CDA

Surgical Treatments
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Oral Devices

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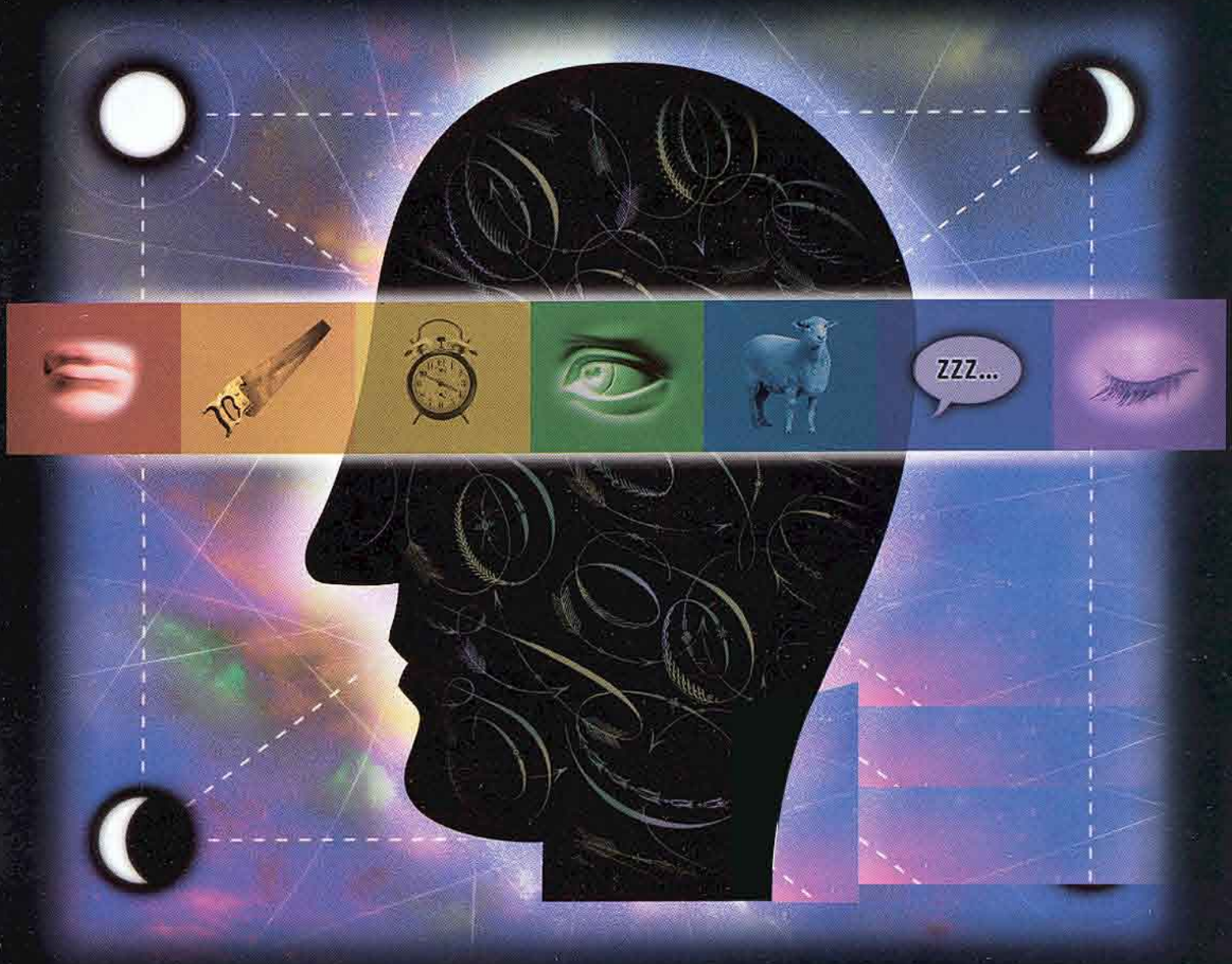
SNORING & SLEEP APNEA



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DEPARTMENTS

FEATURES

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Difficult Demands on Leadership

Jack F. Conley, DDS

We commented recently, with some alarm, that fewer individuals were in the volunteer pool for consideration for leadership positions than might be hoped for, or expected, given an organization the size of California Dental Association. A recent event motivates us to explore the matter of leadership further this month.

The overall demands placed on the dental volunteer leader of today may to some degree explain the reluctance of some individuals to serve, above and beyond the actual demands of the time commitment. As was suggested in this space in June, we receive correspondence from time to time from members who are very straightforward in stating the opinion that they will not serve because they view volunteer service as part of a traditional network that they criticize for not effectively resolving the problems they see facing the dental practitioner of today. The implication is that their time is more profitably spent taking care of the needs of their own business. We will not argue with their priority – one's business is the top priority.

However, the need to maintain a vital network to cope with the needs of the profession also requires a high level of concern and support if we are to have any hope of retaining a profession that is positioned to provide the standard of dental care that we continue to hear most dentists say they stand for and want to provide to the public. ***As we see it, the dental profession has entered a very complex era, one in which the many constituencies within the profession which organized dentistry must try to represent, are asking leadership for representation on a wide ranging variety of issues that are not embraced uniformly throughout the profession.*** It appears that it is becoming increasingly difficult to achieve consensus on issues or agree on desired services because of the increasing influence of divergent values of constituencies within the profession. Age, ethnicity, and sex are just a few of the demographic factors that make it much more difficult for well-meaning volunteers to take positions on issues or make decisions that will receive universal acceptance or approval by those whom they represent.

The debate over the value or importance of the proposed ADA public awareness campaign provides an excellent example of the dilemma we feel that dentistry has been facing for a few years now. It is a dilemma whose most pronounced effect is on the officers, delegates, or committee members serving in local, state, or national positions of responsibility. For some time now, dentistry has attempted various forms of marketing, or Ainstitutional advertising" at the state or national level on behalf of the membership. It was very clear to the 1996 and 1997 ADA Houses of Delegates, that many members of organized dentistry wanted the American Dental Association to place a priority on this activity as a membership

service. It also was clear that this activity is most strongly supported by younger members. Older members with successful practices are not personally interested in the benefits that might be achieved with a successful program, and they do not tend to express strong support for such a program being a major objective for organized dentistry. Further complicating this picture is the reality that many of the younger members who are most supportive of such a program have yet to reach a financial level in practice that makes them totally comfortable with absorbing the significant increase in dues necessary to mount such a campaign.

The dilemma for concerned leaders is that if they support a program they believe is desired by a significant segment of the profession, they also know that they could be forcing an unwanted program or undesirable decision on another major constituency within the profession. Not only is it increasingly difficult to satisfy everyone, the very nature of some of the issues can very easily alienate everyone, as is potentially the case in this current example. Without questions, there have always been divergent positions and opinions in dentistry's political arena. The difference between the past and the present seems to be the differences in the perceived values of membership of the various constituencies within the membership today. These differences make it difficult for leadership to make decisions that will address the values of a majority without disenfranchising what might be another group of significant size that could even leave membership if their values are not satisfactorily addressed.

Getting back to the central point, it seems that given the demands of their own business, most professionals would prefer to avoid expending their effort on issues in which there may be no win-win position. Nationally recognized lecturer Mark Levin told a large audience of CDA component society and state leaders at a recent CDA Leadership Conference that the primary purpose of an association Ais to try to change the environment" on behalf of the membership. That is the primary objective of a volunteer association. Levin told the leaders that AIf you can't do that ... do everything you can to help your members succeed in the Environment that Exists!" That is an extremely difficult task for a volunteer association today.

We return to our hypothesis that some potential leaders may be rejecting this challenge because of the dilemma that has been described. In a fast-paced world, where time is our most valuable commodity, a growing percentage of members would prefer to avoid the aggravation of spending significant time with difficult issues. Unfortunately, such a defeatist attitude must be put aside if dentistry is to survive the challenges that will continue to arise. More dentists must adopt the attitude that the business of their volunteer associations makes up an important part of their personal business. Dentistry as we know it today cannot survive without the organized efforts of our associations.

A large group of dedicated volunteers at the CDA Leadership Conference demonstrated through their participation that they were willing to take up the challenge to improve their responsiveness to the needs and values of the members they represent at the state and local component levels of their profession. It is important that the membership supports leadership efforts and consider increasing those ranks if dentistry is to remain a unified and effective force in determining its own destiny.

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Losing Control — The Deprofressionalization of Dentistry

Jay M. Hislop, DDS

AUTHOR
Jay M. Hislop, DDS, is currently President of the San Joaquin Dental Society. He is a management consultant specializing in critical issues analysis, strategic assessment, planning and goal-setting, and practice systems and operations.

We should be concerned. Jack Conley expressed his dismay over the closing of Northwestern University's dental school in the March 1998 *CDA Journal*. Dr. Conley focused on the failure of the profession to educate university administrators and boards about the importance of dentistry to the American population, and suggests that we be more positive and "aggressive" in ensuring that dentistry continues to receive support as a university educational discipline, and "is worthy of its status as a profession." We need to take these words to heart.

Northwestern based its decision on several factors, but the most chilling from the dental profession's viewpoint is the Northwestern administration's perception that dentistry does not fit into the mission of their medicine and life sciences programs. Dentistry is no longer considered a brainpower pursuit, worthy of elite graduate studies. In the eyes of at least seven university administrations, dentistry is a technical field, not a scholarly pursuit central to the mission of university graduate studies. To them, a DDS is something less worthy than an MD, a PhD, a JD, or even an MBA degree!

On the cover of *U.S. News & World Report* that same month, America's Best Graduate Schools" was the feature title for the cover story. The subtitle was ABusiness, Law, Medicine, Health, Education, Engineering, Public Affairs – 1998 Annual Guide ... Exclusive Rankings." It's their best-selling issue, widely read by parents, teachers, and counselors.Dentistry was not even mentioned in the article. (128:1494-7, 1997) It really should not come as a surprise to us that dentistry has slipped from the rankings of professional prestige and public respect as well. Our status as a profession is compromised each time a dentist places an ad in the yellow pages that panders to patient fears, sells trivial services, or makes inflated claims about how warm and wonderful the dentist is. We are compromised as a profession when a dentist sends a cheap advertising mailer or tabloid with his or her photo along with a coupon for free services or a discount, or rents a billboard for a few months. Worse yet is the indignity of opening the phone book and finding a ADental Referral Service" that claims to provide Aprescreened" doctors, implying that the rest of us are unworthy in some critical way. Or the referral service is a bogus front for one of our colleague's

private practices or clinics, and the state Board of Dental Examiners hasn't the time to investigate the allegations.

We suffer a loss of professionalism when our colleagues run their businesses so poorly that they cannot afford to provide a quality service and so take shortcuts with time, procedures, and supplies.

When a colleague fails to make a careful diagnosis and treatment plan, jumps into treatment without a clear financial arrangement with the patient, and then sends the patient's account to a collection agency, we all suffer a loss of credibility. The colleague who makes overt claims of personal superiority to his or her patients or makes derogatory comments about another dentist, about the patient's existing dental work, or about dentists in general, causes all of us to lose the confidence of that patient. When a large clinic exploits new graduates and produces factory dentistry in an assembly-line process with captive patients who feel forced to go there by the circumstances of their employer's decisions, we are demeaned as a profession. The patients know that the dentist in that clinic has the same license as every other dentist and expect their skills and treatment will be the same. The dentists who work there are not villains. They are often the same kids who just seven or eight years prior were star athletes and scholars at their local high schools. They now have a financial burden that those of us in the profession for more than a decade or so cannot even begin to comprehend.

When, although in the enlightened spirit of encouraging and embracing professional diversity, we sit back and allow the state Board of Dental Examiners, the largest dental licensing agency in the nation, to confer the title ADDS" to licensees who never earned the degree, we lower our professional standards. When the board makes it too easy to obtain a dental license and floods the professional market with practitioners of limited skills and experience, it sends ambiguous messages to the public. The DDS degree has been declared irrelevant by the State of California (AB 1116 passed with the support of the CDA). It is a wonder any university wants to continue granting the degree. There are now, or soon will be, many practitioners using a DDS after their names, who completed what to our sensibilities is nothing more than a technician course in a foreign country. Certainly they weren't ADA-accredited programs. Maybe all those who actually earned their DDS should be able to convert it to a DDS AE (Actually Earned), or a DMD. (What would happen if we all tried to convert our degrees to an MD?) This action by the Assembly is another nail in the coffin of deprofessionalizing the practice of dentistry. Soon, the right to practice dentistry could be entirely an administrative action by the state board, with no reference to training standards developed by the profession itself. The ADA accreditation program would have no teeth. The DDS degree, at least in California as of January 1998, is no longer an honest reference to an earned degree conferred by a faculty of an institute of higher education after diligent effort and scholarly pursuit. It is really a ARDS" – Registered Dental Surgeon. Is the next perilous step to allow hygienists to challenge the state board exam without a dental school degree?

With this the state of affairs in California as of last fall, why would any dean or provost want to continue with a dental program? Perhaps Northwestern University believes the programs will move entirely into the state university systems, and eventually to the community colleges. Conversely, why haven't the deans of the California dental schools put forward a legal challenge to AB 1116? Don't they care that their degree has lost its meaning to the public? Obviously CDA won't take up that torch, since it supported AB1116 in the name of Adiversity."

When we fail to demand and support fundamental research, yet accept and utilize untested products from manufacturers on our patients, we are not behaving as professionals. When our diagnosis is based upon a cursory examination with weak fact-finding and weaker documentation, and our treatment planning is not evidence-based therapy, our patients are not served in a scholarly, professional, or ethical manner. When the products or therapies fail, and the doctor blames the patient instead of managing the case, replacing the restoration, or substituting a more predictable therapy, we all lose credibility. We cannot really answer even the most basic questions about the longevity of our various types of restorative materials with evidence-based conclusions.

When Northwestern looked at the recent contributions from its own dental research, it apparently came up with nothing that demonstrated support of Aexcellence" in its basic health sciences mission.

ADA has provided for its membership a preliminary promotion and survey of the proposed public awareness campaign. The House of Delegates to the ADA will vote on this in October. It is a form of institutional advertising designed to elevate the image of the profession by public awareness marketing techniques. It is important to take a hard look at it and express your opinion to your delegates. Is it the solution to a deteriorating public image? Probably not, because it has no discernible focus. It may help, and at very little cost per member on an annual basis. But it has no identified target audience, and it does not address the problem. Our problem is not a failure to market the profession. We have a problem of what defines the profession.

Is public awareness advertising enough? I doubt it. Much more needs to be done. In the video that promotes the campaign, there is a dentist who says that we can do nothing about our image problem as individuals and must band together and tell our message in the media. But we must also Awalk the walk" if we are going to Atalk the talk." If each individual dentist does not take a hard look at his or her personal professional behaviors, and measure his or her own professionalism against a respected mentor or role model, then the campaign is wasted

money. If we continue to tolerate the transgressions against professionalism that have become uncomfortably common of late, our campaign could even become the subject of public and media mockery. Dentists already dislike the use of dental vignettes in humor, and we despise the seemingly unjustified criticisms of investigative journalism. I for one would loathe seeing the profession become the target of even more serious media criticism.

The message is simple. Behave as a profession and the world can see us no other way. Behave as a professional and the world can see you no other way. Each of us can evaluate and improve our business practices, our marketing practices, the training and education of our staff, our personal appearance and that of our office staff and environment, our financial practices, our diagnostic and treatment presentation methods, our professional demeanor with patients and in the community, our communication skills, our commitment to education and research, our active liaison with our alma mater or local dental institutions, and our commitment to personal and professional growth.

There is nothing we can do as an organization to change the behaviors of others. We cannot institutionalize professionalism and ethics any longer. The Federal Trade Commission won't let us because professional rules of conduct always seem to border on how the FTC defines trade-related issues. We can only overwhelm the negatives by greater insistence on uncompromised professionalism as individuals and among our friends in dentistry. And nothing stops you from picking up the phone and expressing your personal displeasure when you or a family member receive some cheesy bulk mail solicitation from a colleague, or a patient brings one in for you to see. If a doctor gets seven or eight spontaneous calls from peers for actions that demean us collectively, he or she may get the point.

Let's raise the bar and reach for a higher standard of individual professionalism. Then let's tell the public about dentistry.

Editorial Note:

AB 1116 (Keeley) had several components to it, only one of which was the provision allowing graduates of non-accredited dental schools who are currently licensed in California to use the DDS degree. The most important provision from the association's perspective was the elimination by the year 2003 of the restorative technique (bench test) mechanism through which graduates of non-accredited schools have qualified to take the California licensure examination. CDA's support of AB 1116 centered around the objective to require standardized educational achievements from all licentiates in California.

With the passage of AB 1116, graduates of non-accredited dental institutions will be required to either graduate from a U.S. accredited dental school program or a two-year program for international graduates offered by California dental schools. Beginning in 2003, all applicants for licensure in California must have participated in an accredited dental school educational process, either in another state or in California, and they will be required to hold a DDS or DMD degree, which is awarded upon completion of the educational program.

New Front May Open In War Against Caries

Development of an antibody that one day might be used to stop dental caries from forming is being viewed as enticing news.

Full-scale clinical trials to determine the antibody's safety and efficacy will begin soon, casting light on potentially far-reaching implications for the dental profession and for people susceptible to caries formation.

A team of researchers at Guy's Hospital Dental School in London conducted a preliminary clinical trial using the genetically engineered antibody produced by Planet Biotechnology, Inc., a research firm in Mountain View, Calif. Led by Dr. Julian Ma and professor Tom Lehner, the team completed the study in April using a secretory IGA (SIgA) monoclonal antibody named CaroRXJ, produced in genetically altered plants called "plantibodies" designed to prevent infection by the oral bacteria Streptococcus mutans.

The English researchers chose volunteers whose teeth harbored S. mutans. They first decreased the subjects' oral bacterial count, then applied a series of clear and tasteless antibody treatments. According to the researchers, "the bacteria were not able to recolonize the mouth for up to four months following treatment," an effect known as passive immunization.

The preliminary scientific trials were reported in the May issue of Nature Medicine. The trials are being supported in part through Small Business Innovative Research Grants from the National Institute of Dental Research, totaling about $572,000.

And we succeeded in making a secretory IGA antibody by taking genes that encode the antibody and genetically engineering it into a plant," said Elliot L. Fineman, chief executive officer of Planet Biotechnology. "When the plant grows, it produces the antibody, and this is extracted, purified, and is used in the clinical trials."

SIgAs are the antibodies naturally produced by the body to protect oral and other mucosal surfaces against infectious organisms and toxins. The plantibody works by blocking the adherence of the S. mutans bacteria to the teeth.

"Basically, it's just like the principle of a good lawn of grass," said Jim Larrick, MD, PhD, head of research at the Palo Alto Institute of Molecular Medicine, and founder of Planet Biotechnology. "A nice lawn of healthy, growing grass makes it hard for crabgrass to grow. So we reduce all the bacteria in the mouth to very low levels using chlorhexidine mouth wash, and then treat with the antibody. The normal bacterial flora grow back, but the >crabgrass' Streptococcus mutans is unable to recolonize. The antibody blocks the niche where this grows."

The company has received the go-ahead from the Food and Drug Administration to begin full-scale 1/2 (safety/efficacy) clinical trials, which will involve up to 1,000 subjects and are set to begin soon at the University of California, San Francisco, School of Dentistry.

"We're going to try to show both safety and efficacy," Fineman said. "In the work up to now there have been no safety issues and no adverse response in any patients being treated with the antibody."

Fineman said the length of the trial will depend on the accrual rate for patients. He hopes to complete the first phase within 18 months of initiation. If all goes well, the FDA registration trial will begin in a year or so.

If the trials are successful and FDA approves, dentists can expect to see a commercially available product in as little as four years.

We think this product could become part of routine office visits," Fineman said. "Anyone who is of prime cariogenic years, or patients who show a tendency toward caries, would benefit. Patients who have been treated, for example, for head and neck cancer who are at much greater risk for caries would also benefit."

The product would be applied only by a dentist during an office visit. We plan to market our product through dentists, who would apply it to their patients once or twice a year," Larrick said. "The purpose of dentistry is to improve oral health, so this is an ethical product that may have a role in alleviating a lot of pain and suffering."

With the full-scale trials yet to begin, a senior official of the American Dental Association said care must be taken to ensure data from the trials is carefully evaluated before the antibody is declared a success.

"But this work looks very promising, and we're guardedly optimistic about it," said Daniel Meyer, DDS, ADA's associate executive director for its Division of Science. "To us, this is the way the profession should go. Rather than to treat the disease's ravages, it's much better to prevent it from occurring."

While the antibody could eradicate a major cause of caries, it isn't a panacea. "There are other types of bacteria that cause other kinds of decay, such as root caries," said Michael J. Danford, DDS, chair of CDA's Council on Dental Research and Developments. "So this could be a good first step toward helping to eliminate decay."

Bonding With Investments

Municipal bonds are one of the few remaining ways to receive income exempt from regular federal and, in many cases, state income taxes. That is why they are attractive to many investors, especially those in higher tax brackets.

One way for individuals to participate in a portfolio of municipal bonds is through a unit investment trust (UIT). UITs are fixed portfolios of stocks or bonds that are professionally selected, with stated investment objectives. For investors who want an investment that has the potential to reduce taxes and increase spendable income, municipal UITs offer a number of convenient ways to invest in a diversified portfolio of municipal bonds.

Tax-exempt municipal trusts may contain fixed portfolios of quality municipal bonds – obligations issued by states, cities, counties and other political subdivisions. Those trusts offer tax-exempt interest income and may provide taxable equivalent yields that increase with the owner's tax bracket. National portfolios provide month-

ly income exempt from regular federal income taxes. Single-state trusts may allow double or even triple income tax exemption for investors residing in their respective states – an advantage for investors in high-tax states. Insured series trusts appeal to conservative investors seeking a high degree of safety plus tax-exempt income. Portions of the interest income from these types of trusts may be subject to the federal alternative minimum tax.

National Municipal Trusts – National municipal bond UITs hold bonds issued by states and municipalities to finance schools, highways, hospitals, airports, bridges and other public projects. In most cases, income earned on those units is not subject to regular federal income taxes (although all or a portion may be taxed under state and local laws), making them attractive to taxpayers in the higher tax brackets.

State Municipal Bond Trusts – State municipal bond UITs work just like national municipal bond UITs, except that their portfolios contain the issues of only one state. Generally, a resident of that state has the advantage of receiving income free from regular federal, state and local income taxes.

Insured Series ITs – Insured municipal bond trusts contain bonds that are generally rated AAA by Standard & Poor's and offer extra protection through bond insurance. Timely payment of interest and principal on the underlying securities is guaranteed by independent insurance companies. The market value of units of insured trusts is not guaranteed and will fluctuate with changes in market conditions. Unit trusts are sold by prospectus, and terms of the insurance are described in the prospectus.

Municipal trusts have a variety of features. They include:

- Tax-exempt monthly income. While bonds generally pay interest semi-annually, bond UITs provide regular monthly checks. This feature appeals to retired investors and other individuals who prefer a monthly income flow.
- Defined portfolios. Investors in a municipal unit trust are buying a preselected portfolio of bonds. The portfolio is defined so that the securities, maturities, call dates and ratings are all known before you invest.
- Professional selection. Each trusts's portfolio is selected by a team of professional municipal bond analysts and buyers who evaluate a number of factors, including the type of bond and purpose, call features, economic factors, financial position, debt structure, management/governance and various external factors.
- Portfolio supervision. Although the municipal unit investment trust is not managed, analysts generally review and supervise the securities in the portfolio regularly.
- Diversification. Municipal bond portfolios are diversified by issue and bond type to help reduce overall investment risk. To achieve comparable diversification on one's own would require considerable investment capital.
- Low minimum investment. There is a low minimum investment, often as little as $1,000.
- Automatic reinvestment. Investors in municipal trusts may elect to have distributions reinvested at no additional sales charge.
- Daily liquidity. Units may be redeemed at any time. The price received is based on the then-current net asset value of the securities in the portfolio, including a deduction for any remaining deferred sales charge. That is determined by an independent evaluator, based upon the bid price, with no odd-lot penalties. However, depending upon market conditions, the price you receive may be more or less than originally paid.

UITs are available in a variety of maturities to help with various investment objectives, such as to increase current income, reduce taxable income, prepare for retirement, or accumulate a college education fund. Like other investments, unit trusts are subject to market fluctuations and are also sensitive to interest rate changes.

Mr. Gregoriou is an associate vice president for investments for Dean Witter Reynolds Inc. He can be reached at (800) 755-8041. This article does not constitute legal or tax advice. Consult a tax adviser and/or attorney for more information before making tax/legal-related investment decisions.

BC Pill Can Boost Incidence of Dry Socket

Women who use birth control pills are twice as likely to develop dry socket after a tooth extraction, according to a study published recently in the Journal of the Academy of General Dentistry.

"In general, women have a greater chance than men to develop dry socket," says Vicki Grandinetti, DDS, a Chicago general dentist. "But for women who take oral contraceptives, their likelihood of developing a dry socket is twice as likely because of their increased estrogen levels."

The study reports that women who use oral contraceptives experienced a 31 percent incidence of dry socket after molar extractions performed in the first 22 days after their menstrual cycle. Within two to three days after estrogen use was discontinued, the extraction site tissue began the healing process. For women whose molar extractions were performed on days 23 through 28 of their cycle, no incidence of dry socket occurred.

"Unless there is an emergency, women using birth control should try to schedule their extractions during the last week of their cycle, when estrogen levels are inactive," Grandinetti says. "The healing process can then begin immediately."

Pain Control Needn't Be a Shock

Although most children can cope with local anesthetic injections, some children fear needles, and giving them injections presents a challenge to the dentist. For other children, the paresthesia that may linger for hours after the completion of the dental procedure may be more objectionable than the injection itself.

Researchers at the University of Otago, New Zealand, note that interest has been renewed in the past decade in the applications of electronic pain control in dentistry.

The researchers compared the effectiveness of electronic dental anesthesia (EDA) for pain control during restorative procedures with local anesthetic injection (LA) in 32 children, ages 6 to 12 years. Each child had two antimere primary or permanent molars requiring similar-sized Class I or Class II restorations. Pain levels during restorative treatment were assessed using a visual analogue scale. Behavior and heart rates were also recorded.

The authors point out that the use of transcutaneous electrical nerve stimulation (TENS) to help control chronic pain was introduced in 1967. In the mid-1980s, TENS units were modified for intraoral use. Recently introduced is an EDA device that uses extraoral electrodes, eliminating the inconveniences of intraoral electrodes, such as difficulty in application, obstructed field of operation, and easy detachment.

In the New Zealand study, the antimere teeth were restored in two separate visits with random selection of use of EDA or LA. For the control visits, anesthesia was given by infiltration for the maxillary teeth and inferior nerve block for the mandibular teeth. Cavity preparation began after five minutes. Injections were repeated if the anesthesia was not effective.

After both visits, the children were asked their preferred method of anesthesia and the reasons. Twenty said they preferred EDA to LA. Eleven preferred EDA because there was no need for injection; three liked to control the anesthesia; four liked the feeling with EDA; and one preferred EDA because there was no paresthesia after treatment. Twelve children preferred LA because they found LA more effective for pain control.

The authors report that in their study, six treatment procedures using EDA were interrupted because of insufficient pain control, and treatment was completed using local anesthesia. Four of those children showed high pretreatment anxiety. Two children reported "worst pain" for the cavity preparations even after LA was administered, and one reported "worst pain" for cavity preparation of the antimere molar when LA was used.

Researchers conclude that EDA was less effective than LA in controlling pain during cavity preparation in children age 6 to 12. According to the authors, "This study suggests that the effectiveness of EDA is related to children's dental anxiety, the depth of the restoration, operator attitudes, and whether the teeth are permanent or primary. EDA can be a useful adjunct to providing pain control during restorative dental care in children."

Great Expectorations: Saliva-Testing Is Here

Health care professionals soon may shun drawing a patient's blood and instead ask for a sample of spit.

Saliva samples not only can detect the onset of periodontal disease and cavities, but also can reveal the presence of AIDS, Alzheimer's, cystic fibrosis, diabetes, hepatitis, ulcers and depression.

Saliva-testing is being hailed as a more convenient method of monitoring the levels of prescription medications, hormones, tobacco, alcohol, steroids, marijuana, cocaine and opiates.

Roughly 99 percent of saliva is water, the remaining one percent is proteins. Also contained in the mixture are varying amounts of serum products and blood cells, bacteria and bacterial products, and bronchial secretions. The electrolytes in the water portion of saliva are believed to cleanse, buffer, and aid in remineralization and alimentary functions.

Saliva's role in lubricating oral tissues and helping food digestion is well-documented. However, current research indicates it may reveal clues about other developments in the body. As a result, dentists and physicians have shown new interest in saliva. By some estimates, half the country's dental schools now incorporate saliva detection into their curricula.

Saliva testing offers many advantages over blood and urine tests: It's easier to collect and store than other body fluids, and saliva collection techniques are far less invasive than collection of blood or urine.

Saliva testing products and kits for home or office have been used in Scandinavian countries for several years, but await approval from the U.S. Food and Drug Administration.

Early Intervention and Teamwork Breaks Braces' Grip

Since jaw development slows at about age 13, general practitioners who build good relationships with orthodontists can shorten the time of wearing braces and improve children's chances of maintaining sound dental health, reports the Academy of General Dentistry.

Early intervention lets the GP deal with a variety of problems that could cause trouble later. The impact on a child's teeth due to correctable problems such as thumb sucking and tongue thrusting can be reduced or even removed. Dealing with cross bites in the age range of 7 to 11 years can help prevent earaches or headaches later. And early treatment can reduce patients' costs, increase the chance that the GP can perform needed work, and improve the patient's self-image at a younger age.

Children should have an orthodontic screening by age 7, urges the American Association of Orthodontics (AAO). Failure to bring orthodontic care into the picture at an early age could result in a greater need for extraction, a step many practitioners hope to avoid. For maximum results, the AAO encourages clear communication between orthodontists and referring dentists.

AAHD Elects 1998-99 Officers

The American Association of Hospital Dentists (AAHD) has elected Ronald Mito, DDS, of Los Angeles as president for 1998-99. Mito, an active CDA volunteer, is currently associate dean for Clinical Dental Services at UCLA School of Dentistry. Mito's interests include treatment of medically compromised and phobic patients and postdoctoral general dentistry education. Dr. Paul Glassman of the University of the Pacific School of Dentistry was elected first vice president.

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Snoring and Obstructive Sleep Apnea

James E. Eckhart, DDS

It has been a pleasure to participate as contributing editor to this edition of the *Journal of the California Dental Association*, which focuses on dentistry's role in sleep-disordered breathing. The intention was to provide an educational service to compensate for the general absence of this information in dental school curricula.

