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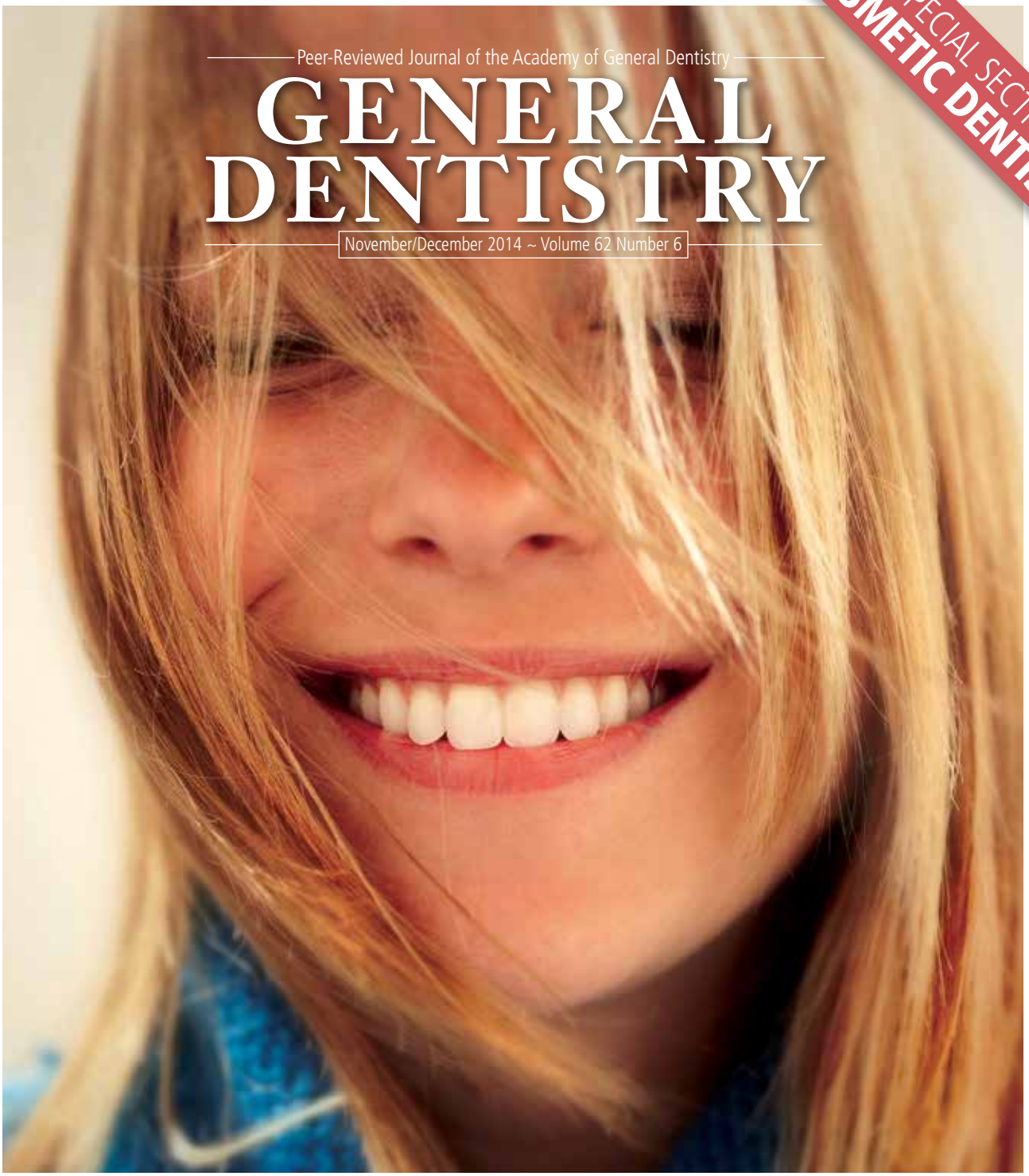
GENERAL DENTISTRY

November/December 2014 ~ Volume 62 Number 6

SPECIAL SECTION
COSMETIC DENTISTRY



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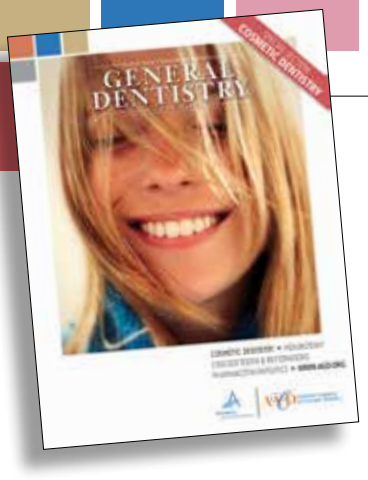

Contents

Special section on cosmetic dentistry

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Coming next issue

In the January/February issue of *General Dentistry*

- p53 expression in oral lichenoid lesions and oral lichen planus
- Local anesthetic calculations: how to effectively treat pediatric patients
- Effect of imaging powders on the bond strength of resin cement

In the November issue of *AGD Impact*

- AGD Special Military Issue: Stories from Region 17
- Redefining Case Presentation

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Dental Materials Update 2015

Johan Figueria, DDS
Communication Protocols with Photography

Edmond R. Hewlett, DDS
Lifelike Composites – It's not Complicated

Brian P. LeSage, DDS, FAACD
CADCAM and Composite Layering

Jacinthe Paquette, DDS
Evolving Concepts of Patient Care

Giuseppe Romeo, MDT
Technical Diagnostic and Esthetic Approach

Richard G. Stevenson III, DDS, FAGD, FACD, ABOD
Occlusal and Polishing Considerations to Minimize Fractures

Robert R. Winter, DDS
Outcome-based Preparation Design


Brucia


Figueira


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Tara Aghaloo, DDS, MD, PhD
Peri-implantitis: Diagnosis, Treatment, and Prevention

Edmond Bedrosian, DDS, FACD FACOMS
Demystifying the options for the compromised maxilla with a fixed prosthesis

Joseph Kan, DDS, MS
Anterior implant aesthetics

Perry Klokkevold, DDS, MS, FACD
Interdisciplinary Implant Treatment in the Aesthetic Zone

Baldwin Marchack, DDS, MBA
Successful Implant Prosthetics: Avoiding Failures and Complications

Peter Moy, DMD
Guided Approach to Implant Dentistry: From work-up to Delivery

Kumar Shah, BDS, MS, FACP
Implant Occlusion and Occlusal Materials

Chandur Wadhwani, DDS, MSD
Implant Restoration 2015 - 5 keys to prevent problems


Agahloo


Bedrosian


Kan


Klokkevold


Marchack


Moy


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Professional growth

The memories and relationships of a long professional career have their high and low points. Hopefully, we remember the former and let the latter fade with time. Our professional growth is attributed to the relationships we developed along our path toward wisdom. As Louis L'Amour wrote,

The best of all things is to learn. Money can be lost or stolen, health and strength may fail, but what you have committed to your mind is yours forever.

It has been said that the largest room in the world is the room for improvement.

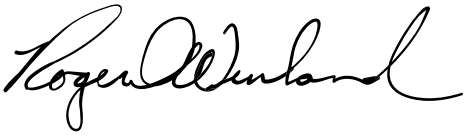
Nature has a beautiful way of illustrating the growth process. There is an inspirational story about how a Chinese bamboo tree seed is planted, watered, and fertilized, yet no growth is visible for the first 4 years. This all changes in the fifth year when the bamboo tree grows 90 feet in 6 weeks. The lesson from this story is that even though no growth is visible, the tree's root system grew and flourished in the first 4 years, giving the tree a strong and sturdy base on which to grow. In many ways this story is a parable for dental school.

On our journey as dental professionals, character is determined by what we accomplish when the excitement of beginning a career is gone. We must maintain the energy to keep growing because our patients deserve nothing less. As B.C. Forbes stated, "It may be all right to be content with what you have, never with what you are." It has also been said that of all the human resources, the most precious is the desire to improve. Most people want to improve themselves, but not too many are willing to pay the price to work at it.

We are all born with abilities, and we are given a number of opportunities to develop them. Life is like a game of cards. We dentists must accept the cards dealt to us, but once those cards are in our hands, we alone decide how to play them to win the game of life. We must decide whether to take a risk and act. Nothing in life provides more opportunities for growth than new experiences, and our lives have the power to touch other lives in either positive or negative ways.

Have you ever been "caught in the doldrums?" The doldrums near the equator abound with everything from thunderstorms to squalls to light variable winds. During sailing times, restless crews on ships often felt helpless waiting for the wind to blow. Sometimes as professionals, we may get caught in the doldrums. We wait for an external force to create change; if nothing presents itself, we may become dispirited and uninspired. Well, personal and professional growth does not suddenly manifest itself. We need to take a conscious step out of the doldrums in order to reach our goals.

David Jordan wrote, "Wisdom is knowing what to do next, skill is knowing how to do it, and virtue is doing it." Claude Pepper observed that, "Life is like riding a bicycle; you don't fall off unless you stop pedaling." You have the potential in you to grow as a professional; you've just got to work to achieve it.



Roger D. Winland, DDS, MS, MAGD
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Reference

Parables of the Kingdom. *Good Seed on Good Soil.* Available at: http://users.stic.net/fortress/pr-kingdomkeys-parables.htm. Accessed September 25, 2014.







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Happiness is something that happens when we chase success.
—August Bruguera

It's an honor to be asked to contribute a few words to your publication. Both the *Academy of General Dentistry* (AGD) and the *American Academy of Cosmetic Dentistry* (AACD) are dedicated to lifelong learning, and they are really more alike than different. Here are a few thoughts on our 2 academies.

What is cosmetic dentistry? For years, dentists and the public have tried to pin down this definition. In my view, every restorative procedure that I perform has an esthetic component. From posterior composites to complete dentures, as restorative general dentists, my team and I pay close attention to "making it look real." As form follows function, if it looks real, there is a good chance that it will function properly as well. One of my mentors said,

Cosmetic dentistry ought to be just good, restorative dentistry.
—Larry Addleson, DDS, AACD

There are several similar associations that offer an advanced award based on an examination, yet there is no *American Dental Association* (ADA)-recognized specialty in *Cosmetic Dentistry*. The world has virtually shrunk as the information age has burgeoned, and cosmetic dentistry has become more mainstream. Any dentist with a license to practice can claim expertise in the art, but the AACD's *Accreditation* award is thought to be the most valid of its kind in the industry (of course I would say that!). The AACD Accreditation program is based on 3 pillars: a written examination, anonymous submission of 5 clinical case types, and finally, an oral examination in which the candidate's cases are defended before a group of 5 seasoned examiners. Advanced recognition of one's skill is the AACD's *Accredited Fellow* award, which requires the submission of 50 documented cosmetic cases.

As you know, the AGD has 2 recognition awards as well, and many AACD members have completed the AGD's *Fellowship* examination and gone on to attain *Mastership*. The AGD's examinations touch on all dental disciplines. In contrast, AACD Accreditation's 5 clinical case types are specific to disciplines that relate to restoration of maxillary anterior teeth, and it incorporates elements of periodontics, prosthodontics, orthodontics, photography, and surgery, as well as dental anatomy, physiology, and occlusion.

Cosmetic dentistry is a specific prosthodontic discipline, requiring… study and practice…
—Daniel Mayeda, DDS, AACD

Although the focus of the AACD may be more limited than that of the AGD, there are many parallels between the 2 organizations.

You're likely to find the same speakers at both AGD and AACD conferences. In fact, the Executive Directors and Executive Committees of both organizations have met formally several times to discuss collaborative efforts.

Both associations offer marvelous opportunities for quality Continuing Education (CE); both have virtual learning programs. The question is, how do we sell ourselves to our future members?

The trend toward virtual CE and inexpensive online courses has challenged the efforts of both academies to hold an annual "brick and mortar"-based event. And there is a multitude of course offerings on many subjects throughout the year. It's a challenge to pick and choose the ones that offer the most bang for the buck.

What if we looked at the problem in the same way that our dental patients of today shop for dentistry? Today's consumer is looking for value, asking about price, and searching for quality. It's time to stress a value-based approach to membership, one that addresses the problems facing young dentists.

According to Marko Vujicic, Chief Economist and Vice President of the ADA's Health Policy Institute, our profession is changing more and more rapidly. In a recent presentation to some of North America's most respected clinicians and researchers, Dr. Vujicic said that 46% of dentists under the age of 35 are employees.[1] This trend is not likely to level off, either.

In the face of staggering student loan debt (averaging more than $240,000 for 2013 graduates), it often makes sense for a new graduate to "get a job," free of management headaches.[2] So why not just get a job and go to work? Can you say, "paid vacation"?

There will always be a percentage of new dentists who "know that they don't know" and who are willing to invest their time and hard-earned money to learn. For both organizations, this is the core of our target market for new members. What have you done to help your academy? Do you mentor, do you enthusiastically support its efforts, do you encourage other professionals to join?

Your academy is ***YOU***, and you are an ambassador. For the health of our profession, help someone to grow as you have been helped. We're all in this together!

James H. Hastings, DDS
President, American Academy of Cosmetic Dentistry

Acknowledgments

Mr. Bruguera is a renowned dental laboratory technician from Barcelona, Spain. Dr. Addleson is an Accredited Fellow and Past President of the AACD (2004-2005), who resides in Kahului, Hawaii. Dr. Mayeda is an Accredited Member and Past President of the AACD (1995-1996), who resides in San Diego, California.

References

1. Vujicic M. *A Profession in Transition.* Presented at the American Academy of Esthetic Dentistry 39th Annual Scientific Session. Santa Barbara, CA: August 7, 2014.
2. Palmer C. ADA urges student loan reforms. *ADA News.* Available at: http://editiondigital.net/publication/?i=204351. Accessed September 25, 2014.

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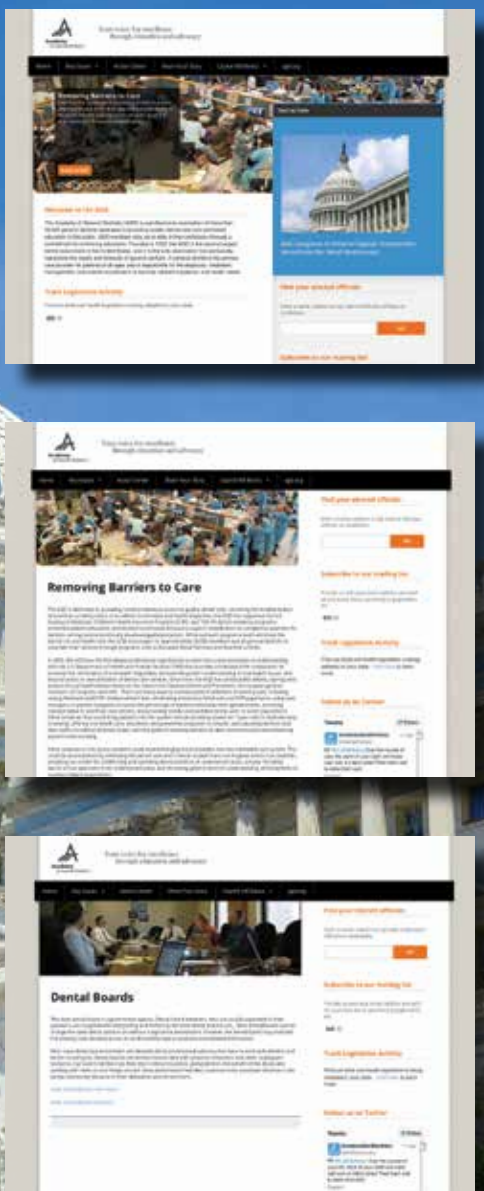

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Perpetuating old myths

I read with dismay the column by Mr. Thomas Viola, "Co-administration of oral contraceptives and antibiotics used in dentistry," in the May/June 2014 issue of *General Dentistry.* Mr. Viola is a pharmacist, not a pharmacologist. I question his expertise in advising dentists on prescribing, as he is neither qualified nor licensed to prescribe.

My 2012 continuing education course, "Pharmacological Aspects of Dental Practice," through Daniel E. Becker, DDS, refutes the concerns raised in this article. We have enough issues to deal with as practitioners that we do not need these old myths to be perpetuated in your journal.

Robert D. Frey, DDS, MAGD
Swanton, OH
(with enclosures)

Response from Mr. Viola

I read with equal dismay the respondent's critique of my column in the May/June issue of *General Dentistry.* A fair examination of the respondent's comments leads me to question his acumen in evaluating the information as presented in such clinical literature without reader bias.

The respondent's concern stems from his contention that my article "perpetuates old myths" and that these "myths" were refuted by Dr. Daniel Becker's work in his 2012 text "Pharmacological Aspects of Dental Practice." The respondent highlighted a section which states "To date, rifampin (an antituberculosis agent) is the only antibiotic having a confirmed interaction with oral contraceptives" and "To date, all human studies measuring the influence of antibiotics on estrogen and progestin serum concentrations have found no interaction with antibiotics other than rifampin." With all due respect to Dr. Becker and his work, while the text that incorporates these passages may be dated 2012, the sources he references for these passages are dated 1971, 1990, and 1994.[1-4]

In my article, I make the following points as clearly as possible:

- "Reports in the literature exist regarding the theory that antibiotics, especially those used routinely in dentistry, can reduce the bioavailability and, thus, the efficacy of contraceptives. Yet, numerous studies have concluded that there is little evidence to support the proposed interaction."
- "There is a lack of scientific evidence supporting the theory that antibiotics, especially those used routinely in dentistry, reduce the effectiveness of oral contraceptives."
- "With the exception of rifampin and rifabutin, studies involving other antibiotics failed to demonstrate a significant effect on serum concentrations of contraceptive components."

These points are based on references from sources dated 2001, 2002, 1999, and 1997.[5-8]

If the respondent's concern stems from the last passage of my article, "Alternative forms of contraception, including barrier methods, may be recommended for those women taking antibiotics and oral contraceptives concomitantly and who may be intolerant of any risk of contraceptive failure," let me remind him that my role in authoring these articles is not to advise dentists on how to prescribe, but to merely present the information as discovered through my research of the applicable clinical literature. Once again, I must question the respondent's acumen in evaluating the information as presented without reader bias.

I take issue with the respondent's statement that, as a pharmacist, I have "questionable" expertise in advising dentists, since I am not "qualified nor licensed to prescribe" myself. To make such a statement clearly undervalues and disqualifies the valuable and life-saving work performed every day by pharmacists in monitoring drug therapy, evaluating clinical drug literature, performing drug utilization reviews for interactions and contraindications, and, yes, even advising prescribers when clinical interventions are necessary, even though pharmacists are not licensed to prescribe.

In conclusion, let me state that it appears the respondent's intention was to imply that if I were a "pharmacologist," I might finally possess the necessary qualifications to advise dentists on prescribing. If this is the case, I find it necessary to inform the respondent that pharmacologists, on the basis of that credential only, are also not licensed to prescribe, unless, of course, they possess some other credential that entitles them to prescriptive authority in their jurisdiction.

References

1. Reimers D, Jesek A. The simultaneous use of rifampicin and other antitubercular agents with oral contraceptives [in German]. *Prax Pneumol.* 1971;25(5):255-262.
2. Back DJ, Orme ML. Pharmacokinetic drug interactions with oral contraceptives. *Clin Pharmacokinet.* 1990;18(6):472-484.
3. Becker DE. Drug interactions in dental practice: a summary of facts and controversies. *Compendium.* 1994;15(10):1228-1242.
4. Miller DM, Helms SE, Brodell RT. A practical approach to antibiotic treatment in women taking oral contraceptives. *J Am Acad Dermatol.* 1994;30(6):1008-1011.
5. Archer JS, Archer DF. Oral contraceptive efficacy and antibiotic interaction: a myth debunked. *J Am Acad Dermatol.* 2002;46(6):917-923.
6. Dickinson BD, Altman RD, Nielsen NH, Sterling ML; Council on Scientific Affairs, American Medical Association. Drug interactions between oral contraceptives and antibiotics. *Obstet Gynecol.* 2001;98(5 Pt 1):853-860.
7. Weaver K, Glasier A. Interaction between broad-spectrum antibiotics and the combined oral contraceptive pill. A literature review. *Contraception.* 1999;59(2):71-78.
8. Helms SE, Bredle DL, Zajic J, Jarjoura D, Brodell RT, Krishnarao I. Oral contraceptive failure rates and oral antibiotics. *J Am Acad Dermatol.* 1997;36(5 Pt 1):705-710.

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Only informed patients can decide what serves them best

Mark Malterud, DDS, MAGD

Minimally invasive biomimetic dentistry (MIBD) can cover the gamut of the dental spectrum. When we review how to best care for a patient, we need to pay close attention to what is desired by the patient and then come up with a treatment plan that will satisfy their desires without compromising our values or ethics in the delivery of care. Educating the patient about the process, procedures, outcomes, and long-term effects of their decisions is the least we can do to help them decide. The following case documents the presenting wishes of a patient and the minimally invasive options that were delivered to satisfy her desire for a nicer smile.

A 27-year-old woman contacted our office upon referral from a friend, who indicated that our office could help with the patient's smile. The patient was very concerned about the discoloration of and spaces between her teeth, both of which created what she considered to be a less than ideal smile. She wanted to have a consultation to begin the process of having veneers created by our office. She informed us that she had been saving for a while and wanted to have a smile of which she could be proud.

A consultation was scheduled for the patient, along with a review of her records to help determine the best treatment. During the consultation, the patient indicated that she disliked the color of her teeth and the white spots on them, as well as the space between her front maxillary incisors. Her desire was a quick fix of veneers on her 6 front maxillary teeth. During the consultation, she also indicated concerns about her overall health and that she really didn't want to have her teeth prepared in any particular way, but was unaware of any other options to meet her esthetic needs.

As is evident in her initial photo, the patient's concerns were valid as she had discolored teeth and an obvious diastema between teeth No. 8 and 9 (Fig. 1). The white spot lesions seemed to indicate fluorosis during the development of the teeth. There were no other restorative needs noted as the patient had just finished her semiannual exam and hygiene appointment; at that point, small lesions on teeth No. 12 and 13 were diagnosed and fillings were completed. The occlusal view of the maxillary arch shows that there was some wear and tear of the posterior teeth as well as restorations on teeth No. 12 and 13 (Fig. 2). Some erosion was noted on the occlusal surfaces of the posterior teeth, so a discussion of habits and health was initiated and the patient disclosed that she frequently drank carbonated soft drinks. A frank discussion ensued about the effects of home care, diet, and oral habits on restorations or any dental treatments we might provide. Upon further discussion, the patient indicated that since childhood, she had frequent medically diagnosed migraines for which she was taking "breakthrough" medications. As shown in Figure 3, her posterior occlusion was well-aligned, and areas on the buccals of the molars indicated where the caries process had likely began. Those areas now showed hard, glassy, smooth, stained remineralization.

Multiple treatment options were presented, but they all began with utilizing an anterior midpoint splint to evaluate the muscle activity and determine whether the years of migraines could have been caused by parafunctional habits, such as primary clenching. Once we determined the efficacy of splint therapy, we could then proceed with other aspects of the patient's care.

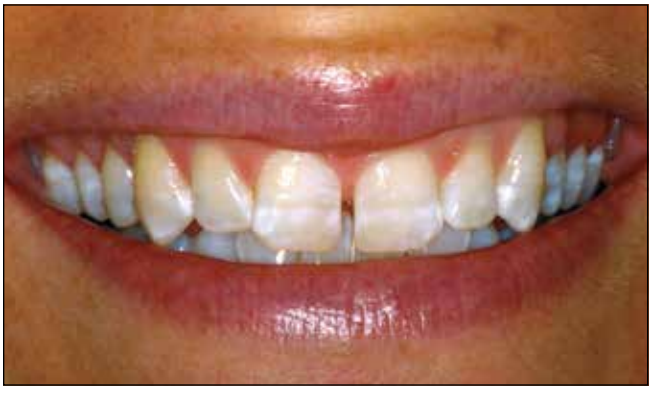

Fig. 1. Smile presentation at initial consultation.

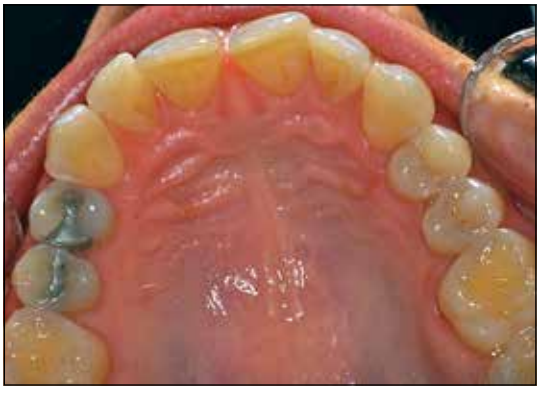

Fig. 2. Pretreatment maxillary occlusal view showing recent amalgam fillings.

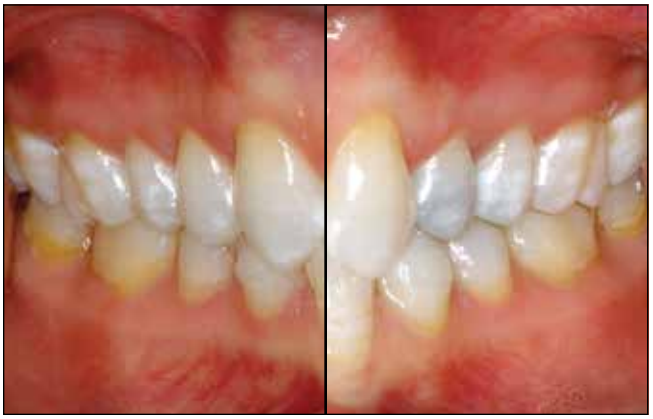

Fig. 3. Anterior views showing areas of hypermineralization along with remineralized buccal surfaces of the posterior molars leaving brown spot lesions.

