactly chicken soup for the soul after all; there's a lot of stress. The mere fact that dentists are wholesale dispensers of stress doesn't mean that we are immune to it ourselves.

The Los Angeles Times, in a recent article, reminds us once again that "various surveys have indicated that the rates of suicide, drug and alcohol addiction and job burnout are higher [with dentists] than those of comparable professions."

Is this a good idea, pointing out to dentists that theirs is an essential profession, trusted perhaps, but on a popularity scale of 1 to 10, rated a weak 3 by the public, just ahead of telemarketers? Is it prudent to remind us that self-destructionwise we are right up there with lemmings and mosh pit devotees? Is a happy dentist simply an unaware dentist?

"Dentists," says the Times, "are sensitive, hard-working and skilled at their craft." When one of us sensitive types comes across these alarming statistics, we become introspective, wondering what price success?

Self: As a dentist, I should be profoundly unhappy. But I'm only moderately morose. Sometimes, like Wednesday afternoon, I'm verging on being marginally content. I need to look into this a little more; I may be missing something.

Impact from this kind of news is so enervating to many of us that the American Dental Association has established programs to address professional and personal burnout. At one such program might be encountered motivational guru Tony Robbins, whose persona has been likened to that of a Cape buffalo on crystal meth. Apparently, his technique of manic enthusiasm is captivating enough to enthrone him on television, a sort of Richard Simmons for the psyche minus the frizzy hair and shorts. At any given moment, day or night, his infomercials are being beamed to the faithful, lusting to exchange their lives of quiet desperation for that of a Power Ball winner.

David K. Okano of Rock Springs, Wyo., who chairs the ADA's dentist well-being advisory committee, claims the reason for dentists' discomfiture is that "compared to physicians, we are much more isolated." You would recognize this truth if you've cooled your heels in an over-crowded ER or an HMO waiting room recently where the activity can sometimes resemble the rush to get on the last helicopter out of Saigon.

The fact that dentists even have a well-being advisory committee is sobering if you were not aware that we needed one. Immured as we are in our operatories,

even lighthouse keepers, monks in isolated monasteries, and firewatchers atop remote towers in national forests seem to enjoy a more flourishing human contact in contrast.

It should be comforting then to know that here and there about the nation are centers devoted to the duality of dentistry. Seattle, Las Vegas, St. Petersburg and Key Biscayne are becoming magnets for a certain breed of dentist. Practitioners pursuing the chimera of perfection can attend one of these institutions to learn from experts how to create the most exquisite dental work possible. Embedded in the technical information is the inclusion of a philosophy that teaches them to regain a balance between work and spirituality. While polishing up their digital skills, their psyche's equilibrium may be getting a makeover as well.

It is probably safe to estimate that of the 200,000 dentists in the nation, maybe only about 15 percent have had the drive, the ambition and the willingness to strive for perfection. So what is the secret here? Is it genes, luck, upbringing? Why is it one kid matures to be a run-of-the-mill, middle-of-the-pack dentist such as -- no offense -- you and another grows up to become a Fauchard or a G.V. Black?

Did Monsieur and Madame Fauchard impart to young Pierre some key to success that put him on the path to becoming No. 1? Or is it something any of us could achieve if we were willing to work hard, keep a positive attitude and perhaps, above all, an extremely positive balance in our checkbooks?

Because the secrets don't come cheap. At one Mecca for the pursuit of excellence, they are divulged in a series of seven "Continuums." Should he or she undertake the whole series, the dentist's coffers, to be on a par with his or her ambitions, must total at least $21,150 to avoid overdraft charges. Eight other courses ranging from "High Impact Management" to "Predictable Complete Dentures" can add another $19,200 to the pursuit. If insolvency is one of the prime reasons for the dentist's discontent, this E-ticket outlay is not going to result in more restful nights.

The renown L.D. Pankey Institute for Advanced Dental Education in Florida was one of the first to recognize that there may be as many holes in the dentist's soul as in his patients' teeth. When the Pankey Institute opened its doors to postgrad students in 1972, it had a single goal in mind: to promote excellence in dentistry. It was further refined to bridge the gap between clinical and managerial skills with the belief that becoming adept in these two areas would redefine "success" in the average dentist's mind.

The "average dentist," if there is such an animal, comes out of school with a limited awareness of the business of dentistry. It's like learning to drive a VW and then being given the keys to a 18-wheeler. The fledgling dentist can exit his cocoon to confront the waiting world many thousands of dollars in debt. This may still be unresolved a decade later. If they equate success with solvency, small wonder that many dentists, mending broken teeth and dentures, find themselves in need of repair.

Somehow it all works out and is worth the candle according to participants. The average five-day course at the Pankey Institute costs $3,200, plus lodging in a nearby condominium. Graduate George Warga, 40, of Winnetka, Ill., says, "I've been here 10 times in the last three years. The visits have kept me in practice." In spite of the fact that each week away from the office costs him an estimated $18,000, Warga says the price is right.

"At age 28, I was going to leave dentistry, but now I'm enjoying myself. I will practice as long as I can." He didn't mention the Power Ball.