Rob Veis, DDS, has provided a dental overview of sleep-disordered breathing, introducing key definitions, giving reasons dentists would want to be involved, outlining a diagnostic protocol, summarizing treatment options, discussing decision-making factors, sketching procedural basics, and suggesting how to obtain more information.

Jerald H. Simmons, MD, has written about sleep physiology and sleep-disordered breathing, expanding on the diagnostic protocols available and how a dentist might participate intelligently.

Lawrence W. Kneisley, MD, has described medical (nondental) treatments for snoring and obstructive sleep apnea.

Michael W. Marshall, DDS, provides information about various surgical options available for snoring and obstructive sleep apnea.

Because there are dentists who frequently and confidently provide low-cost oral appliances for snoring without apparent consideration of the medical significances of snoring, two nationally prominent dental experts were invited to discuss opposite sides of the topic of whether a dentist should treat snoring patients independently of the sleep medicine profession. The reader is encouraged to read both articles and decide the answer for himself or herself.

Lawrence I. Barsh, DMD, is past president of the Sleep Disorders Dental Society and has presented herein some good reasons for cooperation with the sleep medicine community.

W. Keith Thornton, DDS, the current president of the Sleep Disorders Dental Society, has provided logical reasons why dentists might provide sleep-disordered breathing treatment without supervision from sleep medicine, but the reader must read carefully the level of knowledge and competence in his model.

Finally, the contributing editor has presented a study of many commercially available oral appliances, suggesting a set of criteria for evaluating them, and presenting findings of their strengths, weaknesses, and peculiarities.

It is hoped that these articles will enlighten the dental community and increase the level of care of sleep-disordered breathing.

To request a printed copy of this article, please contact CDA Publications Department, (916) 443-3382, Ext. 4640.

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Snoring and Obstructive Sleep Apnea from a Dental Perspective

Rob W. Veis, DDS

ABSTRACT Proper diagnosis and treatment of sleep-related disorders are best handled via a team approach. This team may include a general dentist treating in conjunction with other sleep specialists. However, to provide care, dentists must have a basic understanding of sleep disorders. This paper provides a dental overview of sleep-related breathing disorders, including key definitions, an outline of a diagnostic protocol, a discussion of the factors involved in decision-making, and a summary of the wide variety of treatment modalities.

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Perhaps one in every 10 adults snores. Although snoring has no serious medical consequences for most people, for an estimated one in 100 snorers, habitual snoring is the first indication of a potentially life-threatening sleep disorder called obstructive sleep apnea.[1]

Most physicians have not been trained to deal with sleep-related disorders. It has been estimated that, on the average, only two hours are spent during the four years of medical school teaching medical students about sleep.[2] This has created a significant deficit in our current medical system. Dentists can help correct this problem by participating in the recognition and treatment of sleep disorders.

By adding a few questions relating to sleep to a standard dental screening form, a dentist can recognize a potential sleep disorder. This alone could help guide millions of Americans who suffer from a sleep disorder into a systematic treatment pathway. However, to provide care, dentists must have a basic understanding of the complex process of sleep and sleep disorders.[1]

Sleep Nomenclature

Sleep Architecture

In a normal rest period, the average person will repeatedly cycle through different stages of sleep to create what is called a "normal sleep architecture." The two main divisions of a normal cycle are non-rapid eye movement (non-REM) and rapid eye movement (REM).[3]

Non-REM consists of four stages. The first stage is the transitional stage. This is the time when one is falling asleep, and it usually represents only 5 percent of sleep time. The second stage is the light sleep stage. This stage accounts for 50 percent of normal sleep time. The third and fourth stage of non-REM are the delta and slow-

wave stages. These stages represent about 20 percent of sleep time and are the stages in which one experiences a deep and relaxing state of rest.[3,4]

After the non-REM cycle is complete, one usually enters into REM sleep. Restful sleep continues during this cycle. It is also the period in which one dreams. This stage accounts for the final 25 percent of a normal sleep cycle.[3,4]

To experience a restful night sleep, one must spend sufficient time in deep sleep. A person suffering from sleep apnea cannot do this because they are constantly awakened throughout the night.[4]

Sleep Apnea

Stedman's Medical Dictionary defines apnea as the absence of breathing or the want of breath. When there is a cessation of airflow at the mouth and nose for more than 10 seconds, an apneic episode has occurred. During this time, the individual's oxygen level will drop. If a person experiences 30 or more apneic episodes during a seven-hour sleep period, that person is believed to be suffering from sleep apnea syndrome.[3,5] These episodes can last from 10 to 120 seconds. These apnea events terminate with a partial wakening or an arousal. It is important to understand that these arousals are necessary for the person to begin breathing again. Apnea severity is usually categorized by the frequency of apnea events that occur per hour. This number is called the apnea index.[3] The categories are as follows:

- Mild – Five to 20 episodes per hour;
- Moderate – 20 to 40 episodes per hour; and
- Severe – 40 or more episodes per hour.

It is not unusual for a patient with severe apnea to have as many as 300 apneic episodes per night.

Sometimes there is a decrease in airflow but not a complete blockage. When this happens for 10 seconds or more and there is a decrease in oxygen level of at least 2 percent, an event called a hypopnea has occurred. Hypopneas also lead to an arousal from sleep.[6]

There are three basic classifications of sleep apnea: central, obstructive, and mixed.

- Central apnea. Airflow stops because inspiration efforts temporarily cease. Although the airway remains open, the chest wall muscles make no effort to create airflow. The etiology frequently is encephalitis, brainstem neoplasm, brainstem infarction, poliomyelitis, spinal cord injury, and cervical cordotomy.[3,7] This is a fairly uncommon form of apnea.
- Obstructive sleep apnea. This is the cessation of airflow due to a total airway collapse despite a persistent effort to breathe. An obstruction in the upper airway can occur in three areas.8 They are the nasopharyngeal, oropharyngeal, and hypopharyngeal regions. The nasopharynx is the part of the pharynx that lies above the level of the soft palate. The oropharynx is the division of the pharynx that lies between the soft palate and the upper edge of the epiglottis. The hypopharynx is the division of the pharynx that lies below the upper edge of the epiglottis and opens into the larynx and esophagus.
- Regardless of the level, an obstruction causes breathing to become labored and noisy.1 As pressure to breathe builds, muscles of the diaphragm and chest work harder. The effort is akin to sipping a drink through a floppy straw – the greater the effort, the more the walls collapse. Collapse of the airway walls eventually blocks breathing entirely. When breathing stops, a listener hears the snoring broken by a pause until the sleeper gasps for air and awakens, but so briefly and incompletely that he or she usually does not remember doing so in the morning.[4,7]
- Mixed apnea. This term is used when both central and obstructive episodes are observed during a sleep study. It is usually recorded as a central episode being immediately followed by an obstructive one.[3] When this is seen, the obstructive component is treated first. This usually eliminates the problem; but when it does not, re-evaluating the patient for a central component will have to be done.

Snoring

Many people think that snoring and apnea are the same thing. This is not true. Snoring, which is caused by vibration of the tissues due to air turbulence as the airway narrows, may be a sign that a patient is suffering from apnea. But not all snorers are apneics.

Snoring can be categorized by its severity. On one end of the spectrum is the benign snorer who snores but experiences no physical problems. On the other end lies the snorer who suffers from apnea, and in the middle is the snorer who suffers from upper airway resistance syndrome. In these people, though they may not actually experience apneic episodes, their snoring is so loud and their breathing so labored, that it still wakes them up numerous times throughout the night. This leaves them unrefreshed and tired throughout the day.[6]

Reasons to Treat and Who Should Be on the Team

Patients who suffer from sleep apnea typically suffer from a fragmented sleep pattern, experience excessive daytime sleepiness, and exhibit many other medical conditions. Some of the more common conditions associated with sleep apnea are hypertension; stroke; angina pectoris;9 initiation of a gastroesophageal reflex; frequent nocturnal voiding; hypoxemia; hypercapnia (high blood level of CO_2); susceptibility to atherosclerosis;10 and cardiac changes such as bradycardia, tachycardia, and right heart failure leading to sudden death.[11]

TABLE 1.
Signs and Symptoms of Sleep Apnea in Adults

Heavy snoring
Bed partner reports periods of non-breathing
Gasping or choking during the night
Excessive daytime sleepiness
Frequent arousals during sleep (fragmented sleep)[15]
Nonrefreshed sleep
Restless sleep
Kicking and leg movements
Morning headaches
Personality changes such as becoming irritable, or temperamental
Severe anxiety or depression[16]
Poor job performance
Loss of alertness
Clouded memory
Intellectual deterioration
Occupational accidents[12]
Impotence
Decreased sex drive
Bruxing[17]
Dry mouth upon awakening
Scratchy throat
Alcohol use especially before bedtime
Sedative hypnotics needed to sleep
Regular use of antihistamines
Excessive weight
Age of the patient (generally increases with age)[18]
Cardiac irregularities
High blood pressure[9]

There are also many social reasons to actively treat those who suffer from sleep breathing disorders. These range from husbands and wives who can no longer sleep in the same room, to professional truck drivers who are seeing an increase in accidents and citations for moving violations caused by excessive daytime sleepiness. On-the-job accident rates are also related to sleep apnea.

It has been estimated that the indirect costs of sleep disorders is more than $41 billion a year from lost productivity, $17 billion to $27 billion a year from motor vehicle accidents, $7 billion a year in work-related accidents, and $2 billion to $4 billion a year in home and public accidents.12 Clearly there is a major national problem that needs to be dealt with in an appropriate fashion.

Because sleep apnea can be associated with many other medical problems, and treatment options are so varied, proper diagnosis and treatment are best handled via a team approach. Members of this team may include a sleep specialist, otolaryngologist, internist, orthodontist, oral surgeon, and general dentist. A dentist should play an active role in screening his or her patients, treating them in conjunction with other sleep specialists, and providing them with follow-up therapy.

Diagnosis: Questionnaires, Exams, and Lab or Home Studies

Screening

Adults

To properly screen patients, one must evaluate them for the presence of any physiologic and behavioral predisposing factors indicating obstructive sleep apnea.13 Table 1 lists some of the signs and symptoms that are indicative of an adult who is suffering from sleep apnea. A simple questionnaire can be designed to encompass these points.[13]

Children

Children can also suffer from sleep apnea. Typically such children suffer from growth and development problems. Many of them have underdeveloped maxillas, narrow upper arches, and retruded mandibles. Often they are highly allergic, with their airway completely blocked off due to adenoid and/or tonsillar hypertrophy. If they are already having snoring and breathing problems now, do not ignore them. Table 2 lists some of the signs and symptoms of sleep apnea seen in children.

After having the patient fill out a questionnaire, the practitioner should complete a thorough examination to reveal any anatomical factors. This exam should include the following:

- Complete medical/dental histories;
- Soft tissue/intraoral assessment (this is to include the tongue and the throat);
- Periodontal evaluation;
- Orthopedic/TMJ/occlusal examination;
- Intraoral habit assessment;
- Examination of teeth and restorations;
- Initial dental radiographic survey (to include a baseline lateral cephalometric survey to study the soft palate size, hyoid position, skeletal relationship, and airway opening).
- Diagnostic models;[13] and
- Facial exam to evaluate neck size, weight, possible retrognathia.

While doing the soft tissue/intraoral assessment portion of an exam, the practitioner should evaluate all three regions of the upper airway.

An obstruction in the nasopharyngeal area is usually caused by turbinate hypertrophy, a deviated septum, or an abnormal growth such as a polyp. Although documenting a problem in this region is the job of an otolaryngologist, a dentist can at least check the patient to see if he or she has a patent nasal airway. The patient should hold a finger over one nostril at a time and breathe in.

When evaluating the oropharyngeal region, the practitioner should first check for hypertrophy in the tonsils. Then check the size and position of the tongue as it relates to the soft palate. Finally, look at the size and drape of the soft palate and the uvula. When the soft palate is excessively long, there is a good chance that the patient will suffer from an oropharyngeal blockage.[8]

An obstruction in a hypopharyngeal airway space is harder to detect through oral observation alone. When motor nerve activity stops during REM sleep, the tongue can drop back against the posterior pharyngeal wall and block the airway. Cephalometric films can give some information on whether an airway is likely to be blocked.[21]

A new component of the physical exam has been developed called the chin press/tongue curl maneuver. This maneuver is based on the changes that occur in

the position of a patient's mandible and temporomandibular joint during sleep. During sleep, the patient's mandible and TMJ usually go into the most retruded position. When this occurs, the tongue can drop back against the posterior pharyngeal wall and block the airway. The chin press attempts to replicate this sleep event by guiding the mandible and TMJ into the most retruded position while the patient is supine or reclined in the exam chair.[22,23] This maneuver is performed as follows:[22,23]

- Place the patient in a supine position and instruct him or her to maintain gentle occlusal contact while performing deep nasal respirations. In this occlusal relationship, the degree of respiratory effort during both inspiration and expiration is evaluated. This is considered the baseline respiration with which the chin press/tongue curl respirations are to be compared. If there is difficulty with respiration during this baseline portion of the test, the most likely cause is a nasal airway obstruction.
- With the patient's mandible relaxed, apply gentle pressure on the chin to retro-position the mandible and have the patient close. The goal is to place the TMJ into a centric relation position. The patient is then encouraged to relax his or her tongue and pharyngeal muscles and asked not to prevent any obstruction in the posterior pharyngeal region that may occur during this part of the procedure.
- During this manipulation, the patient is instructed once again to perform deep nasal inspirations and expirations. The responses are graded as follows: no obstruction = 0, partial obstruction = 1, and complete obstruction = 2. The greatest degree of obstruction during inspiration or expiration is used as the scoring response.
- Next, the tongue curl enhancing maneuver is performed. This consists of the same manipulation performed in the chin press with the following addition. The patient places the tip of his or her tongue to the most posterior part of the hard palate. This further retro-positions the mandible increasing the likelihood of obstruction during respiration. The patient again performs deep nasal inspirations and expirations. The responses are again graded in the same manner.

TABLE 2.
Signs and Symptoms of Sleep Apnea in Children.

Hyperactivity[19]
Poor concentration
Abnormal skeletal-facial developmental
Hyponasal quality to their voice
Noisy breathers
Obesity
Frequent upper airway infections
Earaches
Nocturnal mouth breathing[20]
Snoring
Restless sleep
Nightmares
Night terrors
Headaches
Chronic runny nose

Magnetic resonance imaging studies clearly show an airway obstruction occurs when the chin press/tongue curl maneuver is performed on some patients.[22,23] This maneuver has also been shown to correlate with the degree of severity on the nocturnal polysomnogram. It is important to recognize, however, that a negative chin press/tongue curl does not rule out a diagnosis of sleep apnea.[22,23]

Diagnosis of Sleep Apnea

Screening patients by asking them questions, evaluating their medical histories, and performing an intraoral examination should enable a practitioner to make a decision on whether the patient suffers from apnea. If a practitioner suspects that a patient may be experiencing apneic episodes, he or she should refer that patient to a physician immediately. Either a sleep specialist, otolaryngologist, or internist can make sure the patient receives a complete medical work-up and sleep test.

Even after a thorough evaluation by the dentist and the physician, a definitive diagnosis of obstructive sleep apnea can only be accomplished by a sleep test called a polysomnogram. During sleep, a polysomnogram measures ventilation, gas exchange, cardiac rhythm, and the number and length of apneic episodes; assesses oxygen saturation; determines sleep stages; and detects arousals. In the past, this test could only be done in a hospital sleep clinic. Today, there is mobile sleep technology that allows the patient to take this test in the comfort of his or her home.

Treatment

Protocols Possible

It has been said that the best method of treatment is prevention. The dentist can play a role in both the prevention and treatment of snoring and obstructive sleep apnea.

Dentofacial Orthopedics

Early detection of structural abnormalities in the developing child affords the opportunity to intervene with functional therapy, possibly preventing eventual obstructive sleep apnea development. For example, after a thorough orthopedic evaluation, the dentist may then decide to use orthopedic appliances to direct and control a child's growth. Arch development, mandibular repositioning, and controlling vertical dimension may create the intraoral volume needed to accommodate the tongue and prevent its compaction into the hypopharynx.[24]

Many treatment methods have been tried over the years to treat snoring and obstructive sleep apnea. To date, three approaches are most effective. They are

continuous positive airway pressure, surgical techniques, and the use of intraoral appliances. Regardless of the technique used, most patients benefit if a few general behavioral measures are followed.

General Behavioral Measures1

- Lose weight. People with severe sleep apnea are often overweight. Loss of weight will result in reduced adipose tissue volume in the upper airway, decrease the load on the chest wall and abdomen, and improve respiratory muscular efficiency. In mild cases, weight reduction alone may result in a cure. In other cases it enhances the effects of additional therapy.
- Sleep on one's side. Many studies have shown that patients who sleep on their backs have a significantly higher level of sleep disturbance. It is believed that sleeping in the supine position causes a gravitational pull on the tongue forcing it to come in contact with the posterior pharyngeal wall. Therefore, any technique that keeps one sleeping on his or her side could be beneficial.[25]
- One technique is a positional sleep shirt that has a long tube sewn into it. Every time a patient attempts to roll onto his or her back, he or she hits the tube. This forces the patient back onto the side or stomach.
- Avoid alcohol within two to three hours of bedtime. Alcohol is a central nervous system depressant and changes motor activity in the muscles that control normal inspiration. These changes include relaxation of the walls of the upper airway causing it to collapse.[26]
- Avoid certain pharmacological agents. Benzodiazepines, narcotics, barbiturates, and testosterone have all been reported to affect the occurrence of apneic episodes. For example, Flurazepam has been shown to worsen apneic episodes in patients who already suffer from this disease and trigger apnea in patients who have no history of the problem.[27]

Treatment Methods

Continuous Positive Airway Pressure

This technique involves wearing a mask tightly over the nose during sleep. Pressure from an air compressor forces air through the nasal passages and into the airway. This forced air creates a pneumatic splint, keeping the airway open and allowing the person to sleep normally. This is a highly effective therapy and is the most common approach for moderate and severe apnea patients. When it is accepted and used regularly by the patient, it is 100 percent successful at stopping snoring and obstructive sleep apnea.[3]

Surgical Approaches

- Tracheotomy. Surgical treatment of obstructive sleep apnea began with the tracheotomy, which has a 100 percent success rate because it completely bypasses all the sites of upper airway obstruction. This treatment is rarely done today because it is so extreme. When it is done, it does result in an immediate relief of symptoms.
- Nasal reconstruction. A nasal obstruction causes a patient to mouth breathe. When one opens the mouth to breathe, the mandible rotates back and sometimes allows the base of the tongue to drift posteriorly and block the airway. A nasal obstruction also eliminates the use of continuous positive airway pressure as a choice of treatment. Surgical procedures to clear the nasal airway are rendered in order to correct turbinate hypertrophy, septal deformities, and alar collapse, and to remove tumors or polyps.
- Uvulopalatopharyngoplasty. This procedure was introduced by Ikematsu in 1964 and later by Fujita in 1981. This surgical procedure enlarges the pharyngeal air space by excising the soft tissue of the palate, uvula, tonsils, and posterior and lateral pharyngeal walls. When the airway obstruction is only at the oropharyngeal level, this procedure can be quite successful at stopping snoring.[28]
- Laser-assisted uvulopalatopharyngoplasty. This procedure is a modification of the uvulopalatopharyngoplasty surgery. It is accomplished using lasers and is considered a less invasive procedure. It is commonly used to resculpture the soft palate.
- Orthognathic procedures. The position of the hyoid complex, mandible, and tongue, and the size and position of the maxilla all play a role in an obstruction at the oropharyngeal and hypopharyngeal level. The goal of an orthognathic surgical approach would be to make more room for the tongue and/or take the base of the tongue away from the posterior pharyngeal wall.[29]

In patients with a mandibular deficiency, surgical advancement to a normal occlusal relationship can bring the base of the tongue away from the posterior pharyngeal wall. When both a maxillary and mandibular deficiency exist, a bimaxillary advancement surgery will provide more physical room for the tongue as well as increase anterior tension on the tongue musculature.[24]

Patients with normal dental occlusion who need no additional tongue space can undergo a procedure called an anterior inferior genial osteotomy. The genial tubercle is the site of the attachment for the genioglossus muscle. In this procedure, only this part of the mandible is advanced anteriorly. This pulls the tongue forward to improve the hypopharyngeal airway.24 Various procedures have also been designed to reposition the hyoid bone and thereby advance the base of the tongue.[30]

Dental Appliances

Numerous appliances are available to treat snoring and obstructive sleep apnea. They can reposition the tissues by lifting up the soft palate, bringing the tongue forward, or lifting the hyoid bone. As they reposition, some appliances also stabilize these tissues, preventing airway collapse.

Appliances can also increase muscle tone. Specifically, there is an increase in pharyngeal and genioglossus muscle activity.[31,32]

Variations in design range from the method of retention, the type of material being used, the method and ease of adjustability, the ability to control the vertical dimension, differences in mandibular movement, and whether the appliance is lab-fabricated or made in the office.32 The appliance design chosen will depend upon the practitioner's knowledge of these variations and the oral conditions of the patient.

Decision-Making

Clearly, there are many ways to treat obstructive sleep apnea. Which method is best depends upon the severity of the patient's medical risk, the patient's anatomy, the efficacy of the different methods, the cost of each method and the desires of the patient.

For example, although surgical treatment with a tracheotomy is 100 percent effective, with time, a number of complications can result. They are tracheal site infection, physiological problems, granuloma formation, chronic irritation, uncontrolled secretions, bronchial infections, and eventual stenosis.[33] Even more important is the fact that this type of therapy is unwanted by most patients as they cannot accept the idea of a permanent tracheotomy.

Even continuous positive airway pressure, which is considered the therapy of choice by most physicians and is highly effective, is not for everybody. In fact, daily compliance by patients using this treatment is less than 50 percent. The negatives to this treatment are that it is uncomfortable, inconvenient, restricts a patient's movement, and dries out the airway mucosa.[3]

When the airway obstruction is only at the oropharyngeal level, a uvulopalatopharyngoplasty or a laser-assisted uvulopalatopharyngoplasty can be quite successful in stopping snoring and apnea. However, if the obstruction is below the oropharynx, this type of surgery is contraindicated. Most clinical investigations indicate that the success of this surgical approach to correct obstructive sleep apnea is less than 50 percent.[28] This is the case because the level and cause of the obstruction is often misdiagnosed. Removing some of the vibrating tissues may resolve snoring but it does not prevent an obstruction by the base of the tongue. Surgery is not without its complications. Postoperative stenosis, significant postoperative pain, and infection are possible complications of this approach.[28] Patients also often experience nasal regurgitation after this surgery. Furthermore, the morbidity rate for a uvulopalatopharyngoplasty is quite high.

Orthognathic procedures have been shown to be quite successful. In fact, Waite and colleagues have shown a 96 percent improvement in the apnea index when bimaxillary advancement surgery was the primary surgical procedure.[29] However these surgeries are quite invasive and most patients when given a choice will opt for a nonsurgical approach.

Research has shown that many dental appliances are quite effective and can be considered an alternative when choosing a treatment modality. Dental appliances offer several advantages over other therapy choices. They are inexpensive, noninvasive, easy to fabricate, reversible, and quite well accepted by patients.

The basic indications for dental sleep appliances are to treat primary snoring and mild to moderate obstructive sleep apnea. Appliances are particularly appropriate for those patients who cannot tolerate continuous positive airway pressure. When surgery is contraindicated or the patient is unwilling to go through a surgical procedure, appliance therapy may also be appropriate.[34]

The treatment objectives for dental appliance therapy are to stop or reduce snoring, resolve the patient's obstructive sleep apnea problems, get a higher amount of oxygen into the system, and eliminate excessive daytime sleepiness, allowing the patient to function normally.

It should be remembered that fabrication of an appliance in an attempt to stop snoring could cause the practitioner to overlook a serious medical problem such as a nasopharyngeal tumor.

Also, appliances do not work all of the time regardless of the appliance chosen. Although appliances work well for blockages that occur in the hypopharyngeal region, it is possible for there to be an obstruction in more than one region at the same time. When this occurs, the effectiveness of an appliance will be diminished. Even under the best of circumstances, a practitioner may end up trying two, three, maybe even four appliances before finding one that works well and is accepted by the patient.

Based on a complete screening and a thorough examination, any patient who snores and is suspected of being an apnea victim should be medically evaluated before the practitioner proceeds to place an appliance. Armed with the dentist's screening and oral examination results, along with their own physical evaluation, the physician can move the patient to the next appropriate step in treatment This may include immediate use of continuous positive airway pressure in a patient who is severely compromised and a referral to a sleep lab for a complete polysomnogram. A polysomnogram is the only technique to make the distinction between someone who snores and someone who is suffering from apnea.[35] Even when a patient is not suspected of suffering from apnea, it is a mistake to make an appliance without a proper medical evaluation.

Informed Consent, Follow-Up Questionnaire, Common Side Effects

Before placing a dental appliance, the practitioner should provide the patient with an informed consent. This consent should explain all the ramifications of utilizing dental appliances for the treatment of snoring and apnea.

When the patient is suffering from obstructive sleep apnea and is being treated with an appliance, a follow-up

polysomnogram to evaluate the effectiveness of the appliance should be ordered. The patient should not be referred for the second sleep test until there are subjective improvements. When the patient's excessive daytime sleepiness is gone, energy level is back, and snoring has stopped, it is time for the second sleep study.

Periodic evaluation and adjustment of appliances is a must. Most of them need to be titrated to establish the exact mandibular position needed to correct the patient's snoring and apnea. The dentist will also need to make sure that the patient's occlusion remains stable. Even though most of the appliances cap the teeth, anterior flaring and other occlusal changes can still occur. If they are kept clean, these appliances will generally last a long time. However, most of them will need to be replaced from time to time as patients break or wear through them.[32]

During the follow-up visits, the practitioner needs to ask specific questions to evaluate how the patients are progressing. Questions include:[32]

- Can you sleep with the appliance?
- Is it comfortable?
- Are your teeth sore in the morning? For how long?
- Is your bite different from normal in the morning?
- Have you seen any permanent changes in your bite?
- Does your jaw hurt? When? For how long?
- Does your bed partner hear you snore? If so, was it as loud as usual?
- Was any gasping or snoring observed?
- Did you appear to stop breathing at any time?
- Was your breathing any different from prior to the appliance placement?
- Do you wake up often?
- Do you feel more refreshed in the morning?
- How do you feel the rest of the day?
- Do you have any other comments or concerns?

Some of the common side effects that patients may experience with the use of sleep appliances are excessive salivation, discomfort in the teeth, dry mouth and tissue irritation from mouth breathing, temporary disharmonies in the bite, and some pain in the joints. Uncommon complications include TMJ dysfunction and permanent occlusal changes.

Summary

An awareness that snoring is an important sign that a serious medical problem may exist, and the knowledge that one out of every 10 people snore, should be enough information to prod the medical community into action. Dentists can play a role in both the recognition and treatment of sleep disorders. They can start by revising the standard medical and dental questionnaires. The right questions on the questionnaire can trigger a discussion on snoring and apnea. A few questions that can be added are: Do you snore? Do you wake up tired in the morning? Do you dream? Do you become extremely tired or fall asleep during the day? Are you overweight? Can you breathe through your nose? Do you drink alcohol before bedtime?

The practitioner can re-evaluate how he or she does an intraoral examination. He or she should spend the time to look at the oropharyngeal airway space, the hypopharyngeal airway space (via the chin press/tongue curl maneuver), the size of the tongue, the position of the mandible, the vault of the palate, and the nasal airway.

Most important, because sleep apnea can be associated with many other medical problems and treatment options are so varied, proper diagnosis and treatment are best handled by a team approach. The dentist should work closely with other health care professionals. Referral of patients to a physician indicates the dentist's desire to make certain that the patient receives the best care possible.

References

1. Lamberg L, Sleep Apnea Symptoms, Causes, Evaluation and Treatment. American Sleep Apnea Disorders Association, 1988.
2. Rosen RC, Rosekine M, et al, Physician education in sleep and sleep disorders: A national survey of U.S. medical schools. Sleep 16(3):249-54, 1993.
3. Garry J, The Role of the Dentist in Sleep Apnea. Dr. James Garry, 1321 N. Harbor Blvd., #201, Fullerton, CA 92635.
4. Weaver and Millman, Broken sleep. Am J Nurse 88:146-50, 1986.
5. Morgan EJ, Sleep apnea syndrome. WV Medical J 75:14-5, 1979.
6. Guilleminault C, Stoohs R, et al, From obstructive sleep apnea syndrome to upper airway resistance syndrome: consistency of daytime sleepiness. Sleep 15:s13-6, 1992.
7. Waldron RE, Sleep apnea syndrome. AFP 149:149-162, 1985.
8. Rogers R, Sleep disordered breathing, Part I: overview, physiology, clinical management. In, Clinical Dentistry Vol 1, Ch 37, 1995, pp 1-22.
9. Koskenvuo M, Kaprio J, and Partinen M, Snoring as a risk factor for hypertension and angina pectoris. Lancet 1:893-5, 1985.
10. Waller and Bhopal, Is snoring a cause of vascular disease? An epidemiological review. Lancet i:143-6, 1989.
11. Lugaresi, Cirgnotta, et al, Some epidemiological data on snoring and cardiocirculatory disturbances. Sleep 3:221-4, 1980.
12. Stoller MK, The indirect costs of sleep disorders. Clinical Ther 16:873-97, 1994.
13. Meyer and Knudson, Sleep apnea syndrome, part I: diagnosis. J Pros Dent 62:675-9, 1989.
14. Stepanski, Lamphere, and Badia, Sleep fragmentation and daytime sleepiness. Sleep 7:18-26, 1984.
15. Carskadon, Brown, and Dement, Sleep fragmentation in the elderly: relationship to daytime sleep tendency. Neurobiol Aging 3:321-7, 1982.
16. Bonnet MH, Effect of sleep disruption on sleep, performance and mood. Sleep 8:11-9, 1985.
17. Glaros AG, Incidence of diurnal and nocturnal bruxism. J Pros Dent 45:545-9, 1981.
18. Roehrs, Zorick, and Sicklesteel, Age related sleep wake disorders at a sleep disorders center. J Am Geriatric Soc 31:364-70, 1983.
19. Chervin RD, Dillon JE, et al, Symptoms of sleep disorders, inattention and hyperactivity in children. Sleep 20(12)1185-93, 1997.
20. Krieger, Turlot, et al, Breathing during sleep in normal young and elderly subjects: hypopneas, apneas and correlated factors. Sleep 6:108-20, 1983.
21. Riley R, Guilleminault C, et al, Cephalometric analysis and flow volume in obstructive sleep apnea. Sleep 6:303-11, 1983.
22. Simmons JH, Mann CA, and Gulleminault C, The chin press/tongue curl maneuver as part of the physical exam on patients suspected of sleep related obstructive respirations. Somnolgie 1(Suppl 2):1-56.
23. Simmons J, Mann C, et al, Evaluating the upper airway during the physical exam with the chin press maneuver. Sleep Research 24:269, 1996.
24. Cote E, Obstructive sleep apnea, an orthodontic concern. Angle Orthodontist Oct 1988, 293-7.
25. Cartwright RD, Effect of sleep position on sleep apnea severity. Sleep 7:110-4, 1982.
26. Krol, Knuth, and Bartlett, Selective reduction of genioglossal muscle activity by alcohol in normal subjects. Am Rev Respir Dis 129:247-50, 1984.
27. Dolly and Block, The effect of flurazepam on sleep disordered breathing and nocturnal desaturation. Am J Med 73:239-43, 1982.
28. Simmons and Guilleminault, Palatopharyngoplasty operation for snoring and sleep apnea. Otolaryngol Head Neck Surg 92:375-80, 1984.
29. Johnson, Airway changes in relationship to mandibular posturing. Otolaryngol Head Neck Surg 106:143, 1992.
30. Dierks and Keller, Obstructive sleep apnea syndrome, correction by mandibular advancement. So Medical J

83(4)390-4, 1990.
31. Clark G, OSA and dental appliances, uses of dental appliances to treat sleep disorders. J Cal Dent Assoc Oct:26-33, 1988.
32. Rogers R, Sleep disordered breathing, part II: oral appliance therapy. In, Clinical Dentistry, Vol 1, Ch 37A 1-32, 1995.
33. Meyer and Knudson, Sleep apnea syndrome, part II. J Pros Dent 63(3):320-4, 1990.
34. An American Sleep Disorders Association Report, Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances. Sleep 18(6):511-13. 1995.
35. Haponick, Smith, et al, Evaluation of sleep disordered breathing. Is polysomnography necessary? Am J Med 77:671-7, 1984.
36. Clinical Protocol for Dental Appliance Therapy for Snoring and Obstructive Sleep Apnea. Sleep Disorders Dental Society, 11676 Perry Hwy Bldg, Wexford, PA 15090.