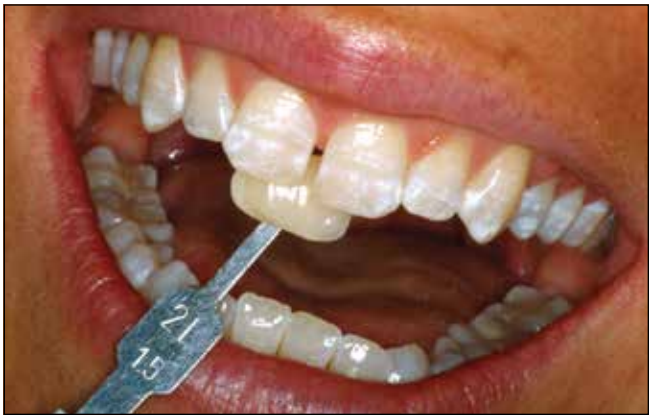

Fig. 4. Color match to the shade guide. Note that the fluorosis stains appear much whiter and opaque.

With a focus on minimally invasive dentistry, the following treatment options were presented to the patient:

- The first treatment option would begin with diagnostic splint therapy, followed by teeth whitening, orthodontics to reduce the deep bite, closing of the diastema, then straightening the upper and lower teeth. With clear aligner therapy proposed, it was estimated that the treatment would be finished in <1 year, and she would not need to have any restorative work done on her teeth unless the tooth whitening didn't blend the white spot lesions in well enough to accomplish her esthetic desires.
- An alternative option involved splint therapy, teeth whitening, and then closing the diastema with bonded resin restorations utilizing virtually no preparations; this would take mere weeks to accomplish.
- A third option—the one the patient initially requested—was to veneer the teeth.

We suggested starting with splint therapy, followed by whitening to try to keep the veneers to a minimum number of teeth and to match the color of the lower anteriors to her upper veneers. This treatment would be completed in <2 months, depending on the success of the whitening process.

Once presented with these options, it was reiterated to the patient that each treatment would began with diagnostic splint therapy followed by a whitening process; at that point, she could make a final decision as to how she wanted to proceed once the whitening effects became evident.

After accepting the initial part of her treatment plan, the patient had impressions taken to fabricate her anterior midpoint stop splints and to fabricate whitening trays that would be used for at-home bleaching after the in-office whitening treatment. An initial shade was taken to use as a baseline to determine the efficiency of both whitening processes (Fig. 4). After the in-office whitening, the patient followed up with home trays for use with a 15% whitening solution for 2 hours/day for an estimated 2 weeks. The immediate results of the in-office whitening were quite dramatic (Fig. 5). However, some regression of the shade shift is a reality, so the follow-up trays for home use were used to complete the whitening process.

Fig. 5. Photograph taken after in-office bleaching procedure. Note the immediate shade has improved dramatically.

Once the patient saw how well the whitening process worked, we sat down to discuss the subsequent treatment alternatives and costs. It became apparent to the patient that, for her personal goals, the treatment involving orthodontics was an excellent and cost-effective option to align her teeth, close the space between her 2 front teeth, and reduce her deep bite.

Orthodontic records were taken and a very detailed vinyl polysiloxane (VPS) impression was taken to submit for digitization to Align Technology, Inc. for their computer-generated ClinCheck, which allowed the patient to visualize the stages of movement that were proposed for her care. The ClinCheck was returned in <2 weeks, and the patient presented for her final decision on which treatment she would choose. After seeing how her teeth were aligned in the final trays of the aligner treatment she decided to pursue the treatment with clear aligner therapy. Her treatment progressed with only a minor midcourse correction due to the bell shape of her natural teeth, which didn't allow the mesioincisal contacts between teeth number No. 8 and 9 to contact far enough gingivally to keep from potentially

Fig. 6. Anterior views. *Top.* Bell shaped contact between teeth No. 8 and 9 that didn't allow the full closure of the diastema. *Bottom.* The minimal amount of enamel recontouring that allowed the teeth to be taken to a more predictable final position.

Fig. 7. Facial view showing smile presentation posttreatment with a closed diastema, reduced deep bite, whitened teeth and the minimized appearance of the white spot lesions.

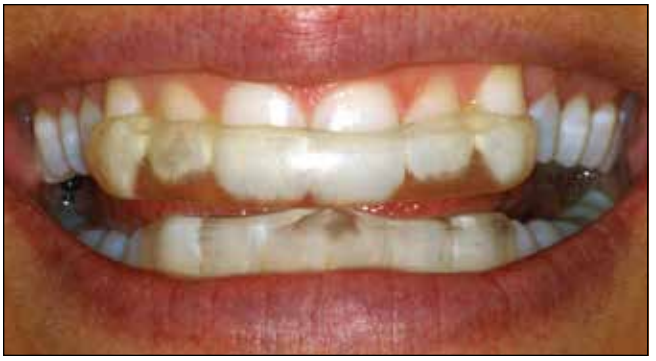

Fig. 8. Facial view showing the specialized retainers that maintained the positions of the teeth and reduced the muscular parafunction that had exacerbated the migraine headaches.

creating a midline black triangle or interproximal gingival void (Fig. 6). Therefore, teeth No. 8 and 9 were treated with a very mild enameloplasty to reshape them in an effort to move the contact closer to the crest of the bone between them, to allow a full fill of soft tissue. A new VPS impression was taken and the final aligners were created to take the case to its final position.

The final alignment satisfied the patient's desire for closure of the gap between her 2 front teeth, along with reducing the deep bite that was present at the start (Fig. 7). Following a minimally invasive philosophy, this treatment created a beautiful smile that the patient can enjoy for a lifetime. To keep the smile stable will require retainers, and in this case—the patient being a migraine sufferer—the retainers were in the form of custom-fabricated anterior midpoint stop splints that were worn only at night (Fig. 8). These splints not only retained the teeth in their proper positions and prevent the wear and tear of her parafunctional activity, but they also reduced both the frequency and intensity of her migrainous episodes.

MIBD is often considered as dentists diagnose and treat tiny lesions with small preparations or save as much tooth structure as possible while restoring teeth back to their full form, function, and esthetics; but it goes much deeper than that. Often when following a MIBD approach, treatment can be completed in a way that requires little or no restorative care. This case is an illustration of what can be accomplished without destroying large amounts of tooth structure and consequently setting the patient up for a lifetime of maintenance and re-restorations. If the patient in this case had received her desired 6 veneers across her front top teeth, she might have embarked upon a lifetime of repair and replacement of these veneers.

With the patient being 27 years old, there are many years of wear and tear that will occur on even her natural teeth. Hopefully the chosen course of treatment will allow her to enjoy her own teeth for years to come; and we know that should she have any issues, there are many options that will allow us to bring each individual damaged tooth back to full form, function, and esthetics with minimal treatment.

As dentistry moves forward, there will always be better materials, equipment, and techniques developing that will allow us to restore teeth more conservatively and predictably. A well-informed patient is the only person that can make the proper decision as to what is best for their given situation and desires. Educating them on the long-term consequences and outcomes of their choices in treatment is our duty as dentists.

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Dr. Malterud is in private general practice in St. Paul, Minnesota. He has practiced some form of MIBD for over 30 years and has been lecturing and publishing on MIBD for more than 18 years.

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Local anesthesia for restorative dentistry

Roger A. Solow, DDS

Dentists must be proficient in multiple routes of local anesthetic (LA) administration so that profound anesthesia can be achieved in a variety of clinical situations. LA for intraoral procedures consists of either nerve blocks that place the anesthetic at the nerve trunk, or infiltration where the anesthetic is placed near the nerve terminus at the root apex. Nerve blocks provide the most intense and longest anesthesia; however, they are more technique-sensitive and incur the risk of direct needle damage, intravascular placement, muscle trismus, hematoma, and delayed onset.

The choice of the type of anesthetic depends on several factors: level and duration of anesthesia desired, tooth site, pulp or soft tissue infection, and the patient's physical and emotional status. LA for the restorative treatment of healthy, asymptomatic teeth may not demand the same level or duration as other procedures, such as endodontic treatment of painful teeth. This article presents the author's rationale for LA selection with adult restorative dentistry.

Routes of anesthesia

Maxillary teeth are covered by porous facial bone that is <1 mm thick at the root apices.[1] These teeth can be predictably anesthetized by buccal infiltration (BI) without the risk of injury from a posterior superior alveolar nerve block if the needle is placed too distal into the vascular plexus.[2] Additionally, 28% of patients have the first molar mesiobuccal root innervated by the middle superior alveolar nerve, thus requiring separate infiltration after the nerve block.[2]

Mandibular anterior teeth and bicuspids can be similarly anesthetized with BI. Thicker buccal cortical bone is the main reason that conventional inferior alveolar nerve blocks (IANB) are used for mandibular molar LA.[3] However, Robertson et al and Jung et al found that BI at the mandibular first molar was faster and more successful than IANB.[4,5] El-Kholey reported a 93% success rate using BI at the first molar to remove impacted third molars.[6] BI avoids the failures of IANB that may be related to a broad sphenomandibular ligament medial to the mandibular foramen, accessory innervation, retromolar foramina, or rare bifid mandibular nerves.[7,8]

Four percent articaine with epinephrine 1:100,000 (A100) is the most diffusible LA and has been shown to be superior to 2% lidocaine with epinephrine 1:100,000 for BI alone, as well as a supplement to IANB.[9-13] The level and duration of anesthesia with A100 is dependent on the dosage.[14,15] Meechan et al demonstrated that BI is much more effective than lingual infiltration for mandibular teeth.[16] Meechan attributed the effects of mandibular BI to anesthetic diffusion through the mental foramen as a type of mental/incisive nerve block.[17] Patients in studies using 27- and 30-gauge needles for BI were unable to sense a difference.[18,19] BI is placed in loose tissue with no large vessels present, and the rate of positive aspiration is neglible.[2,20] Delgado-Molina et al found no difference in positive aspiration between 27- and 30-gauge needles during IANB.[20] Thirty-gauge needles penetrate the mucosa more easily than 27-gauge and slow the flow of anesthetic, which is important for comfort; however, both 27- and 30-gauge needles are acceptable for BI.[19,21,22]

The BI of 1 carpule A100 is typically successful for all maxillary and mandibular anterior teeth or premolars. A BI of 1.5-2 carpules A100 is appropriate for mandibular molars at the first molar site. If the anesthesia is deemed not sufficient (usually when a mandibular molar is involved), then additional BI can be given or

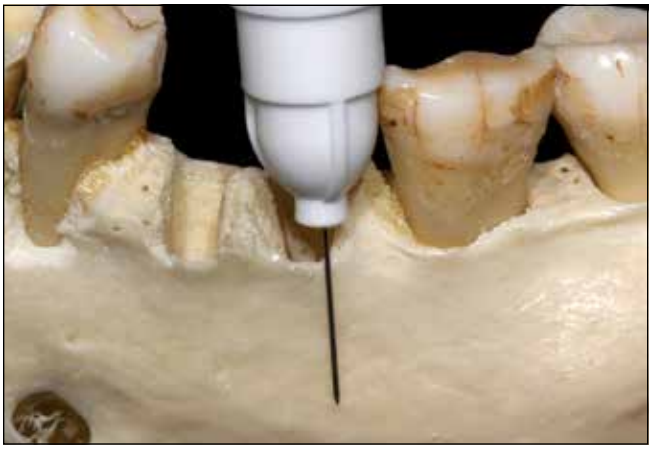

Fig. 1. Position of a 30-gauge extra short needle for buccal infiltration. Needle placement at the depth of the vestibule approximates the root apex.

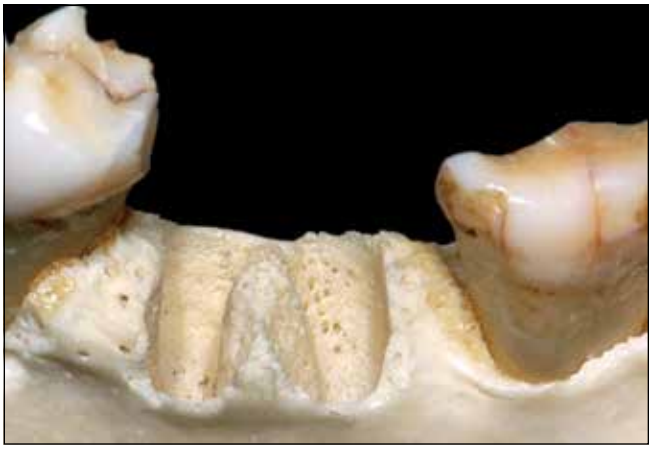

Fig. 2. Mandibular molar site displaying multiple bony crest and cribiform plate nutrient canal foramina.

Fig. 3. Position of a 30-gauge extra-short needle for periodontal ligament injection. The needle is aligned with the long axis and advanced until resistance is felt.

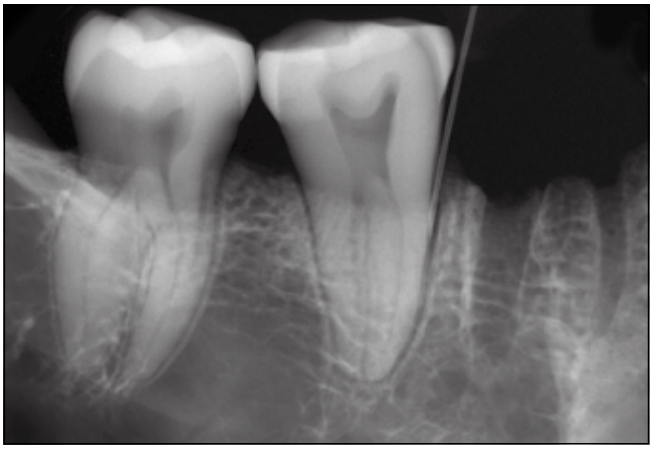

Fig. 4. Dry specimen showing periodontal ligament injection. Note the needle bevel is toward the root and vertically oriented.

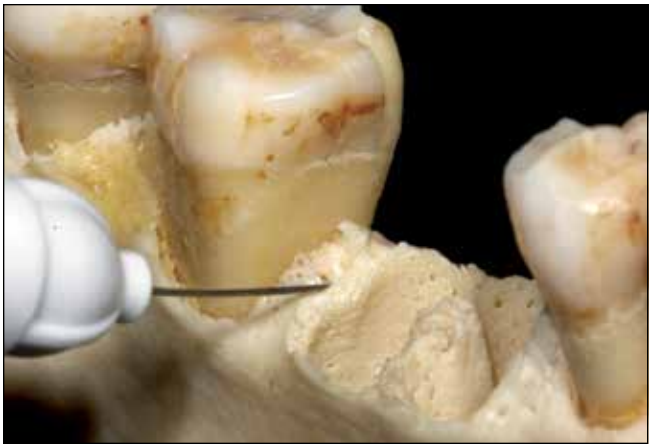

Fig. 5. Position of the 30-gauge extra-short needle for crestal anesthesia. The bevel of is placed at 45 degrees and is adjacent to the crestal bone.

alternate routes can be used: periodontal ligament (PDL), crestal, conventional IANB, or a higher nerve block (such as Gow-Gates or Vazirani-Akinosi). Lingual infiltration of A100 at the mandibular molar has not been shown to be effective.[23]

Technique

First, determine if LA is needed. Many minimally invasive restorations on older patients can be done with a light touch, new bur, and water spray. If the dentin is sensitive to a sharp explorer, then LA is indicated. There are some patients who seldom require LA. If LA is needed, dry the mucosa and place a 20% benzocaine topical gel for 2 minutes. Stretch the mucosa fully and rapidly penetrate the surface at the depth of the vestibule with a 30-gauge extra-short needle so only the bevel is submerged. Topical anesthesia extends several millimeters subsurface, so this critical step is typically painless. Express a single drop of A100 so that the membrane forms a bleb and wait 10 seconds without moving.[24] Repeat this step with another drop. Advance the needle several millimeters apically staying within the anesthetized bleb, aspirate, and express the rest of the carpule over 1 minute. Stop at any time that the patient has any discomfort. Proper placement of the needle approximates the root apex, and the A100 is highly diffusible, so little advancement is needed (Fig. 1). Allow 5 minutes of absorption time before the procedure. If soft tissue anesthesia is needed on the lingual or palatal surface, sequentially introduce A100 in the interdental papilla and then the lingual or palatal. Allow enough time so that each injection occurs into tissue that is already anesthetized, as evidenced by blanching. In most cases, 60-75 minutes of pulpal anesthesia allows most restorative procedures to be performed.[2]

Multiple teeth can be anesthetized sequentially instead of using IANB. The author prefers a 30-gauge extra-short needle because it is less unwieldy than a long needle, which may inadvertently touch the lip or tongue. The extra-short needle can also be used for PDL or crestal anesthesia, if needed, without switching needles. Computerized delivery and vibration devices are effective but not required, as patients often volunteer that they "didn't feel the injection at all" with this slow approach.[2,25]

Both PDL and crestal injections place a small amount of anesthetic (0.2 ml) in a confined space so that it is forced through the small foramina of the nutrient canals in the crest and cribiform plate of the alveolus (Fig. 2). The path of anesthetic is not through the PDL to the apical nerve terminus; it travels through the marrow spaces.[26] These are highly effective injections without the need for perforation of the cortical bone with an intraosseous injection. Proper placement of the needle to obtain resistance during injection is a prerequisite for success.[27,28] For the PDL injection, the needle is vertically aligned with the long axis of the tooth and advanced with the bevel toward the root until resistance is felt (Fig. 3 and 4). In cases of crestal anesthesia, the needle is placed at 45 degrees to the bone crest and advanced until the interproximal bone crest is felt (Fig. 5).

Giffin defined crestal anesthesia as a subperiosteal injection where the needle is adjacent to bone.[29] Malamed described intraseptal anesthesia as similar, but the needle is advanced 1-2 mm into bone.[2] Kaemmerer et al found that injection of anesthetic into the soft tissue papilla remained in the soft tissue and was inferior to PDL injection for pulpal anesthesia.[30] Malamed reported 100% success with PDL injection for tooth extraction and periodontal procedures, and 88% success for restorative procedures using a conventional syringe.[27] Giffin reported a 99% success rate in 6000 crestal anesthesia injections but did not specify the subsequent dental procedures.[29] Taheri Talesh & Solahaye Kahnamouii found 100% success with crestal anesthesia for first, second, and third molar Class I restorations.[31]

Dental care should always be designed individually for each patient's specific needs. Currently, dentists have a variety of materials and routes for predictable LA that can match the requirements of each patient and procedure.

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References

1. Vera C, De Kok IJ, Reinhold D, et al. Evaluation of buccal alveolar bone dimension of-maxillary anterior and premolar teeth: a cone beam computed tomography investigation. *Int J Oral Maxillofac Implants.* 2012;27(6):1514-1519.
2. Malamed SF. *Handbook of Local Anesthesia.* 6th ed. St. Louis: Elsevier Mosby; 2013: 191-195, 190, 66, 258-262, 263.
3. Braut V, Bornstein MM, Lauber R, Buser D. Bone dimensions in the posterior mandible: a retrospective radiographic study using cone beam computed tomography. Part 1—analysis of dentate sites. *Int J Periodontics Restorative Dent.* 2012;32(2):175-184.
4. Robertson D, Nusstein J, Reader A, Beck M, McCartney M. The anesthetic efficacy of articaine in buccal infiltration of mandibular posterior teeth. *J Am Dent Assoc.* 2007; 138(8):1104-1112.
5. Jung IY, Kim JH, Kim ES, Lee CY, Lee SJ. An evaluation of buccal infiltrations and inferior alveolar nerve blocks in pulpal anesthesia for mandibular first molars. *J Endod.* 2008; 34(1):11-13.
6. El-Kholey KE. Infiltration anesthesia for extraction of the mandibular molars. *J Oral Maxillofac Surg.* 2013;71(10):1658.e1-e5.
7. Desantis JL, Liebow C. Four common mandibular nerve anomalies that lead to local anesthesia failures. *J Am Dent Assoc.* 1996;127(7):1081-1086.
8. Shiozaki H, Abe S, Tsumori N, Shiozaki K, Kaneko Y, Ichinohe T. Macroscopic anatomy of the sphenomandibular ligament related to the inferior alveolar nerve block. *J Craniomandib Prac.* 2007;25(30):160-165.
9. Oertel R, Rahn R, Kirch W. Clinical pharmacokinetics of articaine. *Clin Pharmacokinet.* 1997;33(6):417-425.
10. Abdulwahab M, Boynes S, Moore P, et al. The efficacy of six local anesthetic formulations used for posterior mandibular buccal infiltration anesthesia. *J Am Dent Assoc.* 2009;140(8):1018-1024.
11. Brandt RG, Anderson PF, McDonald NJ, Sohn W, Peters MC. The pulpal anesthetic efficacy of articaine versus lidocaine in dentistry: a meta-analysis. *J Am Dent Assoc.* 2011; 142(5):493-504.
12. Haase A, Reader A, Nusstein J, Beck M, Drum M. Comparing anesthetic efficacy of articaine versus lidocaine as a supplemental buccal infiltration of the mandibular first molar after an inferior alveolar nerve block. *J Am Dent Assoc.* 2008;139(9):1228-1235.
13. Kanaa MD,Whitworth JM, Corbett IP, Meechan JG. Articaine and lidocaine mandibular buccal infiltration anesthesia: a prospective randomized double-blind cross-over study. *J Endod.* 2006;32(4):296-298.
14. Martin M, Nusstein J, Drum M, Reader A, Beck M. Anesthetic efficacy of 1.8 ml versus 3.6 ml of 4% articaine with 1:100,000 epinephrine as a primary buccal infiltration of the mandibular first molar. *J Endod.* 2011;37(5):588-592.
15. Pabst L, Nusstein J, Drum M, Reader A, Beck M. The efficacy of a repeated buccal infiltration of articaine in prolonging duration of pulpal anesthesia in the mandibular first molar. *Anes Prog.* 2009;56(4):128-134.
16. Meechan JG, Jaber AA, Corbett IP, Whitworth JM. Buccal versus lingual articaine infiltration for mandibular tooth anaesthesia: a randomized controlled trial. *Int Endod J.* 2011;44(7):676-681.
17. Meechan JG. The use of the mandibular infiltration anesthetic technique in adults. *J Am Dent Assoc.* 2011;142(Suppl 3):19S-24S.
18. Fuller NP, Menke RA, Meyers WJ. Perception of pain to three different intraoral penetrations of needles. *J Am Dent Assoc.* 1979;99(5):822-824.
19. Flanagan T, Wahl MJ, Schmitt MM, Wahl JA. Size doesn't matter: needle gauge and injection pain. *Gen Dent.* 2007;55(3):216-217.
20. Delgado-Molina E, Bueno-Lafuente S, Berini-Aytes L, Gay-Escoda C. Comparative study of different syringes in positive aspiration during inferior alveolar nerve block. *Oral Surg Oral Med Oral Path Oral Radiol Endod.* 1999;88(50):557-560.
21. Hochman MN, Friedman MJ. An in vitro study of needle force penetration comparing a standard linear insertion to the new bidirectional rotation insertion technique. *Quintessence Int.* 2001;32(10):789-796.
22. Lehtinen R. Penetration of 27- and 30-gauge dental needles. *Int J Oral Surg.* 1983; 12(6):444-445.
23. Corbett IP, Kanaa MD, Whitworth JM, Meechan JG. Articaine infiltration for anesthesia of mandibular first molars. *J Endod.* 2008;34(5):514-518.
24. Lee SH, Lee NY. An alternative local anaesthesia technique to reduce pain in paediatric patients during needle insertion. *Eur J Paed Dent.* 2013;14(2):109-112.
25. Difelice MG, Vanderwalle KS, Maller SC, Hancock RH. Effects of a vibratory device on pain from anesthetic injections. *Compend Cont Educ Dent.* 2014;35(4):246, 248, 250-251 passim.
26. Garfunkel AA, Kaufman E, Mamary Y, Galili D. Intraligamentary-intraosseous anesthesia. A radiographic demonstration. *Int J Oral Surg.* 1983;12(5):334-339.
27. Malamed SF. The periodontal ligament (PDL) injection: an alternative to inferior alveolar block. *Oral Surg Oral Med Oral Pathol.* 1982;53(2):117-121.
28. Moore PA, Cuddy MA, Cooke MR, Sokolowski CJ. Periodontal ligament and intraosseous injection techniques: alternatives to mandibular nerve blocks. *J Am Dent Assoc.* 2011;142(Suppl 3):13S-18S.
29. Giffin KM. Providing intraosseous anesthesia with minimal invasion. *J Am Dent Assoc.* 1994;125(8):1119-1121.
30. Kaemmerer PW, Palarie V, Schiegnitz E, Ziebart T, Al-Nawas B, Daublander M. Clinical and histological comparison of pulp anaesthesia and local diffusion after periodontal ligament injection and intrapapillary infiltration anaesthesia. *J Pain Relief.* 2012;1(108): 1-5.
31. Taheri Talesh K, Solahaye Kahnamouii S. Application of crestal anesthesia for treatment of Class I caries in posterior mandibular teeth. *J Dent Res Dent Clin Dent Prospects.* 2011;5(1):17-22.

Predictable replacement of failing porcelain restorations

Wynn Okuda, DMD, FAACD, FICD, FICOI

Clinicians are faced with esthetic dental problems every day. Broken ceramic restorations in the anterior region are a common problem that can occur in general practice. Presently, many porcelain restorations are used to restore the human dentition. Many tooth-colored restorations have been placed over the last 20 years, whether they be porcelain-fused-to-gold (PFG), feldspathic porcelain, porcelain (aluminous) jacket crowns, lithium disilicate or zirconia-based ceramic restorations. Therefore, it would be prudent to realize that many of these restorations will need to be replaced in the years ahead. Further, it is important to have an understanding of how to predictably replace these tooth-colored restorations so that optimal blending of the new restorations can be attained.

For cases in which there is recurrent decay under an existing ceramic restoration, or the majority of the porcelain is broken and the remaining porcelain restoration is compromised, a full replacement of the porcelain restoration should be considered (Fig. 1). Replacing a single porcelain restoration can be more difficult than repairing it with a conservative composite resin.[1,2] Therefore, careful assessment should be taken before considering replacement of a ceramic restoration. However, if a new porcelain restoration is determined to be necessary, there are several important factors that need to be considered.