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Diagnostic Sleep Testing in the Evaluation of Snoring and Sleep Apnea

Jerald H. Simmons, MD

ABSTRACT The dilemma frequently encountered by the dentist wishing to offer an appliance for treatment of snoring or sleep apnea is whether there is a need for extensive testing of patients who present with a complaint of snoring. This article describes diagnostic sleep testing in the evaluation of patients with snoring and sleep apnea.

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Snoring and sleep apnea are extremely common, and patients with these conditions require proper evaluation by the health care system. The dilemma frequently encountered by the dentist wishing to offer an appliance for treatment of snoring or sleep apnea is whether there is a need for extensive testing of patients who present with a complaint of snoring. This article will describe diagnostic sleep testing in the evaluation of patients with snoring and sleep apnea. To understand the role of sleep testing, it is also important to recognize some of the physiology associated with the sleeping process.

Basics of Sleep Physiology

Sleep testing evaluates the state of the patient throughout the night. The standard procedure is to assign a sleep stage (defined below) for each consecutive 30-second period throughout the study.[1] This results in a tabulation of the patient's sleep state throughout the night.

Sleep Stages

The sleep process can be divided into two main stages, rapid eye movement (REM) and non-rapid eye movement (Non-REM). Non-REM sleep normally precedes REM sleep and can be further divided into stages 1, 2, 3, and 4. Frequently stages 3 and 4 are combined and referred to as slow wave, or delta, sleep. Non-REM sleep occupies most of the night.1 There are many important aspects of non-REM sleep, but most of them are beyond the scope of this article. For the purposes of this discussion, two main stages of sleep, REM and non-REM, will be referred to.

REM sleep was first described in 1953.[2] REM sleep is regulated by the brain stem.[3] Features of REM sleep include the following:

- It occurs about every 90 minutes during sleep.
- Each episode becomes longer as the night progresses.
- Vivid dreaming occurs during REM sleep

- Muscle paralysis (hypotonia) occurs, including the muscles of the upper airway.

During REM sleep, the airway muscles are more vulnerable to collapse because of their state of hypotonia. During REM sleep, obstructive respirations are most pronounced, particularly in obese individuals such as those with obstructive sleep apnea.[4,5] This is particularly true when a person is sleeping supine because gravity plays an additional role, which increases the vulnerability of airway collapse. Therefore, if a person is not evaluated while supine in REM sleep, a diagnosis of a sleep-related breathing disorder cannot be excluded.[5]

The progression of sleep that occurs through the night is such that REM primarily occurs during the last half of the night. The first REM episode typically occurs 90 minutes after sleep onset. This REM episode is usually very brief, lasting only several minutes. This is usually followed by a return of Stage 1 and the process repeats. As the brain transitions through the various sleep stages, each REM episode becomes longer, and the slow wave sleep of non-REM becomes shorter. Normally, people have most of their REM in the early morning hours, i.e., 3 to 6 a.m.[4,6]

Sleep Fragmentation

A brief event called a micro-arousal is a disruption of sleep continuity that usually does not result in complete awakening and therefore occurs without the recollection of the patient. Nonetheless, repetitive arousals during the night usually result in daytime sleepiness and fatigue the next day.

Obstructive Respirations

Since sleep studies evaluate the respiratory process, it is important to understand the dynamics of the upper airway that are associated with obstructive respirations during sleep. As air passes through the back of the throat, it can create a negative pressure as a result of the Bernoulli effect. The narrower the airway passage, the greater the negative pressure. When the pressure becomes great enough, the soft tissue of the pharynx, such as the soft palate and uvula, are pulled and begin to vibrate. This vibration causes the snoring sound.[4] When the negative pressure increases to a certain point, the airway may completely collapse. This is what is known as an apneic episode, during which breathing ceases. During this event, the oxygen level typically drops at least 2 percent. The apneic episode terminates in an arousal, which results in increased muscle tone of the airway, opening up the pharynx so breathing is re-established. To be tabulated as an apnea during a sleep study, the event must last a minimum of 10 seconds.[7]

The airway does not need to completely collapse for a significant event to occur. Even with only partial obstruction of the airway, a slight drop in the oxygen level and a brief arousal from sleep can still occur. This event is known as a hypopnea. Like apneas, hypopneas can fragment sleep and cause daytime sleepiness.[4,7]

Recent studies have demonstrated that the spectrum of clinically significant obstructive respirations can include events that are more subtle than hypopneas.8 These subtle events consist of increases in upper airway resistance occurring from a partial blockage of the upper airway. This results in increased work of breathing, triggering an arousal (or micro-arousal) that fragments sleep. This can occur even without a measured decrease in airflow by the routine methods employed in most sleep laboratories.[5,8,9] These events are usually associated with a crescendo increase in snoring, which culminates with the micro-arousal. When this occurs repetitively, it can also cause excessive daytime sleepiness. This is known as upper airway resistance syndrome.8-10 There is frequently no decrease in the blood oxygen level associated with the respiratory events of upper airway resistance syndrome.[8-10]

Measurements of the pressures within the airway can be performed by the addition of a pressure sensors within the airway below the point of obstruction, such as in the esophagus. The pressure sensor can identify a crescendo increase in respiratory effort with each breath, culminating with the micro-arousal. Most laboratories do not use the pressure catheter to establish a diagnosis of upper airway resistance syndrome but instead use the snoring pattern. The snoring increases in a crescendo pattern and leads to an arousal. These subtle events have only relatively recently been recognized as significant and are now known as respiratory arousals. The tabulation of respiratory arousals has only recently been adopted into the practice parameters established and promoted by the American Sleep Disorders Association.5 A person does not have to snore to have upper airway resistance syndrome. In the absence of snoring, the only way to establish the diagnosis is with the esophageal pressure catheter to detect the subtle respiratory-induced micro-arousals.

The spectrum of obstructive breathing ranges from upper airway resistance syndrome to obstructive sleep apnea.8 Anywhere along this spectrum, the degree of obstructive respirations can be considered significant if sleep is disrupted.

It is important to be aware of the cardiovascular consequences of severe obstructive respirations during sleep. Many studies have been done and more are currently under way that are characterizing the ill effects of repetitive obstruction of the upper airway during sleep. The main concerns are the increased risks of hypertension, stroke, and cardiac disease.[11-16] Therefore, even if a patient is not sleepy during the day but suffers from severe obstructive sleep apnea, the apnea should be treated to prevent the development or worsening of these other medical conditions.

Diagnostic Testing for Snoring and Obstructive Sleep Apnea

Sleep testing is performed to identify and quantify the respiratory events described above. In addition, sleep quality is evaluated by measuring the presence of appropriate sleep stages and the absence or presence of frequent arousals.

The most reliable method to identify obstructive sleep apnea is a nocturnal polysomnograph.[5] This test measures a variety of parameters. The most common parameters used for nocturnal polysomnograph testing include the following.

- Right and left eye movements or electro-oculograph;
- Electroencephalograph, two to four separate channels;
- Chin muscle activity, known as electromyography;
- Electrocardiograph;
- Leg electromyography, usually right to left anterior tibialis muscles;
- Snoring or tracheal sounds, measured with a microphone taped to the throat;
- Air flow through the nose and mouth;
- Movement of the chest;
- Movement of the abdomen;
- Oxygen level, measured with a finger oximeter;
- Body position; and
- Esophageal manometry – in some laboratories.

Quantifying sleep into REM and non-REM sleep requires that eye movement, electroencephalograph, and muscle activity be measured.1 There are specific features in the signals from these channels that are used to categorize sleep into the different sleep stages. For example, REM sleep is characterized by a decrease in chin electromyography activity, rapid eye movements on the eye channels, and specific features in the electroencephalograph channels. Arousals, which are brief disruptions in the continuity of sleep, must last at least three seconds.17 Identification of arousals requires electromyograph and electroencephalograph signals for proper tabulation. It is possible to

TABLE 1.

The ASDA Standards of Practice Committee has adopted the following indications for sleep testing for the evaluation of sleep-related breathing disorders.

1) Nocturnal Polysomnography is routinely indicated to establish a diagnosis of sleep-related breathing disorders.
1.1) For most patients a full NPSG is required.
1.2) For patients with a high degree of clinical suspicion for Obstructive Sleep Apnea (OSA) a cardiopulmonary sleep study can be performed, but a negative study must be followed up with a full NPSG study.
2) NPSG is indicated to establish the proper Continuous Positive Airway Pressure (CPAP) in patients with sleep-related breathing disorders.
2.1) Full NPSG with CPAP is recommended for patients with documented sleep-related breathing disorder in whom CPAP is warranted.
2.2) NPSG with CPAP is appropriate for patients with any of the following NPSG results:
a) An Apnea Index (AI, which is the number of apneas per hour of sleep) of at least 20 per hour or an Apnea Hypopnea Index (AHI, which is the number of apneas and hypopneas per hour of sleep) of at least 30 per hour, regardless of the patient's symptoms.
b) AHI of at least 10 per hour in a patient with excessive daytime sleepiness.
c) A respiratory arousal index of at least 10 per hour in a patient with daytime sleepiness.
2.3) A cardiorespiratory sleep study without EEG channels is NOT recommended for CPAP titration (a study which is performed to determine the optimal CPAP pressure).
2.4) For CPAP titration, a split-night study (first half for diagnosis and second half for CPAP titration) is an alternative to one full night of diagnostic NPSG followed by a second night of titration if the following four criteria are met:
a) An AHI of at least 40 per hour documented during a minimum of 2 hours. A split-night may be considered with a AHI of between 20 to 40 based on clinical judgment.
b) CPAP titration is carried out for more than 3 hours.
c) The study documents that CPAP eliminates or nearly eliminates the respiratory events in both REM and Non-REM sleep, including REM sleep with the patient in the supine position.
d) A second full night NPSG CPAP titration is performed if criteria b and c are not met..
3) Preoperative NPSG or cardiopulmonary sleep studies are indicated to evaluate patients for the presence of obstructive sleep apnea before they undergo laser-assisted uvulopalatopharyngoplasty or other related surgical procedures.
4) Follow-up NPSG or cardiopulmonary sleep studies are routinely indicated for the assessment of treatment in the following circumstances:
4.1) After good clinical response to oral appliance treatment in patients with moderate to severe obstructive sleep apnea, to ensure therapeutic benefit.
4.2) After surgical treatment of patients with moderate to severe obstructive sleep apnea, to ensure satisfactory response.
4.3) After surgical treatment of patients with sleep apnea whose symptoms return despite a good initial response to treatment.
5) Follow-up NPSG studies are routinely indicated in the following circumstances:
5.1) After substantial weight loss has occurred in patients on CPAP for treatment of sleep-related breathing disorders to ascertain whether CPAP is still needed at the previously titrated pressure.
5.2) After substantial weight gain has occurred in a patient on CPAP who again is symptomatic despite continued use of CPAP, and to adjust the CPAP pressure to a higher level in order to prevent obstructive respirations.
5.3) When clinical response is insufficient or when symptoms return despite a good initial response to treatment.
6) Follow-up studies are not indicated in patients who continue to do well with treatment.

identify arousals using other parameters such as sudden fluctuations in heart rate, but these are less reliable. Therefore, sleep studies done without electro-oculograph, electroencephalograph, and electromyograph are not reliably able to quantify sleep, stage sleep, or identify arousals. These limited monitoring devices primarily record aspects of respiration. Studies performed with such devices are referred to as cardiorespiratory studies.[5] The parameters included may range from oximetry with the addition of some measure of respiratory effort, to a six-channel unit that measures air flow, snoring, chest movement, oximetry, heart rate, and body position. The current position of the American Sleep Disorders Association is that obstructive sleep apnea is best diagnosed using nocturnal polysomnography.5 In some instances, cardiorespiratory sleep studies can be used to establish a diagnosis but never to exclude a diagnosis of a sleep-related breathing disorder.[5] This is because sleep fragmentation cannot be identified, and therefore subtle breathing disturbances may go undetected.

Diagnostic sleep testing in patients suspected of snoring or sleep apnea is primarily done to identify and quantify the occurrence of apnea, hypopnea, and subtle respiratory arousals that do not fall within the apnea or hypopnea definition but still disrupt sleep. Some patients may present only with a complaint of snoring and deny witnessed pauses in respirations or deny symptoms of daytime sleepiness. This is a common scenario that is handled differently by many in the field. Some practitioners may choose not to study these patients at all, but this approach is short-sighted.

Even though the patient has not been witnessed to have pauses or abnormal breathing during sleep, most likely these observations are made at the beginning of the sleeping period, when a bed partner is still awake. Most patients with obstructive respirations have their worst respirations during REM, and most of the REM sleep occurs during the last portion of the night, as stated earlier.[4] As explained earlier, REM episodes become longer with each sleep cycle as the night progresses. The hypotonia of REM results in vulnerability of the airway to collapse. It is during these early morning hours that the bed partner may also be asleep and therefore unable to witness any apneic events. Therefore, it is not adequate to rely on a bed partner's observation to exclude a diagnosis of sleep apnea. Even if the patient only complains of snoring, a sleep study is important to evaluate the degree of obstructive respiration before embarking on treatment methods.

Many sleep laboratories have a backlog of patients, and the American Sleep Disorders Association advocates the use of cardiopulmonary sleep studies to shorten the waiting period for testing of patients with a high probability of sleep apnea.[5]

The American Sleep Disorders Association published its most recent practice parameters in 1997 for the indications of sleep testing. A summary of these recommendations is outlined in Table 1.5

The American Sleep Disorders Association has proposed an algorithm for the evaluation of snoring. The recommendations state that full nocturnal polysomnography be performed to exclude a diagnosis of a sleep-related breathing disturbance. The cardiorespiratory testing devices are only recommended when there is a high clinical suspicion of obstructive sleep apnea. The algorithm matrix does not recommend a cardiorespiratory sleep study be performed to exclude a diagnosis of a sleep-related breathing disorder, but rather only to establish the diagnosis. Therefore, the recommendation is for negative cardiorespiratory sleep studies to be followed up with full nocturnal polysomnography testing. The algorithm, however, is not designed to accommodate the typical situation encountered by dentists who may embark on treating patients with snoring. In fact, the algorithm states that if there is "simple snoring," then a nocturnal polysomnograph is recommended only if a laser-assisted uvulopalatopharyngoplasty surgery is being considered as a treatment option. No mention is made of appliance therapy. It is stated that the algorithm is a guideline, and in clinical practice it may be necessary to modify the approach to accommodate specific situations.

Evidence is beginning to demonstrate that snoring alone is not a benign process. A recent study evaluated the histological changes within the upper airway muscles in habitual snorers and nonsnoring control subjects.18 The study demonstrated changes within the upper airway muscle tissue of the snoring subjects in a fashion compatible with a neuropathy, such as that caused by trauma. This suggests that the trauma on the tissues of the airway from snoring alone may cause changes that increase the dysfunction of the upper airway muscles, leading to further airway collapse. Therefore, the presence of snoring is an indication for some degree of objective testing.

Future Directions

No clear guideline has been presented for dentists to follow when making decisions on ordering sleep testing. No one would question the need for full nocturnal polysomnography on patients with severe daytime sleepiness, or possible sleep apnea if the patient has a condition that has been shown to be worsened by sleep-related breathing disorders, i.e., heart disease or hypertension. The confounding situation is the patient with a complaint of only snoring and otherwise good health, with no daytime sleepiness. The decision of whether to test this group and the type of testing for this group has not been elaborated on within current practice parameters, other than stating that a sleep study is indicated in this group prior to laser-assisted surgery of the upper airway. Because apnea may be occurring in REM sleep and may go unrecognized by a sleeping bed partner, a sleep study may be indicated to exclude a sleep-related breathing disorder prior to embarking on any treatment of the upper

airway. One approach to be considered is to use cardiorespiratory testing devices for a screening study in the seemingly healthy, snoring, patient prior to fitting a dental appliance. This would allow the identification of moderate to severe apneas, which if present would need proper follow-up attention with a repeat study with the appliance in place. It may be argued that as long as the patient does not have any other conditions that could be worsened by mild sleep apnea, there seems little justification for dentists to perform full nocturnal polysomnograph sleep studies on mild, seemingly healthy, snoring patients. This approach relies on the dentist to take an extensive history from the patient to determine symptoms of daytime sleepiness, or the presence of cardiovascular or cerebral vascular disease. Using specific questionnaires designed to quantify symptoms of daytime sleepiness[19,20] may assist the dentist.

Another suggested approach eliminates pretreatment sleep testing completely in patients with asymptomatic snoring, i.e., snoring without witnessed pauses in breathing, daytime sleepiness, or other cardiovascular disease.21 Neither this approach nor the approach utilizing the cardiorespiratory screening test have been adopted by a consensus panel as of this time, and it is still the standpoint of the American Sleep Disorders Association to perform full nocturnal polysomnograph studies when evaluating to exclude a diagnosis of a sleep-related breathing disorder.

References

1. Rechtschaffen A and Kales A, eds, Manual of Standardized Terminology: Techniques and Scoring System for Sleep Stages of Human Subjects. UCLA Brain Information Service/Brain Research Institute, Los Angeles, 1968.
2. Aserinsky E and Kleitman N, Regularly occurring periods of eye motility and concomitant phenomena during sleep. Science 118:273-4, 1953.
3. Hobson JA, McCarley RW, and Wyzinski PW, Sleep cycle oscillation: reciprocal discharge by two brain stem neuronal groups. Science 189:55-8, 1975.
4. Kryger MH, Roth T, and Dement W, Principles and Practice of Sleep Medicine, 2nd ed. WB Saunders Co, Philadelphia, 1994.
5. Indications for Polysomnography Task Force, American Sleep Disorders Association Standards of Practice Committee, Practice parameters for the indications for polysomnography and related procedures. Sleep 20(6):406-422, 1997.
6. Zulley J, Distribution of REM sleep in entrained 24-hour and free-running sleep-wake cycles. Sleep 2:377-89, 1980.
7. The International Classification of Sleep Disorders, Diagnostic and coding manual. 1990 American Sleep Disorders Association. Allen Press.
8. Guilleminault C, Stoohs R, et al, From obstructive sleep apnea syndrome to upper airway resistance syndrome: consistency of daytime sleepiness. Sleep 15:s13-6, 1992.
9. Srollo P and Saunders M, Significance and treatment of nonapneic snoring. Sleep 16(5):403-8, 1993.
10. Guilleminault C, Stoohs R, and Duncan S, Snoring: daytime sleepiness in regular heavy snores. Chest 99(1):40-8, 1991.
11. Palomaki H, Partinen M, et al, Snoring, sleep apnea syndrome, and stroke. Neurology 42(supp 6):75-82, 1992.
12. Koskenvuo M, Kaprio J, et al, Snoring as a risk factor for hypertension and angina pectoris. Lancet 1:893-5, 1985.
13. Koskenvuo M, Kaprio J, et al, Snoring as a risk factor for ischaemic heart disease and stroke in men. Br Med J 294:16-9, 1987.
14. Podzus T, Greenberg H, and Scharf SM, Influence of sleep state and sleep-disordered breathing on cardiovascular function. In, Saunders NA and Sullivan CE, eds, Sleep and Breathing, 2nd ed. Marcel Dekker, New York, 1994, pp 257-310.
15. Hla KM, Young TB, et al, Sleep apnea and hypertension: a population-based study. Ann Intern Med 120:382-8, 1994.
16. Fletcher EC, The relationship between systemic hypertension and obstructive sleep apnea: facts and theory. Am J Med 98:118-28, 1995.
17. Sleep Disorders Atlas Task Force of the American Sleep Disorders Association, EEG arousals: scoring rules and examples. A preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. Sleep 15:174-84, 1992.
18. Friberg D, Ansved T, et al, Histological indications of a progressive snorers disease in an upper airway muscle. Am J Repir Crit Care Med 157(2):586-93, 1998.
19. Johns MW, A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 14:540-5, 1991.
20. Johns MW, Reliability and factor analysis of the Epworth Sleepiness Scale. 15:376-81, 1992.
21. Perez-Guerra F, How to shorten the sleep laboratory waiting list. ASDA News 5(1):15-6, 1998.

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Medical and Nondental Treatments of Snoring and Sleep Apnea Syndrome

Lawrence W. Kneisley, MD

ABSTRACT The prevalence, pathophysiology, and clinical and polysomnographic evaluation of obstructive sleep apnea are reviewed. The history of the development of nasal continuous positive airway pressure, diagnostic titration of the treatment, abolition of nocturnal apneas, and consolidation of sleep architecture by nasal continuous positive airway pressure and long-term patient compliance with the treatment are discussed. The effects of weight gain and weight loss on the severity of obstructive sleep apnea, and cardiovascular and behavioral complications of obstructive sleep apnea are reviewed. Behavioral treatments are discussed.

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> Blessings on him who first invented sleep.It covers a man all over, thoughts and all, like a cloak. It is meat for the hungry, drink for the thirsty, heat for the cold, cold for the hot.It makes the shepherd equal to the monarch, and the fool to the wise.
>
> From *Don Quixote*
> Miguel de Cervantes Saavedra

The 1995 report of the National Commission on Sleep Disorders Research, commissioned by Congress, estimated that some 10 million adults in the United States suffer from sleep apnea syndrome, most of whom do not know they have it.[1] An epidemiologic study of some 900 middle-aged adults who underwent polysomnography found the prevalence of obstructive sleep apnea to be 9 percent among men and 4 percent among women.[2] Through the use of polysomnography, the pathophysiology of the syndrome of obstructive sleep apnea has become well-understood.[3,4] Sleep apnea is not a single disease but rather a pathophysiologic syndrome resulting from many diseases and abnormalities.[5]

Snoring and sleep apnea syndrome are the two ends of a spectrum of sleep-related compromise of the upper airway. A patient at the mildest end of the spectrum may snore only under certain circumstances, such as when sleeping supine or during REM (rapid eye movement) sleep.

The breathing of patients with greater degrees of upper airway obstruction during sleep is characterized by continuous snoring punctuated by apneas or hypopneas (partial obstructions of air flow) during REM sleep when the tone of the pharyngeal dilating muscles is most reduced or when in the supine position when the

force of gravity on the soft tissues of the tongue and palate further reduces the diameter of the upper airway. At the severe end of the spectrum, the pharyngeal airway of the sleeping patient repeatedly collapses and obstructs completely. The sleeping patient has repetitive obstructive apneas lasting many seconds despite diaphragmatic and intercostal respiratory efforts. Each obstructive apnea is accompanied by drops in arterial oxygen of some 10 percent or 20 percent or more in non-REM sleep and greater drops during REM sleep. Each apnea is ultimately terminated when the patient arouses from sleep. Arousals increase the tone of the pharyngeal dilating muscles, allowing unobstructed breathing and rapid return of arterial oxygen saturation to normal. This process repeats itself hundreds of times during the night. The repetitive arousals from sleep, unappreciated by the patient and often numbering in the hundreds, cause fragmented sleep with a resulting complaint of daytime drowsiness.

Treatments

Nasal Continuous Positive Airway Pressure

Nasal continuous positive airway pressure is the most effective treatment for patients suffering from obstructive sleep apnea syndrome. It is most often used to treat patients whose obstructive sleep apnea is severe or moderate. In some cases, the treatment benefits those whose obstructive sleep apnea is only mild.6

Though arbitrary numbers are at best approximate, most sleep specialists classify an apnea-hypopnea index (the number of apneas plus hypopneas per hour of sleep) of 15 or 20 per hour as mild obstructive sleep apnea, an index of 20 to 50 or 60 as moderate, and an index of greater than 50 or 60 per hour as severe.

Nasal continuous positive airway pressure was developed by Sullivan and colleagues7 in Australia in 1981. Since its approval by the U.S. Food and Drug Administration in 1985, its use to treat patients with obstructive sleep apnea has become widely accepted. The American Sleep Disorders Association has estimated that some 40,000 prescriptions for nasal continuous positive airway pressure were written in the United States in 1991 and that the number has continued to increase. There are now six manufacturers of the equipment worldwide. The continuous positive airway pressure blower delivers air at above atmospheric pressure via an external face mask to the upper airway through the nares. Continuous positive airway pressure acts by providing a pneumatic "splint" to the upper airway. Thus it is a treatment for obstructive sleep apnea , not a cure.

Determination of Pressure Titration

In virtually all sleep centers, a patient that has or is suspected to have obstructive sleep apnea undergoes "titration" to determine the pressure appropriate for the patient. The patient is monitored overnight in a sleep recording labora-

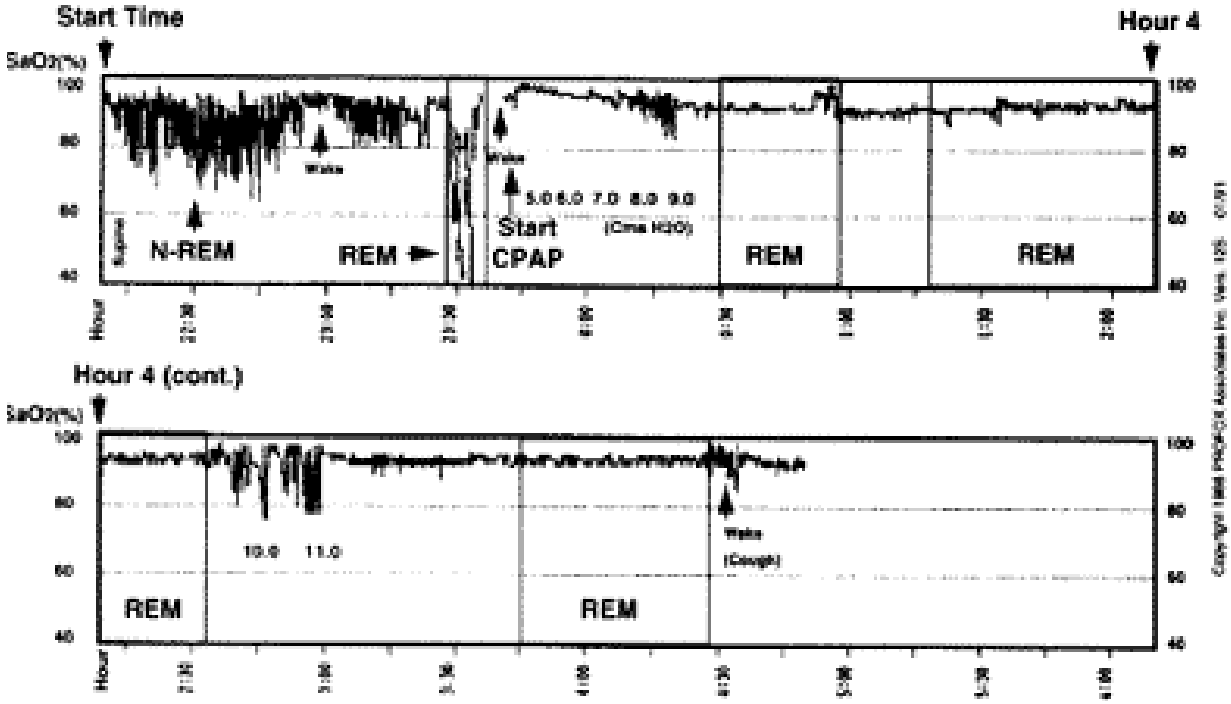

Figure 1. Oximetry/split-night continuous positive airway pressure titration.

tory in the standard fashion. After the procedure is thoroughly explained, the lights are turned off; and the patient's sleep state, body position, oxygen saturation, respiratory rate, heart rate, snoring sounds and pressure are recorded and monitored continuously. The patient is also observed by the technologist using a low-light camera. The treatment mask is placed over the patient's nose, and titration is begun at a low pressure, typically 5.0 centimeters of water. After the patient falls asleep and snoring and apneas appear, the pressure is gradually increased by 1 centimeter of water increments.

The end point of the titration is the abolition of all arousals, snoring, apneas, and hypopneas in both REM and non-REM sleep and steady transcutaneous oxygen saturation values of 95 percent to 98 percent at sea level. The pressure requirement may be several centimeters of water higher in REM sleep than in non-REM sleep. The technologist must be sure that the titration is performed with the patient in the supine position during some portion of the night, most desirably during both REM and non-REM sleep. The procedure takes several hours to measure pressure under all sleep conditions. Within a few minutes of establishing an open upper airway, a patient with severe sleep apnea may begin to have long periods of REM sleep and Stage 4 non-REM sleep.8 This "recovery sleep" pattern after years of sleep fragmentation lasts about a week. Patients often report an immediate increase in alertness after a single night's treatment (Figure 1).

In the past, the patient would undergo a seven- to eight-hour diagnostic recording night in the sleep laboratory and then, if indicated, would have a second recording night for titration of pressure. In recent years, for financial considerations and patient convenience, a single split-night protocol has become widely used to establish the diagnosis of obstructive sleep apnea and to titrate pressure in the same night.9 While protocols vary, the split-night titration is successful in 70 percent to 90 percent of the recording nights.10 In achieving a successful study, much depends on the instruction and preparation given to the patient before the split-night study.11 In many protocols, a patient whose sleep-disordered breathing is observed during the first half of the night to be mild is allowed to sleep without an attempt at continuous positive airway pressure. Patients with moderate or severe obstructive sleep apnea are most likely to successfully complete a split-night titration, though the pressure requirements of some patients whose apnea-hypopnea index is less than 40 per hour may be underestimated by 2 to 3 centimeters of water.[12,13]

Those patients with significant obstructive sleep apnea who are unable to sleep or to tolerate nasal continuous positive airway pressure during the split night are encouraged to return for a full night of titration after full and careful discussion of all the therapeutic options. Patient preferences, clinical symptoms, the severity of polysomnographic findings, and medical co-morbid conditions, considered collectively by both doctor and patient, ultimately determine the treatment(s) of the patient's sleep disorder.

Compliance at Home

Once acclimated to continuous positive airway pressure, most patients usually have little difficulty going to sleep when using the small, quiet modern machines, provided that the nasal mask is comfortable and there are no air leaks. Early objective measures of patients' use at home found only fair compliance.14 However, a more recent 10-year European study that excluded patients who had died or been successfully treated with surgery or weight loss found 79 percent of patients still using home continuous positive airway pressure an average of five to six hours per night.15 Patients with more severe obstructive sleep apnea (an apnea-hypopnea index greater than 15) had better compliance than those whose condition was less severe. Nasal congestion, a common complaint early in the course of use, usually resolves in one to two weeks but can reduce patient use during that time. Patient acceptance of home treatment depends on the skill and care used in fitting the mask, the adjustment of the head strap and hose, and use of air humidification. Written instruction regarding the value and importance of using continuous positive airway pressure and frequent follow-up visits to the sleep physician or technologist are vital in improving patient comfort and achieving long-term patient compliance.16 Of great importance is the face mask, the interface between patient and air source, which has undergone considerable design improvements in the 14 years since its introduction. The early face masks were of a plastic that became hard and uncomfortable after one or two months because of the effect of skin oils. Patients now have a choice of lighter-weight nonhardening silicone "floating" masks, double-layered "bubble" masks, or gel masks, available in many sizes. Modern masks also have quiet, light-weight nonmechanical air valves without moving parts. Another innovation, nasal "pillows," cover only the nares and are preferred by some patients. Masks are held on the face with head straps of simpler design and are attached to lighter and more flexible hoses, which deliver heated or nonheated humidified air.

These interface interchangeably with continuous positive airway pressure machines from many manufacturers. In contrast to the earliest American-made machines, which weighed 10 to 12 pounds and were by today's standards noisy and bulky, modern machines are compact and quiet and have advanced electronics that respond quickly to changes in air flow requirements. Additionally, most machines have a pressure "ramping" capability that allows the patient to fall asleep with the mask in place at a low pressure. Over the ensuing 15 to 30 minutes, the pressure gradually rises to the prescribed level. This design feature contributes greatly to improved patient acceptance of nasal con-

Figure 2. Respironics "Solo" continuous positive airway pressure machine.

tinuous positive airway pressure. Recently, self-adjusting units have been developed by several manufacturers. Experience with a small number of patients suggests that the time of use of the auto-adjusting units may be increased by as much as one hour per night compared to time using conventional units.17 These machines are more expensive than standard ones, and standards for their clinical use are still evolving. In sum, the majority of patients beginning home treatment who are carefully managed continue to use the machine for many years because of the immediate and dramatic improvement in daytime performance and alertness (Figure 2).[18]

Management of the Patient on Home Treatment

Supervision by physicians and sleep center technicians skilled and experienced in the treatment of patients with obstructive sleep apnea and other sleep disorders is essential in maintaining patient tolerance and compliance with home continuous positive airway pressure.

The machines should be calibrated periodically with a water manometer to correct any pressure "drift." Physicians should be alert to the effects of weight gain or weight loss on pressure requirements. A patient who experiences a large weight gain will require an increase of pressure, possibly necessitating a repeat titration sleep study. Where actual home use is uncertain, machines with recording capability may be used to assess a patient's compliance. Some patients who require very high pressures or those with coexisting chronic obstructive lung disease may be unable to tolerate continuous positive airway pressure but may find bi-level treatment more acceptable.19 With bi-level treatment, the inspiratory and expiratory pressures are separately controlled. By lowering the expiratory pressure, the work of breathing is reduced and patient comfort is improved compared to using a fixed high pressure.

Travel and Durability

Modern continuous positive airway pressure units are compact, about the size of a large loaf of bread, weigh 4 to 6 pounds and are easily carried on airplanes, cars, and even motorcycles. They can operate on 110 or 220 volts AC or 12 Volts DC. The units have an average life span of about four to five years, but some have lasted longer. Masks, head straps, and hoses may need replacement after about two years, and the small particle air filters should changed every one to two months.

Weight Loss

Obesity is a predisposing factor for snoring and sleep apnea; 75 percent of obstructive sleep apnea patients are obese.20 While the prevalence of obstructive sleep apnea (based on an apnea-hypopnea index of greater than 15 per hour) in the general population is 4 percent for women and 9 percent for men,[2] the prevalence among obese patients is much higher. Indeed in one university obesity clinic, 40 percent of men and 3 percent of women were found to have obstructive sleep apnea severe enough to warrant treatment.[21]

Recent studies show that upper body obesity carries an increased risk of snoring, sleep apnea, and hypertension. In the population study cited above2 the severity of patients' obstructive sleep apnea correlated better with neck size than with body mass index (height in meters divided by weight in kilograms).22 Among men with a neck circumference of 15 3/4 inches to 17 inches, 18 percent had obstructive sleep apnea. Of those with a neck circumference greater than 17 inches, 31 percent had obstructive sleep apnea.[22] There was a similar relation of increasing neck size to severity of the condition among the women. Weight loss is beneficial in reducing the severity of the condition. Studies of obese sleep apnea patients treated by weight loss using gastric bypass have shown improvement or abolition of obstructive sleep apnea in all.[23] The relationship between weight loss and decline in apnea index is probably not linear, supporting the observation that even modest weight loss may improve snoring and apneas in some patients.[24] The upper airway of patients with obstructive sleep apnea is abnormal in shape: It is elliptical with the long axis oriented anteroposteriorly. This contrasts with the normal airway, whose elliptical shape is oriented transversely.[25] This alteration in airway shape among obese sleep apneaics is probably due to fat deposition in the neck adjacent to the muscles and soft tissues forming the pharyngeal airway. Weight loss probably benefits patients by decreasing the amount of fat surrounding the upper airway, thereby allowing it to dilate more fully to become less compliant and less prone to collapse and obstruct in sleep. With these observations in mind, modest weight loss even to a level above ideal body weight may be reasonable and beneficial in patients with obstructive sleep apnea.

Causes and Complications – Evaluation and Treatment

Predisposing Endocrine Disorders

Hypothyroidism, acromegaly, and testosterone administration can cause, predispose, or exacerbate obstructive sleep apnea.

These should be considered, ruled out, or treated when appropriate.

Drugs, Alcohol and Anesthesia

Alcohol depresses respiratory drive and increases arousal threshold in patients with sleep apnea and has been demonstrated to worsen obstructive sleep

apnea when imbibed before bedtime. Some sedating drugs, such as barbiturates and chloral hydrate, behave similarly. The long-acting benzodiazepine, flurazepam (Dalmane), may worsen obstructive sleep apnea in some patients with pre-existing disease.26 However, the intermediate half-life benzoidazepine hypnotic, temezepam (Restoril),[27] may be safely given to elderly patients with mild obstructive sleep apnea; and the short-acting hypnotic benzodiazepine, triazolam (Halcion), improves sleep architecture in patients with central sleep apnea.28 Obstructive sleep apnea patients undergoing general or light anesthesia or parenteral narcotics merit careful monitoring. Endotracheal intubation may be difficult because of the shape of the airway, combined with a short, thick neck. After extubation, obstructive sleep apnea patients are more prone to experience upper airway obstruction and should be monitored closely, especially while in the supine position.

Cardiovascular Disease

Half of patients with obstructive sleep apnea are hypertensive and may require multiple medications. Apnea patients have an increased prevalence of esophageal reflux,29 accelerated coronary artery disease, 30 frequent cardiac arrhythmias,31 and premature death.[32]

Behavioral Abnormalities

Patients with obstructive sleep apnea are sleepy during the day. Subtle impairments of memory and judgment may be observed.

Behavioral Treatments

Sleep Position Training

Patients whose all-night polysomnograms show snoring and apneas exclusively or predominantly when sleeping in the supine position can benefit significantly by sleeping in the lateral decubitus position. A number of small position-sensitive alarm devices that are strapped to the patient's chest or abdomen have been successful in training the patient to sleep on his or her side. The same can be accomplished by inserting two tennis balls (one is not sufficient) in a sock that is pinned or sewn to the back of the sleeping garment.[33]

Sleep Hygiene

The patient should be counseled to avoid alcohol near bedtime. Erratic sleep schedules, shift work, and trans-meridian flight disrupt the sleep-wake schedule, thereby resulting in reduced total sleep time and increased daytime sleepiness. A patient on home nasal continuous positive airway pressure should take his or her machine on all trips where electric power is available. Those living at or traveling to high altitudes should consider adding a humidifier to the blower to combat drying of the nasal mucosa. These are inexpensive and are available from all machine manufacturers. Nasal and sinus allergies should be promptly treated to maximize compliance.

Elevation of Head of Bed

A patient with congestive heart failure, severe lung disease, or treatment at very high pressures may find nasal continuous positive airway pressure easier to tolerate if the head of the bed is elevated 3 to 6 inches or sometimes more.

Other Mechanical Devices

Nasal obstruction causing increased nasal resistance may produce nasal snoring and can predispose the nasopharyngeal airway to collapse, resulting in obstructive apneas. Nasal dilators may sometimes be helpful in patients with nasal snoring due to narrowing of the anterior nasal passages.[34]

Medications

There are no medications that specifically or uniquely benefit obstructive sleep apnea.

References

1. National Commission on Sleep Disorders Research, A Report of the National Commission on Sleep Disorders Research. Wake up America: a National Sleep Alert, vol 2. US Government Printing Office, Washington, DC, 1995:10.
2. Young T, Palta M, et al, The occurrence of sleep-disordered breathing among middle-aged adults. New Eng J Med 328(17):1230-5, 1993.
3. Agency for Health Care Policy and Research, Polysomnography and sleep disorder centers. AHCPR Publications No. 92-0027. Health Technol Assess Rep 4:1-22, 1991.
4. Guilleminault C, Van Den Hoed J, and Mitler MM, Clinical overview of sleep apnea syndromes. In, Guilleminault C and Dement WC, eds, Sleep Apnea Syndromes, Kroc Foundation Series, Volume 11. Alan R. Liss Inc, New York, pp 1-12, 1978.
5. Kryger MH, Management of obstructive sleep apnea: Overview. In, Kryger MH, Roth T, and Dement WC, Principles and Practice of Sleep Medicine, 2nd ed. WB Saunders Co, Harcourt Brace Co, Philadelphia, 1994, pp 736-47.
6. American Thoracic Society, Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. Am J Respir Crit Care Med 150:1738-45, 1994.
7. Sullivan CE, Issa FG, et al, Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. Lancet 1:862-5, 1981.
8. Issa FG and Sullivan CE, The immediate effects of nasal continuous positive airway pressure treatment on sleep patterns in patients with obstructive sleep apnea syndrome. Electroencephologr Clin Neurophysiol 63:10-7, 1986.
9. Chesson AL, chairman, Polysomnography Task Force, American Sleep Disorders Association, Practice parameters for the indications for polysomnography and related procedures. Sleep 20 (6):406-22, 1997.
10. Iber C, O'Brien C, et al, Single night studies in obstructive apnea. Sleep 14:383-5, 1991.
11. Personal observation.
12. Sanders N, Kern N, et al, Adequacy of prescribing positive airway pressure therapy by mask of sleep apnea on the basis of a partial-night trial. Am Rev Resp Dis 147:1169-74, 1993.
13. Chesson AL, chairman, Polysomnography Task Force, American Sleep Disorders Association. The indications for polysomnography and related procedures (372 refs.) Sleep 20(6):423-87, 1997.
14. Kribbs NB, Pack AI, et al, Objective measurements of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Resp Dis 147:887-95, 1993.
15. Krieger J, Kurtz D, and Petiau C, Long-term compliance with CPAP therapy in obstructive sleep apnea patients and in snorers. Sleep 19(9):S136-43, 1996.
16. Chervin R, Theut S, and Bassetti C, Compliance with nasal CPAP can be improved by simple interventions. Sleep 20 (4):284-9, 1997.
17. Series F, Evaluation of treatment efficacy in sleep apnea hypopnea syndrome. Sleep 19(9) S71-6, 1996.
18. Kribbs NB, Pack AI, et al, Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. Am Rev Resp Dis 147:1162-8, 1993.
19. Reeves-Hauche MK, Hudgel DW, et al, Continuous versus bi-level positive airway pressure of obstructive sleep apnea. Am J Respir Crit Care Med 151:443-9, 1995.
20. Guilleminault C, Natural history, cardiac impact and long-term follow up of sleep apnea syndrome. In, Guilleminault C

and Lugaresi E, eds, Sleep/Wake Disorders: Natural History Epidemiology and Long-Term Evolution. Raven Press, New York, 1983, pp 107-26.
21. Vgontzas A, Tan T, et al, Sleep apnea and sleep disruption in obese patients. Archives Internal Medicine 154:1705-11, 1994.
22. Young T, Palta M, and Badr MS, Prevalence of sleep apnea. [letter]. New Eng J Med 329:1429-30, 1993.
23. Strobel RJ and Rosen RC, Obesity and weight loss in obstructive sleep apnea: A critical review. Sleep 19:(2)104-15, 1996.
24. Browman CP, Sampson MG, et al, Obstructive sleep apnea and body weight. Chest 85: 435-6, 1984.
25. Rodenstein D, Dooms 0, et al, Pharyngeal shape and dimensions in healthy subjects, snorers, and patients with obstructive sleep apnea. Thorax 45:722-7, 1990.
26. Mendelson WB, Garnett D, and Gillin JC, Flurazepam-induced sleep apnea syndrome in a patient with insomnia and mild sleep-related respiratory changes. J Nervous Mental Disease 160:261-4, 1982.
27. Camacho ME and Morin CM, The effect of temezepam on respiration in elderly insomniacs with mild sleep apnea. Sleep 18 (6):644-5, 1995.
28. Bonnet MH, Dexter JR, and Arand DL, The effect of triazolam on arousal and respiration in central sleep apnea patients. Sleep 13(1):31-41, 1990.
29. Guilleminault C, Clinical features and evaluation of obstructive sleep apnea. In, Kryger MH, Roth T, and Dement WC, Principles and Practice of Sleep Medicine, 2nd ed. WB Saunders Co, Harcourt Brace Co, Philadelphia, 1994, 667-77,
30. Hung J, Whitford EQ, et al, Association of sleep apnoea with myocardial infarction in men. Lancet 326:261-4, 1990.
31. Gillis AM and Flemmons WW, Cardiac arrhythmias during sleep. In, Kryger MH, Roth T, and Dement WC, Principles and Practice of Sleep Medicine, 2nd ed. WB Saunders Co, Harcourt Brace Co, Philadelphia, 1994, pp 847-60,
32. He J, Kryger MH, et al, Mortality and apnea index in obstructive sleep apnea: experience with 385 male patients. Chest 94:9-14, 1988.
33. Cartwright R, Ruzica R, et al, A comparative study of treatments for positional sleep apnea. Sleep 14:546-52, 1991.
34. Ulfberg J, and Fenton G, Effect of Breathe Right nasal strip on snoring. Rhinology June 35 (2):50-2, 1997.

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Surgical Options for Obstructive Sleep Apnea

Michael W. Marshall, DDS

ABSTRACT The pathology associated with obstructive sleep apnea is cumulative and progressive. When patients fail to improve with continuous nasal airway pressure or other, less-invasive treatments, surgery should be considered. The initial approach to the surgical patient is identification of all areas of potential obstruction. There are often several sites of obstruction, which can occur anywhere in the upper respiratory tract. One or more procedures may be needed to address these areas. The objective of surgery is to relieve these obstructing sites without interfering with the normal functionality of the upper airway.

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Obstructive sleep apnea is sleep-disordered breathing resulting from an anatomical obstruction of the upper airway. Obstructive sleep apnea should be thought of as a diffuse airway disease that has several potential sites of occlusion. Surgical correction is based on either bypassing those sites, removing the obstructing tissue, or repositioning the tissue and decreasing tissue laxity. In most patients with obstructive sleep apnea, surgery is not a primary treatment option. However, surgery is initially considered in patients with an obvious mass, tumor, or skeletal abnormality that is causing obstruction. Weight loss, nasal continuous positive airway pressure, dental airway appliances, behavioral modification, and pharmacotherapy still remain the initial treatment for patients with this problem.

Surgical correction is indicated in those patients who cannot tolerate other non-invasive treatments or for whom those treatments have been unsuccessful. One of the most effective and widely used forms of nonsurgical treatment is nasal continuous positive airway pressure. Rarely does continuous positive airway pressure fail to help patients with obstructive sleep apnea. Such failure is usually related to lack of compliance. Studies have shown that 40 percent to 50 percent of patients are unable to tolerate the device for long-term use.[1] Continued and efficacious treatment is critical in the patient with sleep-disordered breathing. There is a significant mortality and morbidity associated with obstructive sleep apnea, even for those younger than 50. It is now clear that patients with obstructive sleep apnea have an increased potential for development of cardiovascular complications such as hypertension, cardiac arrhythmias, stroke, and myocardial infarction.[2] The central nervous system is also affected by the disruption and the

lack of deeper planes of sleep critical to proper brain function. When obstruction occurs, the patient awakens or moves to lighter planes of sleep, which disrupts the brain's ability to maintain normal sleep patterns.[3] This resultant sleep fragmentation causes excessive daytime sleepiness (hypersomnolence), which is a major complication of obstructive sleep apnea. Hypersomnolence can impair alertness, cause work and driving accidents, and affect work and social functioning. The pathology associated with obstructive sleep apnea illustrates the necessity for surgical intervention following failure of less-invasive measures.

Evaluation for surgical correction consists of careful examination of the nose, soft palate, tonsils, adenoids, tongue, mandible, and larynx. Nasopharyngoscopy may be helpful in evaluating many of these areas and determining the location of airway closure.[4] Cephalometric radiographs are also routinely used and have been shown to be closely correlated with volumetric computerized tomography studies in determining sites of soft tissue obstruction.[5,6] Cephalometric radiographs are also necessary to identify mandibular deficiencies, genial deficiencies, and retropositioned hyoid bones, all of which can contribute to airway closure during sleep.7-11 These exams are done by both the otolaryngologist and oral and maxillofacial surgeon to use the expertise of both specialties since the problem is not confined to a specific site in the upper airway. Even with extensive clinical and radiographic evaluation, it is often difficult to accurately identify all areas of obstruction. This is a result of the inability to evaluate the patient while he or she is asleep. There are physiologic changes resulting in upper airway closure or narrowing that occur only during sleep.

Once a determination of the site or sites of obstruction has been made, the medical status of the patient must be thoroughly considered. Because of the significant morbidity associated with obstructive sleep apnea, patients may be at high risk for anesthetic or postoperative complications, which would influence the choice of surgical options.

In the past, the only surgical option for treatment of symptomatic obstructive sleep apnea was a tracheotomy. This procedure has a high rate of treatment success, but the associated complications and social stigma fueled the need to develop other, less-morbid surgical options. It was not until 1981 that Fujita utilized the uvulopalatopharyngoplasty for treatment of obstructive sleep apnea in adults.12 Though this treatment was very beneficial for snoring, the success rate for resolving obstructive sleep apnea using this procedure alone was poor. Powell and colleagues in 1983 described the utilization of mandibular and maxillary procedures that addressed other contributing areas of obstruction.13 Combining these procedures in the appropriate patient has lead to increasing success rates. Obstruction can occur at any point from the nose to the larynx and often does at several locations. Surgical intervention now consists of procedures that relieve obstruction in the nasal cavity, nasopharynx, oropharynx, and hypopharynx. Not only do these procedures relieve the obstruction and enlarge the upper airway, they can also help stabilize upper airway soft tissue. Magnetic resonance imaging results have shown a sleep-induced relaxation of upper airway dilator muscles that decreases the anterior-posterior diameter of the upper airway to a critical obstructive size in patients with obstructive sleep apnea.[14] This explains the need for not only enlarging the airway but also reducing tissue laxity. Once the potential sites of obstruction have been determined, the decision as to which surgical procedures to perform must be made keeping in mind the necessity for both airway enlargement and stabilization.

Surgical Options

Nasal Surgery

Nasal obstruction occurs as a result of bony or cartilaginous abnormalities or from reactive soft-tissue changes that decrease airflow and increase airway resistance. The primary goal of nasal surgery in sleep-related breathing disorders is to reduce nasal resistance. This is necessary to reduce negative intrapharyngeal pressure during inspiration. The negative pressure causes the loose pharyngeal walls to collapse, which results in obstruction of air flow.[15] Treatment usually includes septoplasty, turbinectomy, polypectomy, or a combination of these procedures. Series and colleagues have studied the effect of nasal surgery in patients with moderate obstructive sleep apnea.[16] They found a substantial improvement in apneas and sleep-stage changes after surgery in patients with normal cephalometry, but the improvement did not result in resolution of the obstructive sleep apnea. Nasal surgery alone is rarely curative for obstructive sleep apnea patients because nasal obstruction alone does not usually cause sleep apnea. Nasal surgery may also help in improving the tolerance of continuous positive airway pressure.

Tonsillectomy and Adenoidectomy

Tonsils and adenoids contribute to obstruction at the oropharyngeal and velopharyngeal levels, respectively. Tonsillectomy and adenoidectomy alone are usually done only in children with severe snoring with or without a diagnosis of obstructive sleep apnea. Suen and colleagues reported an 85 percent normalization of the respiratory disturbance index after adenotonsillectomy in children with obstructive sleep apnea.[17,18] Thus, this procedure alone may be effective in treating children with obstructive sleep apnea. Respiratory disturbance index or other sleep study parameters alone should not be used to determine resolution or improvement in obstructive sleep apnea, however. Subjective and objective clinical improvement is also needed when assessing the benefits of treatment.

In adults, there is a natural recession of adenotonsillar lymphoid tissue, and as such it is usually not a major contributing factor in obstruction leading to sleep

apnea. It is very common for residual tonsil tissue to be removed at the time of uvulopalatopharyngoplasty to help decrease airway resistance and increase pharyngeal volume.

Uvulopalatopharyngoplasty

Uvulopalatopharyngoplasty was the first surgical procedure specifically utilized to address the palatal abnormalities seen in many patients with obstructive sleep apnea. Uvulopalatopharyngoplasty was originally developed for the treatment of snoring, and because it was noted that snoring is a major component of obstructive sleep apnea, it was thought that alteration of the palate may also influence obstructive sleep apnea. The procedure itself consists of removal of a portion of the glossopalatal arch and soft palate, including the uvula and tonsils, if present (Figure 1).

Figure 1. Uvulopalatopharyngoplasty with removal of soft palate, uvula and lateral pharyngeal tissue.

This is done in hopes of correcting retropalatal collapse and/or narrowing. This procedure has a low morbidity rate but is not without significant complications. Nasal regurgitation, nasal reflux, velopharyngeal stenosis, velopharyngeal incompetence, pharyngeal dryness, speech abnormalities, dysphagia, loss of taste, hemorrhage and postoperative breathing difficulty have all been described.[19] Though Uvulopalatopharyngoplasty is very effective for curing snoring, multiple studies have shown an average of only 50 percent success rate with respect to overall improvement of obstructive sleep apnea.[20,21] The poor success rate appears to be mainly a result of not addressing other concomitant areas of obstruction when considering surgical correction. It is now known, based on more thorough clinical exams and more specific studies such as fast computerized tomography cine, that in many patients not only is the velopharynx a major site of obstruction but so is the hypopharynx. Another reason for the consistent lack of improvement is the variability in which the procedure is performed. Due to the significant potential for development of velopharyngeal incompetence and its associated problems, a conservative removal of soft tissue is often done. It is not uncommon to need further resection if symptoms continue and evidence of velopharyngeal obstruction is still present.

Laser-Assisted Uvulopalatopharyngoplasty

Laser-assisted uvulopalatopharyngoplasty has been utilized as a treatment for both snoring and obstructive sleep apnea.22 The procedure is performed by placing vertical laser incisions lateral to the uvula bilaterally. This produces vertical and fibrotic "trenches" beside the root of the uvula. Further debulking of the bottom and lateral sides of the uvula is then done thereby creating a higher and smaller uvula. This is done during the course of two to five outpatient procedures in which 5 to 8 mm of velum is removed each time to theoretically elevate the new velum to Passavant's ridge.[23] The benefits of this procedure are less morbidity and no need for hospitalization. Laser-assisted uvulopalatopharyngoplasty has been very beneficial for the treatment of snoring, but its efficacy in the correction of obstructive sleep apnea is still controversial. A recent study has shown it to be as effective as uvulopalatopharyngoplasty, though the rate of cure of obstructive sleep apnea was still only 44.7 percent.[24] The authors do not recommend laser-assisted uvulopalatopharyngoplasty in patients with oxygen desaturations of less than 85 percent or a respiratory disturbance index of greater than 20 unless continuous positive airway pressure is also used during the treatment process. For those patients with mild obstructive sleep apnea and/or obstruction only at the velopharynx, this may be a good alternative to uvulopalatopharyngoplasty.

Tongue Surgery

Surgical resection of portions of the tongue has been developed to help enlarge the hypopharyngeal airway space. Though this enlargement can also be accomplished with genial advancement, hyoid suspension, and maxillomandibular advancement, patients with an abnormally large tongue or tongue base may benefit from these reduction procedures. Resection can involve portions of the lateral and/or dorsum of the tongue. For those patients with persistent and severe sleep apnea with evidence of narrowing of the hypopharynx by the tongue base, a midline glossectomy with posterior tongue reduction has also been tried. These procedures have met with variable results and are not without significant complications. Bleeding, dysphagia, and moderate pain on swallowing are common and may persist for two to three weeks. The midline glossectomy has also been combined with an epiglottidectomy in those cases where the epiglottis is contributing to the obstruction.25 Patients treated with this combined procedure run the added risk of chronic aspiration of matter into the trachea. It appears that tongue reduction, particularly posterior resection, should be utilized only after all other modalities of anterior tongue repositioning have failed.

Genial Advancement and Hyoid Suspension

Obstruction at the hypopharyngeal region can be a result of posterior positioning of the tongue; excessive tongue laxity, in which the tongue falls back during sleep and occludes the airway; or abnormal size as described above. If the tongue size is within normal limits and there

Figure 2a. Prior to genial advancement.

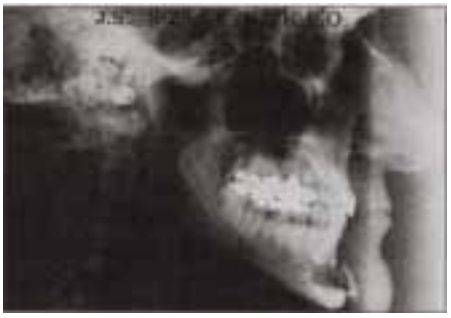

Figure 2b. After genial advancement, the lingual cortex and attached genioglossus and geniohyoid muscles repositioned anteriorly and fixated with titanium screw. Buccal cortex has been removed on the advancing segment. Note the lack of significant soft tissue alteration of the chin.

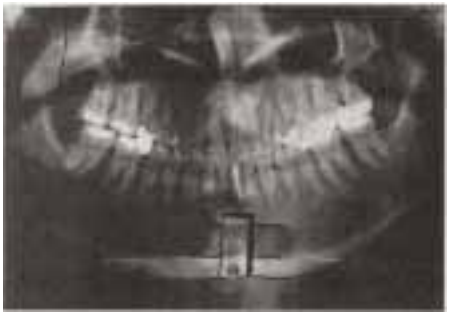

Figure 2c. Panorex after genial advancement.

is evidence of hypopharyngeal obstruction, treatment is directed at opening the posterior airway space and stabilizing the ligamentous and muscular attachments of the tongue. This is done by advancing the attachments of the tongue at the genial tubercles and the hyoid bone.[3,26] The genial advancement procedure consists of a rectangular bicortical osteotomy, which is performed below the apices of the mandibular anterior teeth and does not include the inferior border of the mandible. This creates a free bony segment that includes the genial tubercles and the attached genioglossus and geniohyoid muscles. This is then advanced the full width of the mandible so that the lingual cortex is anterior to the buccal cortex. The rectangular bony segment with muscles attached is then rotated 90 degrees to maintain this anterior position. The buccal cortex of the advanced segment is then removed and the lingual cortex stabilized using a titanium screw (Figures 2a through c). This can result in from 10 to 15 mm of advancement, depending on the thickness of the mandible. This reduces the potential for posterior tongue displacement. The osteotomy can be varied and include the inferior border if lower facial augmentation is needed for esthetics. Genial advancement is often done in conjunction with the hyoid suspension but may also be done alone. On cephalometric exam, if the posterior airway space is within normal limits but there is evidence of hypopharyngeal narrowing due to tongue displacement, the genial advancement alone may correct the obstruction. The most significant complication with this procedure is the potential for mandibular fracture, though leaving the inferior border intact has significantly reduced this problem. Patients will often have a transient paresthesia of the lower incisors. Speech and swallowing are not affected.