Porcelains from different manufacturers have subtle characteristics that make them unique in appearance, so it is important to know the type of porcelain restoration you are replacing.[3] For instance, PFG restorations will appear more opaque than lithium disilicate restorations due to the masking that needs to be done over the metal base of the PFG. By conveying this information to your lab you have an optimal chance of getting the best match and increased success the first time around. If you're not sure, at the very least take preoperative photos of the tooth being restored—as well as the adjacent teeth—using shade tabs with the closest colors to the failing porcelain restoration and the adjacent teeth you are trying to mimic (Fig. 2). The author has found the Vita 3-D MASTER guide (Vident) to be the easiest to match most tooth shades. Sending photos will give your dental laboratory the best chance to analyze the shades needed to match the area being restored.

Whether color matching to a natural adjacent tooth or blending the new restoration to existing porcelain restorations, proper shade selection must be approached first.[4] Through a series of high-resolution photos, the hue, value, and chroma of the restoration are conveyed to the ceramist. Be sure to use a camera that takes at least 2 MB digital files so the lab can fully assess the shades needed to be mimicked. The author uses a Canon 60D camera body with a macro-lens and dual source flash (Canon USA, Inc.).

During removal of the existing restoration, ceramic pieces should be carefully removed, saved, and submitted to the laboratory for shade analysis along with the case (Fig. 3). Having an experienced dental technician is important so that the right color blend, as well as the correct size, contour, and characterization, can be attained in the new restoration.[5]

If the restoration is a bonded ceramic such as a lithium disilicate- or feldspathic-type restoration, it is also important to include a photo of the prepped tooth with an internal stumpf shade (Fig. 4). With these types of porcelain restorations, the chromacity of the internal dentin shade can influence the final external

Fig. 1. Buccal view of existing porcelain restoration with an extensive amount of porcelain fractured off.

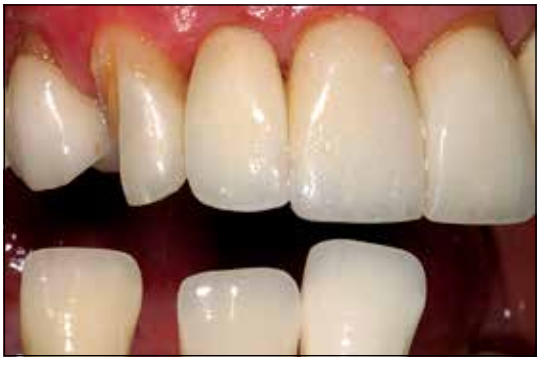

Fig. 2. Photograph showing shade tabs that were closest to the colors of the tooth being restored and the surrounding porcelain restorations.

Fig. 3. Salvaged porcelain pieces sent to the lab for color analysis for the new restoration.

Fig. 4. Photograph of internal stumpf shade sent to the lab for final color considerations.

Fig. 5. Buccal view of esthetic temporary jacket crown created as a transitional restoration.

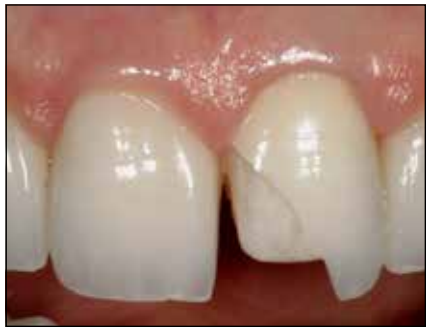

Fig. 6. Anterior view of extensive fracture of existing porcelain restoration on left central incisor.

Fig. 7. Photograph showing the restoration matching the surrounding dentition.

Fig. 8. Anterior view post-restoration.

shade (Fig. 5). Submitting all this information along with the final impression and a detailed lab prescription can lead to predictable and consistent success with porcelain replacement restorations.[6]

Case report

A 56-year-old woman presented with an extensive fracture in her existing porcelain restoration on her left central incisor (Fig. 6). The porcelain restoration was a feldspathic porcelain veneer. First, by using a shade guide, the best shades were selected to match the existing restoration and the surrounding teeth. Digital preoperative photos were taken of the area being restored. Additional photos were taken with matching shade tabs against the teeth (Fig. 7).

During treatment, the remaining broken porcelain pieces were removed and sent to the lab. Although larger porcelain pieces are desired, even small porcelain pieces are helpful when trying to match variations of color, value, and incisal characteristics.

After the tooth preparation was complete, photos of internal shades were taken with reference stump shade tabs. All of these were submitted along with a detailed prescription and a color map.

After receiving the new porcelain restoration from the lab, the color was checked on the prepped tooth with water (glycerin may be used, as well). A visual inspection is important prior to final placement to ensure the external shade matches the surrounding dentition. Using this method, it is the author's experience that 95% of the time, no modifications are needed to adjust for color. The final bonding of the porcelain restoration was accomplished using a total-etch technique.[7] After etching with 37% phosphoric acid (Etch-37 Semi Gel w/BAC, Bisco, Inc.), a dental adhesive (ALL-BOND 3, Bisco, Inc.) and a light-cure luting resin (Choice 2, Bisco, Inc.) was used to bond the porcelain restoration. Next, the restoration was finished with a series of fine finishing diamond burs (Porcelain Finishing Kit, Komet USA, LLC) and polishers (Intraoral Diamond Polishing Kit, Komet USA, LLC). Finally, a micro-diamond polishing paste (Ultradent Products, Inc.) was used to give a final polish (Fig. 8).

Conclusion

There are many different types of porcelain restorations that have been placed throughout the last several decades. In the years to come, many of these existing porcelain restorations will need to be replaced. In order to attain the best esthetic outcomes, a methodical approach to treatment is important. Predictable clinical results can be achieved using the method of porcelain replacement treatment described in this case report.

Author information

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References

1. Kimmich M, Stappert CF. Intraoral treatment of veneering porcelain chipping of fixed dental restorations: a review and clinical application. *J Am Dent Assoc.* 2013;144(1): 31-44.
2. Galiatsatos AA. An indirect repair technique for fractured metal-ceramic restorations: a clinical report. *J Prosthet Dent.* 2005;93(4):321-323.
3. Ubassy G. *Shape and Color: The Key to Successful Ceramic Restorations.* 1st ed. Hanover Park, IL: Quintessence Publishing Co., Inc.; 1993.
4. Muia P. *Four-Dimensional Color System.* Hanover Park, IL: Quintessence Publishing Co., Inc.; 1993.
5. Sieber C. *Voyage: Visions in Color and Form.* Hanover Park, IL: Quintessence Publishing Co., Inc.; 1994.
6. Muia PJ. *Esthetic Restorations: Improved Dentist-Laboratory Communication.* Hanover Park, IL: Quintessence Publishing Co., Inc.; 1993:141.
7. Magne P, Belser UC. *Bonded Porcelain Restorations in the Anterior Dentition: A Biomimetic Approach.* Hanover Park, IL: Quintessence Publishing Co., Inc.; 2002.

Manufacturers

Bisco, Inc., Schaumburg, IL
800.247.3368, www.bisco.com

Canon USA, Inc., Melville, NY
631.330.5000, www.usa.cannon.com

Komet USA, LLC, Rock Hill, SC
888.566.3887, www.kometusa.com

Ultradent Products, Inc., South Jordan, UT
888.230.1420, www.ultradent.com

Vident, Brea, CA
800.828.3839, www.vident.com

Gifts from patients

Toni M. Roucka, DDS, MA, FACD

Well, it's that time of year again for gifts. We exchange them with just about everyone we know: family, friends, staff, colleagues, and business associates. It is rare that we exchange gifts with patients; however, it is quite common for patients to bring gifts to the dental office for the doctor and staff.

There are some ethical considerations to explore regarding the acceptance of gifts from patients. The following case will examine these.

Ava enjoys her visits to her dentist, Dr. Oliver. He is kind, considerate, and very gentle. Ava is one of those "unlucky" dental patients. Despite pretty good oral hygiene, something always seems to go wrong with her oral health. When she was 12, she fell off her bike and fractured her 2 maxillary central incisors and some other teeth. This led to numerous dental problems. Bonding, root canals, crowns, and veneers have all been performed on various teeth at appropriate times over the years by Dr. Oliver. Now it seems there is another problem brewing—resorption. Ava doesn't exactly know which dental treatments will be required in the future, but Dr. Oliver has told her that it will not be a simple fix. They will continue to monitor the teeth over time and intervene as needed. Dr. Oliver warned Ava that she may eventually lose her 2 front teeth.

When Ava looks back on her dental history, she cannot recall how many times she had to visit Dr. Oliver on an emergency basis for issues related to the teeth affected by her accident. She is grateful for every time Dr. Oliver was able to "squeeze her in" his busy schedule. Fortunately, Ava has always had the financial resources to be able to pay for her treatment. First her parents' insurance covered costs; now that she is older with a good job, she is able to pay for her own treatment.

Last November, a new problem occurred. Ava developed a large and very painful sore on her gum tissue in the upper right posterior region. This time the problem seemed to be totally unrelated to anything involving residual issues from the accident. It was 5 pm, the Wednesday before Thanksgiving, and Dr. Oliver's office was already closed for the holiday. Because of his good nature and dedication to his patients, Dr. Oliver came in later that evening to examine Ava and treat the problem. This time it was an easy fix. A large popcorn kernel sheath was lodged in the gingival sulcus adjacent to tooth No. 3 on the lingual side, and the area had a small abscess. Dr. Oliver removed the kernel easily with an instrument, drained the abscess, and ensured Ava she should be fine in 1-2 days. Ava was not only embarrassed but upset with herself for asking Dr. Oliver to come in on a holiday for something so trivial. She paid her bill, and as she left the office, she decided she would do something nice for him in return.

In mid-December, Ava arrived at Dr. Oliver's office with a gift of appreciation for all he had done for her over the years, especially the latest incident right before Thanksgiving. She had thought long and hard about the gift. She wanted to give something he could keep, as opposed to something temporary, such as flowers or a food basket. She also thought it would be fitting to buy something really nice since she has been his patient for so long. Ava settled on a Waterford crystal candy bowl with a nice card of appreciation. She spent $175 on the gift, thinking that it was not too much money for so many years of quality care. She gave the wrapped gift to the office manager and left feeling very satisfied that she had done something really nice for Dr. Oliver. She couldn't wait to hear how he liked it.

When Dr. Oliver opened the gift, he was quite taken aback. He did not know if it was appropriate to accept the gift. Something told him it might be too expensive, as it had come from a patient. Conversely, he did not want to insult Ava by not accepting it, as she was a long-standing patient and hopefully would be for years to come.

What should Dr. Oliver do?

Dr. Oliver is faced with a dilemma that forces him to decide whether to accept the gift from Ava or not, and to consider the following questions:

- How does the acceptance or denial of gifts from patients affect the dentist-patient relationship?
- Should dentists accept gifts from patients at all?
- If so, what level of gift acceptance from patients is appropriate?
- Should a monetary limit be placed on gifts from patients?

The giving and receiving of gifts is a cultural phenomenon. It can also be a very emotionally charged experience on both ends. When someone doesn't like or appreciate a gift we give, particularly when a lot of thought and effort went into its selection, we may become offended. Conversely, when we receive a gift we really don't want, it is just as difficult to deal with. We don't want to hurt the giver's feelings and appear disappointed, yet we don't want to keep the gift, and fear the giver may find out if we return it to the store or give it to someone else. How much money was spent on a gift or how much work went into making it adds another layer of complexity to the situation. In a strictly social context, these circumstances are awkward and difficult enough to deal with. Adding to it the element of patient care in a clinical setting makes it a much more challenging problem.

In this particular scenario, we cannot look directly to the *American Dental Association Principles of Ethics and Code of Professional Conduct* (ADA Code) for specific guidance.[1] The Code does not offer a specific advisory opinion on this topic. However, under the principle of *Nonmalificence* (Section 2.G), the ADA Code does address personal relationships with patients.[1] Although not directly addressing the exchange of gifts with patients, the Code does require dentists to avoid any relationships with patients that may "impair their professional judgment."[1] The acceptance of a gift, depending on what it is and the dentist's perceived value of the gift, as well as the patient's motivation for giving it, may have the potential to alter the professional relationship.

Dentists are also obligated under the ADA Code to be nondiscriminatory in the selection of patients; therefore, most dental offices will probably see patients from many different ethnic and cultural backgrounds.[1] Gift giving and receiving in some cultures is a very common and meaningful occurrence. When presented with gifts, dentists need to balance cultural sensitivity with maintaining professional boundaries.

In the context of the dentist-patient relationship, Ozar & Sokol advocate an *interactive* model, in which the relationship is one of equality, mutual respect, and shared decision making.[2] This model implies that while the dentist has the expertise in the science of dentistry and the provision of care, the patient's values, autonomy, and goals are equally important. If the giving of gifts is significantly important to a particular patient, the dentist may need to consider how the acceptance or refusal of a gift will affect the relationship with that patient before making a decision about the gift. How the dentist can ascertain that poses another challenge.

The medical profession has dealt with this question specifically in the *Code of Medical Ethics of the American Medical Association* (AMA Code).[3] Under Section 10.017, *Gifts from Patients,* there is a statement on how physicians must balance cultural sensitivity with professional obligations.[3] It states that in cases of gifts given in gratitude or as part of a cultural tradition, the doctor-patient relationship may actually be enhanced. Physicians must be cognizant, however, of patients giving gifts with the expectation that they will receive preferential care.[3] This is problematic and could also occur in dentistry. The seeking of preferential care in exchange for a gift could damage the integrity of the relationship and potentially compromise the ethics of both parties.

The AMA Code does not offer any specific guidance, however, with regards to the appropriate cash value of gifts or exactly when it's appropriate to accept gifts, except that "the gift's value relative to the patient's or physician's means should not be disproportionately or inappropriately large."[3] One criterion suggested is that "the physician should determine individually if he/she would be comfortable disclosing acceptance of the gift to colleagues or the public."[3] Dentistry may benefit from approaching the subject of patients giving gifts in a similar manner.

The reality of accepting gifts from patients is that it does change the nature of the relationship whether it happens consciously or not. When a dentist accepts a gift, especially a substantial one, the dynamics of the relationship may shift. Going forward, the dentist may treat the patient differently than other patients.[4] For instance, when the waiting room is full, and the dentist is running behind schedule, how might he treat the gift giver differently than other patients also waiting to be seen? Dentists should be cognizant of the potential for bias or favoritism in such cases. On a different note, by offering a gift, the patient may not necessarily be looking for preferential treatment but might instead be hopeful that the relationship may become more social. Relationship boundary violations could potentially occur as a result.[5]

Andereck states there are 3 possible motives for patients to offer gifts to their doctors: influence, pure beneficence, and appreciation.[6] Since there is no real way to know the intent of the patient presenting the gift, paying attention to previous or subsequent behavior may help. The author also states that some people are just "generous by nature" and advocates for the acceptance of gifts as long as "the patient's motives seem well placed."[6]

Resolution

Dr. Oliver must weigh all of these issues and make a decision as to whether to accept the gift from Ava or not. Since there are few ethical guidelines governing this topic and there was no breach of the law, common sense should prevail. If Dr. Oliver examines this quandary closely, he will find that:

- Ava's intentions appear sincere. She is grateful for the years of care Dr. Oliver has provided to her and is especially thankful he was available for her last Thanksgiving. There is no history of challenges to their dentist-patient relationship.
- The gift was something Ava could afford. It was not disproportionally expensive relative to her or Dr. Oliver's salaries and life circumstances.
- Dr. Oliver feels that rejecting the gift may hurt Ava's feelings.
- Dr. Oliver would not be embarrassed to tell his colleagues, family, or staff about the gift.

All things considered, Dr. Oliver should accept the gift and thank Ava for her kindness. According to Krupa:

Gifts are cherished keepsakes that help remind doctors why they went into medicine in the first place—for the connections they have with patients. In most cases, gifts are wonderful ways of showing appreciation. In a very human way, it is the way we express gratitude.[7]

Author information

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References

1. American Dental Association. *American Dental Association Principles of Ethics and Code of Professional Conduct.* Revised 2012. Available at: http://www.ada.org/sections/about/pdfs/code_of_ethics_2012.pdf. Accessed September 23, 2014.
2. Ozar DT, Sokol DJ. *Dental Ethics at the Chairside.* 2nd ed. Washington, DC: Georgetown University Press; 2002:51-52.
3. American Medical Association. *AMA Code of Ethics 2004.* Revised 2006. Available at: http://www.amsws.org.au/media/docs/images/ama_code_of_ethics.pdf. Accessed September 23, 2014.
4. Marois J. Receiving gifts from patients: a pragmatic shade of grey. *BC Med J.* 2010; 52(3):129-132.
5. Ladouceur R. Should family physicians accept gifts form patients? *Can Fam Physician.* 2013;59(12):1254-1255.
6. Andereck W. Point-counterpoint: should physicians accept gifts from their patients? Yes: if they are given out of beneficence or appreciation. *West J Med.* 2001;175(2):76.
7. Krupa C. *Holiday Gifts from Patients: When do They Spell Trouble?* American Medical News. Available at: http://www.amednews.com/article/20111212/profession/312129941/2/. Accessed September 23, 2014.

Conservative cosmetic dentistry post-trauma

Nicholas Marongiu, DDS ▪ Todd Cochran, AAACD

Traumatized teeth can bleed internally, causing discoloration over time. When this occurs in the smile zone, masking the dark colorations can present challenges when attempting to practice conservative cosmetic dentistry. Implementing nonvital bleaching can significantly improve the dark colorations of the traumatized teeth and support very conservative cosmetic dentistry. Effective communication with the ceramist is essential to ensure the desired results. This article presents a case involving trauma with delayed root canal therapy on tooth No. 9, which produced a very dark front tooth, and the conservative treatment plan chosen to correct it through the use of nonvital bleaching and feldspathic veneers requiring zero or minimal preparation.

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Treatment of only 1 or 2 teeth in the smile zone can present cosmetic challenges in blending with the natural dentition.[1] When treating traumatized dark teeth, the balance between cosmetics and practicing conservative dentistry amplifies these challenges. Historically, dark teeth have been aggressively prepared to allow for restorative materials to block out the dark color. This article will present a case with nonvital bleaching and the fabrication of feldspathic veneers requiring zero or minimal preparation in order to conserve as much healthy tooth structure as possible while producing an excellent cosmetic result.

Case study

A 20-year-old woman presented with a single dark upper front tooth (Fig. 1). She reported that the tooth (No. 9) had been fractured after a fall at age 10 while playing softball. Over the following 10 years, she reported having the tooth filled 4 times, and a root canal completed 6 years after the incident.

Tooth No. 9 presented with evidence of previous internal bleeding due to trauma and was much darker in color than the natural dentition.[2] Additionally, tooth No. 8 presented with a discolored facial composite and a gingival defect in the enamel producing asymmetrical gingival architecture. The incisal edges of the 2 teeth were uneven and the widths were not equal (Fig. 2).

The patient requested a longer lasting cosmetic dental treatment to address the discoloration of tooth No. 9, as well as correction of the asymmetrical gingival architecture of teeth No. 8 and 9.

Clinical evaluation

Complete intra- and extraoral examinations were completed that included an evaluation of the hard/soft tissues, temporomandibular joint, caries risk, periodontal health, occlusion, attrition, orthodontic class, crowding, and condition of existing dental restorations. Radiographs were obtained to evaluate supporting structures and existing dental restorations, to assess caries, and to verify the integrity of the existing root canal of tooth No. 9 (Fig. 3). A cosmetic photo series was acquired with study models for records of preoperative conditions and evaluation with the ceramist (Fig. 4).

Tooth No. 9 presented with an existing root canal without any obvious pathology and a mesial incisal lingual facial composite with a chipped mesial incisal corner. The existing shade of tooth No. 9 was D-3 (VITA Classical shade guide, Vident), relative to the remaining natural dentition of B-1 (Fig. 5). The existing facial composite on tooth No. 8 was stained and breaking down along the margins. The mesiodistal widths and edge positions of the 2 teeth were asymmetrical. No mobility was observed. Periodontal pockets were

Fig. 1. Photograph of smile at initial consult showing darkened teeth No. 8 and 9.

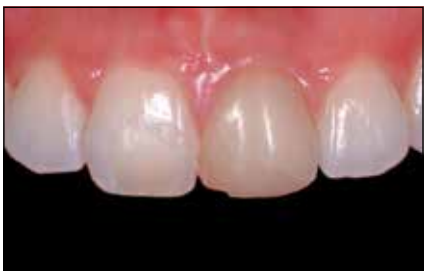

Fig. 2. Retracted close-up view showing discolorations and chipped composite.

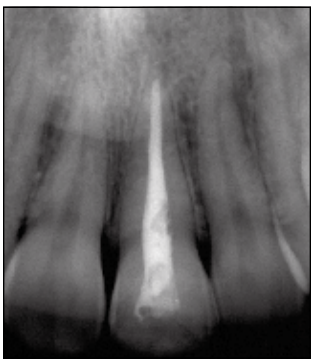

Fig. 3. Preoperative radiograph of teeth No. 8 and 9.

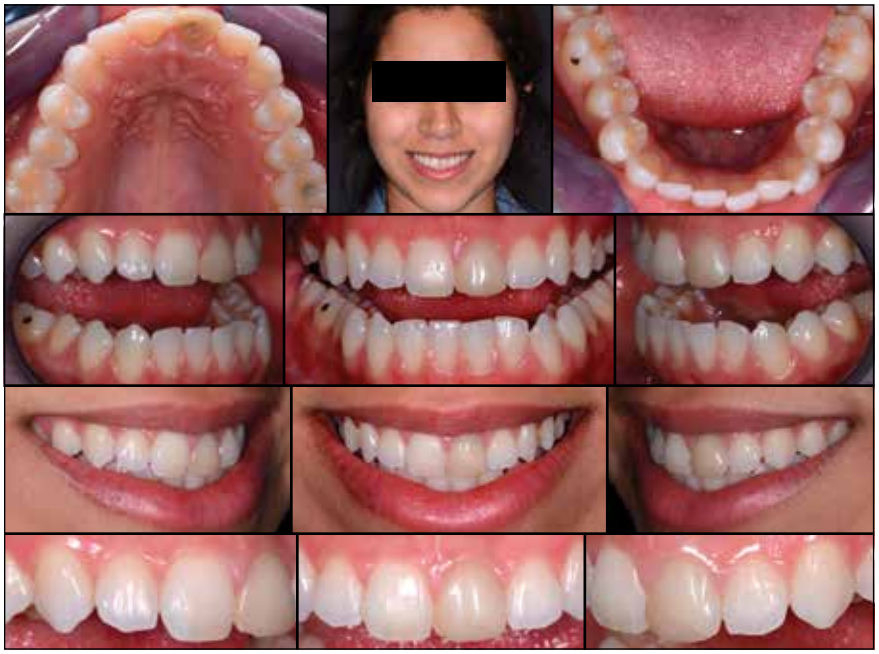

Fig. 4. Preoperative cosmetic photo series of patient's mouth.

Fig. 5. Existing shade of tooth No. 9 (D-3).

Fig. 6. Anterior view of mandibular crowding.

2-3 mm. All other existing restorations were in good condition. The patient was in class I molar and canine occlusion and presented with postorthodontic lower anterior crowding with wear on tooth No. 24 (Fig. 6).

Given the mandibular crowding and signs of wear, the patient was scheduled initially to correct the crowding and prevent traumatic occlusion.[3-5] Tooth No. 9 needed to be treated with nonvital bleaching prior to the final restoration in order to determine the minimum restoration thickness needed to conserve as much natural tooth structure as possible, to preserve natural enamel strength, and to provide full enamel bonding.[6] Lastly, the final restorations on teeth No. 8 and 9 needed to be fabricated in porcelain and bonded to recreate proper incisal edge position, symmetry, and color, as well as to provide the needed strength for long-term predictability.[7-9]

Treatment planning

The anterior mandibular crowding was discussed with the patient and a limited orthodontic treatment vs enameloplasty of the lower incisors was proposed. Treatment options to address the patient's chief complaint of discoloration were proposed as a more aggressive preparation to block out the dark color as opposed to nonvital bleaching and a more conservative preparation. The replacement of composite on teeth No. 8 and 9 instead of porcelain veneers was discussed, as well as the advantages and limitations in treating only tooth No. 9. Treating both central incisors would produce symmetry in edge positions and tooth widths. The patient elected to correct the mandibular anterior crowding and proceed with nonvital bleaching and porcelain veneers on both teeth No. 8 and 9.

According to the treatment plan, the correction of the mandibular anterior crowding would be completed with a minor interproximal reduction of the lower incisors and the use of a series of Essix ACE thermoplastic aligners (DENTSPLY International).[10] The nonvital bleaching of tooth No. 9 would be achieved with Opalescence Endo 35% hydrogen peroxide (Ultradent Products, Inc.).[11-15] Then, the existing composite would be removed from teeth No. 8 and 9 and feldspathic veneers (Creation CC, Creation Willi Geller International GmbH) requiring zero or minimal preparation would be fabricated.

This treatment plan addressed the patient's chief complaint while being as conservative as possible, thereby maintaining the strength of the natural enamel to provide long-term predictability.[7] The Essix aligners were chosen to minimize the cost of the anterior crowding correction. The nonvital bleaching was selected to bring tooth No. 9 closer in color to the natural dentition, while minimizing the reduction of the natural tooth structure. Feldspathic porcelain was chosen for its optical characteristics, strength upon enamel bonding, and ability to fabricate very thin restorations.

Clinical technique

After correction of the mandibular anterior crowding, nonvital bleaching was initiated on tooth No. 9. Septocaine (4%) with 1:100 k epinephrine (Septodont, Inc.) was administered, and a rubber dam was placed to isolate tooth No. 9. The existing lingual composite access fill and the gutta percha were removed from the chamber and canal to a depth 2 mm below the crestal bone.[1] The canal was sealed up to the crestal bone with a resin-modified glass ionomer, Ketac Nano (3M ESPE).[16,17] Opalescence Endo was placed in the chamber along with a small cotton pellet. The lingual access was temporarily sealed

Fig. 7. Anterior view showing composite removed from tooth No. 8 and nonvital bleaching of tooth No. 9.

Fig. 8. Shade photo of hydrated teeth.