The hyoid suspension more directly addresses the posterior positioning of the tongue and also helps in reducing tongue laxity. The procedure consists of releasing the infrahyoid muscles (omohyoid, sternohyoid, thyrohyoid), then advancing the hyoid both anteriorly and superiorly by suspending it from the inferior border of the mandible.[13,26,28] This stabilizes the base of the tongue in a more anterior position and enlarges the pharyngeal space (Figure 3). A more recent modification of this procedure is to suture the hyoid, after infrahyoid release, to the thyroid cartilage.[29] Complications include dysphagia, which is usually transient; injury to the superior

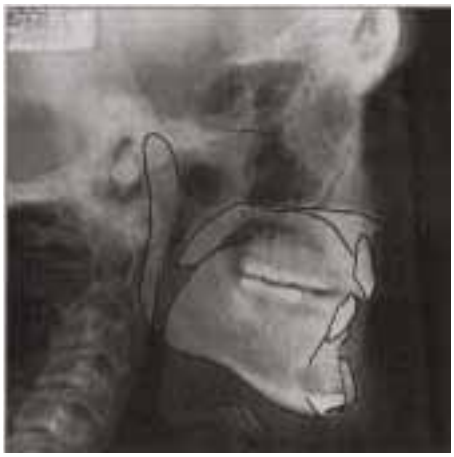

Figure 3. Pre and post-operative position of hyoid bone after suspension from mandibular symphysis.

laryngeal nerve; and possible aspiration if the thyrohyoid membrane is violated. Even when the genial advancement is combined with the hyoid suspension, speech and swallowing are usually not affected after the initial edema has resolved.

Maxillofacial Surgery

Maxillary and mandibular advancement procedures are commonly done for correction of dentofacial deformities, but these procedures are also therapeutic for the treatment of obstructive sleep apnea. In the patient with a dentofacial deformity and subsequent sleep-disordered breathing, it is clear that correction of the anatomic abnormality should be addressed initially. In the normocephalic patient, maxillomandibular advancement procedures are usually considered only after failure of other surgical procedures that attempt to more directly alter the specific area of obstruction as described above. In these refractory patients, maxillomandibular advancement procedures have been shown to have a high rate of success.[30,31,34] Advancement of the facial skeleton has the benefit of expanding the pharyngeal airway space both anteriorly and laterally since all muscle attachments of the tongue and soft palate are maintained. This provides a significant volumetric increase in pharyngeal space without disturbing the functions of speech or swallowing (Figures 4a and b).

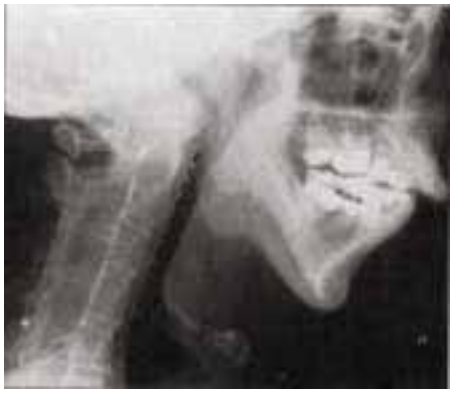

Figure 4a. Narrowed posterior airway space and hypopharyngeal obstruction due to mandibular retrognathia and associated posterior tongue position.

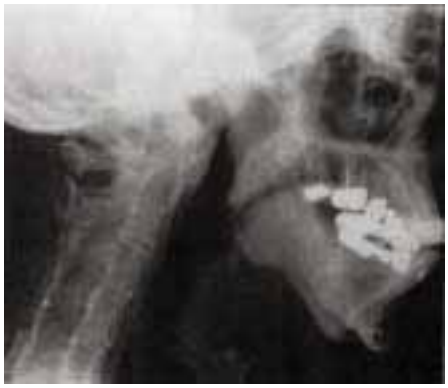

Figure 4b. Enlargement of posterior airway space with mandibular and genial advancements.

Figure 5a. Preoperative photo.

Figure 5c. Postoperative photo.

Figure 5b. Preoperative cephalometric X-ray

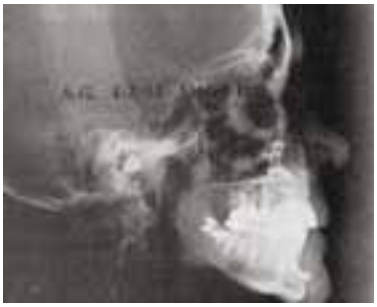

Figure 5d. Postoperative cephalometric X-ray after 6 mm maxillary and mandibular advancement in a patient with a pre-existing Class I occlusion. Note the increase in posterior airway space and acceptable esthetics.

Figure 6. Tracheotomy placed solely for the treatment of obstructive sleep apnea.

The most difficult part of treating this type of patient is preserving a functional occlusion and maintaining acceptable esthetics. If time permits, orthodontic treatment of the Class I patient would include mandibular premolar extraction with retraction of lower anteriors.

If orthodontics is not an option, surgical dentoalveolar retraction can be done as a separate pretreatment procedure. In many patients, however, maxillomandibular advancement of 5 to 8 mm can be accomplished without significant esthetic alteration (Figures 5a through d). Commonly used osteotomy procedures, such as the high Le Fort osteotomy, bilateral sagittal-split osteotomy, and the use of rigid internal fixation have significantly improved the ability to advance the mandible and maxilla with predictable stability.[30,31]

Tracheotomy

Most practitioners agree that a tracheotomy results in 100 percent cure for patients with obstructive sleep apnea (Figure 6).

It has been shown that a tracheotomy results in normalization of apneic indices and resolution of cardiac arrhythmias.[32] It is understandable that if the entire upper airway is bypassed, there is no potential for obstruction. For most patients, however, a tracheotomy is not an acceptable treatment. In patients with severe obstructive sleep apnea and resultant cardiac compromise, a tracheotomy may be the only surgical option. Clinical urgency would dictate the necessity for a tracheotomy rather than other, less-morbid treatment alternatives. Not only can a tracheotomy result in physical complications such as recurrent bronchitis, bleeding, and stenosis, but there are also significant psychosocial problems.[32] Depression, spousal rejection, and problems with work may occur, thus this procedure is reserved for only the most refractory cases. Depending on the severity of the case, a tracheotomy may also be performed as a temporary procedure until it is clear that the airway is unobstructed from swelling as a result of other upper airway procedures.

Discussion

The determination as to which surgical procedure(s) to perform in the obstructive sleep apnea patient is more complex than merely a consideration of polysomnographic (sleep study) results. These results must be weighed along with the current symptomatology of the patient, naso-

pharyngoscopy results, a clinical exam, a cephalometric evaluation, and the current health status of the patient to determine the most appropriate and potentially successful surgery. Certainly, if anatomic abnormalities exist, such as retrognathia, micrognathia, or microgenia, then the focus of treatment should be directed in these areas. But several obstruction sites can contribute to obstructive sleep apnea, and in the majority of patients, both the soft palate and tongue are obstructing. This helps explain the initial poor overall improvement with uvulopalatopharyngoplasty alone. At one time, it was the only recommended procedure for the treatment of obstructive sleep apnea, but it did not address possible hypopharyngeal obstruction. Treatment failures who went on to have genial advancements and hyoid suspensions showed significant improvements. Now that more information is becoming available as to the contributing areas of pathology, more careful evaluation of the upper airway is done thus improving treatment success by addressing all areas of obstruction. Unfortunately, surgical treatment is still in its infancy in regard to these patients. Even with careful evaluation and improved techniques for site-specific treatment of possible areas of obstruction, surgical results are still not highly predictable. It is with this in mind that patients are informed of the potential need for multiple, staged procedures. Practitioners must always weigh the potential morbidity and mortality of the procedures with the potential for improvement.

In the patient with simple snoring or mild to moderate obstructive sleep apnea without evidence of hypopharyngeal obstruction, the surgical options are more clear. Laser-assisted uvulopalatopharyngoplasty or uvulopalatopharyngoplasty are the treatments of choice. The laser-assisted uvulopalatopharyngoplasty is a less morbid procedure and can be done in an outpatient setting, but its efficacy in treating obstructive sleep apnea is still under study. The amount of pharyngeal tissue removed is much more limited than in the uvulopalatopharyngoplasty, and as such may not entirely address pharyngeal narrowing leading to obstruction. If the patient is significantly symptomatic, then consideration should be given for the more aggressive uvulopalatopharyngoplasty.

Figure 7a. Preoperative photo.

Figure 7b. Preoperative cephalometric X-ray of patient with rheumatoid arthritis and associated TMJ involvement resulting in mandibular displacement and hypopharyngeal obstruction.

Figure 7c. Postoperative photo. Patient underwent maxillary advancement and impaction, mandibular advancement and genial advancement.

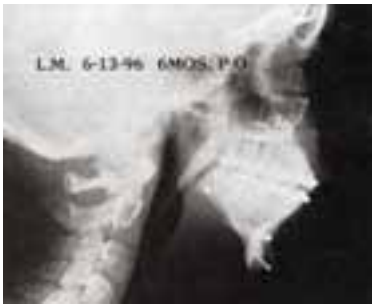

Figure 7d. Postoperative cephalometric X-ray.

If the posterior airway space is not abnormal but tongue laxity or size indicates a potential for obstruction, then genial advancement can be utilized. With the genial attachments brought from lingual to buccal, the muscle and ligaments are tensed the thickness of the symphysis and thus will decrease the distance the tongue can fall posteriorly during sleep. If there is no significant improvement and there is evidence of hypopharyngeal obstruction continues, the hyoid suspension can be done.

If hypopharyngeal obstruction exists, then genial advancement and hyoid suspension would be the treatments of choice. When significant posterior airway space narrowing is present, then both genial advancement and hyoid suspension give the best potential for success since these two procedures anteriorly reposition the attachments of the tongue, thus moving the base of the tongue forward, and decrease tongue laxity. Using both these procedures, the hyoid can be advanced as much as 20 to 30 mm as measured on the cephalometric X-ray. Unfortunately, the amount of anterior tongue advancement is not proportional to the amount of bony advancement. Relapse does occur but typically not back to baseline. If problems still persist, then maxillofacial surgery is considered.

When a skeletofacial abnormality is present in the patient with obstructive sleep apnea, treatment should initially be directed at correcting the abnormal anatomy. This may be a developmental

abnormality, such as the patient with Pierre-Robin syndrome and resultant retrognathia, or may be a result of systemic disease, such as rheumatoid arthritis with associated condylar resorption causing posterior mandibular repositioning and tongue displacement (Figures 7a through d).

The use of mandibular and/or maxillary advancement procedures in these patients is a primary treatment option. Currently, the Class I occlusion patient with significant, symptomatic obstructive sleep apnea that has failed other surgical procedures is now considered for these same procedures. It has been the experience of the author and others that patients, refractory to other forms of surgical intervention, have highly successful and predictable improvements utilizing maxillofacial advancement procedures.[13,30,31,33,34] These procedures have also been considered as primary surgical treatments. Waite and colleagues reported on a series of patients in which maxillomandibular advancement surgery was the primary surgical option.[34] Ninety-six percent of patients were subjectively and objectively improved with desaturations of less than 90 percent greatly decreased. Sixty-five percent of patients achieved a reduction in the respiratory disturbance index to a level of less than 10. These results were based on repeat polysomnograms obtained at one week and six weeks. It is typically recommended that follow-up polysomnograms be obtained at six months after surgery to assure full resolution of swelling and stabilization of healed tissues. It should be stressed, however, that the polysomnogram is not the only determinant of surgical success. Patients may have an improvement in their polysomnographic parameters to near normal levels but still continue to experience daytime sleepiness or other problems related to obstructive sleep apnea. If the patient is still symptomatic and there is evidence of continued hypopharyngeal obstruction, then tongue reduction surgery may be an option.

When all else has failed or the patient's risks from surgery are too great, a tracheotomy should be considered. This is the one procedure with a 100 percent cure rate for obstructive sleep apnea. This procedure should be reserved for only the most extreme cases because of its significant associated morbidity.

Surgical candidates present with both diagnostic and surgical challenges. To effectively treat these patients, more than one procedure may be needed simultaneously. The amount of tissue reduction or anatomic advancement has to be balanced with maintaining the other functions of the pharynx, such as speech and swallowing. The severity of the existing disease and the patient's current health status may exclude staging of surgeries. These factors, along with the variable success rates associated with the current surgical procedures, make the permanent correction of obstructive sleep apnea a continued effort.

References

1. Dickens QS, Jenkins NA, et al, Long-term nasal CPAP use. Sleep Res 18:233, 1989.
2. Bowman CP, Newman JP, and Winslow DH, Nasal CPAP therapy for obstructive sleep apnea syndrome: determinants of long-term compliance. Sleep Res 18:206, 1989.
3. Riley RW, Powell N, and Guilleminault C, Current surgical concepts for treating obstructive sleep apnea syndrome. J Oral Maxillofac Surg 45:149-57, 1987.
4. Rojewski TK, Clarke RW, et al, Video-endoscopic determination of the mechanisms of obstruction in obstructive sleep apnea. Otolaryngol Head Neck Surg 92:127, 1984.
5. Hudgel DW, The treatment of obstructive sleep apnea. Chest 109:1346-58, 1996.
6. Riley R, Powell N, and Guilleminault C, Cephalometric roentgenograms and computerized tomographic scans in obstructive sleep apnea. Sleep 9:514-5, 1986.
7. DeBerry-Borowiecki B, Kukwa A, and Blanks RH, Cephalometric analysis for diagnosis and treatment of obstructive sleep apnea. Laryngoscope 98:226-34, 1988.
8. Riley R, Guilleminault C, et al, Cepahlometric analyses and flow-volume loops in obstructive sleep apnea patients. Sleep 6(4):303-11, 1983.
9. Hochban W and Brandenburg U, Morphology of the viscerocranium in obstructive sleep apnoea syndrome-cephalometric evaluation of 400 patients. J Cranio Maxillo Fac Surg 22:205-13, 1994.
10. Strelsow V, Blanks R, et al, Cephalometric airway analysis in obstructive sleep apnea syndrome. Laryngoscope 98:1149-58, 1988.
11. Bacon W, Turiot JC, et al, Cephalometric evaluation of pharyngeal obstructive factors in patients with sleep syndrome. Angle Orthod 60(2):115-22, 1989.
12. Fujita S, Conway W, et al, Surgical correction of anatomic abnormalities of obstructive sleep apnea syndrome; uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg 89:923, 1981.
13. Powell N, Guilleminault C, et al, Mandibular advancement and obstructive sleep apnea syndrome. Bull Eur Physiopathol Respir 19:607, 1983.
14. Collard P, Rombaux P, and Rodenstein DO, Why should we enlarge the pharynx in obstructive sleep apnea? Sleep 19(9) S85-7, 1996.
15. Kales A, Vela-Bueno A, and Kales J, Sleep disorders: sleep apnea and narcolepsy. Annals Internal Medicine 106:434-43, 1987.
16. Series F, St. Pierre S, Carrier G, Effects of surgical correction of nasal obstruction in the treatment of obstructive sleep apnea. Am Rev Respir Dis 146:1261-5, 1992.
17. Sher A, Schechtman KB, and Piccirillo J, The efficacy of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. Sleep 19(2):156-77, 1996.
18. Suen JS, Arnold JE, and Brooks LJ, Adenotonsillectomy for treatment of obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg 121:525-30, 1995.
19. Haavitso L and Suonpaa J, Complications of uvulopalatopharyngoplasty. Clin Otolaryngol 19:243-7, 1994.
20. Janson C, Gislason T, et al, Long-term follow-up of patients with obstructive sleep apnea treated with uvulopalatopharyngoplasty. Arch Otolaryngol Head Neck Surg 123:257-62, 1997.
21. Pepin J, Veale D, et al, Critical analysis of the results of surgery in the treatment of snoring, upper airway resistance syndrome (UARS), and obstructive sleep apnea (OSA). Sleep 19(9):S90-100, 1996.
22. Utley DS, Shin EJ, et al, A cost-effective and rational surgical approach to patients with snoring, upper airway resistance syndrome, or obstructive sleep apnea syndrome. Laryngoscope 107:726-34, 1997.
23. Kamami Y, Outpatient treatment of sleep apnea syndrome with CO2 laser, LAUP: Laser-assisted UPPP results on 46 patients. J Clin Laser Med Surg 12(4):215-19, 1994.
24. Walker RP, Grigg-Damberger MM, and Gopalsami C, Uvulopalatopharyngoplasty versus laser-assisted uvulopalatopharyngoplasty for the treatment of obstructive sleep apnea. Laryngoscope 107:76-82, 1997.
25. Mickelson SA and Rosenthal L, Midline glossectomy and epiglottidectomy for obstructive sleep apnea syndrome. Laryngoscope 107:614-9, 1997.
26. Johnson NT and Chinn J, Uvulopalatopharyngoplasty and inferior sagittal mandibular osteotomy with genioglossus advancement for treatment of obstructive sleep apnea. Chest 105:278-83, 1994.
27. Ramirez SG and Loube DI, Inferior sagittal osteotomy with hyoid bone suspension for obese patients with sleep apnea. Arch Otolaryngol Head Neck Surg 122(9):953-7, 1996.
28. Riley RW, Powell NB, and Guilleminault C, Obstructive sleep apnea syndrome: A surgical protocol for dynamic upper airway reconstruction. J Oral Maxillofac Surg 51:742-7, 1993.
29. Riley RW, Powell NB, and Guilleminault C, Obstructive sleep apnea and the hyoid: a revised surgical procedure. Otolaryngol Head Neck Surg 111:717-21, 1994.
30. Nimkarn Y, Miles PG, and Waite PD, Maxillomandibular advancement surgery in obstructive sleep apnea syndrome patients: long-term surgical stability. J Oral Maxillofac Surg 53:1414-8, 1995.
31. Standards of Practice Committee, American Sleep Disorders Association, Practice parameters for the treatment of obstructive sleep apnea in adults: The efficacy of surgical modifications of the upper airway. Sleep 19(2):152-5, 1996.
32. Riley RW, Powell NB, and Guilleminault C, Maxillary, mandibular, and hyoid advancement for treatment of

obstructive sleep apnea: a review of 40 patients. J Oral Maxillofac Surg 48:20-6, 1990.
33. Metes A, Direnfeld V, et al, Resolution of obstructive sleep apnea following facial surgery. J Otolaryng 20(5):342-4, 1991.
34. Waite PD, Wooten V, et al, Maxillomandibular advancement surgery in 23 patients with obstructive sleep apnea syndrome. J Oral Maxillofac Surg 47:1256-61, 1089.

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Dentistry's Role in the Recognition and Treatment of Sleep-Breathing Disorders: The Need for Cooperation with the Medical Community

Laurence I. Barsh, DMD

ABSTRACT While oral appliance therapy for the treatment of sleep-disordered breathing can be an exciting and rewarding adjunct to the practice of dentistry, it is essential that dentists realize that snoring and obstructive sleep apnea are medical and not dental problems. Sleep-disordered breathing and its sequelae are diseases that should remain in the purview of the medical community. While the dentist can identify patients with sleep-breathing disorders and participate in their treatment, it is essential to emphasize that sleep-breathing disorders are potentially life-threatening diseases whose diagnosis and treatment are the domain of the medical profession. Accepting dentistry's position as part of a treatment team, ongoing review of scientific literature, cooperation with medical colleagues, and attendance at educational meetings dedicated to the study of sleep-related disorders are essential to proper and ethical dental participation in the treatment of sleep-disordered breathing.

AUTHOR

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The Washington Post, March 5, 1997, ran a story by Leef Smith. The article read, in part:

"A Man Who Fell Asleep at Wheel Charged in Deaths of Two in Va."

"A Fredericksburg, Va., man who told police that he fell asleep at the wheel of his car has been charged with hit and run and two counts of involuntary manslaughter for his role in a crash that killed two motorists in Prince William County."

A follow-up story described the sentencing as follows:

"Drowsy Driver Gets Five-Year Sentence"

"Judge Sends Message After Reckless Act Killed Two in Pr. William"

The judge said he was exceeding state sentencing guidelines, which recommend a maximum term of six months, because of the nature of the crime and to send a message.

"If you're tired and falling asleep as you drive, you need to get off the road, the judge said. For drivers who may consider getting on the road when they're drowsy, he said, "the lesson from this case is you go to jail."

TABLE 1.
The Muscles of the Pharynx and Their Actions

Muscle	Action
Digastricus	Elevates hyoid, depresses mandible
Genioglossus	Protrudes tongue (inf. fibers), depresses tongue (mid. fibers)
Geniohyoid	Elevates hyoid, depresses mandible
Levator veli palatini	Elevates soft palate
Musculus uvulae	Shortens the uvula
Palatoglossus	Elevates and retracts the tongue
Palatopharyngeus	Elevates larynx
Salpingopharyngeus	Elevates larynx
Styloglossus	Retracts and elevates tongue
Stylohyoid	Elevates and retracts hyoid
Stylopharyngeus	Elevates larynx
Tenor veli palatini	Opens auditory tube, tenses soft palate

Subsequent interviews with the driver's defense attorneys, the prosecutor, and the driver's wife revealed the following. Despite the fact that the driver had complained of fragmented sleep and insomnia to his primary care physician, the physician failed to diagnose obstructive sleep apnea because the patient was of average height and weight. After the accident, the driver's attorney recruited a pulmonologist who prescribed an overnight sleep study, which disclosed severe obstructive sleep apnea.

Of the general adult population in middle age, 93 percent of women and 82 percent of men with moderate to severe sleep apnea syndrome have not been clinically diagnosed. Sleep apnea is even less likely to be diagnosed in the older population.1 Unrecognized sleep-disordered breathing is linked to motor vehicle accident occurrence in the general population and may account for a significant proportion of motor vehicle accidents.[2]

The question then arises: Who is responsible for recognition and diagnosis of obstructive sleep apnea?

A posting was made recently to an international sleep discussion forum on the Internet by a physician asked to act as an expert witness in several malpractice cases, that read, in part:

"I am interested in experiences of other sleep physicians interacting with the legal community. I have reviewed three medical cases wherein lack of recognition of sleep apnea by the attending physician allegedly led to brain damage or death, and have been asked to state whether that lack of diagnosis constituted a breach of the standard of care in each case."

Snoring, a common malady that affects people of all ages but especially middle-aged men and elderly men and women who are overweight[3,4] has been identified as a risk factor for hypertension, ischemic heart disease and stroke.[5] Although not all people who snore have obstructive sleep apnea, snoring is a cardinal symptom of obstructive sleep apnea and, thus, may be associated with increased morbidity and mortality. Furthermore, snoring in some patients without apnea has been associated with significant sleep disturbance and excessive sleepiness. This condition has been referred to as upper airway resistance syndrome and is characterized by repeated nocturnal arousals without recognizable hypopneas or apneas.[6]

Is the dentist medically qualified and legally capable of making the diagnosis of obstructive sleep apnea and upper airway resistance syndrome and differentiating between primary snoring and these conditions?

Because snoring is so common, both the medical profession and the public have regarded it as merely an annoyance to one's bed partner or as a source of humor. With the realization that an oral appliance could stop snoring, the natural assumption by the dental profession was, therefore, that treatment of snoring could be incorporated into the dental practice simply and easily. Because snoring was considered so benign, the sequela to this assumption was that a physician did not have to be involved in the diagnosis of the problem and that the dentist could exclude obstructive sleep apnea from the diagnosis of primary snoring. Recently, however, snoring has become considered as prima facie evidence of an upper airway obstruction,7 a risk factor for cardiovascular disease,7-11 and a recognized precursor of obstructive sleep apnea.[12]

Because of these factors, the assumption that snoring is innocuous has been proven false, and snoring is now considered one stage of a complex continuum of disease states culminating in obstructive sleep apnea. Understanding the complicated pathophysiology of obstructive sleep apnea is essential to proper treatment and underscores the necessity of dentist-physician interaction. David Rapoport of the Department of Medicine, New York University School of Medicine, stated:

"Upper airway obstruction in OSAS [obstructive sleep apnea syndrome] results from interaction between subtle anatomic airway narrowing, increased wall collapsibility due to sleep-induced loss of baseline muscle tone, and insufficient inspiratory phasic dilator muscle contraction to oppose the negative intraluminal pressure resulting from diaphragmatic contraction."[13]

The signs and symptoms of obstructive sleep apnea and the distinction between obstructive sleep apnea and simple snoring may appear to be easily recognizable, thus tempting the dentist to treat

without physician involvement. However, the pathophysiology of both snoring and obstructive sleep apnea is infinitely more complex and less well understood. Because of this, the sequelae of improperly or inadequately treated obstructive sleep apnea syndrome can have an adverse effect on the general health of the patient.

There may be several causal factors contributing to the collapse of the airway. Potentially pathogenic anatomic configurations may include overdevelopment of the soft palate and/or tongue, constriction of the lumen (especially in the buccopharyngeal region), and poor compliance of the pharyngeal walls to distension and rebound during the respiratory cycle. A further possible contributory factor is the existence of localized regions of turbulent or near-turbulent airflow, which could either restrict flow directly or alter the normal patterns of air pressure within the lumen and contribute to collapse.

"Balance of Pressures" Concept

According to the "balance of pressures concept" discussed by Isono and Remmers:[14]

"The size of the pharyngeal lumen depends on the balance between outward forces developed by actively contracting muscles and inward forces resulting from subatmospheric luminal pressure during inspiration."

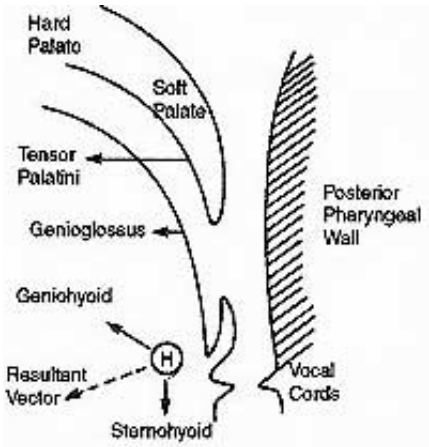

Figure 1. A depiction of the action of various muscles on pharyngeal structures. The tensor palatini moves the soft palate ventrally. The genioglossus acts to displace the tongue ventrally. The geniohyoid and sternohyoid act on the hyoid bone (H) to move it.[14]

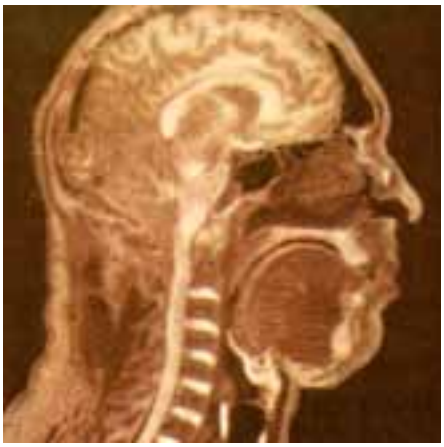

Figure 2. Sagittal view. Visual Human Project. The posterior border of the tongue makes upper the anterior wall of the pharynx. This portion of the upper airway is collapsible. An enlarged tongue also forces the soft palate and uvula toward the posterior wall of the pharynx and further obstructs the upper airway.

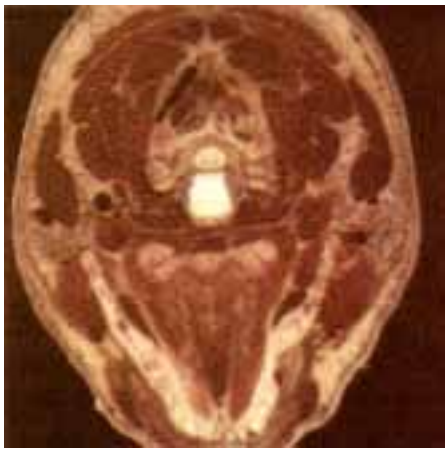

Figure 3. Coronal View. Visible Human Project. This view is taken at the floor of the mouth. Clearly visible are the lingual tonsils and the airway.

Neuromuscular Factors

Control of the size of the upper airway and stiffness of the walls depends on the relative contraction of a host of paired muscles known as the pharyngeal dilator muscles (Table 1). Contraction of these muscles promotes ventral movement of the soft palate, mandible, tongue, and hyoid bone as shown in Figure 1. It is generally accepted that inspiratory motor output to the muscles of the pharynx and related structures stiffens the pharynx and enlarges its lumen. The activity of the pharyngeal muscles is highly dependent upon a variety of factors within the central nervous system, particularly on the brain stem level. Wakefulness conveys a supervisory function that ensures airway patency. Sleep onset and pharmacologic agents that depress the brain stem respiratory network interact with the dilating effects produced by the musculature thus implicating the nervous system as a secondary contributor to the development of obstructive sleep apnea and hypopnea.[14]

Anatomic Factors

The size and configuration of the upper airway plays a major role in determining whether the patient will have obstructive sleep apnea. Gain or loss of fat in the pharyngeal and/or related areas like the tongue can lead to a change in size or configuration of the passive pharyngeal airway. According to Isono and Remmers,[14] anatomic and structural abnormalities compromise the size of the pharyngeal airway in patients with obstructive sleep apnea (Figures 2 and 3).

Mechanical Factors Influencing Upper Airway Closure

Mechanical factors that influence upper airway closure depend upon complex mathematical formulae beyond the scope of this discussion. The comments that follow are included to paint a complete picture of the complexity of the situation and intended to illustrate that multiple theories and multiple causative factors are involved when discussing why a pharynx collapses and why obstructive apnea occurs. For a more complete discussion, the reader is referred to Isono and Remmers' chapter in *Principles and Practice of Sleep Medicine*[14] and the numerous references provided.

- Compliance. The compliance of the pharynx is defined as the ratio of change in the cross-sectional area of the pharynx to change in the transmural

pressure (P_{tm}), which is the difference between the luminal and tissue pressure ($P_{tm} = P_l - P_{ti}$). Using this definition, it has been demonstrated that multiple collapsible segments exist in the passive pharynx of patients with obstructive sleep apnea and that the nasopharynx is more compliant than the oropharynx and hypopharynx in many of these patients.

- Surface adhesive forces. Surface adhesive forces between opposed luminal surfaces may contribute to airway closure. These same forces may also make re-establishment of a patent pharynx more difficult. As the pharyngeal airway narrows, the mucous film lining the airway thickens and further narrows the airway.
- Pharyngeal luminal pressure. Two types of physical phenomena cause a reduction in the luminal pressure as gas flows through a tube. These are loss of energy by work done in overcoming flow resistive aspects of the upper airway and conversion of energy from static to kinetic caused by increase in the velocity of airflow when lumen size decreases.
- Effects of geometry of the pharynx on pressure and flow. This discussion adopts a fluid mechanical approach to the airflow in the pharynx. This allows one, according to Isono and Remmers, to infer that the true driving force for fluid flow between two points is not the difference in pressure but the difference in total fluid energy. This fluid mechanical approach could account for the mechanisms of pharyngeal narrowing extending downward during inspiration.
- Site and patterns of pharyngeal narrowing. Using endoscopic methods of evaluation, Remmers has determined that there are primary and secondary sites of pharyngeal narrowing, and 75 percent of patients had more than one site of narrowing. The soft palate is the most common site of narrowing in the pharynx of patients with obstructive sleep apnea. A posterior movement of the tongue and uvula causes narrowing of the oropharynx. Primary narrowing of the hypopharynx is rare in obstructive sleep apnea patients.[14]

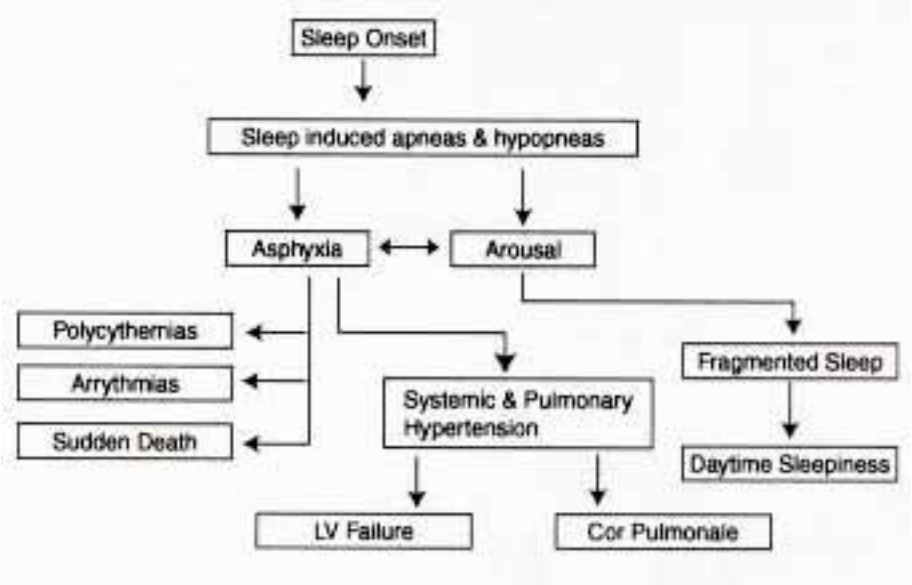

Figure 4. Sequelae of sleep disordered breathing include systemic and pulmonary hypertension, polycythemia, cardiac arrhythmias, fragmented sleep, and the possibility of sudden death.[35]

Study of the pathophysiology of snoring and obstructive sleep apnea is not included in the dental curriculum of any accredited dental school as yet, nor is the subject of sleep disorders and the diagnosis and treatment of sleep-disordered breathing included in the dental practice act of any state. Despite dentistry's ability to offer a treatment modality – oral appliance therapy – that often provides the patient with a necessary solution to his or her medical problem, the diagnosis and prescription for that treatment still remains the prerogative of the medical community.

The sequelae of undiagnosed and incompletely treated sleep apnea are medical in nature and are disease states with which the dentist is unprepared to cope. In addition to an increased rate of motor vehicle accidents among the sleep apneic population,15 systemic hypertension has been reported in up to 50 percent of patients with sleep apnea.16 In fact, mean morning blood pressure has been shown to increase almost linearly with an increasing severity of obstructive sleep apnea in both obese and non-obese patients.[17]

Bradycardic arrhythmias have been associated with sleep apnea and ventricular tachycardia has been noted with severe hypoxemia.18 Myocardial ischemia has been noted in patients with underlying coronary artery disease. The infarction may be triggered by the hypoxemia and the concurrent bradycardia and hypertension.19 There is also evidence that there is an increased risk of stroke in patients who snore, have a history of observed apneas, or present with daytime sleepiness.20 A summary of the sequelae of sleep-disordered breathing is shown in Figure 4.

The fact that the diagnosis and treatment of sleep apnea is beyond the licensure limitations of dentistry has prompted a dental malpractice insurance carrier to make the following ruling:

"Dentists providing sleep apnea therapy to patients (including Snore Guard therapy) are required by [the] insurance underwriter to ascertain that the patient has seen a physician about the problem and that the physician

has determined that a dental appliance is the proper course of action."

As an alternative, the underwriter has requested that any dentist working with patients without physician referral should present the protocol followed to the underwriter for approval. (Personal communication – CNA Insurance – 1998).

"A simple, cost-effective screening tool for obstructive sleep apnea is a goal that many investigators are seeking but have not as yet achieved."

"Several features of obstructive sleep apnea suggest that it may be an appropriate disease for screening programs for general populations and more specific high-risk groups. Preliminary data suggest that obstructive sleep apnea represents an important health problem in terms of high prevalence, increased levels of morbidity and mortality, and increased public safety risk. Furthermore, the chronicity of the disease and the relatively low levels of recognition of the disorder in the medical community suggest a potential for lead-time gains for screening programs. Specific groups that might be considered for screening programs include commercial vehicle operators, hazardous duty personnel, and certain groups of medical patients. The purpose of this clinical commentary is to consider the issues of population and specific group screening for obstructive sleep apnea by reviewing the general principles of screening for chronic disease and then applying these principles specifically in the case of obstructive sleep apnea. More extensive outcomes data relating levels of severity of the disorder to its potential adverse outcomes are needed and will assist in tailoring appropriate screening programs and determining the cost-effectiveness of screening various populations."[21]

Because snoring was once considered so harmless, an oral appliance manufacturer might suggest and a dentist might assume that a medical history would be sufficient to distinguish between primary snoring and obstructive sleep apnea. Based on this assumption, the dentist might decide to treat his or her snoring patient with an oral appliance without consulting a physician or ordering a sleep test, which would then be reviewed by a physician. A number of studies[22-29] state that while a detailed medical history may be an excellent screening tool, it is an insufficient instrument on which to base a final diagnosis and treatment plan. In this instance, the malpractice insurer quoted above might well refuse malpractice coverage for the dentist while he or she utilizes oral appliances for the treatment of snoring and obstructive sleep apnea.

The protocol of one appliance suggests that a dentist can screen for obstructive sleep apnea utilizing detailed medical histories and an overnight pulse oximetry reading, consulting with a physician only after treatment has been instituted. The use of pulse oximetry alone as a screening tool is still controversial31 as interpretation of the findings is difficult and should be approached by only those trained and experienced in interpreting the results. Furthermore, the levels of oxygen desaturation do not always correlate linearly with the severity of the apneas.

"The consequence of using screening oximetry was that a significant number of patients with sleep disorders that cause excessive sleepiness would remain undiagnosed and untreated. ... Screening oximetry was most successful in detecting patients with a high likelihood of having OSA [obstructive sleep apnea]... or those with more severe disease. ... In patients with milder disease, normal results of nocturnal oximetry did not mean the absence of SDB [sleep-disordered breathing]."[31]

The author continued by quoting other researchers on the same subject:

"Three other studies evaluated oximetry vs. PSG [polysomnography] with respect to severity of disease and found that oximetry was a suitable screen for patients with moderate and severe OSA but was inadequate for patients with milder cases. Only one study looked specifically at patients with milder forms of disease, including UARS [upper airway resistance syndrome]. ... [In this study] oximetry detected all patients with moderate and severe OSA. However, 30 percent of the patients who were diagnosed normal by oximetry were found to have UARS on PSG. ... They concluded that oximetry was a poor screening tool since normal results did not rule out disease and all abnormal results required further PSG study."[31]

In the patient base that dentists are likely to see – those with mild to moderate obstructive sleep apnea – pulse oximetry is the least accurate. Since both the medical sequelae and the results of excessive daytime sleepiness, that is, drowsy driving, can occur in mild to moderate disease; utilization of pulse oximetry as a tool to determine necessity of medical consultation leaves the dentist liable for missed diagnosis and, consequently, inadequate treatment.

Once one assumes responsibility for the diagnosis and treatment of a patient with a medical problem, one must also be prepared to deal with the diagnosis and treatment of a wide array of peripheral causative factors and sequelae to the original disease state. The cardiac ramifications as well as the results of excessive daytime sleepiness have been discussed previously. In the case of obstructive sleep apnea, there are other medical problems that must be managed coincident with the management of the obstructive sleep apnea per se.

- Weight management. Weight gain is associated with narrowing of the pharyngeal aperture as fat lines the pharynx. In addition, increased upper body fat contributes to difficulty in breathing.
- General fitness. Accompanying the loss of muscle tone with aging, the muscles of the pharynx weaken and are less able to maintain pharyngeal dilation opposing the factors discussed that tend to narrow the airway.
- Smoking cessation. The doctor who assumes care of the patient with primary snoring or obstructive sleep apnea must

be prepared to counsel and treat that patient for smoking addiction as well as to be prepared to diagnose or refer for evaluation of the sequelae of cigarette smoking, including respiratory disease, coronary heart disease, and cancer.

- Alcohol consumption. While moderate levels of alcohol consumption have been reported in the media as being healthful, alcohol consumption before bed can lead to relaxation of the pharyngeal musculature and the development of obstructive sleep apnea.
- Nasal obstruction. Congestion from upper respiratory disease or allergic reaction may account for the development of transient snoring and obstructive sleep apnea; however, nasal polyps and tumors can block the air passage as well. The doctor who assumes care of the patient with obstructive sleep apnea must be prepared to diagnose and treat or refer for treatment patients with tumors of the nasal passage and adenoids.
- Pharyngeal tumors. These can include tumors of the tonsils and pharynx whose presence must be eliminated in the diagnosis of primary snoring and obstructive sleep apnea.
- Gastroesophageal reflux. Increasing thoracic pressure due to efforts to breathe against a closed airway can cause regurgitation of stomach contents into the oral cavity.
- Iatrogenic factors. Prescription medications including tranquilizers, sedatives, and muscle relaxants can increase the chance as well as the intensity of obstructive sleep apnea. Control of medication becomes a primary responsibility for the doctor treating obstructive sleep apnea.

Once one assumes care of a patient with a medical problem, one is bound by the standard of care, which is defined as:

"The uniform standard of behavior upon which the theory of negligence is based. The standard of care requires the actor to do what the ">reasonable person of ordinary prudence' would do in the actor's place. ... If the actor's conduct falls below the standard that a reasonable person would conform to under the circumstances, the actor may be liable for injuries or damages resulting from his or her conduct."[32]

The most common definition of standard of care is "how similarly qualified practitioners would have managed the patient's care under the same or similar circumstances."[33]

Negligence refers to conduct that falls below the standard established by law for the protection of others against unreasonable risk of harm.[32] The dentist who chooses to diagnose and treat primary snoring and obstructive sleep apnea without the participation of a medical colleague must ask himself or herself whether he or she is violating the standard of care.

According to legal experts:

"The medical malpractice plaintiff must establish the appropriate standard of care. In theory, establishing the standard of care and establishing the breach of that standard are legally separate. In reality, unless there is a factual question about what the defendant did, the proof of the standard of care also proves the defendant's breach. For example, assume that the defendant admits that she did not counsel the patient about prenatal testing. If the patient can establish that the standard of care was to offer this testing, the defendant breached the standard."[33]

The parallels to the situation wherein a dentist treats a patient's snoring or apnea with an oral appliance without consulting a physician about the necessary sleep testing are obvious, leaving the dentist negligent.

Would a reasonable person assume the responsibility for the diagnosis and treatment of a medical problem for which he or she was not educated or licensed to treat?

The use of oral appliances for the treatment of sleep-disordered breathing can be an exciting adjunct to the practice of general dentistry. The ability to participate in the treatment of a potentially life-endangering disease provides a sense of satisfaction unmatched by other aspects of dentistry. Cooperation with colleagues in the medical community is essential before undertaking this challenging new aspect of dental practice. Additionally, the dentist must be willing to commit to the study of oral appliance therapy for sleep-disordered breathing with the same intensity and integrity as is applied to the rest of one's practice.

Accepting dentistry's position as part of a treatment team, ongoing review of scientific literature, participation with sleep specialists in sleep laboratories, and attendance at educational meetings dedicated to the study of sleep-related disorders are essential to proper and ethical practice.

References

1. Young T, et al, Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. Sleep 20(9):705-6, 1997.
2. Young T, et al, Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. Sleep 20(8):608-13, 1997.
3. Bloom JW, Kaltenborn WT, and Quan SF, Risk factors in a general population for snoring. Importance of cigarette smoking and obesity. Chest 93(4):678-83, 1988.
4. Schmidt-Nowara WW, et al, Snoring in a Hispanic-American population. Risk factors and association with hypertension and other morbidity. Arch Intern Med 150(3):597-601, 1990.
5. Young T, et al, Population-based study of sleep-disordered breathing as a risk factor for hypertension. Arch Intern Med 157(15):1746-52, 1997.
6. Guilleminault C, Stoohs R, and Duncan S, Snoring (I). Daytime sleepiness in regular snorers. Chest 99:40-8, 1991.
7. Orr W, Sleep apnea syndromes, a primer of diagnosis and treatment. In, a Primer of Diagnosis and Treatment, Quan S, ed. CNS Inc, Chanhassen, Minn, 1991.
8. Koskenvuo M, Kaprio J, and Partinen M, Snoring as a risk factor for hypertension and angina pectoris. Lancet i:893-896, 1987.
9. Koskenvuo M, Kaprio J, and Telakivi T, Snoring as a risk factor for ischaemic heart disease and stroke in men. Br Med J 294:16-9, 1987.
10. Partinen M and Palomaki H, Snoring and cerebral infarction. Lancet ii:1325-6, 1985.
11. Shepard J Jr., Hypertension, cardiac arrhythmias, myocardial infarction, and stroke in relation to obstructive sleep apnea. Clin Chest Med 13(3):437-58, 1992.
12. Lugaresi E, et al, Snoring: pathogenic, clinical, and therapeutic aspects. In, Kryger M, Roth T, and Dement W, eds, Principles and Practice of Sleep Medicine. WB Saunders, Philadelphia, 1994, pp 621-9.
13. Rapoport D, The upper airway in the control of breathing, 1997, http://www.med.nyu.edu/Research/D.Rapoport-res.html.

14. Isono S and Remmers J, Anatomy and physiology of upper airway obstruction. In, Kryger M, Roth T, and Dement W, eds, Principles and Practice of Sleep Medicine. WB Saunders, Philadelphia, 1994, pp 642-56.
15. Findley LJ, Levinson MP, and Bonnie RJ, Driving performance and automobile accidents in patients with sleep apnea. In Phillipson EA and Bradley TD, eds, Clinics in Chest Medicine. WB Saunders, Philadelphia, 1992, pp 427-35.
16. Fletcher E, The relationship between systemic hypertension and obstructive sleep apnea: facts and theory. Am J Med 98(2):118-28, 1995.
17. Strohl K, Novak R, and Singer W, Insulin levels, blood pressure and sleep apnea. Sleep 17(7):614-8, 1994.
18. Guilleminault C, Connolly S, and Winkle R, Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. Am J Cardiol 52:490-4, 1983.
19. Hung J, et al, Association of sleep apnoea with myocardial infarction in men [see comments]. Lancet 336(8710):261-4, 1990.
20. Palomaki H, et al, Snoring, sleep apnea syndrome, and stroke. Neurology 42(7 Suppl 6):75-81; discussion 82, 1992.
21. Baumel MJ, Maislin G, and Pack AI, Population and occupational screening for obstructive sleep apnea: Are we there yet? Am J Respir Crit Care Med 155(1):9-14, 1997.
22. Viner S, Szalai JP, and Hoffstein V, Are history and physical examination a good screening test for sleep apnea? Ann Intern Med 115(5):356-9, 1991.
23. Carroll JL, et al, Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. Chest 108(3):610-8, 1995.
24. Lindblom SS, Adults who snore. Postgrad Med 101(6):171-2, 174, 183-4 passim, 1997.
25. Bonekat HW and Krumpe PE, Diagnosis of obstructive sleep apnea. Clin Rev Allergy 8(2-3):197-213, 1990.
26. Schafer J and Lenders H, [Anamnesis and polysomnography in patients who snore and in obstructive apnea syndrome: a comparative study in 140 patients]. Laryngorhinootologie, 69(11):595-9, 1990.
27. Fischer J, Jackowski M, and Raschke F, [Evaluation of an anamnesis questionnaire for the diagnosis of sleep apnea in patients with chronic diseases of the respiratory organs]. Pneumologie 45 Suppl 1:205-8 1991.
28. Stacchi R, Weiss M, and Brandli O, [Can obstructive sleep apnea syndrome be predicted based on anamnesis and clinical examination findings?]. Schweiz Rundsch Med Prax 84(9):247-54, 1995.
29. Kehrer P and Nicod LP, [Screening for sleep apnea syndrome]. Schweiz Rundsch Med Prax 83(37):1039-43, 1994.
30. Series F, et al, Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hypopnea syndrome [see comments]. Ann Intern Med 119(6):449-53, 1993.
31. Epstein L and Dorlac G, Cost-effectiveness analysis of nocturnal oximetry as a method of screening for sleep apnea-hypopnea syndrome. Chest 113(1):97-102, 1998.
32. Gifis S, Law Dictionary. Barrons, New York, 1984.
33. Lerner S, Establishing medical standards of care & breach without experts. 'Lectric Law Library, 1996, http://www.lectlaw.com.
34. Weigard L, The clinical sequelae of obstructive sleep apnea. Sleep Med Rev 1(5), 1993.

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Should the Dentist Independently Assess and Treat Sleep-Disordered Breathing?

W. Keith Thornton, DDS

ABSTRACT Sleep-disordered breathing is a chronic problem of the inappropriate mechanical collapse of the upper airway. Symptoms range from mild occasional snoring to severe obstructive sleep apnea. The standard of care for the diagnosis and treatment of sleep-disordered breathing by sleep medicine has been the use of the polysomnogram and continuous positive airway pressure. This approach is burdensome, costly, and ineffective due to lack of compliance with or rejection of treatment. Oral appliances are highly effective in managing the mild snorer to the moderate sleep apneic and are approaching the efficacy of continuous positive airway pressure with the severe apneic. The dentist can and should manage these patients. However, the dental practitioner must acquire sufficient training and knowledge to appropriately treat these patients.

AUTHOR

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Dentists have been criticized by sleep specialists and in professional literature for independently assessing and treating sleep-disordered breathing, ranging from snoring to sleep apnea. For instance, Rosalind Cartwright, PhD, a sleep specialist, said that dentists were Ademedicalizing@ sleep-disordered breathing.1 In addition, she called into question the motivation of otolaryngologists and pulmonary physicians for using home monitoring equipment and self-titrating continuous positive airway pressure devices, thereby "despecializing" sleep-disordered breathing. She assumes that only a multidisciplinary team of otolaryngologists and pulmonologists headed by a sleep specialist can properly provide therapy. Today, many dentists in the United States, Canada, England, and Scotland are successfully treating patients with sleep-disordered breathing. A discussion of whether dentists should treat these patients should be based on what is best for the patient, patient preference, and outcomes. To reach a conclusion, several factors must be analyzed and evaluated. These are:

- The anatomy and physiology of the upper airway and the pathophysiology of sleep-disordered breathing;
- The diagnosis and treatment of sleep-disordered breathing; and
- A proposed standard of care.

The Anatomy and Physiology of the Upper Airway and the Pathophysiology of Sleep-Disordered Breathing

Sleep-disordered breathing is the inappropriate mechanical collapse of the upper airway during sleep. This ranges from slight or infrequent partial collapse to total collapse resulting in a continuum of symptoms from mild snoring to obstructive sleep apnea.

Understanding normal physiology and anatomy of the upper airway from the nose to the glottis is critical to managing the mechanical collapse of the airway. The pharynx acts as a collapsible tube to control breathing, swallowing, and phonation. Because the airway and alimentary canal cross in the pharynx, a very sophisticated mechanism causes this flexible tube to collapse during swallowing,[2] protecting the airway. As swallowing begins, the mandible retrudes, generally into centric occlusion, forming a stable bony base for muscular contraction of the tongue, elevation of the hyoid bone, and contraction of the muscles of the soft palate and constrictor muscles of the pharynx. Once swallowing is complete, the airway is returned to normal by the relaxation of the constrictor muscles, the contraction of the dilator muscles (stylopharyngeal and palatopharyngeal muscles), and a posturing of the mandible straight forward in a "physiologic rest position" or breathing position. The mandibular plane at rest is parallel to the maxillary plane. A forward movement of the mandible brings the condyles down the eminence of the fossa, causing the posterior aspect of the mandible to move away from the maxilla (Christensen's phenomenon). The chin point moves an equal distance down and forward, creating a space between the maxillary and mandibular teeth known as "freeway space" in dentistry.

This position of the mandible has three distinct functions. First, the movement away from the maxilla stretches and tightens the lateral walls of the pharynx and the superior constrictor muscle through the attachment to the medial pterygoid plate, the pterygoid mandibular raphe, the floor of the mouth, and the tongue.[3] Schwab and colleagues[4] point out the importance of the lateral collapse of the pharynx in obstructive sleep apnea patients. Secondly, the volume of the oral cavity increases, allowing room for the tongue. Thirdly, the forward chin point brings the genial tubercles forward to provide a fixed bony base. The tongue then rests behind the upper incisors and independently forms an air seal with the soft and hard palate. A simple test to confirm the mandible/tongue position is to try breathing only through the nose with the jaw rotated back and open. Another test is to part the lips while the tongue is in the proper position, confirming the ability of the tongue to form an air seal. The description of the neuromuscular activity of the tongue in both the nonapneic and apneic individual has been well-described by Mezzanote and colleagues.[5] Thus, the function of the mandible and the temporomandibular joint is similar to that of a tent pole, holding the airway open in a protrusive position and allowing it to collapse in a retruded and/or rotated position. A test to confirm this is to try to swallow or snore in a protruded position.

Virtually every health care professional and many lay individuals have a clear understanding of the role the mandible plays to reverse the collapse of the airway when they are trained in the "ABCs" of cardiopulmonary resuscitation. The "A" represents airway management, clearing the airway of any foreign object, followed by performing a "jaw thrust" maneuver. Only when the mandible is placed in the proper position to open the airway can ventilation, or breathing, "B," be performed easily and successfully. These same techniques apply to managing a patient's airway during conscious sedation or to ventilating a patient with a bag in general anesthesia.[6] The pathophysiology of sleep-disordered breathing and treatment relate directly to this mechanism of airway management. Sleep-disordered breathing occurs because of the increased narrowing of the airway (flow-resistive load), which can be from two basic causes. The first and primary cause is the position of the mandible and tongue. A sleep-disordered breathing patient's decrease in baseline neuromuscular activity during sleep results in rotation of the mandible into a functionally retruded craniofacial position, partial mouth breathing, and the collapse of the tongue into the pharynx.7 The reactivation of the neuromuscular control reverses the collapse as indicated by the chin point electromyogram. The position of the mandible can be affected by underlying craniofacial anatomy, soft tissue anatomy, increasing age, pathological changes, medications, alcohol, food, fatigue, and sleep stage (atonia during rapid eye movement sleep).[8-12] A supine sleep position also negatively affects the position of the mandible by allowing it to rotate back and open.[13] The second major cause of airway narrowing is obesity. Fatty deposition in the neck, pharyngeal tissues, and tongue tend to narrow the lumen of the airway and decrease the likelihood of the neuromuscular mechanism and jaw holding the airway open enough to prevent obstruction.[14] Obesity also requires more effort for the patient to ventilate (mass load), although this is separate from the mechanical collapse of the airway. Other causes of airway narrowing, such as a deviated septum or allergies, must be evaluated and may be contributing factors but are not the primary causes of the underlying problem.

Diagnosis and Treatment of Sleep-Disordered Breathing

Traditional Medical Approaches

Sleep-disordered breathing is a continuum from mild, occasional snoring to severe obstructive sleep apnea. Yet, the vast amount of time, effort, and money is spent on the differentiation between the apneic and the nonapneic patient using the polysomnogram for diagnosis. The result is that the diagnosed obstructive sleep apnea patient is treated with

continuous positive airway pressure with there being little or no treatment for the failed continuous positive airway pressure patient or the non-obstructive sleep apnea patient. Historically, this treatment paradigm developed in the United States for three reasons. The first was the discovery of sleep apnea by psychiatrists and neurologists during routine experimentation on sleep in the sleep lab setting.[15] The second was the discovery of a way to manage the disorder via the use of continuous positive airway pressure.[16] The third was the high prevalence and the serious consequences of the disorder.[9] Both the diagnosis and treatment of obstructive sleep apnea required the use of the polysomnogram, causing the proliferation of sleep laboratories for clinical purposes. This occurred during a time of adequate funds for new therapies and created a new specialty, sleep medicine, supported by a billion-dollar sleep industry. The focus of this sleep medical-industrial complex has been the improvement of the systems surrounding the polysomnogram-continuous positive airway pressure approach. Millions are spent on mask and continuous positive airway pressure technology to improve compliance. A paucity of resources is allocated to other promising technology. Dr. Cartwright was an early advocate of oral appliance therapy. Her 1988 paper on the Tongue Retaining Device invented by psychiatrist Charles Samuelson cited great promise for oral appliances.[17] The results of treatment of 24 patients were that only five of the 24 remained unimproved (the five included the most obese cases). Her conclusions were that "treatment can proceed in a logical fashion starting with the less invasive treatments for both mild and severe cases and with careful clinical management, most patients will reach acceptable levels of control. Only a few will require the more cumbersome CPAP [continuous positive airway pressure] or more invasive surgical treatments." These therapies would be appropriate "for those who do not respond to a trial of habit change and TRD [Tongue Retaining Device] treatment."

"An appliance ... has an early place in the treatment of these [apneic] patients either alone or as an adjunct to other measures."

However, to date little has been done within the sleep community to further oral appliance technology.

Comparison of Treatment Alternatives

Blinded, crossover, outcome studies on all approaches should be the basis for any discussion of the proper management of sleep-disordered breathing. Since these are not available, continuous positive airway pressure therapy and oral appliance therapy will be compared with published data. Surgical treatment is excluded from this discussion since surgery is usually recommended for sleep-disordered breathing only after the more conservative therapies have been tried. Conservative therapies such as weight loss and positional therapy are also excluded since these should be instituted no matter what other approaches are tried. To date, most of the published studies relate to polysomnograms and continuous positive airway pressure. As explained previously, for lack of access to sleep labs and funding, much less has been done with oral appliances. Any analysis of treatment should include efficacy, effectiveness, cost, availability of medical resources, quality of life, and prevention.

Efficacy is the capacity to produce a desired effect. Continuous positive airway pressure is currently the gold standard for efficacy in normalizing an abnormal polysomnogram, not necessarily the gold standard for managing sleep-disordered breathing. Continuous positive airway pressure has two distinct functions. The first is airway management and the second is ventilation. Less has been written describing the ventilatory effects such as increase in vital capacity or an increase in the pressure gradient across the alveoli. The new autopaps appear to be sophisticated ventilators. However, the effect of continuous positive airway pressure to act as a pneumatic splint preventing the mechanical collapse of the pharynx has been well-described by Sullivan.[16] Two facts reveal that it is doubtful that this is the most efficacious way to manage an airway. The first is that continuous positive airway pressure does not work with the jaw rotated back and open, which is the usual sleep position for most individuals with sleep-disordered breathing. According to Mark Forester, chief technologist at Presbyterian Sleep Institute in Dallas, more than 90 percent of the institute's patients leak air through the mouth, thereby requiring chin straps for continuous positive airway pressure to be effective. Therefore, a significant part of the efficacy of continuous positive airway pressure is the position of the mandible, whether this is accomplished by an external jaw positioning device and/or an increase in the baseline neuromuscular activity of the jaw positioning muscles and tongue to close the jaw and effect an air seal between the tongue and soft palate. The second fact is the difficulty in ventilating a patient with air pressure alone while managing a patient in anesthesia or CPR. Until the jaw and head are in the proper position, ventilation, even with great pressure, is difficult.

The efficacy of continuous positive airway pressure to normalize polysomnograms is well-documented. Although the results of most of the published studies on oral appliances have shown that they are not as efficacious in normalizing polysomnograms, the results of studies on the newer, adjustable appliances are approaching that of continuous positive airway pressure. In an abstract of preliminary results based on 38 patients by Pancer and Hoffstein,[18] the average respiratory disturbance index was 42 before oral appliance therapy. After therapy, most of the symptoms were eliminated, and the average respiratory disturbance index was 11, with virtually all events being mild hypopneas. An abstract by Roberts, Jamieson, and Becker19 cites similar results with continued improvement beyond the maximum range of protrusion of the

mandible. Loube has shown normalization of polysomnographic parameters with and without an oral appliance in a patient with upper airway resistance syndrome.[20] More-definitive studies are now under way on oral appliance therapy with upper airway resistance syndrome.

Effectiveness is not the same as efficacy. Effectiveness is efficacy over time to obtain the desired results. Based on this definition, continuous positive airway pressure is highly ineffective for the treatment of sleep-disordered breathing, even obstructive sleep apnea, due to cost, initial rejection of treatment, and compliance. Guilleminault found a 2 percent compliance with continuous positive airway pressure in a large series of patients with upper airway resistance syndrome.[16] Similar results have been shown with the nonapneic snorer. Therefore, continuous positive airway pressure is virtually excluded as a viable therapy in more than 70 percent of the patients with sleep-disordered breathing. A number of studies have also shown the lack of compliance with continuous positive airway pressure in the sleep apnea patient, even those with severe apnea.[21] A seminal study, done by Kribbs and Pack, utilized covert monitoring to determine continuous positive airway pressure use.[22] Of 35 patients, only 16 (46 percent) met the minimum criteria of four hours of use on 70 percent of days monitored. If a conservative criterion of seven hours of sleep based on normative data of middle-aged adults is used, only two of 35 achieved this result at least five of seven days. Their conclusion is that "frequent, long-duration, quality sleep is a relatively rare occurrence in OSAS [obstructive sleep apnea syndrome] patients treated with CPAP [continuous positive airway pressure] ... that actual CPAP use by OSAS patients falls short of providing quality sleep all night, every night."