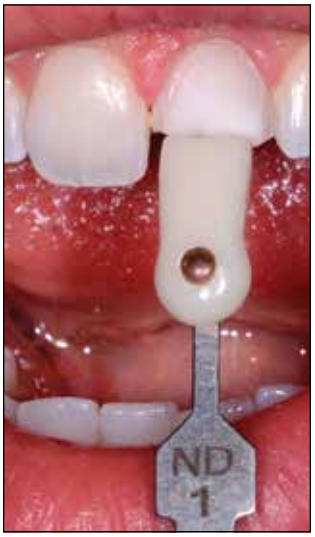

Fig. 9. Stump shade photo of tooth No. 9.

with Ketac Nano (3M ESPE). Two rounds of nonvital bleaching were completed for 3 days each cycle to achieve the desired whitening; this brought tooth No. 9 from shade D-3 to A-1, as measured by the VITA Classical shade guide (Vident). The stained composite on the facial of tooth No. 8 was also removed (Fig. 7).

The lingual access was then restored by administering the Septocaine and placing a rubber dam to isolate tooth No. 9. The chamber was cleaned out to the previously placed Ketac Nano level, rinsed, and dried. Scotchbond Etchant 35% phosphoric acid (3M ESPE) was applied to the enamel around the lingual access for 5 seconds, then applied to the chamber for an additional 15 seconds, rinsed, and blotted dry with cotton to remove excess water. Two coats of Adper Single Bond Plus Adhesive (3M ESPE) were applied and agitated with a microbrush for 15 seconds. The Single Bond was air-thinned for 5 seconds and light-cured for 10 seconds using an Elipar S10 (3M ESPE) curing light. B1B Filtek Supreme Ultra nanocluster composite (3M ESPE) was layered in 2 mm increments to full contour with incremental curing times (20 seconds each).

On the day of preparation for the veneers, a preoperative cosmetic photo series was acquired along with shade photos of the hydrated teeth for the ceramist. The shade B1 from the VITA Classical shade guide was photographed along with higher and lower valued shade references (Fig. 8). Local anesthetic was not needed due to the nonvital bleaching of preparation of teeth No. 8 and 9. A preoperative impression was acquired with a plastic impression tray and Star VPS vinyl polysiloxane (Danville Materials). The existing class IV composite was removed and tooth No. 9 was prepared for a veneer using a coarse round end taper friction grip high speed diamond bur (799.11, Premier Products Co.). The stump shade of tooth No. 9 was recorded and photographed as *ND1* using the IPS Natural Die Material shade guide (Ivoclar Vivadent, Inc.) (Fig. 9).

An Expasyl gingival extraction system (Kerr Corporation) was placed in the gingival sulcus of teeth No. 8 and 9, and a small cotton pellet was used to tamp the Expasyl down. After 2 minutes, the Expasyl was rinsed, and the teeth dried. Imprint 3 light body vinyl polysiloxane (3M ESPE) was injected into the sulcus of teeth No. 8 and 9 and around the other anterior maxillary teeth. A metal full arch impression tray loaded with Provil Novo Putty Fast Set (Heraeus Kulzer) was seated over the maxillary teeth and held in place for 4 minutes. The impression was then removed and all margins and critical areas were verified. The opposing impression was captured in a similar manner with a metal full arch tray, putty, and a polyvinyl siloxane (PVS) wash. A full arch interocclusal bite record was recorded with Blu-Mousse super-fast bite set registration material, a thixotropic PVS (Parkell, Inc.).

A Kois Dento-Facial Analyzer (Panadent Corporation) was used to facilitate the transfer of the occlusal plane and facial midline registrations to the ceramist. Tooth No. 9 was then temporized using a spot etch direct temporary technique. Tooth No. 9 was spot etched with Scotchbond Etchant for 5 seconds, and rinsed and dried. Adper Single Bond Plus Adhesive was applied, air-thinned and light-cured for 10 seconds using the Elipar S10 curing light. The preoperative impression was loaded with Protemp Plus shade A1 (3M ESPE) in the area of tooth No. 9, then seated in the patient's mouth for 5 minutes. Upon removal of the preoperative impression, the excess temporary material was removed, the impression was occlusion-verified, and then polished with Sof-Lex polishing discs (3M ESPE).

The impressions, interocclusal bite record, all pre- and perioperative photos, and Kois Dento-Facial Analyzer records were sent to the ceramist.

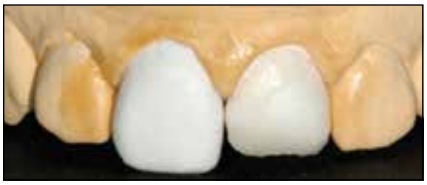

Fig. 10. Opaque bake of veneer on refractory dies of teeth No. 8 and 9.

Fig. 11. Layering of feldspathic porcelain on refractory dies of teeth No. 8 and 9.

Fig. 12. Veneers of teeth No. 8 and 9 polished on stone model.

Fig. 13. Veneers on black surface displaying translucency and characteristics. *Left.* Tooth No. 9. *Right.* Tooth No. 8.

Fig. 14. Veneers on black surface displaying translucency and characteristics. *Top.* Tooth No. 9. *Bottom.* Tooth No. 8.

Fig. 15. Veneers completed on stone model.

Laboratory fabrication

Prior to beginning the case, the clinician and ceramist met to discuss the projected treatment plan and options—including materials and techniques—that could achieve the best result. The photos reviewed included the preoperative full cosmetic photo series, shade photos with multiple shade references (to illustrate chroma, value, and internal characterization), and stump shade photos.

The models were poured in Type IV GC Fujirock EP stone (GC America, Inc.) and mounted for review. This case involved the fabrication of a no-preparation veneer on tooth No. 8 and a minimal preparation veneer on tooth No. 9. Feldspathic porcelain was chosen for the desired esthetics. The refractory veneer technique was used to build this case, a Gellar model was fabricated, and dies were duplicated with Polypour (GC America, Inc.), a pourable vinyl polysiloxane duplicating material. The refractory dies were then poured using Nori-Vest (Kuraray America, Inc.) and cured under pressure. The dies were processed strictly according to the manufacturer recommendation, then seated back into the Gellar model.

Willi Gellar Classic Creation (Creation Willi Geller International GmbH) was the chosen porcelain type for the case. The first "bake" was carried out using a 50% mixture of CL-O and HT-51 as a bonding layer to the refractory and was fired 30°C higher than the typical temperature, which is 920°C, to ensure a smooth bond to the refractory material.

The next bake was an opacious dentin layer to mask the line of the preparation and to begin mimicking the adjacent tooth shade (Fig. 10). The firing temperatures were increased 15°C to accommodate the mass of refractory material. The third bake was California White Dentin and E-57, CL-O, TI-4, and TI-2 (Creation Willi Gellar International GmbH). Mamelon characterization was created by mixing MI-61 with INN-1 and INN-2 and fired. E-57, TI-1, TI-2, HT-51 and SI-02 were layered on the incisal and mid facial areas and HT-51 and HT-52 were layered in the cervical area (Fig. 11). A slight halo was added using a mixture of California White Dentin and E-57 on both restorations for a perfect match. The final firing was a slight correction firing that added slight white stain characterizations to mimic the natural dentition.

The restorations were completed on the refractory dies using diamond impregnated silicone wheels (Axis Dental) and Legabril diamond polish paste (Metalor Technologies USA Corporation) (Fig. 12).

The veneers were divested using glass beads for the bulk of the material and finished with aluminum oxide at very low pressure—less than 1.5 bars. After divesting, the internal surface of the veneers were etched with IPS Etching Gel 5% hydrofluoric acid (Ivoclar Vivadent, Inc.) for 20 seconds then placed in an ultrasonic cleaner for 10 minutes (Fig. 13-15). The case was then packed and sent to the clinician.

Final cementation

Septocaine was administered to ensure patient comfort during delivery and finishing of veneers. The temporary veneer was removed from tooth No. 9 by making a vertical cut through the veneer with the 799.11 diamond bur and using a crown spreader to remove the material. Teeth No. 8 and 9 were then cleaned with pumice and a slow speed rubber cup. The 2 veneers were initially tried in one at a time to verify fit, and then tried in together to verify passive seat and proximal contacts. Once verified, the restorations were tried

Fig. 16. Try-in of veneers on teeth No. 8 and 9 with translucent try-in paste.

Fig. 17. Photograph of postoperative smile.

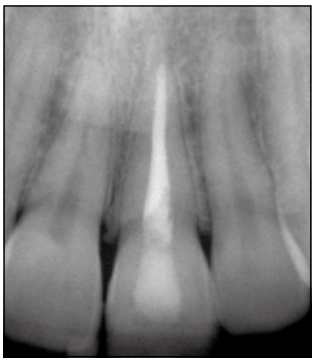

Fig. 18. Postoperative radiograph of teeth No. 8 and 9.

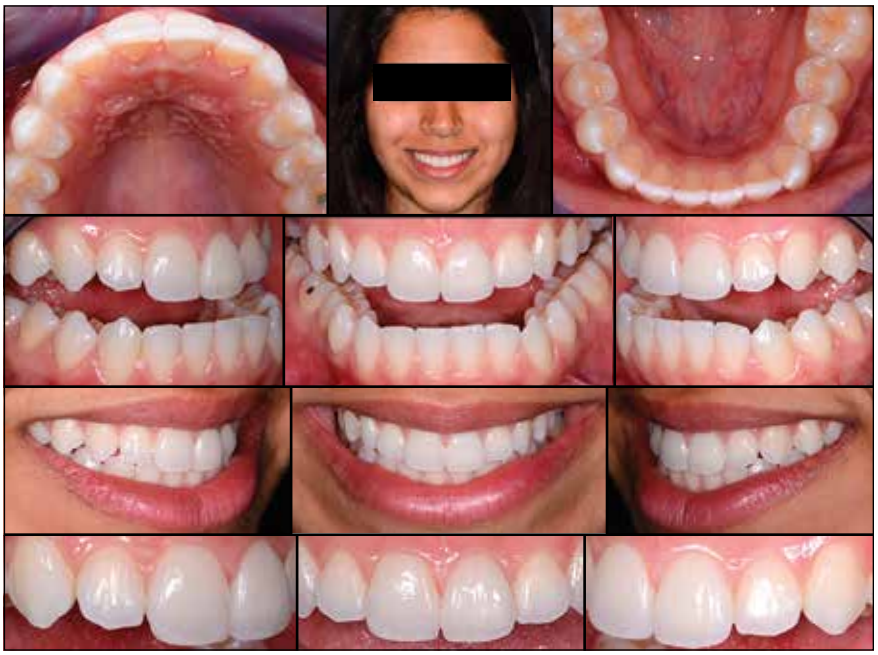

Fig. 19. Postoperative cosmetic photo series of patient's mouth.

in with Rely X Translucent Try-In paste (3M ESPE) to verify incisal edge position, symmetry, midline, hue, chroma, and value (Fig. 16). The veneers were then removed, thoroughly rinsed with water spray, then cleaned with Ivoclean (Ivoclar Vivadent, Inc.) for 20 seconds.

A fourth generation etch-and-rinse bonding system was used for optimal bond strength.[18,19] A single coat of silane RelyX Ceramic Primer (3M ESPE) was applied to the internal surface and allowed to evaporate. Adper Scotchbond Multi-Purpose Adhesive (3M ESPE) was applied to the silane-treated surfaces and air-thinned. Scotchbond Etchant was applied to teeth No. 8 and 9 for 15 seconds, then rinsed with water spray. Excess water was blotted away from teeth No. 8 and 9, leaving the tooth surface moist. Adper Scotchbond Multi-Purpose Primer (3M ESPE) was then applied to teeth No. 8 and 9 and gently dried for 5 seconds. Two coats of the Adper Scotchbond Multi-Purpose Adhesive were applied to teeth No. 8 and 9, and gently air-thinned. The veneers were then loaded with RelyX Translucent Veneer Cement (3M ESPE) and seated with gentle pressure. A small diameter tacking tip was used on the Elipar S10 curing light to spot-cure the veneers in place on the facial surface. The excess cement was removed with microbrushes. The regular curing tip was reapplied to the curing light and each veneer was light-cured for 10 seconds. Glycerin was applied to margins to eliminate the air-inhibited layer and each veneer was then light-cured for an additional 20 seconds. A No. 12 scalpel blade was used to remove excess cement under microscope and Sof-Lex polishing strips and discs were used to finish the margins. The occlusive, excursive, and protrusive movements were checked.

The patient returned 2 weeks later for follow-up, at which point a postoperative cosmetic photo series and radiographs were taken (Fig. 17-19).

Conclusion

Through use of nonvital bleaching, very conservative cosmetic treatment can be used to treat dark teeth. Following proper protocol to seal the canal space is essential to provide for the long-term health of teeth undergoing nonvital bleaching. Communication with the ceramist and understanding of material options is crucial in providing beautiful, natural looking restorations. Conservative tooth preparation allows for complete enamel bonding, maximizing long-term predictability while conserving natural tooth structure.

Author information

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References

1. Sesemann MR. Successful strategies for matching one or two indirect restorations to natural dentition. *Gen Dent.* 2011;59(6):448-456.
2. Sane J, Ylipaayalniemi P. Dental trauma in contact team sports. *Endod Dent Traumatol.* 1988;4(4):164-169.
3. Little RM. Stability and relapse of dental arch alignment. *Br J Orthod.* 1990;17(3):235-241.
4. Freitas KM, de Freitas MR, Henriques JF, Pinzan A, Janson G. Postretention relapse of mandibular anterior crowding in patients treated without mandibular premolar extraction. *Am J Orthod Dentofacial Orthop.* 2004;125(4):480-487.
5. Little RM. Stability and relapse of mandibular anterior alignment: University of Washington studies. *Semin Orthod.* 1999;5(3):191-204.
6. Magne P, Douglas WH. Cumulative effects of successive restorative procedures on anterior crown flexure: intact versus veneered incisors. *Quintessence Int.* 2000;31(1):5-18.
7. Calamia JR, Calamia CS. Porcelain laminate veneers: reasons for 25 years of success. *Dent Clin North Am.* 2007;51(2):399-417.
8. Calamia JR. Etched porcelain facial veneers: a new treatment modality based on scientific and clinical evidence. *N Y J Dent.* 1983;53(6):255-259.
9. Karlsson S, Landahl I, Stegersjo G, Milleding P. A clinical evaluation of ceramic laminate veneers. *Int J Prosthodont.* 1992;5(5):447-451.
10. Ricketts DN, Smith BG. Clinical techniques for producing and monitoring minor axial tooth movement. *Eur J Prosthodont Restor Dent.* 1993;2(1):5-9.
11. Rotstein I, Walton RE. Bleaching discolored teeth: internal and external. In: Fouad A, Torabinejad M, Walton RE, eds. *Endodontics: Principles and Practice.* 4th ed. New York: Saunders Elsevier; 2008:391.
12. Abbott P, Heah SY. Internal bleaching of teeth: an analysis of 255 teeth. *Aust Dent J.* 2009;54(4):326-333.
13. Plotino G, Buono L, Grande NM, Pameijer CH, Somma F. Nonvital tooth bleaching: a review of the literature and clinical procedures. *J Endod.* 2008;34(4):394-407.
14. Boksman L, Jordan RE, Skinner DH. Non-vital bleaching—internal and external. *Aust Dent J.* 1983;28(3):149-152.
15. Freccia WF, Peters DD, Lorton L, Bernier WE. An in vitro comparison of nonvital bleaching techniques in the discolored tooth. *J Endod.* 1982;8(2):70-77.
16. Madison S, Walton R. Cervical root resorption following bleaching of endodontically treated teeth. *J Endod.* 1990;16(12):570-574.
17. Carrasco LD, Froner IC, Corona SA, Pecora JD. Effect of internal bleaching agents on dentinal permeability of non-vital teeth: quantitative assessment. *Dent Traumatol.* 2003;19(2):85-89.
18. Yazici AR, Celik C, Ozgunaltay G, Dayangac B. Bond strength of different adhesive systems to dental hard tissues. *Oper Dent.* 2007;32(2):166-172.
19. Hamouda IM, Samra NR, Badawi MF. Microtensile bond strength of etch and rinse versus self-etch adhesive systems. *J Mech Behav Biomed Mater.* 2011;4(3):461-466.

Manufacturers

Axis Dental, Coppell, TX
800.346.3636, www.axisdental.com

Creation Willi Geller International GmbH, Meiningen, Austria
43.5522.76784, www.creation-williegeller.com

Danville Materials, San Ramon, CA
800.827.7940, www.danvillematerials.com

DENTSPLY International, York, PA
800.877.0020, www.dentsply.com

GC America, Inc., Alsip, IL
800.323.7063, www.gcamerica.com

Heraeus Kulzer, South Bend, IN
800.435.1785, www.heraeus-dental-us.com

Ivoclar Vivadent, Inc., Amherst, NY
800.533.6625, www.ivoclarvivadent.us

Kerr Corporation, Orange, CA
800.537.7123, www.kerrdental.com

Kuraray America, Inc., New York, NY
800.879.1676, www.kuraraydental.com

Metalor Technologies USA Corporation, Attleboro MA
508.226.4470, www.metalor.com

Panadent Corporation, Colton, CA
800.368.9777, www.panadent.com

Parkell, Inc., Edgewood, NY
800.243.7446, www.parkell.com

Premier Products Co., Plymouth Meeting, PA
610.239.6000, www.premusa.com

Septodont, Inc., Lancaster, PA
800.872.8305, www.septodontusa.com

Ultradent Products, Inc., South Jordan, UT
888.230.1420, www.ultradent.com

Vident, Brea, CA
800.828.3839, www.vident.com

3M ESPE, St. Paul, MN
888.364.3577, solutions.3m.com

Shade evaluation of ceramic laminates according to different try-in materials

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The porcelain laminate replaces the visible portion of enamel with a ceramic, which is attached to the dental surface. To enhance cosmetic results, a preliminary color matching procedure is performed prior to cementing the veneers. This procedure can be performed using water, water-soluble gel, or try-in paste. The different shades of cement and try-in pastes are intended to obtain better color and esthetics of the final restoration. This study sought to evaluate the shade of ceramic veneers produced by different try-in materials. Forty bovine teeth and 40 ceramic discs (0.6 mm thick) were prepared. The samples were divided into 4 groups (n = 10). For Group 1 samples, no material was used between the tooth and the ceramic, Group 2 interposed samples with water, Group 3 used a water-soluble gel, and Group 4 used try-in paste (value 0). The color was measured with a spectrophotometer, obtaining L*, a*, and b* values to calculate the color difference (ΔE*). The data were subjected to normality tests and 1-way ANOVA. No significant statistical differences were found among the groups, indicating that the different try-in materials had similar effects on the color of the ceramic laminates.

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Key words: dental veneers, color perception tests, resin cements

Dental ceramics stand out due to their excellent optical properties, biocompatibility, durability, and close resemblance to the natural appearance of teeth.[1] The porcelain veneer replaces the visible portion of enamel with a ceramic laminate, which attaches itself securely to the dental surface.[2] New ceramic systems offer esthetics and high resistance, because they are reinforced with leucite and lithium disilicate, which allow for the development of minimally invasive ceramic laminates.[3] This technique utilizes thin ceramic veneers (0.3-1mm thick) with little or no preparation of the tooth structure necessary.[4]

The color of the cement used when placing these translucent ceramics may compromise the final esthetic result.[5,6] In this context, the resin cements offer several different colors and opacities. The adhesive systems and resin cements allow for an effective interaction between the ceramic and the tooth structure, as described in the literature.[7-9] The success of a ceramic restoration depends primarily on the durability of the bond between the ceramic and resin cement, as well as that between the cement and tooth structure.[10] The resin cements used specifically for luting ceramic veneers usually are activated by visible light. Advantages of these cements include their color stability and longer working time, compared to chemically cured and dual-cured resin cements.[11]

To enhance esthetic results, the ceramic is placed over the substrate prior to cementing the veneers, at which point the color obtained is evaluated. This placing can be done with water, glycerin gel, or try-in.[12] According to Al Ghazali et al, the try-in pastes that accompany resin cements allow dentists to predict the final esthetic result before cementation.[13] The purpose of the different shades of resin cements and try-in pastes is to improve the color and esthetics of the final restoration. The correct correlation between try-in and resin cement colors under ceramics is critical, especially when using extremely thin and translucent ceramics.[14]

This study sought to evaluate the shade of ceramic veneers resulting from different try-in materials. The null hypothesis was that there were no differences among the ceramic colors obtained with the try-in materials tested.

Materials and methods

Teeth preparation

Forty bovine teeth were used in this study. Initially, these teeth were stored in 0.05% thymol solution and kept in deionized water for 24 hours to remove the thymol. The roots were separated from their respective crowns with a refrigerated low-speed diamond saw, and the whole pulp was removed and discarded. The buccal surface was flattened on an enamel surface with 180 grit sandpaper (3M ESPE) mounted on the turntable of a polishing machine (DP-10, Struers, Inc.).

Veneer preparation

Initially, a wax pattern was made (11 mm wide x 8 mm thick). Forty ceramic discs (IPS Empress, Ivoclar Vivadent, Inc.)—color ETC0—were injected to obtain a ceramic block. Next, the blocks were placed in a cutting machine (IsoMet, Buehler) used at a speed of 250 rpm to produce 40 discs, each 1 mm thick. The discs were then polished manually with humid 800- and 1200-grit abrasive paper (3M ESPE) to obtain a thickness of 0.6 mm each. The thickness standardization was registered using a digital electronic caliper (Mitutoyo America Corporation).

Try-in application

The 0.6 mm ceramic discs were placed on the prepared dental substrate. The try-in materials were placed between the ceramic discs and the tooth substrate. A pilot study was conducted to standardize the application of the try-in materials.

Samples were distributed among 4 groups (n = 10). In Group 1, no material was used between the substrate and the ceramic. For Group 2, the try-in material was 0.2 ml of water, measured with a disposable syringe. Try-in material for Group 3 was a water-soluble gel (KY Jelly, Johnson & Johnson) while a try-in paste (Variolink

Table 1. Mean values and standard deviations (SD) of ΔE* for all groups.

Group	ΔE*
1	11.03 (2.59)[a]
2	10.57 (3.14)[a]
3	10.40 (1.97)[a]
4	11.90 (5.87)[a]

ΔE* indicates change in color

Similar letters denote no statistical difference ($P > 0.05$).

Table 2. Mean values and standard deviations (SD) of all color values obtained before and after the test using spectrophotometry ($P = 0.05$).

Group	L_o*	L*	a_o*	a*	b_o*	b*
1	98.69 (1.34)[A,a]	97.43 (1.77)[A,b]	-0.47 (0.55)[A,a]	-0.57 (0.33)[A,b]	22.68 (3.31)[A,a]	12.00 (1.50)[A,b]
2	97.69 (2.68)[A,a]	95.08 (2.37)[A,B,b]	-0.09 (0.78)[A,a]	-0.53 (0.44)[A,b]	24.02 (4.03)[A,a]	14.19 (2.75)[A,b]
3	97.77 (2.12)[A,a]	92.54 (2.57)[B,b]	-0.51 (1.18)[A,a]	-0.93 (0.71)[A,b]	23.3 (2.99)[A,a]	14.42 (2.68)[A,b]
4	95.47 (6.68)[A,a]	94.85 (3.08)[A,B,b]	-0.36 (1.91)[A,a]	-0.8 (0.79)[A,b]	24.41 (5.72)[A,a]	13.06 (1.75)[A,b]

L* indicates lightness

a* indicates hue (saturation in the red-green axis)

b* indicates hue (saturation in the blue-yellow axis)

L_o*, a_o*, and b_o* are the values measured first on the tooth substrate.

L*, a*, and b* are the values measured once the ceramic was in position over the substrate.

Similar uppercase letters denote no statistical differences for comparisons between columns for the same coordinates and similar lowercase letters denote no statistical differences for comparisons between lines for the same coordinates ($P > 0.05$).

Veneer, Ivoclar Vivadent, Inc.) was used for Group 4; for both Groups 3 and 4, the materials were placed between the substrate and the ceramics and pressed in a special appliance developed for this study to standardize the thickness of the materials with a pressure of 1 kg. Although not measured directly, it was assumed that the film thickness was relatively uniform after the pressing due to the uniform loading conditions.

Color measurements

All color measurements were performed with an spectrophotometer (Vita Easyshade Spectrophotometer, Vident). According to the International Commission on Lumination (CIE), color measurements make it possible to evaluate the degree of perceptible color change based on 3 coordinates: L*, a*, and b* (known as the *CIE-L*a*b* system*).[6] Color was measured in this study using the CIE-LAB system parameters, with L* indicating the lightness from 0 (black) to 100 (white), while a* and b* indicate the hue (a* represents the saturation in the red-green axis and b* in the blue-yellow axis). The color readings on each specimen were performed against a white standard background.

The measurements were performed twice: once in the tooth substrate (L_o*, a_o*, and b_o*) and once with the ceramic in position over the substrate (L*, a*, and b*). Three measurements were made in each instance and the mean of the coordinates obtained were considered for statistical analysis.

The color change (ΔE*) is used commonly to represent the difference in color between 2 measurements. A ΔE* was obtained between the substrate and the try-in values to evaluate the color difference obtained with the try-in materials and the ceramics over the substrate. The color change can be calculated using the formula:

$$\Delta E^* = [(\Delta L^*)^2 + (\Delta a^*)^2 + (\Delta b^*)^2]\ 0.5,$$

where ΔL* is the variation of L*, Δa* is the variation of a*, and Δb* is the variation of b*.

Statistical analysis

Data were organized in a database for statistical analysis with software SPSS 17.0 for Windows (IBM Corporation) with a significance level of 0.05. A Shapiro-Wilk test was used to evaluate the normality of parametric data distribution. One-way ANOVA was used to make comparisons between groups, while a paired t-test was used to make comparisons within groups.

Results

The data obtained for the different groups tested are shown in Tables 1 and 2. There was no statistical difference among the ΔE* found for the groups tested ($P = 0.804$). The ΔE* for Group 4 was higher than the other groups while Group 3 samples had the lowest mean. When comparing the different values obtained for each coordinate (L_o*, a_o*, b_o*) among the substrate measurements, no statistical significant difference was found for L_o*, a_o* and b_o* ($P = 0.294$, $P = 0.372$, and $P = 0.795$, respectively). A comparison of the same coordinates obtained from the try-in materials among the groups showed a statistically significant difference in L* values only between Groups 1 (control) and 3 (water-soluble gel) ($P = 0.02$). Finally, the comparison between L_o* vs L*, a_o* vs a*, and b_o* vs b* showed a statistical significant difference for all pairs ($P = 0.000$, $P = 0.006$, and $P = 0.000$, respectively).