Another study reviews the diagnostic and treatment process for all patients seen in a major sleep center during an 11-year period.[23] An assumption was made that all the patients who were diagnosed by history to have obstructive sleep apnea were patients with symptoms of sleep-disordered breathing severe enough to warrant a referral to a sleep center. The compliance results of the patients contacted were extrapolated to those patients not contacted. Not only did this reveal that continuous positive airway pressure compliance was a problem, but it also revealed that the diagnostic and treatment paradigm has major shortcomings. Twenty-five percent of the population did not return for a diagnostic night of polysomnogram and 57 percent of the obstructive sleep apnea patients rejected or quit wearing the continuous positive airway pressure. Based on these results, less than 12 percent of this sleep-disordered breathing population is being treated with continuous positive airway pressure. If covert monitoring of the ones who said they were using the continuous positive airway pressure were done, the results would be even more abysmal. Dinges has stated, "Studies that have objectively monitored the nightly duration of CPAP [continuous positive airway pressure] use have consistently produced an average duration that appears to be below the duration of sleep considered essential for alert functioning."[24]

Although no comparable study has

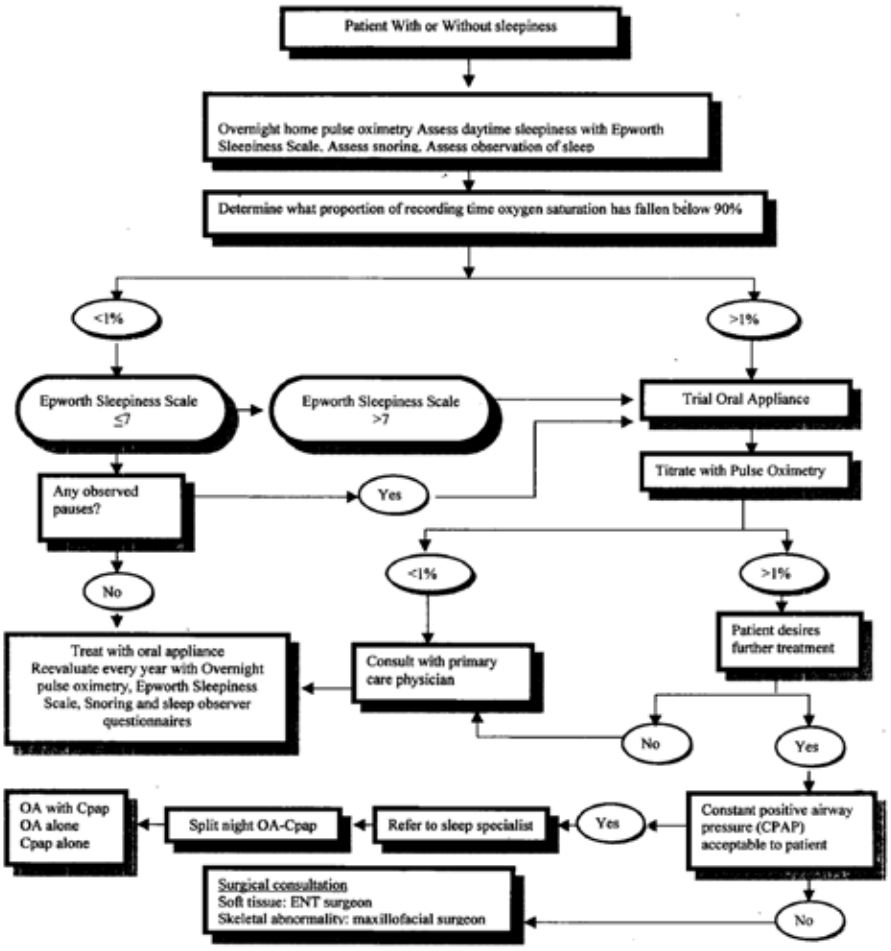

Figure 1. Assessment and Treatment Alsorithm for Sleep-Disordered Breathing.

been done on oral appliances, logic would point to a much greater use of oral appliances than continuous positive airway pressure. In a follow-up telephone survey done by this author on 208 patients who had worn an oral appliance for more than six months, 81 percent were still wearing it an average of 25 days per month.[25] Twelve patients (6 percent) used the appliance as needed to control snoring only. Factors sighted for discontinued use included 17 (8 percent) who either lost weight or lost their bed partner, eight (4 percent) with pain, and three (1 percent) with no complaint other than difficulty sleeping with the appliance. Certainly, if preference indicates the greater likelihood of increased use, oral appliance therapy would be the clear winner. In a crossover study by Clark,[26] a post-treatment telephone interview revealed an overwhelming 95 percent preference of oral appliances over continuous positive airway pressure. Only one of 21 patients was using continuous positive airway pressure. Seventeen were using an oral appliance every day, while two were using it intermittently. The preferences were even more notable, considering the fact that continuous positive airway pressure was more efficacious by polysomnogram than the oral appliance. If continuous positive airway pressure is limited to the treatment of the few sleep apneics who will wear it and if most of these would prefer oral appliances, the most logical noninvasive treatment modality for all sleep-disordered breathing patients is the oral appliance. Continuous positive airway pressure should be used only after trial oral appliance therapy fails from noncompliance or lack of symptom improvement. Dr. Perez-Guerra, a pulmonologist and sleep medicine physician, recommends the following similar approach. "(1) Fully asymptomatic snorers without associated co-morbidity (particularly if not overtly obese) are referred for oropharyngeal surgery or a dental appliance without preceding polysomnography; (2) snorers with an Epworth Sleepiness Scale >12, body mass index >28, and who do not desire consideration for nasal CPAP will be referred for oropharyngeal surgery or a dental appliance without preceding polysomnography; (3) patients with suspected severe sleep-disordered breathing, particularly when serious co-morbidity exists, will undergo split-night studies. In the future, most patients likely to require nasal CPAP will have objective documentation of sleep-disordered breathing by the simplest ambulatory monitoring. This will probably consist of the flow/time profile utilizing nasal cannulae with or without simultaneous oximetry; and (4) no nasal CPAP re-titrations will be performed in patients who remain asymptomatic."[27]

Other determining factors of treatment point to oral appliance therapy. The cost of oral appliance therapy is significantly less than continuous positive airway pressure, particularly if the cost of titration of the continuous positive airway pressure with a polysomnogram and the failure rate of compliance is considered. Assessing quality of life, few if any would choose continuous positive airway pressure over an oral appliance unless the oral appliance were ineffective. Responses on questionnaires list the common problems with continuous positive airway pressure as inconvenience, stuffy or runny nose, poor sleep, disturbed sleep, less intimacy with bed partner, claustrophobia, facial irritation, and expense.[28] Although the same questions were not asked in the Pancer, Hoffstein study, 61 percent of respondents said they were very satisfied and 39 percent said they were moderately satisfied with the oral appliance, with no one being moderately dissatisfied.18 Finally, the prevention of obstructive sleep apnea seems to be tilted in favor of the oral appliance and a dental intervention. As in the treatment of the craniofacial anomaly of the child with the long face syndrome with orthodontics, functional appliances, and/or orthognathic surgery or the early treatment of snoring with oral appliances before obstructive sleep apnea develops, dental therapy seems to hold great promise.

A Proposed Standard of Care

Diagnosing Sleep-Disordered Breathing

If oral appliance therapy is the preferred treatment for the majority of sleep-disordered breathing patients, then the next question is who should diagnose the condition and what diagnostic tests should be used. The answer includes establishing a diagnostic hypothesis, progressively ruling out specific disease (sensitivity), and then confirming a specific disease (specificity). Sleep medicine specialists would require a polysomnogram or equivalent for everyone suspected of obstructive sleep apnea.[29] However, many are now recommending first applying clinical screening programs to differentiate the likely obstructive sleep apnea patient from the non-obstructive sleep apnea patient to reduce the cost of diagnosis and treatment.[30,31] These screening tests can essentially rule out obstructive sleep apnea while the polysomnogram diagnoses the presence or absence. The same screening program can be used to titrate the adjustable oral appliances and then rule out obstructive sleep apnea if the tests are negative while wearing the appliance. The screening program includes questionnaires for sleepiness such as the Epworth Sleepiness Scale;[2] questions of the bed partner including information on pauses, snorting, or gasps;[33] and overnight pulse oximetry, which can be used to rule out obstructive sleep apnea. If oxyhemoglobin saturation does not fall below 90 percent for more than 1 percent of the night, then the likelihood of obstructive sleep apnea is less than 2 percent.[34,35] If all the tests are negative, obstructive sleep apnea is ruled out. If any are positive with the oral appliance, then a consultation and polysomnogram or other study to determine the respiratory disturbance index is warranted. Although there is criticism of the polysomnogram in the medical community concerning arbitrary cutoff points for normals and obstructive sleep apnea, lack of standard-

ization of testing methods with uncertain validity and reliability, and variable definitions of abnormal breathing events and syndromes,[36] the American Sleep Disorders Association continues to call the polysomnogram the gold standard. Pack has questioned whether "the gold standard (PSG) is really gold. Are we treating a disease or perpetuating an expensive test? Are there other tests that define the disease better?"[37] Although the polysomnogram remains the purview of the sleep specialist, the dentist can be well-prepared to perform all the other requirements of screening, including patient history, physical exam, and overnight pulse oximetry.

The Dentist as the Primary Practitioner

Three factors should be considered before a dentist decides to treat sleep-disordered breathing. These include legal, professional-liability, and ethical issues. The first two are relatively easy to assess. The last requires much more thought, particularly in developing a standard of care for a new therapy.

Legally, the treatment of any disease process or condition is based on both state and federal laws. Under most state laws, the practice of dentistry is related to conditions and diseases that affect the teeth, jaws, temporomandibular joint, or related structures including their function and dysfunction.[38] If sleep-disordered breathing can be treated through dental therapies such as oral appliances and orthognathic surgery, then sleep-disordered breathing is within the scope of dentistry. The overlap of treatment modalities in medicine and dentistry for a particular diagnosis occurs frequently. The treatment given is based on patient complaints, symptoms, severity of the disease, efficacy and effectiveness of the treatment, referral patterns, the specialty and bias of the professional consulted, costs, and insurance coverage. Examples of this dental-medical overlap include diagnosis and treatment of nocturnal bruxism, temporomandibular disorders, myofacial pain, and headaches. A direct correlation can be made between nocturnal bruxism and sleep-disordered breathing. Both are sleep disorders, have symptoms that are confused with or mimic other disorders, are chronic, and need to be managed over time. Both can be effectively managed in the majority of cases with noninvasive oral appliance therapy.

The Food and Drug Administration oversees and regulates the sale of devices for the treatment of conditions such as sleep-disordered breathing. The dental branch of the FDA, not the medical branch, regulates oral appliances. At a recent FDA conference on oral appliances, consideration was given to making them available over the counter.[39] Although they are still restricted to prescription by a physician or dentist, the possibility exists that they will become an over-the-counter product if data is presented confirming their safety, lack of significant morbidity, and effectiveness.

A second concern for the dentist treating sleep-disordered breathing is whether the practitioner's malpractice insurance will cover this treatment. Statements have been made that dentists are at risk if a patient has a stroke, myocardial infarction, or motor vehicle accident while being treated with an oral appliance. As with any other potential act of malpractice, many factors concerning liability would be considered, including circumstances, informed consent, standard of care in the dental community, and treatment documentation. Three malpractice carriers, AAOMS National Insurance Co. RRG, Fortress Insurance Co.,[40] and CNA[41] do not restrict the treatment of sleep-disordered breathing nor do they charge more for coverage. The sleep specialist treating obstructive sleep apnea would have the same risks as the dentist, particularly since there is known failure of continuous positive airway pressure because of noncompliance. With the improvement of the results with oral appliance therapy, the documented preference of oral appliance therapy over continuous positive airway pressure, and the requirement by the American Sleep Disorders Association that accredited sleep labs have a dental consultant, it is the sleep specialist who faces a real issue in the area of informed consent from patients.

Finally, the ethical issues of sleep-disordered breathing demand three things of the dentist: having adequate and appropriate training, giving adequate information on sleep-disordered breathing, and doing no harm. The dentist has a duty to have enough knowledge and understanding of sleep-disordered breathing to educate the patient on treatment options, morbidity of the treatment, and outcomes. Fortunately for the patient, oral appliance therapy has little morbidity, is reversible, inexpensive, and with the newer adjustable appliances highly effective. It is the overwhelming treatment of choice. Simple questionnaires and testing can quickly determine those patients who are oral appliance therapy treatment failures or are being undertreated. Proper referral of the more severe patient or the patient who may not have sleep-disordered breathing can be effected with little delay in treatment and no harm done to the patient. The potential harm that a dentist can render is not maximizing the benefit of oral appliances for the patient if he or she attempts to treat sleep-disordered breathing with little knowledge, training, and skill. The highly inconsistent results and numerous failures of both dentists and many oral appliances in the past have given oral appliance therapy a poor reputation among many sleep physicians. If a dentist is going to diagnose and treat sleep-disordered breathing, he or she must be able to place the jaw in the most effective treatment position. This necessarily requires home or sleep lab titration of the device utilizing objective measurements of results such as pulse oximetry, home sleep study, or polysomnogram. This also requires the use of adjustable oral appliances, whether they are used as trial devices or treatment appliances. Just as continuous positive airway pressure is

adjustable and titrated, so too must oral appliances be adjustable and titrated.[19]

Any patient complaining of sleep-disordered breathing can and should be treated by the dentist if the dentist determines that the patient has the condition and can be helped with an oral appliance. This may include the patient whose chief complaint is snoring; the continuous positive airway pressure failure; the patient who refuses to undergo a polysomnogram; the patient who refuses to wear a continuous positive airway pressure device; the continuous positive airway pressure compliant patient who desires an alternative or a substitute while traveling; the orthognathic, uvulopalatopharyngoplasty or laser-assisted uvulopalatopharyngoplasty surgical candidate; and the surgical failures. Unlike patients of sleep medicine, which treats obstructive sleep apnea patients only, every patient can receive therapy since oral appliances can manage the occasional mild snorer and the patient with severe obstructive sleep apnea. The oral appliance becomes as much a part of the diagnosis as it does treatment at a fraction of the cost and time of the polysomnogram-continuous positive airway pressure treatment routine. This is analogous to using the "smart CPAPs" to both diagnose and treat obstructive sleep apnea.[42]

The dentist should follow a standard of care for the sleep-disordered breathing patient that will ensure appropriate treatment, especially if the practitioner chooses to treat the patient prior to an assessment by a sleep specialist. The routine evaluation should include sleep questionnaires; medical history; physical examination of the head, neck and pharynx; and overnight pulse oximetry. If pathology of the upper airway is ruled out and the chief complaint is snoring with no symptoms of sleepiness and no oxygen desaturations, then the definitive treatment is the oral appliance. If the patient snores, is sleepy, but has no desaturations then the likely diagnosis is upper airway resistance syndrome and the treatment of choice is an oral appliance. Follow-up questionnaires for sleepiness must be completed after oral appliance therapy, and failures should be referred to their physicians for evaluation. If the patient desaturates greater than 1 percent of the night below 90 percent, then obstructive sleep apnea is a possibility. At this point, a consultation is warranted with the patient's physician with the permission of the patient. Treatment options must be thoroughly explained. If oral appliance therapy is chosen by the patient, then after therapy if desaturation is less than 1 percent of the night below 90 percent, obstructive sleep apnea is practically ruled out, particularly if there are no remaining symptoms. If the patient is still desaturating or continues to have symptoms, then with the patient's understanding and permission, a referral should be made to his or her primary care physician for further analysis and referral to either a maxillofacial surgeon, an otolaryngologist, or a sleep physician.

There is a new standard of care for the sleep community. If the sleep physician is going to offer comprehensive therapy, then he or she must include oral appliances as part of the evaluation and treatment routine,[43] whether this is independent of continuous positive airway pressure, combined with continuous positive airway pressure, or as a substitute when continuous positive airway pressure cannot be worn or is inconvenient to wear. One tenet of the American Sleep Disorders Association is that a "decision on an individual patient regime must combine objective evaluation of severity and patient preference."[44] At least one sleep medicine group has a full-time dentist on staff, and limits the use of oral appliances only because of the lack of insurance coverage or the refusal of the patient to pay for the therapy if insurance does not. The group, Sleep Medicine Associates of Texas, includes three board-certified sleep physicians, including the current president of the American Sleep Disorders Association, Dr. Wolfgang Schmidt-Norwara. Will this lead to physicians becoming the primary source for oral appliance therapy as concern some dentists? It is doubtful. Long-term management of oral appliance therapy requires dental intervention. This is best accomplished by the dental professional, who focuses on periodic recalls, prevention, early intervention, accessibility, affordability, and appropriate referral when warranted. The oral cavity, dentition, and temporomandibular joint must be healthy for the patient to benefit from long-term oral appliance therapy. Until a simpler, more cost-effective and user-friendly treatment is found, patients with sleep-disordered breathing are best served by the dental community.

References

1. Cartwright R, Who should treat sleep apnea and how? Chest 111:266-7, 1997.
2. Bosma J, Deglutition: pharyngeal stage. Physiol Rev 37:275-300, 1957.
3. Sicher H, The muscles of the soft palate and pharynx. In, Sicher H, Oral Anatomy, 4th ed. CV Mosby Co, Chicago, 1965, pp 298-305.
4. Schwab RJ, Gupta KB, et al, Upper airway soft tissue anatomy in normals and patients with sleep disordered breathing. Significance of the lateral pharyngeal walls. Am J Respir Crit Care Med 152:1673-89, 1995.
5. Mezzanote W, Tangel D, and White D, Waking genioglossal electromyogram in sleep apnea patients versus normal controls (a neuromuscular compensatory mechanism). J Clin Invest 89(5):1571-9, 1992.
6. Morikawa S, Safer P, and DeCarlo J, Influence of the head-jaw position upon upper airway patency, Anesthesiology 22:265-70, 1981.
7. Clark G, Kobayashi H, and Freymiller E, Mandibular advancement and sleep disordered breathing. J Cal Dent Assoc 24(4):49-61, 1996.
8. Coccagna G, et al, The bird-like face syndrome (acquired micrognathia, hypersomnia, and sleep apnea). In, Guilleminault C, Dement W, eds, Sleep Apnea Syndromes. Alan R Liss, New York, 1978, pp 259-71.
9. Young T, Palta M, et al, The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 328:1230-5, 1993.
10. Issa FG, and Sullivan C, Alcohol, snoring, and sleep apnea. Neurol Neurosurg Psychiatry 45:353-9, 1982.
11. Suratt PM, Turner B, and Wilhoit SC, Effect of intranasal obstruction on breathing during sleep. Chest 90(3):324-9, 1986.
12. Wetter DW, Young TB, et al, Smoking as a risk factor for sleep disordered breathing. Arch Intern Med 154(19):2219-24, 1994.
13. Cartwright R, Ristanovic R, et al. A comparative study of positional sleep apnea. Sleep 14(16):546-52, 1991.
14. Davies RJ and Stradling JR, The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnoea syndrome. E Respir J 3(5):509-14, 1990.
15. Dement W, History of sleep physiology and medicine. In, Kryger M, Roth T, and Dement W, eds, Principles and Practice

of Sleep Medicine, 2nd ed. WB Saunders, Philadelphia, 1994.
16. Sullivan C, Issa F, et al, Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1:86-865, 1981.
17. Cartwright R, Stefoski D, et al, Toward a treatment logic for sleep apnea: The place of the tongue retaining device. Behav Res Ther 26(2):121-6, 1988.
18. Pancer J and Hoffstein V, Evaluation of Adjustable Mandibular Positioning Appliance for Treatment of Obstructive Sleep Apnea, Sleep Disorders Dental Society Report, Sept:1-5, 1997.
19. Roberts DH, Becker P, and Jamieson A, Determining efficacy of a split night oral appliance and CPAP titration protocol using polysomnography, Annual Meeting Abstract Book, American Professional Sleep Society, 1998 (in publication).
20. Loube D, Andrada T, et al, Successful Treatment of Upper Airway Resistance Syndrome with an Oral Appliance, Sleep Disorders Dental Society Report, Dec:1-7, 1997.
21. Engleman HM and Douglas NJ, CPAP Compliance. Sleep 16(8):S114, 1993.
22. Kribs N, Pack A, et al, Objective measurement of nasal CPAP patterns of use by patients with obstructive sleep apnea. Am Rev Res Dis 147:887-95, 1993.
23. Kramer M and Bailey S, The Diagnostic and Treatment Process for Obstructive Sleep Apnea: A Clinical Experience, Annual Meeting Abstract Book, American Professional Sleep Society, 1996, p 346.
24. Dinges D, University of Pennsylvania Conference on Sleep Apnea, Course Notebook, Feb 1997.
25. Thornton WK and Roberts DH, Nonsurgical management of the sleep apnea patient. J Oral Maxillofac Surg 54:1103-8, 1996.
26. Clark GT, Blumenfeld I, et al, A crossover study comparing the efficacy of continuous positive air pressure with mandibular positioning devices on obstructive sleep apnea patients. Chest 109(6):1477-83, 1996.
27. Perez-Guerra F, How to shorten the sleep laboratories' waiting lists. ASDA News 5(1):12-5, 1998.
28. Hoffstein V, Viner S, et al, Treatment of obstructive sleep apnea with nasal continuous positive air pressure, patient compliance, perception of benefits, and side effects. Am Rev Respir Dis 145(4PT1):841-5, 1997.
29. Standards of Practice Committee of the American Sleep Disorders Association, Practice parameters for the use of portable recording on the assessment of obstructive sleep apnea. Sleep 17(4):372-7, 1994.
30. Jamieson A and Becker P, Snoring: its evaluation and treatment. Hospital Medicine March 1996.
31. Baumel MJ, Maislin G, and Pack A, Population and occupational screening for obstructive sleep apnea: Are we there yet? Am J Respir Crit Care Med 155:9-14, 1997.
32. Johns MW, A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 14(6):540, 1991.
33. Kump K, Whalen C, et al, Assessment of the validity and utility of a sleep-symptom questionnaire. Am J Respir Crit Care Med 150:735-41, 1994.
34. Gyulay S, Olson L, et al, A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. Am Rev Respir Dis 147:50-3, 1993.
35. Series F, Marc I, et al, Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hypopnea syndrome. Ann Intern Med 119:449-53, 1993.
36. American Sleep Disorders Association Task Force, Descriptions and Definitions of Abnormal Sleep Related Breathing Events, The Chicago Criteria for Measurements, Definitions, and Severity Ratings of Sleep Related Breathing Disorders in Adults (Draft), Jan 1998:6-8.
37. Pack A, University of Pennsylvania Conference on Sleep Apnea, Course Notebook, Feb 1997.
38. State Board of Dental Examiners, Texas Dental Practice Act, 1995.
39. Food and Drug Administration, Center for Device and Radiological Health, Office of Device Evaluation. Meeting of the Dental Products Panel, Nov 3-5, 1997.
40. Personal conversation with Dr L Jack Bolton, chairman of the board, AAOMS National Insurance Co RRG and Fortress Insurance Co.
41. CNA Insurance Co, professional liability contract, 1997.
42. Bradley PA, Mortimore IL, and Douglas NJ, Comparison of polysomnography with ResCare Autoset in the diagnosis of the sleep apnoea/hypopnea syndrome. Thorax 50:1201-3, 1995.
43. Schmidt-Norwara W, OA for OSA. Chest 109(5):1275, 1996.
44. American Sleep Disorders Association, slide series, 1992.

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Comparisons of Oral Devices for Snoring

James E. Eckhart, DDS

ABSTRACT A study was performed on 19 types of oral appliances for snoring/obstructive sleep apnea. The appliances were categorized into two groups, mandibular advancers and tongue advancers. A set of limited criteria was developed by which an appliance could be evaluated, and the criteria were weighted. Evaluations were then performed on the appliances, and they were rated according to satisfaction of criteria. The rating table should help a dentist in selecting an appliance that will be accepted by the patient and effective in treating snoring/obstructive sleep apnea.

AUTHORS

James E. Eckhart, DDS, is an orthodontist who also treats TMJ and snoring.

A dentist considering treating a patient for snoring might not be aware of the broad variety of oral devices available. Even if aware, he or she might still be perplexed at which appliances might be best in different circumstances. This paper is intended to familiarize the dentist with the choices and to help in the selection of snoring treatment devices. The patient should be provided with an appliance that is comfortable and does its job. If this is done, the field of medicine may more quickly increase its acceptance of dentistry's helpful role in sleep medicine. However, for medicine to accept dentistry's role, dentistry must understand enough sleep medicine to meet the standards of care set by medicine.1 A dentist entering this field must be aware that snoring itself has certain morbidities[2-8] and that it may be a sign of upper airway resistance syndrome or obstructive sleep apnea, whose symptoms, associated morbidities, and diagnostic protocols the dentist must also know. This paper does not directly address the question of efficacy at treating obstructive sleep apnea with oral devices, which can best be evaluated by polysomnography after delivery of an oral device. No polysomnographies were performed as part of this study. However, this paper does offer guidance regarding which appliances are most favorable from both the dentist's and patient's viewpoints relative to comfort, convenience, and the stopping of snoring. The more highly rated devices might be considered preferable for treating snoring and perhaps as logical starting places for selection of appliances to treat obstructive sleep apnea.

For this study, it was decided to exclude oral devices that depend on contact with the soft palate or dorsal surface of the posterior tongue, because they produce gagging responses and do not compete in any important way with

the devices studied herein. It was also decided to exclude appliances that are not offered commercially and to exclude a few commercially available appliances that are relatively ineffective or require too much effort compared to the benefit to be gained. Further, this study may have examined an appliance from one vendor that is available from more than one vendor, and might therefore present only a partial view of the truth about an appliance.

Categories of Appliances

Effective, readily accepted oral appliances for snoring/obstructive sleep apnea work by moving either the mandible or the tongue forward. The following classification of appliances fit the existing commercially available appliances.

Mandibular Advancers

One-Piece

- Boil-and-bite – Snore Guard (Figures 1a and b) and Snore Free (Figures 2a and b).
- Lab-fabricated – MIRS (Figures 3a and b), SNOAR (Figures 4a and b), Mandibular Repositioner (Figures 5a and b), Elastomeric (**Figure 6**a and b), and NAPA (Figures 7a, b, and c).

Two-Piece

- Boil-and-bite – Thera Snore (Figures 8a, b, and c).
- Lab-fabricated – Silent Nite (Figures 9a and b), Herbst (Figures 10a and b), Restore (Figures 11a, b, c, and d), TAP (Figures 12a, b, c, and d), PM Positioner (Figures 13a and b), Silencer (Figures 14a and b), EMA (Figures 15a and b), Klearway (Figures 16a, b, anc d), and Hilsen (Figures 17a, b, and c).

Tongue Advancers

- Off-the-shelf – Snore X (Figures 18a, b, and c).
- Lab-fabricated – Tongue Retainer Device (Figures 19a and b).

One-Piece

BOIL-AND-BITE – SNORE GUARD (FIGURES 1A AND B) AND SNORE FREE (FIGURES 2A AND B).

Figure 1a. Snore Guard, front view.

Figure 1b. Snore Guard, side view.

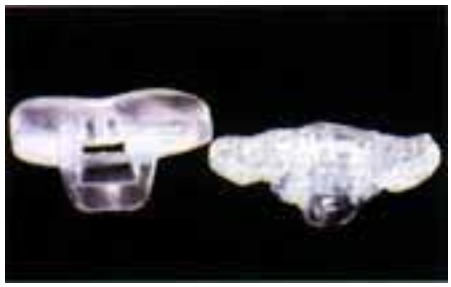

Figure 2a. Snore Free, front view.

Figure 2b. Snore Free, side view.

LAB-FABRICATED – MIRS (FIGURES 3A AND B), SNOAR (FIGURES 4A AND B), MANDIBULAR REPOSITIONER (FIGURES 5A AND B), ELASTOMERIC (FIGURE 6A AND B), AND NAPA (FIGURES 7A, B, AND C).

Figure 3a. MIRS, front view.

Figure 3b. MIRS, side view.

SNOAR, front view.

Figure 4b. SNOAR, side view.

Figure 5a. Mandibular Repositioner, front view.

Figure 5b. Mandibular Repositioner, side view.

Figure 6a. Elastomeric, front view.

Figure 6b. Elastomeric, side view.

Figure 7a. NAPA, front view.

Figure 7b. NAPA, side view.

Figure 7c. NAPA, top view.

Two-Piece

boil-and-bite – thera snore (figures 8a, b, and c).

Figure 8a. Thera Snore, front view.

Figure 8b. Thera Snore, side view.

Figure 8c. Thera Snore, front view (disarticulated).

Lab-fabricated – Silent Nite (Figures 9a and b), Herbst (Figures 10a and b), Restore (Figures 11a, b, c, and d), TAP (Figures 12a, b, c, and d), PM Positioner (Figures 13a and b), Silencer (Figures 14a and b), EMA (Figures 15a and b), Klearway (Figures 16a, b, anc d), and Hilsen (Figures 17a, b, and c).

Figure 9a. Silent Nite, front view.

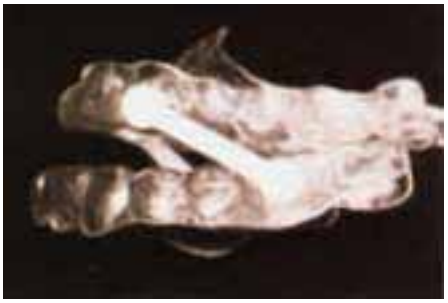

Figure 10a. Herbst, front view.

Figure 10b. Herbst, side view.

Figure 11a. Restore, front view.

Figure 11b. Restore, side view.

Figure 11c. Restore, top view (disarticulated).

Figure 11d. Restore, front view (closeup).

Figure 12a. TAP, front view.

Figure 12b. TAP, side view.

Figure 12c. TAP, rear view. (disarticulated).

Figure 12d. TAP, top view (disarticulated).

Figure 13a. PM Positioner, front view.

Figure 13b. PM Positioner, side view.

Figure 14a. Silencer, front view.

Figure 14b. Silencer, side view.

Figure 15a. EMA, front view.

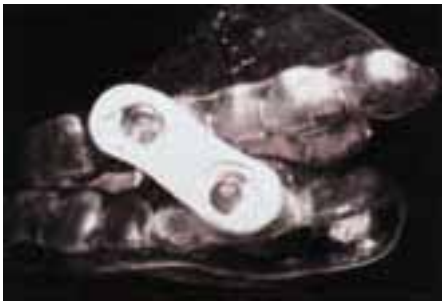

Figure 15b. EMA, side view.

Figure 16a. Klearway, front view.

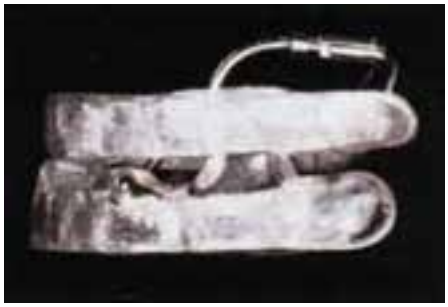

Figure 16b. Klearway, side view.

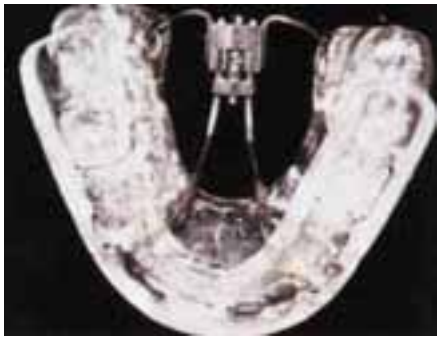

Figure 16c. Klearway, inferior view.

Figure 17a. Hilsen, front view.

Figure 17b. Hilsen, side view.

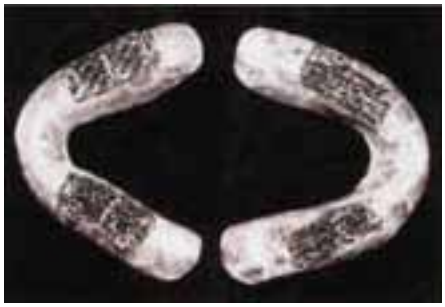

Figure 17c. Hilsen, top view (disarticulated).

Tongue Advancers

Off-the-shelf – Snore X (Figures 18a, b, and c).

Figure 18a. Snore X, front view.

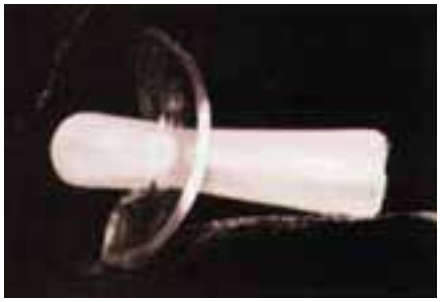

Figure 18b. Snore X, side view.

Figure 18c. Snore X, inferior view.

Lab-fabricated – Tongue Retainer Device (Figures 19a and b).

Figure 19a. Tongue Retaining Device, front view.

Figure 19b. Tongue Retaining Device, side view.

Description of Categories

Mandibular Advancers

In most cases, these devices are secured in the mouth by being teeth-borne to the upper teeth. Most also attach snugly to the lower teeth; however, three of the boil-and-bite types do not secure snugly to the lower teeth but rely instead on an inclined plane behind the lower anterior alveolar process to hold the mandible forward (Figure 20). The amount of forward repositioning varies with the appliance and the clinician. Also, the amount of vertical opening of the mandible varies with the appliance. The rationale behind these appliances is that since the tongue attaches to the back of the mandibular symphysis, moving the mandible forward also pulls the tongue forward, thereby moving the backside of the tongue away from the posterior wall of the throat (Figure 21). Further, because the palatoglossus muscle attaches from the side of the tongue to the soft palate, pulling the mandible and tongue forward also pulls the soft palate forward, separating it from the back of the throat (Figure 22).

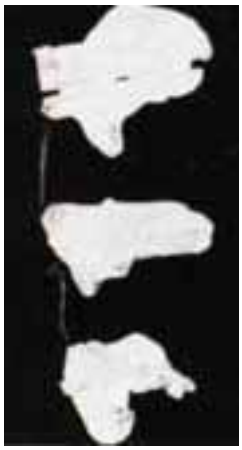

Figure 20. Inclined planes behind lower anterior alveolar process. From top: Thera Snore, Snore Free, and Snore Guard.

The mandibular advancers may be made of elastomer material or hard acrylic, or thermoplastic. They may create their retention on the teeth by friction fit of plastic in undercuts, which is most common, or by clasps.

They may use many different mechanisms to create and adjust the mandibular advancement, and the amount of advancement varies from a little to more than the patient can actively produce, depending on the patient's severity of snoring and ability to tolerate jaw joint stretch. Frequently an advancement begins at about 70 percent of a patient's maximum active protrusive ability.

Figure 21. Anatomy of tongue and throat.

Figure 22. Palatoglossus muscle.

Tongue Advancers

These devices are secured in the mouth by forming a suction on the tip of the tongue via a suction bulb that is braced against either the teeth or the lips. The size of the suction bulb is selected by measuring the thickness and width of the anterior quarter of the tongue when protruded past the teeth.

The rationale for these appliances is similar to the mandibular advancers in that the tongue and soft palate are advanced away from the back wall of the throat. These tongue advancers can be used when the jaw joints do not tolerate stretching or when there are insufficient teeth to support a mandibular advancer. These devices tend to take longer to become accustomed to than mandibular advancers

Criteria of Evaluation of Oral Appliances

Substantial experience in treating patients with oral snore devices and comparison of the features of more than 20 devices were used to develop a set of criteria to help dentists in selecting an appliance.

Those criteria are presented here for both the experienced and novice snore clinician to consider.

Reliability at Stopping Snoring

This is the most important criteria. If it is not met, the patient is not satisfied, the dentist has wasted his or her time and lost the patient's confidence, and the profession has lost credibility in the eyes of medicine. Medicine has additional criteria, which include reduction of obstructive sleep apnea, reduced oxygen desaturation, and decrease in daytime sleepiness; but these were not evaluated in the present study. A dentist must keep in mind that these medical criteria must be considered in the care of some patients, in addition to the criteria discussed in this paper. The evaluation of the medical criteria are discussed elsewhere.[9,10]

In general, the adjustable two-piece mandibular advancers are most reliable. They can be adjusted easily to increase the mandibular advancement until airway patency is sufficient.

Many of the one-piece lab-fabricated mandibular advancers are reliable, because even if the bite needs further advancing, they are easily cut apart and luted back together. An exception to this is the Elastomeric device, which is not easily cut apart nor re-luted.

Titratability

This term describes the easy ability to fine-tune the amount of mandibular advancement to keep the airway open without excessively stretching the temporomandibular joint. This criterion has some similarity with the first criterion of reliability, but this criterion specifically

monitors the ease and the gradiency of adjustments. A high score here means fewer remakes and easier delivery.

In general, the adjustable two-piece mandibular advancers score well. The TAP and Restore devices are designed to be titrated while the patient is asleep in the sleep lab without the devices being removed from the mouth (Figure 23). The EMA and Silent Nite appliances, using straps with prepunched holes, do not have as smooth a gradient of adjustments as the screw devices, but the resiliency of the EMA's straps compensates for this disadvantage (Figure 24).

Figure 23. Accessible titration mechanisms. From top: TAP and Restore.

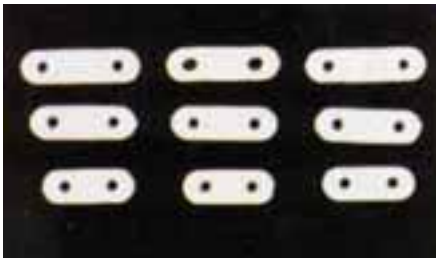

Figure 24. Strap selections for EMA.

None of the one-piece mandibular advancers, nor the tongue advancers, are easily titratable. Some clinicians may point out that the boil-and-bite devices could be reheated and advanced, but with the exception of the two-piece Thera Snore, this is not particularly easy.

Simplicity of Delivery

To be considered simple to deliver in this study, an appliance should not require more than 30 minutes of chairtime, including initial impressions, whether delivered at the first appointment, such as boil-and-bite devices or Snore X, or at the second appointment, like the other devices.

For most of the one-appointment deliveries, the boil-and-bite devices require a source of very hot water and considerable trimming and polishing of the soft material once molded. The simplicity of the single appointment delivery is offset by the amount of chairtime fitting and adjusting of the appliance, compared to the ease of trying in a completed lab-fabricated appliance at a second appointment.

Most of the commercially available devices scored highly in simplicity of delivery; and, as the practitioner gains experience, even those that might not be easy for a novice to deliver would become fairly easy.

Low Bulk

The absence of bulk makes an appliance more comfortable and therefore more likely to be worn. The feeling of excess bulk can be from the size of the appliance, the extent of its flanges, or the protuberance of its working parts. The perception of bulkiness may be experienced in the lips, cheeks, or tongue. This criterion is more subjective than the first ones and depends on variations in individual appliances as well as tolerance from the individuals wearing them. Others evaluating similar appliances might report different findings than presented here.

The low-bulk appliances are evenly divided among the one-piece and two-piece mandibular advancers.

Lip Seal

Similar to the low-bulk criterion but sufficiently different to warrant being a criterion of its own is the concept of lip seal. Appliances that allow the lips to close are more likely to be accepted by patients and are easier to get used to. Appliances that pinch the lips or have protuberances into or beyond the lips and appliances made at excessive vertical opening violate this concept, and although acceptable to some patients, are not as universally agreeable (Figure 25).

Figure 25. Violations of lip seal. Clockwise from upper left: NAPA, TRD, Snore X, Restore, TAP, and SNOAR.

Tongue Space

For the tongue to be able to move away from the back wall of the throat, it helps if the area lingual to the incisors and between the upper and lower incisors is kept free of any appliance. This provides for no irritation to the tip of the tongue and allows it to advance, which it will do if the space is provided (Figure 26). Appliances that provide an open space between the upper and lower incisors are superior for the above reason, rather than because they allow oral breathing, which many snorers do not need once the pharyngeal airway is held open.

Figure 26. Lingual view of anterior tongue space freedom. From top: Hilsen, PM Positioner, and Mandibular Positioner.

Non-interference With Sleep

Some devices can be worn and easily accommodated, without initially disturbing sleep. This criterion is similar to the low-bulk criterion but sufficiently different to warrant being a distinct criterion. These findings are very subjective and not necessarily generalizable to a large population; but some appliances, by their presence, cause many awakenings during the night and tended either to be bulky or to violate lip seal or tongue space. Most of these problems could be overcome by repetitive nights of wearing, but the investigation was intended to discover which appliances were easy to adapt to.

Temporomandibular Joint or Tongue Symptoms Easily Adjusted

This criterion is similar to the reliability of stopping snoring criterion and to the titratability criterion. If the TMJ gets sore from being stretched, the appliance

needs to be easily adjustable and adjustable unilaterally if necessary.

The developer of the TAP appliance states that unilateral adjustability of TMJ stretch has not been necessary in more than 1,000 cases treated, provided that posterior occlusion is not allowed on the appliance. However in the author's experience, in some cases the ability to adjust the bite did help the TMJ.

The two tongue advancing devices, since they do not stretch the mandible, are exempt from the TMJ criterion, as TMJ symptoms would not be expected from these two devices. However, these two devices can cause soreness to the tongue tip due to the suction, and this can also cause difficulty in accommodation.

Low Cost

Low purchase price is also a factor that influences a practitioner's thinking. Appliance cost varies from $60 to $500. Most fall between $100 and $200. If an appliance costs less than $100, it was arbitrarily considered low-cost. The cost of chairtime to deliver these appliances should also be considered, and that is discussed under simplicity of delivery.

Lateral Freedom

A frequently mentioned criterion, but a poorly studied one, perhaps less necessary than commonly believed, is lateral freedom. It is argued that bruxers need lateral freedom. The author has provided lateral freedom to many patients but found they were happier when the appliance was converted to a one-piece by luting the two splints together. This property needs to be considered for some patients but probably not most.

Weighting of Criteria

The criteria discussed above are not all of equal value or importance. It is probable they are of decreasing importance in the order they were discussed, but this opinion could vary for some patients and for other clinicians. The weighting of the criteria might be as shown in Table 1.

Other criteria that were not studied but which might deserve consideration include reliability at stopping obstructive sleep apnea, durability, and avoidance of bite changes or teeth movement.

Circumstances can change the hierarchy of criteria. An edentulous patient would probably need a TRD. A TMJ patient might move criterion 8 higher on the list. A bruxer might move No. 10 higher. If the doctor wants to try a patient's tolerance to a type of device without great expense, criterion 9 might move higher. If a dentist wants to advance the mandible extremely far, as in a severe obstructive sleep apnea case, then reliable retention to the teeth and absence of TMJ symptoms would both be the most important goals, and the TAP appliance would be more likely to be considered.

Table 1.

Weighting of Criteria.

Possible	Points
1. Reliability of stopping snoring	4
2. Titratability	2
3. Simplicity of delivery	2
4. Low bulk	2
5. Lip seal	2
6. Tongue space	1
7. Non-interference with sleep	1
8. TMJ or tongue symptoms easily adjusted	1
9. Low cost	1
10. Lateral freedom	1

Evaluation of Appliances

Nearly all the vendors of the appliances included in this study provided an appliance at no charge in order to facilitate this study. This study is a report of the author's evaluation of each appliance on himself. The author was considerably experienced in treating snoring, with experience in using several devices prior to this study.

Those who object to these findings on the basis that it is not a randomized clinical trial of sufficient magnitude are welcome to perform such a study, but in the meantime these findings are offered.

Also, these findings may be disputed by some clinicians who prefer to use devices not highly rated here. In some cases (TRD, Snore Guard, TAP, Klearway, Mandibular Repositioner, Silencer, TheraSnore, EMA), there is data and published literature elsewhere showing efficacy of appliances not highly rated here.[11-18] It is to be hoped that interested individuals will use this evaluation to choose from among the better appliances and provide even better treatment to more patients in order to continue improving dentists' confidence and medicine's respect.

Table 2 lists the appliances along with their evaluations.

The Hilsen appliance was extremely easy to deliver and very comfortable and effective. The edge of the Velcro was bothersome to the tongue until it was trimmed with an acrylic bur.

The SML Herbst was well-designed. The buccal hooks and Herbst axle positions were carefully chosen to be maximally comfortable to the lips. It lacked posterior bite blocks, so the vertical dimension was small. Its retention and advanceability probably would make it good for obstructive sleep apnea.

The EMA appliance had great lateral freedom. The rubber straps came off too easily. They are interchangeable with straps of different length and tension.

The Silent Nite appliance also had excellent lateral freedom. It was of very low bulk but probably had insufficient tooth retention for severe mandibular advancement in obstructive sleep apnea cases. The spare nylon straps could probably be more useful if shorter ones were available (the lab only provides one shorter replacement pair than the pair they install, and this only allows one protrusive adjustment). The absence of posterior bite blocks makes this appliance unsuitable for bruxers.

The PM Positioner was comparatively bulky and extremely tight-fitting on the teeth initially. It would probably be a good choice for obstructive sleep apnea cases requiring firm retention during greater mandibular advancement.

Table 2. Evaluation of Appliances

Possible Points	4	2	2	2	2	1	1	1	1	1
	Reliable No Snore	Titrate	Simple Delivery	Low Bulk	Lip Seal	Tongue Space	Non Disturb Sleep	TMJ & Tongue	Low Cost	Lateral Freedom
Elastomeric	2	-	2	-	2	-	-	-	1	-
EMA	3	1	2	1	2	1	1	1	-	1
Herbst,GLO	3	2	2	-	2	1	1	1	-	-
Herbst,SML	3	2	2	2	2	1	1	1	-	-
Herbst,Speciality	2	2	2	-	2	1	1	1	-	-
Hilsen	3	2	2	2	2	1	1	1	-	-
Klearway	3	2	2	-	2	-	-	-	-	1
Mand. Repositioner	2	-	2	2	2	1	1	1	-	-
MIRS	2	-	2	2	2	1	1	1	1	-
NAPA	2	-	2	1	-	1	1	1	-	-
PM Positioner	3	2	2	-	2	1	1	1	-	-
Restore	3	2	2	1	1	1	-1*	-	-	1
Silencer	3	2	-	2	2	-	1	-	-1*	1
Silent Nite	2	1	2	2	2	1	1	-	1	1
SNOAR	-	-	2	-	-	1	-	-	-	-
Snore Free	-	-	2	2	2	1	-	1	1	-
Snore Guard	-	-	1	-	2	-	-	-	1	-
Snore X	-	-	2	-	-	1	-	-	1	-
TAP	4	2	1	-	1	-	1	-	-	1
Thera Snore	1	2	1	2	2	-	-	1	1	-
TRD	-	-	2	-	1	-	-	1	-	-

The MIRS appliance was of low bulk and pleasant to wear. Its mandibular ramp seemed unnecessary because the mandible was held forward by friction fit on the lower teeth.

The GLO Herbst had an unnecessary transpalatal arch and uncomfortable position of the hooks and axles but was a good appliance, with sufficient retention and advanceability to probably be good for obstructive sleep apnea.

The Specialty Herbst was effective but had its hooks and axles in uncomfortable positions. It also would probably be effective for obstructive sleep apnea.

The Mandibular Repositioner was an excellent appliance, but because it is one-piece, it requires great accuracy with the wax bite and lab procedure. It was quite comfortable. It was made with clasps that were probably unnecessary because of the tightness of the acrylic.

The Silencer required fairly complex procedures at the impression appointment and was by far the most expensive. It was a very well-made appliance.

The Klearway was comfortable on the teeth with good lateral freedom, but the transpalatal advancement screw was quite bothersome to the tongue.

The Thera Snore had no firm retention on the lower teeth but relied on a lingual ramp to nudge the mandible forward. This was quite uncomfortable and ineffective compared to other devices.

The TAP fit the teeth extremely well and was capable of great advanceability and retention, probably making it good for obstructive sleep apnea. Its lingual hook

mechanism was bothersome to the tongue, and its titration knob protruded out the lips.

The Restore fit the teeth very well and would probably be good for obstructive sleep apnea. Its anterior hook mechanism sometimes pinched the lips.

The Snore Free was soft and comfortable but also had a lingual ramp instead of lower teeth retention and was therefore less effective at holding the mandible forward. Its price is low.

The NAPA was quite similar to the Mandibular Repositioner, except that it had a beak-like flat tube protruding through the lips. The beak seemed unnecessary.

The Elastomeric appliance could have benefited from a larger air hole (which the tongue could protrude into). It was comfortable and effective but very difficult to grind, which was necessary because the flanges rubbed the gums.

The TRD was comfortable but much more difficult to get used to than the mandibular advancers.

The Snore Guard seemed too small to be effective and relied on a lingual ramp to hold the mandible forward, instead of retention on the lower teeth.

The Snore X was simple to wear but produced a very sore tongue tip the next day.

The SNOAR appliance was made at a very large vertical opening, making lip seal impossible and making the appliance difficult to wear.

Discussion

The criteria for evaluating oral devices presented in this paper do not include efficacy in reducing obstructive sleep apnea or daytime sleepiness, and certainly the field of medicine would be interested in such results if available. This paper focuses more on snoring.

The criteria for selection presented here also do not include durability of the appliances, primarily because durability has not been studied yet. However, the following comments reflect the author's experience.

To keep the appliances closed, the Herbst appliances need vertical elastics that must be changed occasionally. The vendors did not supply elastics. Nor did the Herbst vendors supply advancing shims unless requested. The EMA appliance needs its elastic straps changed occasionally (frequency unknown). The Hilsen seems to fit more loosely over time, and the Velcro strips seem to partly tear loose but they are secured with monofilament thread. The PM Positioner is somewhat subject to breakage where the jackscrews are attached to the splints, especially if the appliance is removed improperly. Silent Nite splints are somewhat thin and have a history of some breakage, which may be resolved by now. The Silent Nite nylon straps are also thicker than they were previously, and they are now adjustable by the dentist in that straps of different lengths are provided along with instructions on how to change them. Also, the Silent Nite can be made of different (thicker and softer) material if the doctor notifies the lab that the patient is a bruxer. The Restore jackscrew broke off once when the appliance was assembled incorrectly but it was easily repaired with self-cure acrylic. The NAPA sometimes produced a whistling sound through its beak. The TAP is very much improved and simpler to deliver.

Another factor that has not been studied here is the tendency for appliances to move teeth or to change the bite permanently. It has been suggested at Sleep Disorders Dental Society meetings that the thermoplastic appliances may be more likely to move teeth than their rigid plastic cousins. It has also been said that shallow bites or open bites are more likely to change from wearing these devices than deep bites.

There is a wide range of behavior of the thermoplastics used in these devices, from the very soft boil-and-bite devices to the medium-soft acrylic in the Hilsen, to the very firm material in the PM Positioner. Devices using thermoplastic include PM Positioner, Hilsen, Silencer, Klearway, Thera Snore, Restore, Snore Free, TAP, and Snore Guard.

There have been people who have advocated using the less expensive devices as trial devices to see if the patient can tolerate a dental appliance, and then replacing the trial device with a more permanent long-term device. For example, it is often suggested that a Snore Free be used to see if a patient can tolerate and benefit from mandibular advancement. Also, the Snore X was developed to see if a patient can tolerate and benefit from a tongue advancing device. In reviewing the table of ratings, one can re-evaluate those suggestions. The Snore Free appliance is so inferior to the best mandibular advancers, particularly in reliability at stopping snoring and in not disturbing sleep, that a patient who did not do well with the Snore Free still might do fine with a better appliance, and the trial appliance therefore served no purpose.

However, in the case of the Snore X, which is a trial device preceding a TRD, the ratings are so close that there is no reason not to do the Snore X first. If a person could not tolerate nor benefit from a Snore X, there is some likelihood that that might be true of a TRD too.

Table 3 is a list of snore appliance vendors. There are probably additional vendors for the Herbst, Mandibular Repositioner, and PM Positioner, but this is a listing of vendors who were kind enough to provide appliances to be studied. No deliberate exclusion of other vendors was intended

References

1. Schmidt-Nowara W, Lowe A et al, Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. Sleep 18:501-10, 1995.
2. Mateika JH, Mateika S, et al, The effect of snoring on mean arterial blood pressure during non-REM sleep. Am Rev Respir Dis 145:141-6, 1992.
3. Palomaki H, Snoring and the risk of ischemic brain infarction. Stroke 22:1021-5, 1991.
4. Zaninelli A, Fariello R, et al, Snoring and risk of cardiovascular disease. Int J Cardiology 32:347-52, 1991.
5. D'Alessandro R, Magelli C, et al, Snoring every night as a risk factor for myocardial infarction: a case-control study. Br Med J 300:1557-8, 1990.
6. Norton PG and Dunn EV, Snoring as a risk factor for disease: an epidemiological survey Br Med J 291:630, 1985.
7. Partinen M and Palomaki H, Snoring and cerebral infarction. Lancet Dec 14, 1985:1325.
8. Lugaresi E, Coccagna G, and Cirignotta F, Snoring and its clinical implications. Sleep Apnea Syndromes Alan R. Liss Inc,

Table 3. Snore Appliance Vendors

Anti-snore Appliance	Price	Company Name	Address	City	State	Zip	Phone	Lab/Off-shelf
Elastomeric Sleep Appliance	$95	Great Lakes Ortho	199 Fire Tower Dr. P.O. Box 5111	Tonawanda	NY	14151	800-828-7626	L
EMA Elastic Mand. Advancer	$125	EMA Lab	1019 Foster Ave.	Coeur d'Alene	ID	83814	208-667-9497	L
Herbst	$150	Great Lakes Ortho	199 Fire Tower Dr. P.O. Box 5111	Tonawanda	NY	14151	800-828-7626	L
Herbst	$215	Space Maintainers Lab	P.O. Box 4184	Van Nuys	CA	91409	800-423-3270	L
Herbst	$130	Speciality Appliances	P.O. Box 105224	Atlanta	GA	30348	800-522-4636	L
Hilsen Adjustable Positioner	$215	Dr. Kenneth Hilsen	555 North Ave.	Fort Lee	NJ	07024	201-592-1818	L
Klearway	$250	Great Lakes Ortho	199 Fire Tower Dr. P.O. Box 5111	Tonawanda	NY	14151	800-828-7626	L
Mandibular Repositioner	$115	Space Maintainers Lab	P.O. Box 4184	Van Nuys	CA	91409	800-423-3270	L
MIRS Mand. Incl. Repos. Splint	$80	Space Maintainers Lab	P.O. Box 4184	Van Nuys	CA	91409	800-423-3270	L
NAPA Noct. Arwy. Patncy. Appl.	$134	Great Lakes Ortho	199 Fire Tower Dr. P.O. Box 5111	Tonawanda	NY	14151	800-828-7626	L
PM Positioner	$200	Harrison Lab	10740 Meridian Ave., N, Suite G3	Seattle	WA	98133	800-525-5913	L
Restore	$179	Brabant's	1616 29th St. #200	Sacramento	CA	95816	800-786-8848	L
Silencer	$500	Integrated Health Technologies	Unit C 17761 66th Ave.	Surrey	B.C., Canada	V357X1	888-575-1333	L
Silent Night	$79	Glidewell Lab	4141 MacArthur Blvd.	Newport Beach	CA	92660	800-374-7874	L
SNOAR	$104	Micro Dental Lab	6665 Amador Plaza Rd.	Dublin	CA	94568	800-229-0936	L
Snore Free	$64	Space Maintainers Lab	P.O. Box 4184	Van Nuys	CA	91409	800-423-3270	OH (heat)
Snore Guard	$195	3 Snore Guard	1100 N.W. Loop 410, Suite 718	San Antonio	TX	78213	800-680-9361	OH (heat)
Snore X	$50	Snore X	2188 Peralta Blvd.	Freemont	CA	94536	800-276-6739	O
TAP Thornton Adj. Positioner	$195	Oral Appliance Tech. Inc.	6131 Luther Lane #224	Dallas	TX	75225	214-691-4270	L
TheraSnore	$64	Distar Inc.	34748 Eubank N.E.	Albuquerque	NM	87111	800-477-6673	OH (heat)
TRD Tongue Ret. Device	$116	Professional Positioners	2525 Three Mile Rd.	Racine	WI	53404	800-742-6640	L

New York, 1978, pp 13-21.
9. ASDA Report, Practice parameters of the treatment of snoring and obstructive sleep apnea with oral appliances. Sleep 18(6):511-3, 1995.
10. Levy P, Pepin JL, et al, Management of simple snoring, upper airway resistance syndrome, and moderate sleep apnea syndrome. Sleep 19(9):S101-10, 1996.
11. Cartwright R, Ristanovic R, et al, A comparative study of treatments of positional sleep apnea. Sleep 14:546-52, 1991.
12. Schmidt-Nowara WW, Mead TE, and Hays MB, Treatment of snoring and obstructive sleep apnea with a dental orthosis. Chest 99:1378-85, 1991.
13. Thornton WK and Roberts DH, Nonsurgical management of the obstructive sleep apnea patient. J Oral Maxillofacial Surgery 54:1103-8, 1996.
14. Lowe AA, Ozbek M, et al, An adjustable oral appliance for obstructive sleep apnea, abstract presented at APSS, Nashville, Tenn, 1995.
15. Menn SJ, Loube DI, et al, The mandibular repositioning device: role in the treatment of obstructive sleep apnea. Sleep 19(10):794-800, 1996.
16. Perlstrom JR, Raphaelson M, and Alpher EJ, Oral appliance therapy for obstructive sleep apnea syndrome: progressive mandibular advancement during polysomnography. Sleep 21 Supplement:105, April 15, 1998.
17. Szakacs Z, Koves P, and Klenk G, Experiences with semiadjustable form of mandibular advancement oral appliances. Sleep 21 Supplement: 121, April 15, 1998.
18. Frantz D and Kuna S, Effect of oral elastic mandibular advancement device in treatment of obstructive sleep apnea, APSS Abstract Book No 668:334, June 10-15, 1997.

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Snore and You Sleep Alone

Robert E. Horseman, DDS

What is the greatest scourge known to mankind today? No, it's not the common cold. We have given up on the common cold except as source of revenue for manufacturers of sneezing, coughing, runny nose, fever and malaise medications. Long after even the cockroaches have departed this earth, the common cold will still be with us, defying nuclear holocaust, Armageddon, and black holes to eradicate it.

The scourge I refer to is snoring, the cause of more marital discord than indiscriminate channel-changing or wrong-end-of-the-toothpaste-squeezing. Snoring has sold more twin beds and peopled more two-bedroom homes than has ever been recorded.

At night, I lie in my customary fetal position, blankie drawn up snugly about my ears, thinking about a terrific idea I have for a comic strip featuring an engineering nerd and his pet dog. The nerd's loftiest ambition is to survive his mind-numbing life in a cubicle, while his dog is busy trying to take over the world by posing as a business consultant. I am considering the money-making possibilities of this when I receive a sharp poke between the shoulder blades.

"Stop snoring!" my helpmate demands.

"Snoring? Who's snoring? I'm wide awake," I point out.

"You're snoring," she insists. I deny even the possibility of this and return to my meditations only to receive, two minutes later, another blow, considerably sharper than the first. This tableau has become a nocturnal ritual, leaving me with enough contusions to qualify for abused spouse protection.

I decided to do some research on snoring to buttress my position. Centuries ago, it seems, snoring was thought to be the result of demons within the skull trying to get out at night. To test the validity of this theory, snorers frequently had their skulls clove by dedicated researchers; and, sure enough, the snoring stopped as the demons escaped.

More recent studies have shown that snoring is the direct result of breathing; and scientists discovered that if you could stop a snorer from breathing long enough, the problem would disappear. Also disappearing is the stereotype of the typical snorer: a man (women do not snore as they do not sweat as they do not grow hair in their ears) lies flat on his back, mouth open, from which arises a line of little "z's" terminating in a balloon containing a log being cut by a saw. The descriptive words for this act look something like "snor-r-f," "bla-a-ff." and sometimes "y-o-on-n-k."

Meet the new breed of snorer. Contrary to the stereotype, I can demonstrate the ability to snore while lying on my

side, mouth clenched shut while thinking that I am wide awake. If I apparently can't distinguish between being awake or asleep, I may have a problem more serious than just snoring. Besides my sounding like an International Harvester during the height of the season, my bride claims that occasionally I go for long minutes without breathing at all, followed by an explosive snort to make up for lost time.

My research shows this to be a phenomenon known as sleep apnea that is considered by students of sleeping disorders to be a serious problem. Having always been the type of person who will face his problems whenever there appears to be no other way out, I have sent away for a device known as an oral proprioceptive stimulator. This is a plastic appliance to be worn in the palate at night and resembles a flipper without teeth, but with a movable flap at the distal of the soft palate that pushes the base of the tongue down while the wearer attempts to keep his dinner down.

The theory behind its operation is something I don't have time to understand, nor the capacity to do so. I bought it as an antisnoring machine; and although the jury is still out, I think the portents are good. My wife says she thinks it may be working. She came in from the other room and woke me up to tell me this. I was pleased, as you can imagine I would be, to be awakened at 2 a.m. with this kind of information. As it turns out, being aroused periodically is not a bad idea if you wish to avoid one other nocturnal problem, that of drowning in your sleep. My salivary glands, which seem to be the last of my glands to show the ravages of time, are producing upwards of 50 gallons of saliva every night in a frantic effort to wash out the appliance before morning.

I think young people who are out tomcatting around all night when they have the natural ability to sleep straight through from 10:30 p.m. until 9 a.m., would do well to listen to the laments of their elders who can never remember having had this blessing once. Grab as many zeds as you can while you're young, kids, there will be plenty of time at night later to consider other pursuits like wondering if there is any Alka-Seltzer in the cabinet or trying to determine what time the luminous dial on the clock says without finding your glasses first.