Discussion

According to O'Brien et al, the color selection process is complex, due to the different characteristics of the color components, types of lighting used (that is, natural or artificial), and the subjectivity of the viewer's perception and experience.[15] Color selection also involves an assessment of dental shape, gloss, surface texture, translucency, gingival color, adjacent teeth, and tooth position in the arch.[15]

Until recently, color selection by visual perception with standardized scales was the most common method used in dental clinics.[16,17] However, this method of color selection has some disadvantages, such as the scales differing by manufacturer, resulting in a lack of systematization. In addition, earlier color scales were built in materials that are different from the restoratives now

used, and the thicknesses of the teeth used in the guides did not simulate clinical conditions. These disadvantages have led researchers to discontinue using this method as a model of comparison.[16,17]

Color was measured and compared in this study using the CIE-LAB system and a spectrophotometer. Spectrophotometers have been used industrially and in research to measure the colors of materials and substrates and also to determine the reflectance and transmittance factors of an object, as a function of the wavelength and electromagnetic radiation.[17] According to van der Burgt et al, spectrophotometry remains the most accurate method, however, the authors concede that it is expensive and technically complex.[18]

The data in the present study did not show significant differences among the L_o^*, a_o^*, and b_o^* values found in the different tooth substrates, indicating a standardization of the substrate color. It was important to confirm that the measured color for the ceramic was not affected by the tooth's color. The color of the ceramic specimens was also standardized by using 1 ceramic block to make all the discs. The try-in material was therefore the only way a group's color could be affected.

The results of this study revealed that the colors obtained with the ceramic in position were similar for all tested try-in materials, confirming the null hypothesis. These results corroborate with the 1997 study by Balderamos et al, who found that a preliminary color matching procedure can be done with either water, water-soluble gel, or try-in pastes.[12]

The present study used a try-in paste with a value of 0 and found that replacing this paste with water, water-soluble gel, or no material at all does not change the shade obtained. There was no statistical difference among the ΔE* values for the 4 groups. This result was expected because the value 0 try-in paste was a neutral color that should not change the color of the ceramic. Additional research is necessary to compare other colors of try-in pastes (with high or low values) to determine the ability of these materials to influence the final color of the ceramic veneers.

Analyzing the L*, a*, and b* values before and after the ceramic trial revealed that all groups demonstrated a statistically significant decrease in luminance (lower L*), less redness (lower a*) and less yellowness (lower b*) after the try-in materials were used. Considering that the try-in materials used were mostly transparent, the color differences observed were due to the ceramic chosen. The same situation is described in the study by Balderamos et al, which confirmed the effect of porcelain opacity on the resultant shade of veneer and substrate systems, and concluded that porcelain veneers provide a masking effect when luted with resin composite cements.[12]

Considering the L* value, the highest values were observed in Group 1 (mean L* = 97.43 ± 1.77). This group interposed no material between the porcelain veneer and the substrate; as a result, the color measured was that of a dry laminate over enamel. It is likely that the absence of try-in material decreases the color interaction between tooth and porcelain veneer, which may suggest that the high L* obtained was due to the brightness of the laminate. Groups 2-4 had lower L* values, possibly because the interposed material between laminate and substrate produced some color interaction.

According to Xing et al, ΔE*values higher than 2 may be considered a noticeable color change.[14] A 2010 study by Al Ghazali et al found that 3.3 units of color difference had not been noticeable for 50% of the observers using the visual method.[13] A recent in vivo study reported that a ΔE* of 5.5 units was necessary for the color difference to be perceived by the human eye.[19] Nevertheless, when observing the mean ΔE* values for all groups, the present study reported an apparent difference in color between the substrate and the tested groups in all cases. This concurs with other studies that showed ceramic veneers can be used for color correction.[4,11,20]

Conclusion

In all cases, the different try-in materials (try-in paste, water, water-soluble gel, and no material at all) produced similar color changes in the ceramic veneers.

Author information

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References

1. Kelly JR, Nishimura I, Campbell SD. Ceramics in dentistry: historical roots and current perspectives. *J Prosthet Dent.* 1996;75(1):18-22.
2. Horn HR. Porcelain laminate veeners bonded to etched enamel. *Dent Clin North Am.* 1982;27(4):671-684.
3. Anusavice KJ. *Phillips' Science of Dental Materials,* 11th ed. Philadelphia: Elsevier; 2003.
4. Azer SS, Rosenstiel SF, Seghi RR, Johnston WM. Effect of substrate shades on the color of ceramic laminate veneers. *J Prosthet Dent.* 2011;106(3):179-183.
5. Prata RA, Oliveira VP, Menezes FC, Borges GA, Andrade OS, Goncalves LS. Effect of 'Try-in' paste removal method on bond strength to lithium disilicate ceramic. *J Dent.* 2011;39(12):863-870.
6. Turgut S, Bagis B. Colour stability of laminate veneers: an in vitro study. *J Dent.* 2011;39(Suppl 3):e57-e64.
7. Lacy AM, LaLuz J , Watanabe LG, Dellinges M. Effect of porcelain surface treatment on the bond to composite. *J Prosthet Dent.* 1988;60(3):288-291.
8. Kamada K, Yoshida K, Atsuta M. Effect of ceramic surface treatments on the bond of four resin luting agents to a ceramic material. *J Prosthet Dent.* 1998; 79(5):508-513.
9. De Castro HL, Passos SP, Zogheib LV, Bona AD. Effect of cement shade and light-curing unit on bond strength of a ceramic cemented to dentin. *J Adhes Dent.* 2012;14(2):155-160.
10. Alqahtani MQ, Aljurais RM, Alshaafi MM. The effects of different shades of resin luting cement on the color of ceramic veneers. *Dent Mater J.* 2012;31(3):354-361.
11. Archegas LR, Freire A, Vieira S, Caldas DB, Souza EM. Colour stability and opacity of resin cements and flowable composites for ceramic veneer luting after accelerated ageing. *J Dent.* 2011;39(11):804-810.
12. Balderamos LP, O'Keefe KL, Powers JM. Color accuracy of resin cements and try-in pastes. *Int J Prosthodont.* 1997;10(2):111-115.
13. Al Ghazali N, Laukner J, Burnside G, Jarad FD, Smith PW, Preston AJ. An investigation into the effect of try-in pastes, uncured and cured resin cements on the overall color of ceramic veneer restorations: an in vitro study. *J Dent.* 2010;38(Suppl 2):e78-e86.
14. Xing W, Jiang T, Ma X, et al. Evaluation of the esthetic effect of resin cements and try-in pastes on ceromer veneers. *J Dent.* 2010;38(Suppl 2):e87-e94.
15. O'Brien WJ, Nelson D, Lorey RE. The assessment of Chroma sensitivity to porcelain pigments. *J Prosthet Dent.* 1983;49(1):63-66.
16. Sproull RC. Color matching in dentistry. I. The three-dimensional nature of color. *J Prosthet Dent.* 1973; 29(4):416-424.
17. Paul S, Peter A, Pietrobon N, Haemmerle CH. Visual and spectrophotometric shade analysis of human teeth. *J Dent Res.* 2002;81(8):578-582.
18. van der Burgt TP, ten Bosh JJ, Borsboom PSF, Kortsmit WJ. A comparison of new and conventional methods for quantification of tooth color. *J Prosthet Dent.* 1990; 63(2):155-162.
19. Douglas RD, Steinhaurer TJ, Wee AG. Intraoral determination of the tolerance of dentists of perceptibility and acceptability of shade mismatch. *J Prosthet Dent.* 2007;97:200-208.

20. Dozic A, Kleverlaan CJ, Meegdes M, van der Zel J, Feilzer AJ. The influence of porcelain layer thickness on the final shade of ceramic restorations. *J Prosthet Dent.* 2003;90(6):563-570.

Manufacturers

Buehler, Lake Bluff, IL
800.283.4537, www.buehler.com

IBM Corporation, Armonk, NY
800.426.4298, www.ibm.com

Ivoclar Vivadent, Inc., Amherst, NY
800.533.6825, www.ivoclarvivadent.us

Johnson & Johnson, Skillman, NJ
800.690.1826, www.jnj.com

Mitutoyo America Corporation, Aurora, IL
888.648.8869, www.mitutoyo.com

Struers, Inc., Westerville, OH
440.871.0071, www.struers.com

Vident, Brea, CA
800.828.3839, www.vident.com

3M ESPE, St. Paul, MN
800.364.3577, solutions.3m.com

Achieving highly esthetic anterior restorations with ideal assessment, communication, and technique

Scott Finlay, DDS, FAGD, FAACD ▪ Nelson Rego, CDT, AAACD

Although all cases should be approached comprehensively, restoring a limited segment in the esthetic zone presents challenges particularly related to *microesthetics*. Microesthetics are those criteria related to the subtle intricacies of shade, textures, translucencies, and surface effects that make teeth look like teeth. These are the criteria that aid dentists in fooling the eye and allowing restorations to blend invisibly into the smile. Completing a comprehensive assessment of a patient ensures that the restorative foundation will remain biologically and structurally predictable, durable, and above all, esthetically pleasing. Starting esthetic treatment without first doing a comprehensive assessment will result in a compromised result. Within the criteria of microesthetics, the utilization of a common nomenclature and quantitative means of communication between the restorative dentist and the laboratory ceramist are at the core of success. The use of prototypes during the provisionalization phase and progressive techniques in digital photography are invaluable tools. Along with traditional techniques in acquiring proper shade selection, the use of cross-polarization filters has been proven to be an effective way to eliminate spectral artifacts typically found in flash photography. Additionally, the use of a color-corrected master die system provides the ceramist a method to calibrate shades on the lab bench by capturing images—via the cross-polarization filters—that are similar to what is observed clinically.

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One of the most challenging clinical situations in dentistry is the restoration of a limited segment in the esthetic zone. Often complicating this task is varying hybrid restorative preparation designs that require differing laboratory modalities to create restorations that blend into the surrounding dentition. Ceramic systems that utilize a common veneering ceramic offer a great advantage in harmonizing the restorations with the full smile. An ideal material replicates natural tooth characteristics through a combination of opalescent, translucent, and light diffusion properties; and replicates the polychromatic optical properties observed in natural teeth. These restorations also require a material that exhibits biocompatibility, high strength, and durability. Although restorative success primarily hinges upon proper prosthetic design, it behooves the clinician and the ceramist to utilize the advancements in modern materials to ensure a predictable, durable, and beautiful result.[1]

The clinician's desire to emulate nature and the patient's expectations of enhanced esthetics have raised the bar on what is considered a successful restoration. The foundation of a successful result lies within the ability of the restorative team's evaluation and the interpolation of essential criteria related to smile design with a common vision and nomenclature. The *American Academy of Cosmetic Dentistry* (AACD) is the largest cosmetically focused dental academy in the world. The AACD offers a credentialing program that defines a platform for evaluation of smile design based upon commonly accepted standards as supported by the preponderance of literature. The credentialing process provides a common nomenclature when assessing and communicating criteria related to smile design. A smile design analysis is divided into 3 sections to conceptually guide the pretreatment evaluation.[2] The analysis begins with the broadest strokes of smile design, then progressively narrows the focus to each individual tooth in order to replicate nature. This analysis starts from the perspective of *global esthetics,* an approach that focuses on those criteria that are observed in unretracted smiles and how the smile orients to the face. As the analysis narrows, the focus narrows on the elements of *macroesthetics*. Macroesthetics relates to the shapes and contours of teeth and their relationship to each other. The final phase of analysis focuses on *microesthetics*, which are those criteria related to the subtle intricacies of shade, textures, translucencies, and surface effects that essentially make teeth look like teeth. These are the criteria that aid the practitioner in fooling the eye and allowing restorations to blend invisibly into the smile.

Although the intended restorative focus may be limited in many cases, the final diagnosis must be comprehensive in evaluating the biological, structural, functional, and esthetic needs of the patient. The assessment begins with the evaluation of the temporomandibular joint (TMJ), which provides a precise location from which the design process can begin. The understanding of this sophisticated system allows for the optimization of esthetics by beginning with the end in mind, but with a focused discipline and an understanding that allows the clinician to modulate the desired contours to be in harmony with such functions as occlusion. Failure to comprehensively evaluate the system will not only put restorations in harm's way, but also detrimentally affect the prognosis and success of the case.[3]

The vision of the restorative dentist and ceramist team needs to be synergistic. They need to continually find new ways to creatively communicate in a progressive and quantitative way to optimize function and esthetics in order to take advantage of the advancements in contemporary materials. Predictability can only be achieved with mutual accountability and the adherence to an effective protocol.

Evaluating and communicating case specifics

Restorations are provided to the patient in what can easily be categorized as an

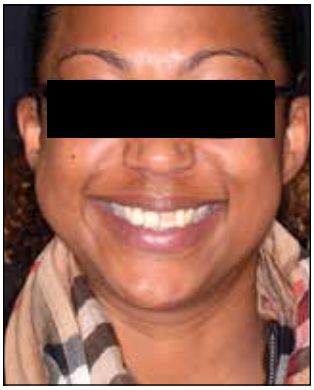

Fig. 1. Preoperative full face view of the patient's smile.

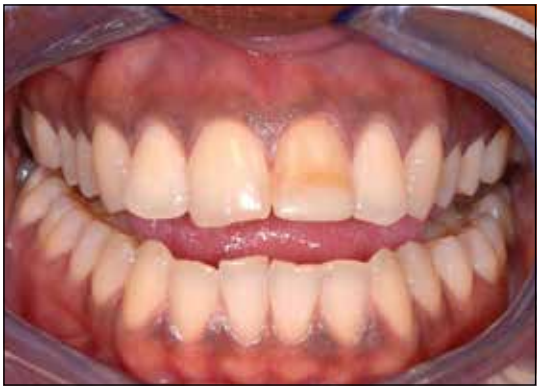

Fig. 2. Preoperative 1:2 frontal retracted view.

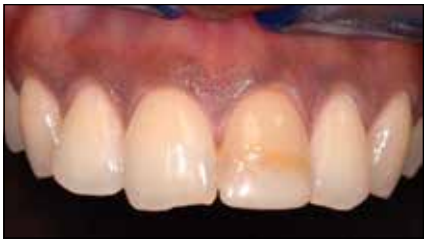

Fig. 3. Preoperative 1:1 frontal retracted view.

incredibly hostile oral environment. Any restoration will be subjected to thermal changes, staining agents, and hundreds of pounds of force. Careful evaluation and diagnosis of risk assessment are essential to a treatment plan in order to manage these etiologies of dental deterioration.

Diagnostic photographs are an essential tool in documentation and communication between the clinician and the laboratory. Image composition needs to be reproducible from different perspectives: angle, magnification, and exposure. Digital photography enhances laboratory communication and brings to life the stone models on the lab bench. A variety of photographic techniques can enhance the evaluation of shade comparisons to the surrounding dentition, intensity of characterization, degrees of translucency and opacity within the incisal edge, as well as the incisal edge position as it relates to provisional prototypes and the intended final restorations.[4,5]

Determining the proper shade requires an understanding of the science of color, including the influences of surface finish and texture.[6] In the experience of this restorative dentist/ceramist team, cross-polarization filters provide an invaluable tool for shade calibration. This photographic technique enhances the visual detail of enamel defects and eliminates specular reflections that can be caused by saliva, or a surface finish that was created by the illumination source. It involves the use of polarization filters that are oriented on the flash and lens at 90 degrees to each other.[7] These specular reflections are essentially artifacts in the image that mask the underlying idiosyncrasies of the tooth. Eliminating these artifacts provides a unique opportunity to calibrate the shade from the operatory to the lab bench. In the lab, the ceramist can extend this application with the utilization of a color-corrected die system and a cement simulator to recreate what is observed clinically.

Accurately mounted diagnostic wax-up models are an attempt to implement the desired changes to the patient's smile from a 2-dimensional perspective to a virtual 3-dimensional simulator—the articulator. From the modulated contours of the diagnostic wax-up, stents can then be fabricated to guide conservative preparations and the creation of prototypes. These prototypes are the most important quality control step relative to functional and global/macro esthetics in the restorative process.[8] These prototypes are tested and detailed clinically to ensure that they meet the functional, phonetic, and esthetic needs of the patient. These refined contours are then communicated to the dental laboratory with an impression and the creation of an approved provisional model. From this approved provisional model, the laboratory technician can create 2 key indices to ensure functional success: the incisal edge matrix and a custom incisal guide table. These indices will clearly define the exact incisal edge position of the final restorations and a guidance that is in harmony with the envelope of function.[9]

Case report

A 34-year-old woman presented with discolored direct composite restorations on teeth No. 8 and 9 (Fig. 1). The patient reported damaging her anterior teeth 10 years earlier when she fell after fainting at work (Fig. 2). She had composite restorations placed to repair teeth No. 8 and 9 at that time, with additional endodontic treatment on tooth No. 9 (Fig. 3). The patient also presented with interproximal resin restorations placed to restore previous caries. She desired to improve the appearance of her anterior teeth with the most durable option possible.

Treatment planning

The clinical examination included hard and soft tissue evaluations, complete radiographic survey, diagnostic photographic series, TMJ exam, occlusal survey (T-Scan, Tekscan, Inc.), and mounted diagnostic models. It was determined that the patient was in generally good oral health, with adequate oral hygiene and a healthy periodontium. The patient demonstrated a full range of motion. The joints loaded incrementally without symptoms, and there was a slide from centric relation (0.5 mm horizontal and 1 mm vertical) to centric occlusion.

Although proposed as an ideal treatment option, the patient declined orthodontics to better position her teeth. Any shortcoming in the treatment recommendations introduces a level of compromise; however, the resulting consequences of this particular limitation were deemed insignificant by the study clinician.

Fig. 4. View of the diagnostic wax-up on the articulator.

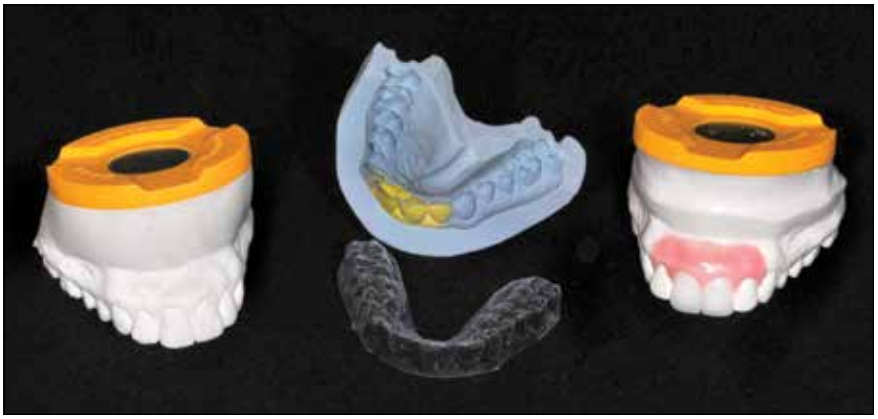

Fig. 5. *Left.* Pre-op model. *Center.* Clear reduction guide and temporary matrix. *Right.* Diagnostic waxup.

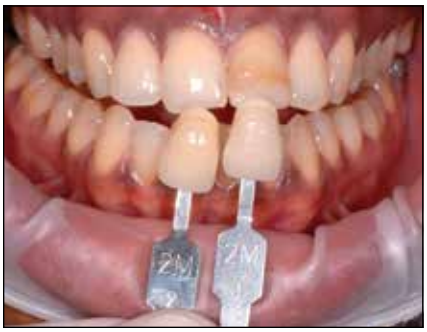

Fig. 6. Final shades were selected before preparation to avoid dehydration.

Fig. 7. A cross-polarization device was used for additional shade information.

Fig. 8. A cross-polarization device was used to determine the prep shade.

After dental prophylaxis, the patient's teeth were whitened using a tray system. Accurately mounted diagnostic study models were evaluated on a semi-adjustable articulator. Treatment options were then explored to achieve the desired improvements to the patient's smile, which included an equilibration to optimize and balance occlusal forces and meet the requirements for a stable occlusal design, as well as a diagnostic wax-up to simulate the intended changes in contour for teeth No. 8 and 9 (Fig. 4).

The diagnostic wax-up was used to fabricate a Siltech matrix (Ivoclar Vivadent, Inc.) that the clinician used—along with a clear .020 inch tray—to create the new provisional and to properly evaluate the amount of reduction (Fig. 5).

The restorative treatment plan included a conservatively prepped lithium disilicate (IPS e.max, Ivoclar Vivadent, Inc.) layered veneer on tooth No. 8. A core repair and a lithium disilicate layered porcelain jacket crown was planned for tooth No. 9.[10] Provisionalization for teeth No. 8 and 9 would be accomplished utilizing the "lock-on technique" and a self-curing composite (Telio, Ivoclar Vivadent, Inc.).

The final restorative shades should be determined prior to preparation in order to avoid dehydration of the tooth surfaces (Fig. 6). The clinician in this study observed the natural shade under a variety of light sources and intensities, being sensitive to the properties of metamerism. After viewing the shade in ideal daylight, a color-corrected lighting of 5500 Kw/a color rendering index near 95% was also employed to observe the natural shade. The initial shade value was first selected, the clinician taking care to control the level of light reaching the rods by squinting. Brief glances of 5 to 7 seconds helped to avoid oversaturating and desensitizing the cones of the clinician's eyes. An 18% grey card was used as a "middle grey" reference to reset the color balance and minimize the "blue fatigue" effect of the eye. A limited field of view also helped eliminate visual distractions that may have interfered with the interpolation of the shade.[11,12]

A cross-polarization device was utilized to eliminate spectral reflections and artifacts in the images used to communicate color, and multiple shade guides were compared to avoid distraction from the shade variations (Fig. 7). A spectrophotometer (Vita Easyshade, Vident) was also used to provide additional information and verification of the ideal shade.[13]

After shade selection, the preparation design was driven by the need to cover the surfaces of the teeth in order to meet the functional and esthetic needs of this case while minimizing the removal of

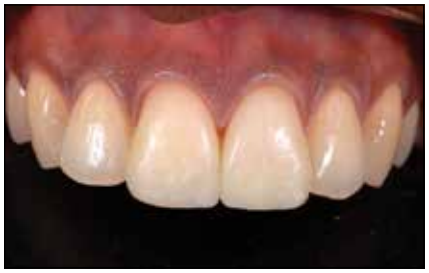

Fig. 9. Provisionals were fabricated using an esthetic material.

Fig. 10. A custom incisal guide table was fabricated from the protrusive check bite.

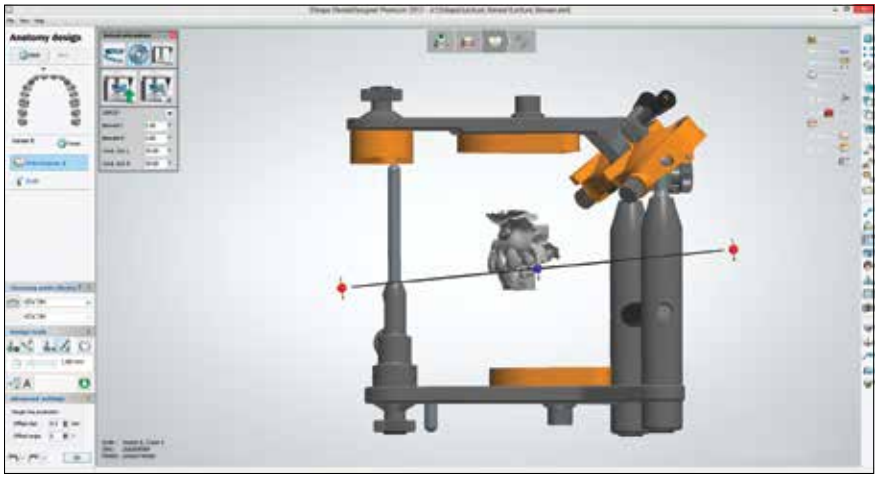

Fig. 11. Software was used to scan the models into the digital articulator.

tooth structure. A conservative preparation of 0.3 mm was created for the veneer on tooth No. 8. The preparation shade was then recorded to facilitate the selection of the color-corrected die material in the lab to assist in the calibration of the final shade (Fig. 8).[14] The completed equilibration was verified in the diagnostic models prior to treatment, to ensure the balance of the occlusal contacts and ensure occlusal stability.

Provisionalization

The mounted models and diagnostic wax-up provided a blueprint for the smile design changes and fabrication of the provisional matrix.[15] The provisional matrix intimately captured all of the wax-up details and contours—as well as the relationship to the opposing arch—by creating indexing on the intaglio surface of the matrix. When creating this matrix, the putty was placed on the upper member of the articulator prior to the final set. The articulator was then closed to engage the opposing arch, which recorded an indexing of the mandibular teeth on the bottom of the provisionalization stent. This feature of indexing ensured the complete seating and even distribution of the hydrostatic pressure of the composite material as the stent was re-introduced to the patient's mouth, allowing the patient to close and engage these indices.

Spot etching was used on the incisal aspect of tooth No. 8 to help retain the provisional, and the etchant was kept several millimeters away from the cavosurface to prevent interference with the seating of the final restoration. A bonding agent (ExciTE F DSC, Ivoclar Vivadent, Inc.) was placed on this etched area prior to the seating of the provisional matrix.

A bilaminar technique was used to establish a natural gradation of shade: A1 for the cervical half and BL3 for the incisal half. Once the matrix was inserted, the lighter shade of Telio provisional material was injected into the incisal half, and the warmer chroma was injected into the cervical half. The bisacryl was left in place for 3 minutes to allow for a complete cure, with the intention of enabling the provisional to lock into place. The matrix was then removed and any flash was eliminated.

Additional characterization of the provisional was accomplished utilizing several shades for characterization (IPS Empress Direct Color, Ivoclar Vivadent, Inc.). Blue tint was diluted with an unfilled resin and then applied to the incisal aspect to simulate translucency; white opaque was similarly diluted to create the milky hypocalcification observed in the adjacent teeth. A final glaze was then placed (Fig. 9).

The patient was instructed to use peroxyl, a Waterpik (Water Pik, Inc.), and a toothbrush. The patient was scheduled for another appointment 48 hours postinsertion, at which time the prototypes were re-evaluated for esthetics and function, and an impression was taken to record the approved contours of the provisional.[16]

Laboratory fabrication

The model of the approved provisional was mounted on a semiadjustable SAM articulator (SAM Prazisionstechnik GmbH) and used to create a custom incisal guide table (PATTERN RESIN LS, GC America, Inc.) that accurately replicated the lingual surfaces of the provisional, ensuring that the envelope of function would not be adversely affected (Fig. 10).

The final restorations were fabricated based on the approved provisional restorations, incisal edge matrix, and custom incisal guide table. The models were scanned into the digital articulator (3Shape A/S) (Fig. 11). After the digital protrusive and excursive movements were

Fig. 12. The wax-ups were milled after the digital protrusive and excursive movements were calculated.

Fig. 13. Wax-ups were invested and pressed using a lithium disilicate material.

Fig. 14. The restorations were cut back and layered using multiple shades of porcelain.

calculated based on the mounted cases, the final restorations were milled in wax using a 4-axis milling machine (Wieland Mini Mill, Ivoclar Vivadent, Inc.) (Fig. 12). The waxed units were marginated, sprued, and pressed using IPS e.max lithium disilicate (Ivoclar Vivadent, Inc.) in a No. 2 shade.[17] After a careful inspection of the marginal integrity, the restorations were cut back and layered with multiple shades of veneering ceramic, contoured, and layered with final incisal powders (Fig. 13-15).

Color correct dies (IPS Natural Die Material, Ivoclar Vivadent, Inc.) were used to calibrate the shade, as the new restorations were very thin (0.3 mm) and the underlying shade of the remaining tooth structure influenced the final perceived shade. The study ceramist selected a natural die material to virtually assist the clinician chairside. The restorations were photographed on the natural die material with glycerin, and the images were then captured with and without the cross-polarization filters and the respective shade tabs (Fig. 16). These images were compared to those captured by the clinician in order to verify the final shade.

The incisal edge matrix created from the approved provisional was used to verify the incisal edge position of the final restorations in both a vertical and horizontal component (Fig. 17 and 18).

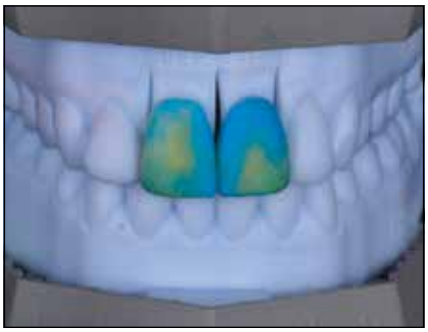

Fig. 15. The restorations were layered with final incisal powders.

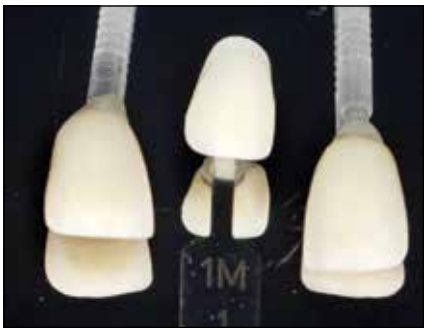

Fig. 16. Stump dies and the shade tab were photographed with a polarization filter for shade verification.

Fig. 17. An incisal edge matrix was fabricated from the approved provisionals.

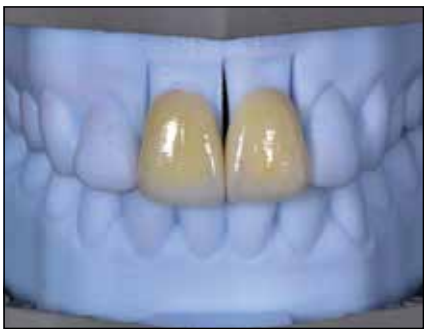

Fig. 18. Definitive restorations on the 3-dimensional printed model.

Final restoration try-in and cementation

The restorations were returned to the study clinician. The provisionals were removed, preparations cleaned, and restorations tried-in in order to evaluate fit, esthetics, and contacts. A neutral try-in paste was utilized as a cement simulator, and the shade was evaluated clinically and photographically. Both methods were used with and without a cross-polarization filter.[18]

Once approved, the restorations were cleaned with Ivoclean paste (Ivoclar Vivadent, Inc.), and a priming agent (Monobond Plus, Ivoclar Vivadent, Inc.) was applied. The preparation surfaces were cleaned and etched. A light-curing veneer cement (Variolink Veneer, Ivoclar Vivadent, Inc.) was applied to tooth No. 8, as the restoration thickness was deemed conservative enough to not require a dual-curing luting material. An alternative cement (Variolink II, Ivoclar Vivadent, Inc.) was applied to tooth No. 9, as the restoration thickness benefited from a dual-cure luting agent. The restorations

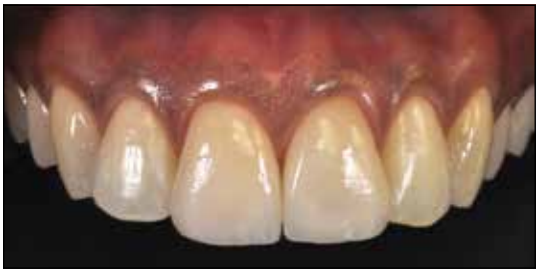

Fig. 19. Immediate postoperative view of seated restorations.

Fig. 20. Final postoperative view of the patient.

were seated and light-cured according to the manufacturer's instructions. Excess cement was removed, and the occlusion was checked and verified (Fig. 19). The patient was satisfied with the new esthetics and function of her smile (Fig. 20).

Conclusion

There are many challenges when managing restorations in the smile zone. The keys to success involve the proper assessment, technique, and communication to ensure a common vision for both the restorative dentist and the ceramist. In this case, the patient required 2 independent, highly esthetic restorations with differing restorative requirements. It was only after a complete exam and an adherence to a set protocol for diagnosis and treatment planning—utilizing photography, mounted models, and a diagnostic wax-up—that a dental blueprint was created that ensured a predictable outcome. Many of the elements of microesthetics and shade determination that allow restorations to invisibly dissolve into the smile zone can be challenging; however, the use of a deliberate shade-matching protocol—including color-corrected lighting, cross-polarization filters and color-corrected master dies—has proven to be invaluable. Precisely contoured prototype restorations are the key to predictability for functional esthetics. The use of a versatile ceramic system allows not only the conservation of tooth structure, but also the harmonization of different preparation designs to create a functional, durable, and esthetically pleasing result.

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Disclaimer

The authors have no financial, economic, commercial, and/or professional interests related to topics presented in this article.

References

1. Pieger S, Salman A, Bidra AS. Clinical outcomes of lithium disilicate single crowns and partial fixed dental prostheses: a systematic review. *J Prosthet Dent.* 2014; 112(1):22-30.
2. Finlay S. *AACD Guide to Accreditation Criteria: Contemporary Concepts in Smile Design.* Madison, WI: American Academy of Cosmetic Dentistry; 2014.
3. Dawson PE, Cranham JC. Aesthetics and function: conflict or complement? *Dent Today.* 2007;10:80, 82-83.
4. Elter A, Canikliogl B, Deger S, Ozen J. The reliability of digital cameras for color selection. *Int J Prosthodont.* 2005;18(5):438-440.
5. Dunn JR, Hutson B, Levato CM. Photographic imaging for esthetic restorative dentistry. *Compend Contin Educ Dent.* 1999;20(8):766-778, 770-772, 774 passim.
6. Terry DA, Snow SR, McLaren EA. Contemporary dental photography: selection and application. *Compend Contin Educ Dent.* 2008;29(8):432-436, 438, 440-442 passim; quiz 450, 462.
7. McLaren EA, Schoenbaum T. Combine conventional and digital methods to maximize shade matching. *Compend Contin Educ Dent.* 2011;32(Spec No 4):30, 32-33.
8. Kim E, Son T, Lee Y, Jung B. Development of polarization dental imaging modality and evaluation of its clinical feasibility. *J Dent.* 2012;40(Suppl 1):e18-e25.
9. Cranham, JC, Finlay, SW. Timeless principles in esthetic dentistry. *J Cosmet Dent.* 2012:28(3):60-68.
10. Small BW. Pretreatment wax-ups and provisionals for restorative dentistry. *Gen Dent.* 2005;53(2):98-100.
11. Kois JC, McGowan S. Diagnostically generated anterior tooth preparation for adhesively retained porcelain restorations: rationale and technique. *J Calif Dent Assoc.* 2004;32(2):161-166.
12. Nakhaei M, Ghanbarzadeh J, Keyvanloo S, Alavi S, Jafarzadeh H. Shade matching performance of dental students with three various lighting conditions. *J Contemp Dent Pract.* 2013;14(1):100-103.
13. Bahannan SA. Shade matching quality among dental students using visual and instrumental methods. *J Dent.* 2014;42(1):48-52.
14. Witkowski S, Yajima ND, Wolkewitz M, Strub JR. Reliability of shade selection using an intraoral spectrophotometer. *Clin Oral Investig.* 2012;16(3):945-949.
15. Sesemann MR. Successful strategies for matching one or two indirect restorations to natural dentition. *Gen Dent.* 2011;59(6):448-456.
16. Helvey GA. Provisional material considerations: creating a blueprint of the final restorations. *Compend Contin Educ Dent.* 2014;35(1):56-60.
17. Reshad M, Cascione D, Kim T. Anterior provisional restorations used to determine form, function, and esthetics for complex restorative situations, using all-ceramic restorative systems. *J Esthet Restor Dent.* 2010;22(1):7-16.
18. Chaiyabutr Y, Kois JC, Lebeau D, Nunokawa G. Effect of abutment tooth color, cement color, and ceramic thickness on the resulting optical color of a CAD/CAM glass-ceramic lithium disilicate-reinforced crown. *J Prosthet Dent.* 2011;105(2):83-90.

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A minimally invasive smile enhancement

Fred H. Peck, DDS

Minimally invasive dentistry refers to a wide variety of dental treatments. On the restorative aspect of dental procedures, direct resin bonding can be a very conservative treatment option for the patient. When tooth structure does not need to be removed, the patient benefits. Proper treatment planning is essential to determine how conservative the restorative treatment will be. This article describes the diagnosis, treatment options, and procedural techniques in the restoration of 4 maxillary anterior teeth with direct composite resin. The procedural steps are reviewed with regard to placing the composite and the variety of colors needed to ensure a natural result. Finishing and polishing of the composite are critical to creating a natural looking dentition that the patient will be pleased with for many years.

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The phrase *minimally invasive dentistry* has been around for many years in our profession.[1] The trend toward conservative treatment has gained momentum in recent years, and preserving tooth structure should be of the utmost importance in the daily treatment of our patients. Aggressive removal of tooth structure is detrimental to the long-term health of the tooth and, ultimately, the patient, and should only be chosen when other options are not available. Each patient should be presented with all options prior to any dental treatment, especially those which are elective.[2,3] The following case report portrays how one can be conservative in both diagnosis and treatment when restoring a smile.[4]

Case report

The patient was a 22-year-old man who was dissatisfied with his smile (Fig. 1). Specifically, he did not like the spaces between his upper anterior teeth; however, he was content with their color (Fig. 2 and 3). A complete dental examination was performed including photographs, full mouth X-rays, as well as periodontal, occlusal, and restorative evaluations. It was determined that joint loading was normal, there was no tooth mobility, no signs of wear, and no occlusal issues.[5] The patient had moderate gingivitis, no decay, and structurally sound teeth. His primary concerns were the diastemas between his anterior teeth, and the size and shape of these teeth.

Several options were discussed with the patient. Orthodontics would align the teeth, but not solve the size discrepancies. Other options included porcelain veneers or direct resin bonding.[6] After reviewing the options, the decision was made to restore teeth No. 7-10 with direct resin bonding.[7] The position of the patient's teeth appeared to allow for a no-prep type of restoration; this would be verified after a diagnostic model evaluation.[8]

Diagnostic models were taken utilizing an alginate substitute material (Algin-X Ultra, DENTSPLY Caulk), ensuring the ability to obtain excellent gingival margins. A Kois facial analyzer (Panadent Corporation) was used to mount the models on an articulator (Panadent Corporation).[9] A diagnostic wax-up was completed on the plaster models and a full arch putty was made, along with an incisal edge putty matrix (Template, Clinician's Choice Dental Products, Inc.).[10] The patient was scheduled to try in the proposed treatment shape for contour and incisal edge position (Fig. 4 and 5). The patient approved the provisional workup and was scheduled for treatment.[11]

The goal of the treatment was to increase the width of the 4 incisors to impart a more natural look with the correct proportions of width to length ratios.[12] The height of the gingival levels were also discussed with the patient. Ideally, the gingival heights should be even with the central incisors and cuspids. The lateral incisor gingival free margins can be equal to the central incisors or slightly lower. The gingival discrepancies were discussed with the patient, but he was content to leave the tissues as they were.[13] Evening out the free gingival margins

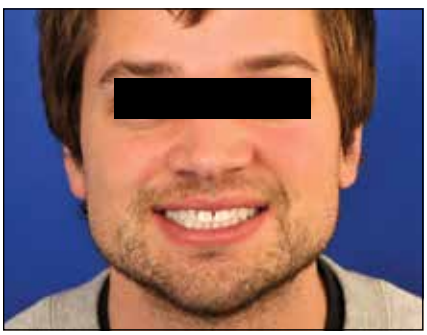

Fig. 1. Full face photograph pre-restoration.

Fig. 2. Unretracted smile pre-restoration.

Fig. 3. Retracted view of mouth pre-restoration.

Fig. 4. Smile with putty matrix try-in using BIS-GMA resin from diagnostic wax-up.

Fig. 5. Retracted view of BIS-GMA resin from putty matrix of wax-up.

Fig. 6. Photograph showing preliminary color matching of composite resin.

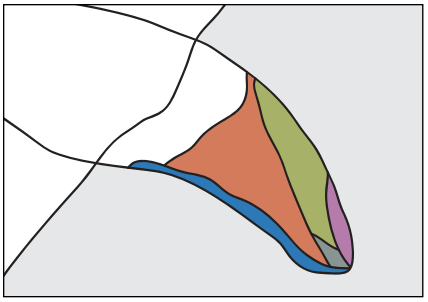

Fig. 7. Cross-section color shade map of desired composite resin shades.

Fig. 8. Sample cutback of an incisal putty on a typodont model.

Fig. 9. Handpiece reduction and thinning of lingual composite shelf on typodont model.

Fig. 10. Photograph showing the dentist closing half of the mesial diastema on a single tooth and verifying the midline is perpendicular to the horizon and not canted.

would have required a combination of periodontal crown lengthening and the orthodontic extrusion of tooth No. 7 to lower the margin. It is important that these discussions take place prior to any treatment to avoid patient dissatisfaction.

The patient returned for a multi-hour appointment. The composite chosen was Estelite Omega (Tokuyama Dental America, Inc.), which, in the author's opinion, has excellent handling properties.

The desired color was selected after a quick mock-up of the color layers over the unetched tooth surface. Trying out the proposed colors before final selection is highly recommended to avoid ending up with the wrong color combination at the end of treatment (Fig. 6). A shade map was then made of all the proposed colors for future reference (Fig. 7).[7] The teeth were isolated with cotton rolls, and a Premier Comfort View small retractor (Premier Products Co.) was used. A retraction cord was placed in the sulci of the teeth that were to be restored. A size 0 retraction cord (Ultradent Products, Inc.) was dipped in ViscoStat Clear 25% aluminum chloride gel (Ultradent Products, Inc.). Using a clear retraction gel helps prevent discoloration of the final composite restorations.

The teeth were air-abraded with a Danville Prep Start (Danville Materials) utilizing 27μ aluminum oxide, then lightly roughened with a coarse Sof-Lex disk (3M ESPE). The central incisors were treated first. Each tooth was isolated with a Mylar strip to keep the phosphoric acid off of adjacent teeth. Then the tooth was etched with 37% phosphoric acid. A bonding agent (ALL BOND UNIVERSAL, Bisco, Inc.) was used over the prepared tooth surface. At this time, the Mylar strip was removed. The putty matrix was held against the lingual surface. A very thin layer of composite was placed in the lingual putty matrix as the lingual shell (Fig. 8). The selected shade was MILKY WHITE (Tokuyama Dental America, Inc.). Prior to curing, a 12B Bard-Parker surgical blade (Aspen Surgical Products) was utilized to cut between the uncured composite and the adjacent tooth, along the matrix. This prevented the thin lingual shelf from bonding to the adjacent tooth and fracturing when detached from the tooth surface. If the lingual shelf is too thick, it can be thinned with a bur, then the bonding protocol can be repeated before proceeding (Fig. 9). In order to fill in the appropriate width of the diastema and to simulate the dentin shade while blocking out the darker appearance from the back of the mouth, a more opaque shade was used (DA-1, Tokuyama Dental America, Inc.) over the MILKY WHITE shade (Fig. 10).

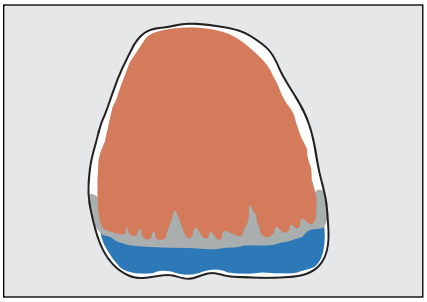

Fig. 11. Color diagram showing dentin shade, lingual shelf, and translucent resin in developmental lobes.

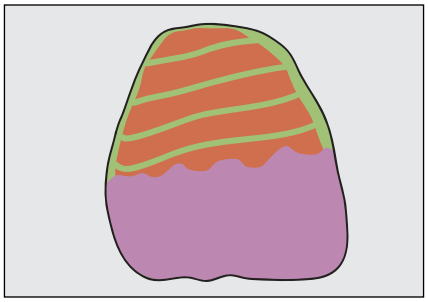

Fig. 12. Color diagram showing enamel layer and value modifying outermost layer.

Fig. 13. Metal matrix band (.001 inch thick) to protect adjacent teeth during polishing phase.

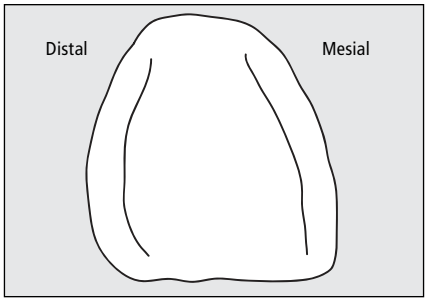

Fig. 14. Diagram of desired line angles.

Fig. 15. Pencil drawing of planned facial contours on the tooth.

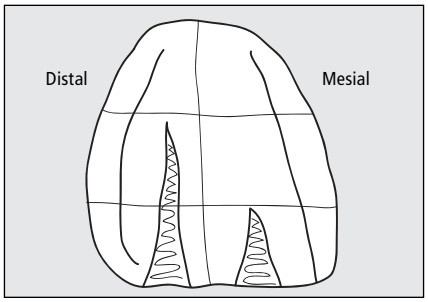

Fig. 16. Diagram of facial contours and line angles.

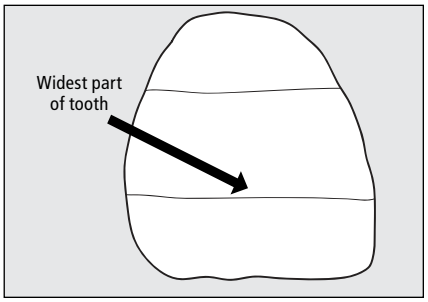

Fig. 17. Diagram of 3 horizontal segments of facial surface.

The cervical area was covered with a more translucent enamel shade (EB-1, Tokuyama Dental America, Inc.) to impart some chroma and simulate a more intense color at the marginal area. The cervical portion was built out to full contour for 2-3 mm from the margin, then beveled toward the incisal. The middle and incisal thirds were covered with the enamel shade BL-2 (Tokuyama Dental America, Inc.). Prior to curing the composite in the incisal third, indentations were placed to simulate the developmental lobes that are seen in natural dentition (Fig. 11).[14,15] The composite was cured and a translucent shade (Tokuyama Dental America, Inc.) was placed in the depressions and cured. The final color placed was MILKY WHITE, which acted as a value-modifying layer over the incisal third to slightly reduce the value (Fig. 12).

The goal of adding composite should be to place just enough composite without too much bulk and overcontouring. It is always better to add small increments at a time. If each shade is overbulked, then the final shape would be too large. Excess reduction will then be necessary and this will result in removal of the outermost shades, with a resultant loss in color variations. The tooth will then appear monochromatic in color.

The last step of applying the composite was the final cure. It is very important to cure the oxygen-inhibited layer. This was accomplished in this case by covering the composite with a water soluble glycerin gel that easily washed off.[16]

The initial contouring was started on the central incisor. Ideally, the only time a bur should be used in the initial stages is to smooth the cervical margins adjacent to the retraction cord. If too much composite is placed, then a fine diamond bur can be used to remove the excess. A thin (.001 inch) metal Tofflemire band was placed on both interproximal surfaces to prevent damage to the adjacent teeth while polishing (Fig. 13). A coarse Sof-Lex disk was used with the cutting surface facing the back of the contra-angle handpiece. This prevented the mandrel from scratching the tooth. A pencil line was drawn on the facial surface toward the mesial and distal where the line angles were desired. The facial aspect between the lines was adjusted first, and excess composite was removed from the lingual. This stage of adjustment is necessary to ensure that the lingual/incisal and facial/incisal line angles are lined up with the desired shape in the putty matrix from the diagnostic wax-up.

After the tooth was contoured to a proper outline shape with the coarse disk, medium and fine disks were used for further polishing. Any overhangs were removed with the surgical

Fig. 18. Unretracted smile showing completed resin bonding several weeks after restoration.

Fig. 19. Retracted view of mouth with final polish completed several weeks after restoration.

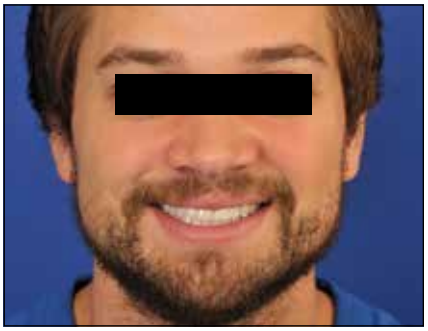

Fig. 20. Final full face photograph several weeks after restoration.

Bard-Parker blade No. 12B. The speed of the electric handpiece—set at a contra angle—was 40,000 rpm.

The finishing of a composite restoration takes precision and time. One must ensure that all surfaces are polished completely and no sharp angles or overhangs are present. Dentists who are new to restoring anterior teeth with direct resin veneers may make the mistake of not giving the finishing stage a high priority, when it is at this critical stage that close attention to detail produces a final restoration that appears as a natural, polished tooth.[17]

Additional lines were drawn on the tooth to begin placing the facial anatomy (Fig. 14-17).[7] The tooth was divided into thirds from cervical to incisal, and a triangular line was placed where ridges and depressions were desired. The facial anatomy was placed with a Brasseler 8888.012 diamond bur (Brasseler USA). The electric handpiece was then turned down to 2500 rpm and the corrections were done with a very delicate touch, in an effort to avoid gouging the composite. When viewing the final restoration, the light should be reflecting off of the line angles and other highlights of the tooth, known as *reflective* areas. The depressions on the facial surface of the tooth do not reflect any of the light, and are known as *deflective* areas. It is very important to remember when placing the facial anatomy of a central incisor that teeth No. 8 and 9 should be mirror images of each other.

After the depressions were placed in the facial surface, additional faint lines were placed in the surface which served to break up the light reflections and make the finished tooth look more natural. A super fine disk was then used to put a smoother surface on the majority of the tooth. The finished interproximal surfaces will also prevent the bonding of the adjacent tooth from sticking to the finished tooth.

Attention was then turned to restoring the opposite central incisor. The central incisors are the cornerstone of the smile and should always be completed first. Determining the length of the interproximal area is always addressed after both central incisors are completed. According to the 50-40-30 rule, the length of the connector between the centrals should be 50% of the height of a single central, assuming both central incisors are identical.[18] So if an ideal tooth is 10 mm long, the interproximal connector size would be 5 mm. This can be increased to simulate an older smile, or less with a more youthful smile. The incisal embrasure can be altered for the desired length. The connector size between the lateral and central would then be 40%, and 30% for the lateral incisors and cuspids. The incisal embrasures increase as one progresses to the posterior.

The lateral incisors were then completed, knowing that the gingival heights were going to be uneven, based on the patient's decision not to work on the tissues. Laterals do not need to be mirror images of each other, but optimally should be similar. In this patient's case, the author would have liked the tissue heights to be closer in size. That is why it was so important that everything was explained to the patient in advance so there were no unrealistic expectations.[18]

The final disk polishing was completed and photos were taken to evaluate the shape and other details of the restorations.[19] The patient was scheduled to return to the office approximately 3 weeks later, after the tissues had healed from the placement of the retraction cords. At the follow-up visit minor modifications were made to the incisal embrasures and the facial anatomy (Fig. 18). The final step in completion of the composites was polishing the teeth with a Cosmedent FlexiBuff wheel utilizing Enamelize composite polishing paste (Cosmedent) (Fig. 19).[20] The author has found that only small amounts of this polishing paste are needed to get a high gloss shine. The patient returned a few weeks later for final photos (Fig. 20).

Discussion

The patient received a treatment that drastically improved not only his smile, but his self-confidence. His existing tooth structure was maintained, which was especially important due to his young age. Leaving the enamel intact, the treatment ensured that the teeth remain strong with minimal flexure during masticatory forces.[21] Any future repairs, if necessary, could be readily accomplished.

Composite resin veneers can have the esthetics of natural teeth, be minimally invasive, and provide an excellent treatment for many patients. Composite resin veneers have several advantages as an esthetic treatment over porcelain restorations. For instance, when these veneers are applied over dark or tetracycline-stained teeth, the control of color rests solely in the hands of the dentist.

Porcelain veneers rely on a laboratory technician using only a description and photography. The result may be multiple visits in order to match the final colors needed. With composite veneers, the dentist can easily visualize and apply the exact colors necessary to achieve the desired outcome. The matching of adjacent teeth is accomplished easier and more accurately when using multiple colors and tints in the restoration. Shape and contour can also be determined with the surrounding lips and facial features.

The restoring dentist has a variety of restorative materials of which to choose. Many brands of dental resin composites exist that have similar properties, but their ease of handling and color shades can be very different. The dentist must find the composite he/she is most comfortable with and understand the interaction of the various colors in order to achieve an optimal result. There is no simple way to accomplish this task, but an understanding of the material strength, and the color interaction of the materials with the existing tooth color is a great start. Further skills in contouring the tooth anatomy, as well as proper finishing and polishing, further ensure a successful esthetic result. Composite resin placement to re-create an anterior tooth can be a time-consuming process, but proper planning can reduce the time and ensure a predictable outcome.

Conclusion

Many patients seek out dental care to improve their smiles and hopefully raise their self-esteem. Treatment planning for cosmetic dentistry can involve a variety of treatment options. Dentists must always remember to keep the patient's best interests in mind. As direct resin bonding can be a very difficult procedure to master and can be very time consuming, a referral and co-treatment with another dental professional may be the most ethical option for the patient. However, with proper training and a commitment to learn, any dentist can master the skills required to effectively place direct resin bonding in the esthetic zone with excellent results. There are many situations in which direct resin is the best treatment modality, especially in the cases of younger patients—such as teenagers—who should not have tooth structure removed if there is a way to avoid it. There is only a fixed amount of enamel in permanent teeth, and dentists as a profession must respect that. Banking tooth structure for the patient's future is essential. Today's dentists should be thinking about being minimally invasive in all treatments.

Author information

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References

1. Prieto LT, Araujo CT, de Oliveira DC, de Azevedo Vaz SL, D'Arce MB, Paullilo LA. Minimally invasive cosmetic dentistry: smile reconstruction using direct resin bonding. *Gen Dent.* 2014;62(1):e28-e31.
2. Margeas, Robert C. Augmentation versus amputation: implementing noninvasive and minimally invasive protocols for maximum success. *Dent Today.* Available at: http://www.dentistrytoday.com/aesthetics/10633-augmentation-versus-amputation-implementing-noninvasive-and-minimally-invasive-protocols-for-maximum-success. Accessed September 3, 2014.
3. Heyman HO. The artistry of conservative esthetic dentistry. *J Am Dent Assoc.* 1987;Spec. No. 14E-23E.
4. Dietschi D, Argente A.A comprehensive and conservative approach for the restoration of abrasion and erosion. Part II: clinical procedures and case report. *Eur J Esthet Dent.* 2011;6(2):142-159.
5. Dawson PE. *Evaluation, Diagnosis and Treatment of Occlusal Problems.* 2nd ed. St. Louis: Mosby; 1989.
6. Beier US, Kapferer I, Burtscher D, Dumfahrt H. Clinical performance of porcelain laminate veneers for up to 20 years. *Int J Prosthodont.* 2012;25(1):79-85.
7. Fahl N. Mastering composite artistry to create anterior masterpieces—Part 2. *J Cosmetic Dent.* Available at: http://www.aacd.com/proxy/files/Dental%20Professionals/Students/Dental%20Student%20Resources/26-4_fahl.pdf. Accessed September 3, 2014.
8. Wells D.Beautiful, *Healthy No-Preparation Veneers: Ridiculous or Reality?* Presented at The 84th Annual Meeting of the Academy of Restorative Dentistry. Chicago, 2014.
9. Kois JC. *Treatment Planning I Manual.* Available at: http://koiscenter.com/. Accessed September 9, 2014.
10. Gurrea J, Bruguera A. Wax-up and mock-up. A guide for anterior periodontal and restorative treatments. *Int J Esthet Dent.* 2014;9(2):146-162.
11. Peck FH.A conservative approach to restoring a canted smile. *Dent Today.* Available at: http://www.dentistrytoday.com/articles/186-mid-aesthetics/2667-a-conservative-approach-to-restoring-a-canted-smile. Accessed September 3, 2014.
12. Chiche GJ, Pinault A. *Esthetics of Anterior Fixed Prosthodontics.* Chicago: Quintessence; 1994.
13. Kokich VO, Kokich VG, Klyak HA Perceptions of dental professionals and laypersons to altered dental esthetics: asymmetric and symmetric situations. *Am J Orthod Dentofacial Orthop.* 2006;130(2):141-151.
14. Fahl N Jr. A polychromatic composite layering approach for solving a complex Class IV/direct veneer/diastema combination: part II. *Pract Proced Aesthet Dent.* 2007;19(1):17-22.
15. Chan, D. *Composite Artistry Hands on Seminar*, AACD meeting. Seattle, 2013.
16. Suh BI. A new resin technology: a glaze/composite sealant that cures without forming an oxygen-inhibited layer. *Compend Contin Educ Dent.* 2003;24(8 Suppl):27-29.
17. Gonulol N, Yilmaz F. The effects of finishing and polishing techniques on surface roughness and color stability of nanocomposites. *J Dent.* 2012;40(Suppl 2): e64-e70.
18. Morley J, Eubank J. Macroesthetic elements of smile design. *J Am Dent Assoc.* Available at: http://www.saudident.com/album/data/media/2/smile_design.pdf. Accessed September 3, 2014.
19. Blitz N, Steel C, Willhite C. *Diagnosis and Treatment Evaluations in Cosmetic Dentistry: a Guide to Accreditation Criteria.* Madison, WI: American Academy of Cosmetic Dentistry; 2005.
20. Mopper, B, Louisiana State University Esthetic Continuum Seminar [lecture]. *Direct Resin Bonding.* September, 1999.
21. Magne P, Belser U. *Bonded Porcelain Restorations in the Anterior Dentition.* Hanover Park, IL: Quintessence; 2002.

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Comparison between visual and instrumental methods for natural tooth shade matching

Welson Pimentel, DDS, MSc ▪ Rodrigo Tiossi, DDS, PhD

Tooth shade matching in daily clinical practice is still a cause of discomfort for many professionals due to the subjectivity of the process and the need for advanced training; this discomfort may lead to unsatisfactory results for the clinician and the patient. Instrumental methods were developed to simplify daily color matching procedures and to provide better esthetic outcomes. This study compared the accuracy of shade matching by both visual and instrumental methods to determine whether the instrumental method would significantly improve the process. Visual shade matching was performed by 4 dentists using a classic shade guide; instrumental shade matching was performed with a spectrophotometer by a previously calibrated examiner. Shade matching was conducted in a dental clinic under controlled illumination on the middle third of the right central incisor tooth of 30 subjects. Data were analyzed by Cohen's Kappa inter-rater agreement and by the equality of 2 proportions test ($\alpha = 0.05$). Results showed statistically significant differences between the groups (76.7% ± 11.1% and 32.4% ± 7.8% for the instrumental and visual methods, respectively). Shade matching by clinicians using the instrumental method presented more agreement, and was more effective than shade matching by clinicians using the visual method.

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Key words: tooth color, spectrophotometry, visual shade selection, dental porcelain, fixed partial denture

Dental shade matching is a challenging and difficult task for clinicians. Unlike dental shape, size, proportion, and texture, dental shade selection is affected by environmental light conditions, age, variable viewer interpretation, previous eye exposure, and eye fatigue. Even experienced clinicians can erroneously select and evaluate the color of teeth and restorations.[1] Visual shade selection relies on the clinician's interpretation of color and the communication of the selected color to the technician. Visual shade guides and spectrophotometers were developed to facilitate and standardize the communication between dentists and technicians.[2]

The correct tooth color selection is essential for the successful esthetic outcome of a restoration.[3] The use of commercially available visual shade guides is the standard for tooth color matching of prosthetic restorations. However, visual color assessment is subjective and relies on a series of visual evaluations that are communicated between 2 or more professionals using shade guides that do not always represent the entire gamut of natural teeth.[1,4-6] The variable of viewer interpretation is another difficulty of visual shade matching for oral rehabilitations.[7-9]

Other factors that could compromise the esthetic outcome and the final shade of ceramic restorations include differences between the dental shade guides and the dental porcelain, nonuniform porcelain layer thickness, porcelain processing variables, wrong shade interpretation, and uncontrolled environmental light conditions.[1,10-18] Spectrophotometers and colorimeters have been developed in an attempt to overcome the difficulties and complexity of visual shade matching.[6] Instrumental measurements can quantify color and enable communication to be more uniform, more rapidly obtained, and more precise than visual measurements.[6,19] However, instrumental shade matching is not error-proof.[6] Translucent optical differences between teeth and dental ceramics can modify the standardized illuminating light used for color determination.[6,20] The software used during instrumental shade matching is capable of predicting some of the optical differences, but complete compensation is difficult to achieve and could lead to errors.[6] The color database that is available in the chosen software also influences the accuracy of shade matching.[21]

This study evaluated the accuracy of both visual and instrumental tooth shade matching in an effort to determine whether the instrumental method would provide significant assistance in the color selection process. The tested null hypothesis was that there would be no significant differences between the visual and instrumental methods.

Materials and methods

The trial protocol for this study was approved by the ethics committee of the Sao Leopoldo Mandic School of Dentistry, Brazil (Protocol No. 2007/0145). Four dentists with different levels of experience (3 professors, 1 master's candidate) voluntarily participated in this study for the visual selection method. Each participant was screened for color deficiencies using Ishihara's plates for color blindness.[6] The study sample consisted of 30 volunteer dental students from the Sao Leopoldo Mandic School of Dentistry who presented with a sound right central incisor and without any severe enamel anomaly or pigmentation.

Visual shade matching was performed separately on each patient by each dentist that participated in the study. The 4 dentists were classified as D1, D2, D3, and D4. The visual assessment of tooth shade was done under a corrected light source with a color temperature of 5500 K.[6] Dentists were instructed to use the middle third of the tooth for color selection with the aid of a shade guide (VITA VITAPAN Classical Dental Shade & Color Guide, Vident) (Figure). A previously calibrated examiner performed the instrumental measurement. Three measurements (SP1, SP2, and SP3) were performed on each patient, on the same incisor, for the instrumental

Figure. Tooth color shade guide used in the study.

Table 1. Color selection distribution found by the different examiners.

	D1		D2		D3		D4	
Selected color	Total count	%	Total count	%	Total count	%	Total count	%
B1	4	13	8	27	5	17	6	20
A1	14	47	10	33	16	53	17	57
C1	1	3	5	17	1	3	2	7
B2	0	0	1	3	0	0	1	3
A2	8	27	2	7	6	20	2	7
D2	0	0	1	3	0	0	0	0
C2	0	0	1	3	1	3	0	0
B3	0	0	0	0	0	0	1	3
A3	2	7	2	7	0	0	0	0
A3.5	0	0	0	0	1	3	1	3
C3	1	3	0	0	0	0	0	0

Abbreviations: D1, Dentist 1; D2, Dentist 2; D3, Dentist 3; D4, Dentist 4.

Table 2. Color selection distribution found by the instrumental method.

	SP1		SP2		SP3	
Selected color	Total count	%	Total count	%	Total count	%
B1	3	10	3	10	3	10
A1	17	57	15	50	16	53
C1	2	7	4	13	4	13
A2	6	20	4	13	3	10
D2	0	0	2	7	2	7
A3	1	3	1	3	1	3
D3	1	3	1	3	1	3

Abbreviations: SP1, spectrophotometer shade matching 1; SP2, spectrophotometer shade matching 2; SP3, spectrophotometer shade matching 3.

shade matching with a dental spectrophotometer (SpectroShade Micro, MHT S.r.l. A Socio Unico).

Data were collected and submitted for statistical analysis. Cohen's Kappa inter-rater agreement and the equality of 2 proportions test compared the visual and instrumental tooth color assessment ($\alpha = 0.05$).

Results

Table 1 presents the proportion distribution of the tooth colors that were selected by the different examiners using the visual method. The selection of the tooth color A2 was different between examiners D1 and D3 and examiners D2 and D4. Table 2 presents the colors selected in the consecutive instrumental measurements. No significant differences between the consecutive instrumental measurements were found and the agreement between measurements was determined to be significant ($P < 0.001$). The agreement found for the different instrumental measurements was higher than that found for the different examiners using the visual color assessment ($P < 0.001$) (Table 3).

Discussion

The accuracy of dental shade matching between visual and instrumental methods for tooth color selection was evaluated in this study. The results support rejection of the tested null hypothesis, since the instrumental method presented significantly better agreement than the visual method.

Accurate color selection is a complex and significant aspect of successful esthetic outcomes for dental restorations.[5,14] Small color differences will lead to dissatisfied patients and dentists.[8] Shade matching has always been a source of challenge for the clinician.[19] Despite not presenting the entire gamut of natural tooth color, the Vita classical shade guide used in this study was selected because of its widespread use in dental clinics.[4,7]

With the introduction of new technologies comes speculation about reliability. A 1998 study by Okubo et al found that the chosen colorimeter was precise in 50% of shade matching cases, whereas the visual method showed a precision rate of 48% with no significant differences between the methods.[4] The differences between the results found by Okubo et al and the present study could be due to the fact that the instruments used were different. However, other studies found results that were similar to those of the present study,

Table 3. Kappa inter-rater agreement test results.

	% (SD)	*P* value
Spectrophotometer	76.7 (11.1)	<0.001
Examiners	32.4 (7.8)	<0.001

Abbreviation: SD, standard deviation.

with significant differences between the visual and instrumental methods.[7-9,22-25] A study by Paul et al used 3 operators that independently assessed the best match to the middle third of unrestored maxillary central incisors of 30 patients.[7] The same tooth color was measured 3 times by a reflectance spectrophotometer. The agreement between the operators was only 26.6%, whereas the agreement between each spectrophotometric assessment was 83.3%. Another study compared conventional visual and spectrophotometric shade assessment for porcelain-fused-to-metal crowns in 10 patients.[8] The shade evaluation matched for all 3 visual shade selections in only 2 cases. By comparison, the spectrophotometric shade selections matched in 9 of 10 cases. Two studies compared the reliability of shade-matching instruments.[24,26] All the tested devices presented high reliability, with values ranging from 85% to 99%.[24,26] This further supports the use of spectrophotometers for dental shade matching.[26,27]

The shade matching between the 2 methods that were tested also found a better agreement when lighter (more value) or darker (less value) colors were present. These results are in agreement with a study by Lagouvardos et al, which found that darker shades presented more reliable and valid shade matching results, and that intermediate shades were less distinguishable between each other.[15] Individual color perception and variable viewer interpretation influence the reproducibility of a dental restoration. A successful tooth shade selection relies on adequate knowledge of color science and control of the variables that influence the visual color assessment.

The perception of color is subjective and different between viewers, and is also affected by environment light conditions. The instrumental analysis with the spectrophotometer can be a valuable aid to the clinician, providing better color agreement and standardization to contribute to a better esthetic outcome of a dental restoration. It should be stated that the use of only 1 visual shade guide was a limitation of this study. Future studies are recommended to compare differences between other visual shade guides and different instrumental equipment.

Conclusion

Within the limitations of this study design, and based on the results that were found, it can be concluded that dental shade matching using an instrumental method was more reliable and repeatable compared to the visual method. The use of an instrumental method is therefore recommended for adequate tooth color selection.

Author information

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References

1. Chu SJ, Devigus A, Paravina RD, Mieleszko AJ. *Fundamentals of Color: Shade Matching and Communication in Esthetic Dentistry.* Hanover Park, IL: Quintessence Publishing Co., Inc.; 2010.
2. Yap AU, Sim CP, Loh WL, Teo JH. Human-eye versus computerized color matching. *Oper Dent.* 1999;24(6):358-363.
3. Ozat PB, Tuncel I, Eroglu E. Repeatability and reliability of human eye in visual shade selection. *J Oral Rehabil.* 2013;40(12):958-964.
4. Okubo SR, Kanawati A, Richards MW, Childress S. Evaluation of visual and instrument shade matching. *J Prosthet Dent.* 1998;80(6):642-648.
5. Hajto J. *Anteriores - A Beleza Natural dos Dentes Anteriores.* Sao Paulo: Santos; 2008.
6. Alsaleh S, Labban M, AlHariri M, Tashkandi E. Evaluation of self shade matching ability of dental students using visual and instrumental means. *J Dent.* 2012;40(Suppl 1):e82-e87.
7. Paul S, Peter A, Pietrobon N, Hammerle CH. Visual and spectrophotometric shade analysis of human teeth. *J Dent Res.* 2002;81(8):578-582.
8. Paul SJ, Peter A, Rodoni L, Pietrobon N. Conventional visual vs spectrophotometric shade taking for porcelain-fused-to-metal crowns: a clinical comparison. *Int J Periodontics Restorative Dent.* 2004;24(3):222-231.
9. Judeh A, Al-Wahadni A. A comparison between conventional visual and spectrophotometric methods for shade selection. *Quintessence Int.* 2009;40(9):e69-e79.
10. Sproull RC. Color matching in dentistry. Part I. The three-dimensional nature of color. 1973. *J Prosthet Dent.* 2001;86(5):453-457.
11. Matthews TG. A method for shade selection (II). *Quintessence Int Dental Dig.* 1980;11(3):67-70.
12. Schwabacher WB, Goodkind RJ. Three-dimensional color coordinates of natural teeth compared with three shade guides. *J Prosthet Dent.* 1990;64(4):425-431.
13. Donahue JL, Goodkind RJ, Schwabacher WB, Aeppli DP. Shade color discrimination by men and women. *J Prosthet Dent.* 1991;65(5):699-703.
14. Joiner A. Tooth colour: a review of the literature. *J Dent.* 2004;32(Suppl 1):3-12.
15. Lagouvardos PE, Diamanti H, Polyzois G. Effect of individual shades on reliability and validity of observers in colour matching. *Eur J Prosthodont Restor Dent.* 2004;12(2):51-56.
16. Dozic A, Kleverlaan CJ, Meegdes M, van der Zel J, Feilzer AJ. The influence of porcelain layer thickness on the final shade of ceramic restorations. *J Prosthet Dent.* 2003;90(6):563-570.
17. O'Brien WJ, Kay KS, Boenke KM, Groh CL. Sources of color variation on firing porcelain. *Dent Mater.* 1991;7(3):170-173.
18. Davison SP, Myslinski NR. Shade selection by color vision-defective dental personnel. *J Prosthet Dent.* 1990;63(1):97-101.
19. Chu SJ, Trushkowsky RD, Paravina RD. Dental color matching instruments and systems. Review of clinical and research aspects. *J Dent.* 2010;38(Suppl 2):e2-e16.
20. Ishikawa-Nagai S, Ishibashi K, Tsuruta O, Weber HP. Reproducibility of tooth color gradation using a computer color-matching technique applied to ceramic restorations. *J Prosthet Dent.* 2005;93(2):129-137.
21. Brewer JD, Wee A, Seghi R. Advances in color matching. *Dent Clin North Am.* 2004;48(2):v, 341-358.
22. Derdilopoulou FV, Zantner C, Neumann K, Kielbassa AM. Evaluation of visual and spectrophotometric shade analyses: a clinical comparison of 3758 teeth. *Int J Prosthodont.* 2007;20(4):414-416.
23. Fani G, Vichi A, Davidson CL. Spectrophotometric and visual shade measurements of human teeth using three shade guides. *Am J Dent.* 2007;20(3):142-146.
24. Kanawati A, Richards MW. Repeatability of a dental shade-matching instrument when compared to traditional visual methods of shade evaluation. *Gen Dent.* 2009;57(4):323-327.
25. Della Bona A, Barrett AA, Rosa V, Pinzetta C. Visual and instrumental agreement in dental shade selection: three distinct observer populations and shade matching protocols. *Dent Mater.* 2009;25(2):276-281.
26. Kim-Pusateri S, Brewer JD, Davis EL, Wee AG. Reliability and accuracy of four dental shade-matching devices. *J Prosthet Dent.* 2009;101(3):193-199.
27. Khurana R, Tredwin CJ, Weisbloom M, Moles DR. A clinical evaluation of the individual repeatability of three commercially available colour measuring devices. *Br Dent J.* 2007;203(12):675-680.

Manufacturers

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Diagnosis, treatment, and risk management for a victim of domestic abuse: a case report

Richard W. Featherstone, DDS, AAACD, FAGD, AFAAID

A 37-year-old woman was referred to the author by the *American Academy of Cosmetic Dentistry*'s "Give Back a Smile" program. The program helps facilitate dental treatment and support services for adults who have suffered dental injuries to the smile zone as a result of domestic or sexual violence. The woman presented with a broken jaw and fractured teeth. This case report describes the treatment planning decisions and a successful outcome.

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Key words: diagnosis, treatment, implants, GBAS, bridges

A 37-year-old woman was referred for treatment by the *American Academy of Cosmetic Dentistry*'s (AACD) "Give Back a Smile" (GBAS) program, which is designed to provide treatment and support services at no charge to adults whose smiles have been damaged as a result of domestic abuse.[1] The patient's chief complaints were partial edentulousness and the discoloration of her remaining teeth (Fig. 1). While many of the patient's dental problems were a result of several instances of blunt force to the face, the patient also reported bruxing in times of stress and that she had a 10 cigarettes/day smoking habit. She was 3 months pregnant and not taking any medications. Cold stimulation to the teeth produced pain. The patient also reported occasional pain in the right jaw, with no joint sounds.

Diagnosis and risk assessment

A panoramic radiograph strongly suggested a Type VI intracapsular fracture as described by Spiessl & Schroll (Fig. 2).[2] This was considered to be a result of the patient receiving a blow to the jaw. Healing of the fractured condyle shortened the right ramus by approximately 8 mm; as a result, the bite was collapsed on the right side (Fig. 3). Complications in the treatment plan were expected due to the multiple missing teeth, the impacted condylar fracture, and the lack of research guidelines on treating occlusion after fractures.

Additional photographic, radiographic, and clinical records were made, along with impressions, face-bow, and bite registrations for diagnosis and for creating a wax mock-up with an articulator (Artex, Jensen Dental Incorporated). An outline of diagnostic information and risk assessments was derived by examining 4 key elements: temporomandibular joint (TMJ) disorder, periodontitis, caries and structural defects, and esthetics.[3]

TMJ disorder and functional issues

Examination of the TMJ and masseter muscles revealed no discomfort. Slight anterior pressure in the ear canal upon jaw opening revealed joint clicking on the right side. The jaw opening was normal at 54 mm but was accompanied by deviation. An attempt to place the joints in centric relation with bimanual manipulation revealed interference at tooth No. 12. Upon closure, the jaw moved markedly to the left and forward 2 mm to a more centered position. There was discomfort (described by the patient as "tension") in the right side during joint loading with bimanual manipulation.[4]

Periodontal disease

The initial examination revealed 6 mm periodontal pockets between the maxillary left molars and 5 mm pockets between the maxillary premolars, with bleeding on probing. The diagnosis was moderate periodontitis.

Caries and structural elements

Caries was severe. It was determined that teeth No. 7, 10, 13, 15, 27, and 28 were nonrestorable due to decay or fracture. Pulp testing revealed that teeth No. 7, 9,

Fig. 1. Anterior view of the patient's mouth at the initial visit.

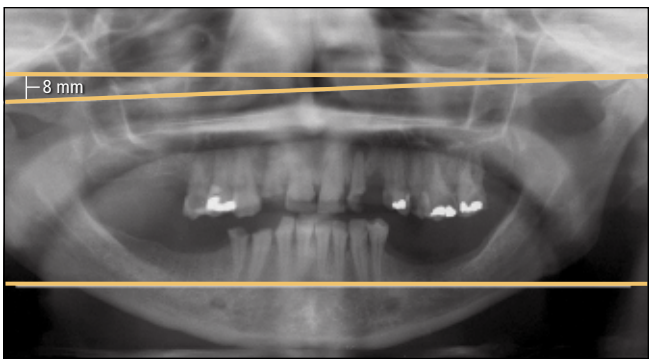

Fig. 2. A panoramic radiograph showing a collapsed ramus.

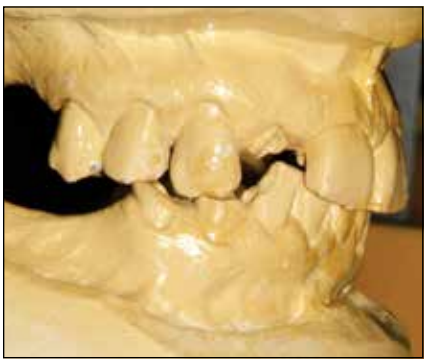

Fig. 3. A model demonstrating the patient's collapsed bite.

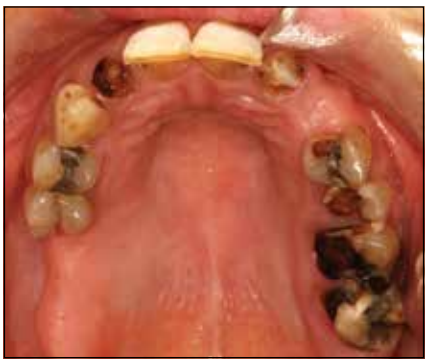

Fig. 4. Occlusal view of maxilla, revealing caries destruction.

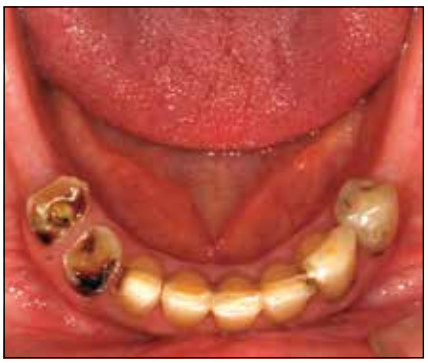

Fig. 5. Occlusal view of mandible, showing teeth No. 27 and 28 worn to the gum line.

10, and 27 were nonvital. The radiograph revealed a periapical lesion on tooth No. 10. The patient's susceptibility to caries could further compromise the treatment plan (Fig. 4 and 5).

Esthetics

The patient's upper lip rose to 2 mm below the gingival margins when forming the "e" sound. This was an acceptable level, requiring no treatment.

Treatment plan

The goals of treatment were to improve the patient's long-term quality of life and social confidence, preserve maxillary and mandibular bone mass, and provide both adequate function and a high level of esthetics. The challenge was to employ treatment that would serve the patient's interests while conforming to GBAS guidelines.[5] The patient had 6 structurally sound teeth remaining in the mandible. Only 7 teeth would remain in the maxilla after the necessary extractions were performed. It was determined that treatment would involve placing maxillary fixed prostheses and a mandibular immediate hybrid implant prosthesis with tilted implants.

Maxilla

Teeth No. 7, 10, 13, and 15 were extracted and endodontic therapy was performed on teeth No. 9 and 14. Bridges were made for teeth No. 3-8 and 9-14, to provide the maximum amount of bone preservation, esthetics, and function. The patient's periodontal health and foundational bone level were manageable.

High caries risk required the patient to be able and willing to maintain the bridgework. The patient responded well to hygiene instructions, and it was felt that she would continue long-term maintenance. Another concern was the use of a cantilever at the tooth No. 3 position. Upon review, it was determined that the risk for fracture or stress on anterior abutments would be low, because the cantilever would be attached to 4 strong teeth, none of which had root canal fillings or posts. The bridge extended to the central incisor, providing 24 mm anteroposterior and 15 mm mediolateral support, as measured at the long axes of teeth No. 8 and No. 4, thus providing an ample amount of resistance to cantilever forces.[6]

Mandible

Treatment involved extracting the remaining teeth, performing a 5-7 mm vertical reduction alveolectomy, and placing 4 implants and an interim fixed appliance in a single visit. This approach provided the patient with immediate function and long-term bone maintenance from that time forward. The longer posterior implants (16 mm) were angled so that the 30 degree multi-unit attachments would exit the tissue near the same position as a molar vertical implant, which negated the need for a long cantilever. There has been impressive documentation on the success of immediately restored fixed hybrid dentures in edentulous mandibles.[7-9] This treatment plan was proven to be effective in a 2012 review article documenting 462 patients in 10 studies.[7] Other retrospective/longitudinal studies reviewed cases involving 410 patients, with success rates comparable to other implant-supported prosthetic procedures.[8,9] This is particularly impressive as the majority of these cases used NobelActive prostheses with a miniscule 15 Ncm of torque on very small screws to retain the prostheses. Misch expressed serious misgivings regarding this technique and the low torque in 2011; however, the cases have proven successful.[10]

The author of this case report has had previous experience with this operation, including managing another fixed hybrid implant patient who functioned successfully with 3 implants for 9 months while bone grafting healed around a fractured facial plate at the right posterior position.

The only treatment obstacle remaining for the patient was finding donors for the mandibular implants, the 2 prosthetic appliances, and the maxillary fixed prostheses in order to reduce costs of this *pro bono* case. The 3 donors listed in the Acknowledgments section agreed to donate materials and labor.

Treatment options and considerations

Full or partial dentures

The options of full or partial dentures—including 2 implant-retained mandibular overdentures—were rejected because of the resultant edentulism which would eventually lead to ≤60% bone loss.[11] At 37 years old, the patient was determined to be too young to risk that amount of bone loss.

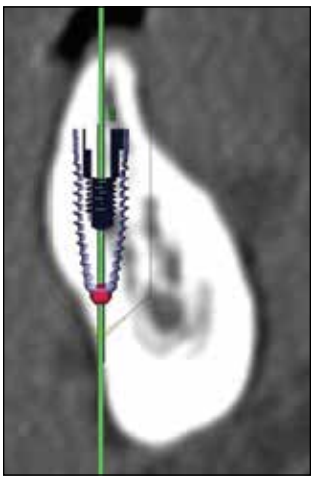

Fig. 6. A rendering of one of the narrow implants (10 mm long) used in the procedure.

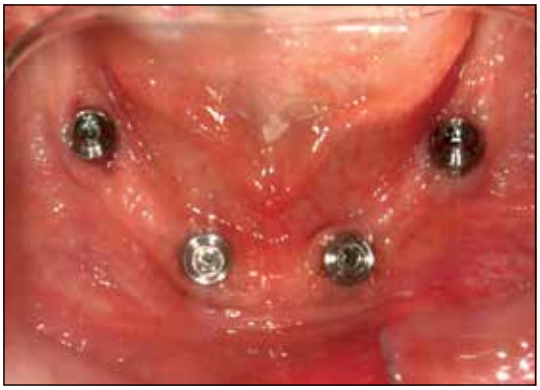

Fig. 7. Occlusal view of angled abutments with healthy surrounding tissue.

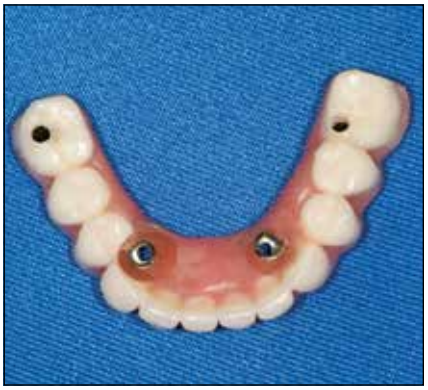

Fig. 8. A photograph of the preliminary mandibular fixed hybrid appliance.

Reconstruction

One viable option involved selective extractions followed by crowns and bridges on implants and natural teeth. This would involve at least 7 implants, crown lengthening, and placing crowns on every tooth. It was the most complicated, risky, and time-consuming option; in addition, the scope of this treatment plan went well beyond GBAS guidelines, which state: "The program is designed to focus on standard of care smile-zone dentistry, not full mouth reconstruction."[5]

Implant-supported dentures

Treatment of edentulism with implants and prosthetic appliances has been documented in the literature.[11,12] These studies reveal that implants protect bone over the long term, which helps preserve bone mass. Many options are possible for bone preservation, such as cantilevered bar overdentures, overdentures or bridges without cantilevers, overdentures attached to individual implants/mini-implants, and immediate maxillary and mandibular temporary hybrid implant prostheses, followed by fixed milled bar hybrid dentures. These options would have fulfilled the treatment plan's objectives. Some are complex and time consuming, and some require long-term maintenance, such as replacing worn removable attachment apparatuses. Other options that involved cantilevers were more prone to restoration fracture and screw loosening/breakage.[13]

One option that was considered for the mandible involved adding implants posterior to the mental nerve. This procedure would have been risky since the left mandible was resorbed already. Figure 6 shows a narrow implant (10 mm in length) in this position, which was very close to the mylohyoid depression. In addition, the crest was too narrow to allow 2 mm of bone on the buccal and lingual sides. As a result, it was decided that placing long, tilted implants following the ostectomy was the simplest and best solution.

Another option involved extracting the 7 remaining viable maxillary teeth and placing an implant-supported hybrid denture similar to that planned for the mandible. However, this procedure would require an invasive reduction of bone height; in addition, there is evidence that implant-supported maxillary prostheses do not fare as well as mandibular prostheses.[14] Studies using machined Branemark implants showed that the denser bone of the mandible is more amenable for long-term implant success compared with the softer Type III or IV bone common to the maxilla.[14-16]

The plan of treatment used in this study went beyond the GBAS-suggested treatment protocol regarding the esthetic zone and constituted reconstructions.[5] However, the author found it difficult to compromise the patient's long-term health and well-being while staying within those guidelines, and thus chose the more appropriate, less complex treatment.

Treatment

Extractions, crown buildups, and endodontic therapy were performed over 2 visits. Two months after extractions in the esthetic zone, 2 maxillary bridges were prepared for teeth No. 3-8 and No. 9-14. Teeth No. 13 and 15 were extracted. Prototype resin bridges created from a wax mockup (Yeti Dentalprodukte GmbH) were placed.

The intaglio surfaces of the pontics were shaped into a modified oval with a flattened bottom to maximize papillae support.[17]

Seven months after initial presentation, the patient returned for treatment of the mandible. Impressions were made for an immediate denture. A bite relation was created with a Jankleson Myomonitor (Myotronics) to record myofunctional occlusion since the fractured right condyle was painful in the retruded, manipulated centric relation. A computed tomography scan was deferred until after the patient delivered her baby. The scan provided information used in the planning positions, lengths, and angulations of the implants. Following extractions and an ostectomy, 4 implants—measuring 4.7 x 16 mm with 4.5 mm platforms (Implant Direct, LLC)—were placed using an

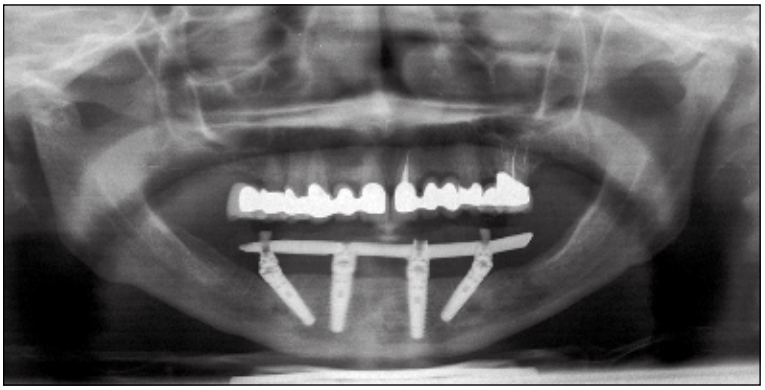

Fig. 9. A panoramic radiograph of the implants and milled bar.

Fig. 10. Post-treatment photograph of patient with improved smile.

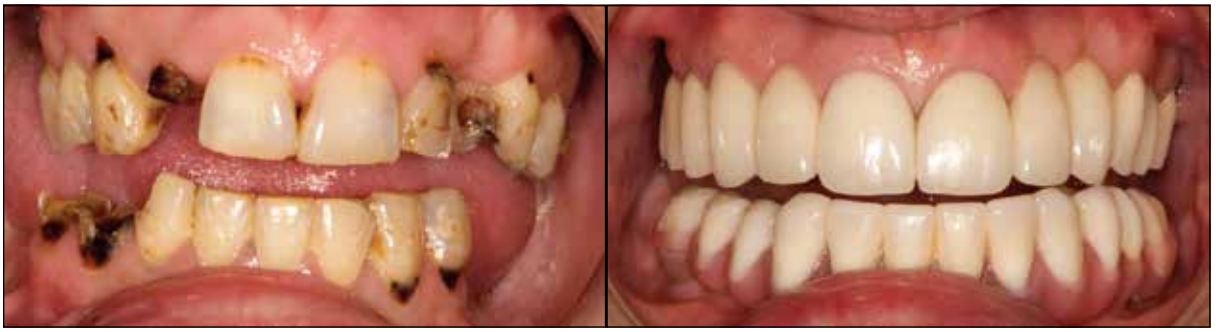

Fig. 11. Anterior views of the patient's mouth. *Left.* Pretreatment. *Right.* Post-treatment.

All-on-4 surgical guide (Nobel Biocare USA, LLC) to aid with angulations. Two 30-degree and 2 15-degree angled abutments were screwed into the posterior and anterior implants, respectively. An impression was made of the mandibular implants and sutured ridge to fabricate the implant replica model. The mandibular appliance had been prepared by a technician from impressions of the mandible made at a previous office visit. The denture was hollowed out in the implant temporary abutment areas, attached to the implants with resin, and removed so that resin could be added and trimmed by the attending technician. Following the implant manufacturer's instructions, the mandibular hybrid, all-acrylic denture was screwed in at 30 Ncm and adjusted for occlusion to the prototype maxillary bridgework.

One month later, the impressions and bite registration were made for the final maxillary prostheses. An image of the bite stick was sent to the laboratory to confirm horizontal alignment. That same month, the mandibular hybrid denture was removed; at that time, it was observed that the implant tissue around the implants was very healthy. The patient's home care regimen included a Water Flosser (Water Pik, Inc.). She had been diligent about cleaning along the intaglio surface of the denture (giving special attention to the implant sites) and maintaining the prototype maxillary bridgework (Fig. 7 and 8). Bleeding on probing was nearly nonexistent.

The following month, the bridges and teeth No. 3-8 and No. 9-14 were seated, with only minor adjustments made to the acrylic mandibular teeth. At a follow-up visit the next month—9 months after initial presentation—the patient's periodontal health remained excellent. Four months later, the patient returned for an oral hygiene checkup and to have impressions made for the final mandibular prosthesis. The patient continued to maintain her oral health.

Results

A panoramic radiograph of the completed case shows prescribed implant angulation and no bone loss (Fig. 9). Fig. 10 shows the patient with an improved smile, and Fig. 11 shows the before and after anterior views of the patient's mouth.

Summary

This article describes the diagnosis, treatment plan, and options considered for a patient whose fractured jaw and supererupted broken teeth complicated the planning. The risks outlined for caries and periodontal disease were mitigated by the patient's willingness to perform oral hygiene functions on a daily basis, and therefore were reduced to "low risk." The risk of future TMJ disorders in the patient will continue to be moderate due to the fractured ramus.

This case establishes the fact that, when given the opportunity, people can recover from facial damage due to domestic abuse.

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References

1. The American Academy of Cosmetic Dentistry Charitable Foundation. *Give Back a Smile.* Available at: http://www.aacd.com/index.php?module=cms&page=610. Accessed September 3, 2014.
2. Spiessl B, Schroll K. Gelenkfortsatz-und kieferkopfchenfrakturen. In: Nigst H, ed. *Spezielle Frakturen-und Luxationslehre.* Stuttgart, Germany: Thieme; 1972.
3. Kois JC. New challenges in treatment planning - part 2. *J Cosmetic Dent.* 2011;27(1):110-123.
4. Dawson PE. *Functional Occlusion: From TMJ to Smile Design.* St. Louis: Mosby; 2007:86-90.
5. The American Academy of Cosmetic Dentistry Charitable Foundation. *Give Back a Smile Volunteer Dentist Procedures and Expectations.* Available at: http://www.aacd.com/index.php?module=cms&page=631. Accessed August 12, 2014.
6. Himmel R, Pilo R, Assif D, Aviv I. The cantilever fixed partial denture—a literature review. *J Prosthet Dent.* 1992;67(4):484-487.
7. Del Fabbro M, Bellini CM, Romeo D, Francetti L. Tilted implants for the rehabilitation of edentulous jaws: a systematic review: *Clin Implant Dent Relat Res.* 2012; 14(4):612–621.
8. Babbush CA, Kutsko GT, Brokloff J. The all-on-four immediate function treatment concept with NobelActive implants: a retrospective study. *J Oral Implantol.* 2011; 34(4):431-445.
9. Malo P, de Araujo Nobre M, Lopes A, Moss SM, Molina GJ. A longitudinal study of the survival of All-on-4 implants in the mandible with up to 10 years of follow up. *J Am Dent Assoc.* 2011;142(3):310-320.
10. Misch CE. Enhancing outcomes with block bone grafts. Presented at: 60th Annual Meeting of the American Academy of Implant Dentistry; Las Vegas. 2011.
11. Zarb GA, Schmitt A. The edentulous predicament. I: a prospective study of the effectiveness of implant-supported fixed prostheses. *J Am Dent Assoc.* 1996; 127(1):59-65.
12. Attard NJ, Zarb GA. Long-term treatment outcomes in edentulous patients treated with overdentures: the Toronto study. *Int J Prosthodont.* 2004;17(4):425-433.
13. Lee H, Yu A, Flores-Mir C. Limited evidence that cantilevers are associated with slightly lower survival rates of implant-supported fixed partial dentures. *J Am Dent Assoc.* 2010;141(11):1371-1372.
14. Jaffin RA, Berman CL. The excessive loss of Branemark fixtures in type IV bone: a 5-year analysis. *J Periodontol.* 1991;62(1):2-4.
15. Jemt T, Linden B, Lekholm U. Failures and complications in 127 consecutively placed fixed partial prostheses supported by Branemark implants: from prosthetic treatment to first annual checkup. *Int J Oral Maxillofac Implants.* 1992;7(1):40-44.
16. Friberg B, Jemt T, Lekholm U. Early failures in 4641 consecutively placed Branemark dental implants: a study from stage I surgery to the connection of completed prostheses. *Int J Oral Maxillofac Implants.* 1991;6(2):142-146.
17. Eubank J. Phased treatment for complete dentistry. *Dent Today.* 2008;27(6):68-73.

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Direct or indirect composite veneers in anterior teeth: which method causes higher tooth mass loss? An in vitro study

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There is little information in the literature regarding the relationship between preparations made for direct and indirect veneers and the loss of tooth structure required for each technique. This in vitro study sought to quantify the different mass losses from preparation techniques used for direct and indirect veneers. Thirty artificial teeth were weighted using a digital balance and placed in a dental manikin in the position corresponding to the right maxillary central incisor. Five clinicians—all experts in esthetic dentistry—were asked to perform conventional preparations for both a direct composite resin veneer and an indirect ceramic veneer. After preparations, specimens were weighted again in the same digital balance. Teeth undergoing veneer preparations demonstrated a statistically significant mass loss compared to unprepared teeth. Indirect ceramic veneer preparations produced more mass loss than direct composite veneer preparations ($P < 0.01$).

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Keywords: dental veneers, dental cavity preparation, tooth structure, esthetics, ceramic, composite resin

Modern dental patients are increasingly concerned about esthetics, alignment, and whiter teeth without discolorations or surface alterations.[1] Anterior teeth play a crucial role in facial esthetics.[2] In some clinical situations, more conservative techniques (such as dental bleaching or conventional composite restorations) are unable to produce a satisfactory esthetic result, and veneer preparations may be required. Although veneer placement has the potential for esthetic benefits, their preparation requires removing sound tooth structure.[3] According to the concept of minimally invasive dentistry, the preservation of sound tooth structure is important and techniques that result in greater preservation of dental structure may be preferred.[4-6]

With the evolution of adhesive systems and photo-activated composite resins, less invasive adhesive restorative techniques can be developed that remove comparatively little sound dental structure. The main disadvantages involved in these adhesive restorations include the need for highly sensitive techniques, the complex application of materials, and the need for correct marginal sealing against deleterious factors that could compromise longevity.[4]

Direct composite veneers are a popular adhesive technique for improving esthetics in discolored, decayed, or fractured anterior teeth. Direct composite veneers involve a buildup of sequential layers over the buccal tooth surface. This technique may be performed in a single session, without the need for laboratory work, and involving a reduced removal of sound tooth structure when compared to indirect ceramic veneers.[7] This technique also offers both restoration longevity and satisfactory esthetic results.[8,9]

Indirect ceramic veneers are another esthetic restorative technique available for anterior teeth. This technique traditionally requires some preparation that includes tooth structure loss, such as removing contact points and incorporating the incisal edge to allow adequate space for taking an impression and veneer adaptation.[4,6,8,10] To preserve the maximum amount of tooth structure, some authors have advocated a minimal preparation even for indirect ceramic veneers.[11,12] For young patients with large pulp chambers, the preparation of conventional ceramic veneers could damage the pulp dentin complex.[13] However, the esthetic results obtained with ceramic veneers is excellent and the longevity of this treatment tends to be higher than that for direct veneers.[14]

Both techniques (direct and indirect veneers) have been described in the literature.[3,8] However, there is a paucity of information regarding the amount of tooth structure that must be removed during these clinical procedures.[3,8] This study sought to quantify the amount of tooth loss needed for direct composite veneers and indirect ceramic veneers, and to detect if there were significant differences in tooth loss between both techniques.

Materials and methods

To perform this study, artificial right central incisors were selected and divided in 2 groups according to the veneer technique preparation: direct (Group 1) or indirect (Group 2).

Sample size calculation

A pilot study was performed to define the sample size. Three samples for each group were prepared to identify the mean and standard deviations, with a 95% confidence interval. With an estimated error of 15% and a standard deviation of 0.029 g, a sample size of 14.3 teeth per group was obtained.

Specimen selection

Thirty artificial teeth corresponding to the right maxillary central incisor were selected, numbered, and weighted in a digital balance. Before weighting, the specimens were stored at 37°C for 24 hours. Fifteen specimens were allocated randomly to both groups.

Specimen preparations

The veneers were prepared for 5 previously calibrated experts in esthetic dentistry with similar clinical experience. The experts were blinded in relation to the objectives of the study. Each dentist prepared 6 artificial teeth, 3 for each group.

The specimens were prepared with the teeth mounted in a dental manikin (P Oclusal Produtos Odontologicos Ltda ME) to better simulate clinical conditions. For specimen preparation, each dentist received the same preparation kit, including a high-speed turbine, a low-speed micromotor, mirror, explorer probe, and diamond burs (KG Sorensen), Soflex disks (3M ESPE), polishing strips (3M ESPE), silicone points, and a manual instrument for finishing margins.

When the dentists received the specimens and instrument kits, a hypothetical clinical situation was explained to them: "A patient who has stated a desire for improved esthetics has a nonvital right maxillary central incisor, well-aligned in the arch. The tooth shows a moderate discoloration and it is not responding to bleaching treatment." Two restorative options were offered to the dentists: a conventional direct veneer preparation for composite resin, and a conventional indirect veneer preparation for ceramic.

After direct and indirect preparations, the teeth were collected, washed, dried with a paper towel, and maintained at 37°C for 24 hours; at that point, they were weighted again in the same digital balance. The structure loss was determined by comparing the weight before and after preparations.

Statistical analysis

A paired t-test was used to compare differences between groups, with the confidence level set at $P < 0.01$.

Results

Data related to the weight in different groups are listed in the Table, along with mass loss and statistical analysis. The results showed that unprepared artificial teeth had similar weights ($P = 0.208$). Differences were observed between unprepared teeth and prepared teeth in both groups ($P < 0.001$), with the indirect veneer specimens exhibiting the highest mass loss ($P < 0.001$).

Table. Mean weight and standard deviation (SD) for unprepared and prepared teeth (in grams).

Group (n = 15)	Unprepared teeth	Prepared teeth	Mass loss rate (%)
1 (direct veneer)	0.688 (0.003)^{Aa}	0.607 (0.012)^{Bb}	11.8
2 (indirect veneer)	0.690 (0.004)^{Aa}	0.561 (0.027)^{Cb}	18.7

Different uppercase superscript letters indicate statistically significant differences between columns ($P < 0.01$).
Different lowercase superscript letters indicate statistically significant differences between rows ($P < 0.01$).

Discussion

This study showed that both veneer preparations resulted in a significant loss of tooth structure; the loss increased when an indirect ceramic veneer was prepared.

Different methods have been described to measure the amount of tooth structure removed for indirect ceramic preparations.[3] The present study employed a methodology that had been used previously to determine the loss of tooth structure when preparing different esthetic treatments for anterior teeth.[3] Hussain et al evaluated mass loss in naturally extracted canine and incisor teeth that were submitted to different endodontic and restorative treatments.[5] It is important to note that the use of naturally extracted teeth is becoming more difficult not only because of ethical concerns, but also because the concept of preventive dentistry has caused a significant decrease in the need to extract teeth. Artificial teeth appear to be a reliable alternative for this kind of study. At present, no study has used a similar approach to compare mass loss for veneer preparation.

Tooth discoloration was the only criteria used in this study to indicate the need for a veneer preparation. Other criteria that could influence the preparation (such as tooth decay or bad alignment) were not included, because it would be difficult to reproduce and standardize them in a manikin.

The preparations were performed using an air/water spray, as resin is a material that can absorb water. Before weight measurements were performed, teeth (prepared and unprepared) were stored for 24 hours at 37°C. According to Edelhoff & Sorensen, weight stabilization for artificial teeth is obtained after storage for 24 hours in similar conditions.[3]

Stappert et al measured differences in the amount of tooth loss between different veneer preparations in terms of deepness in millimeters.[15] Representing tooth loss in mm can allow for better visualization, using the top of the burs as a guide. However, the analysis of mass loss, while not feasible clinically, is useful for research purposes, as it allows for the quantification of the necessary amount of tooth structure that needs to be removed with the different veneer preparations. Measuring mass loss is quite simple and can be reproduced easily in different laboratories for testing similar conditions.[5] To ensure standardization, only 1 tooth type was used (right maxillary central incisor).

The main reason for the difference in weight observed is due directly to the more aggressive designs required for ceramic restorations. When an indirect veneer was prepared, the incisal border was removed and proximal contacts were invaded also; such steps were not necessary for direct composite preparations. There are several advantages to preparing a direct composite veneer instead of a ceramic veneer, including lower cost, immediate results, reduced time required for the preparation/restoration, good clinical performance, and the elimination of laboratory work.[8,9] A deeper preparation—such as those commonly found in ceramic veneers—can also cause pulpal problems and postoperative sensitivity.[16-18] However, the more aggressive preparation with a ceramic restoration is associated with better esthetics and higher longevity.[10,14,19-21]

This study's ability to determine mass loss precisely reinforces the need for more conservative preparations whenever possible, to avoid removing additional tooth structure, which may weaken the teeth in the long term.

Conclusion

Within the limitations of this in vitro study, it was possible to conclude that both preparation types caused tooth mass loss, and that the loss was greater for the indirect veneer preparations.

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References

1. Meireles SS, Santos IS, Bona AD, Demarco FF. A double-blind randomized clinical trial of two carbamide peroxide tooth bleaching agents: 2-year follow-up. *J Dent.* 2010;38(12):956-963.
2. Sadowsky SJ. An overview of treatment considerations for esthetic restorations: a review of the literature. *J Prosthet Dent.* 2006;96(6):433-442.
3. Edelhoff D, Sorensen JA. Tooth structure removal associated with various preparation designs for anterior teeth. *J Prosthet Dent.* 2002;87(5):503-509.
4. Tyas MJ, Anusavice KJ, Frencken JE. Minimal intervention dentistry: a review. FDI Commission Project 1-97. *Int Dent J.* 2000;50(1):1-12.
5. Hussain SK, McDonald A, Moles DR. In vitro study investigating the mass of tooth structure removed following endodontic and restorative procedures. *J Prosthet Dent.* 2007;98(4):260-269.
6. Prati C. In vitro and in vivo adhesion in operative dentistry: a review and evaluation. *Pract Periodontics Aesthet Dent.* 1998;10(3):319-327.
7. Aschheim KW, Dale BG. *Esthetic Dentistry: A Clinical Approach to Techniques and Materials.* Philadelphia: Lea & Febiger; 1993.
8. Wakiaga JM, Brunton P, Silikas N, Glenny AM. Direct versus indirect veneer restorations for intrinsic dental stains. *Cochrane Database Syst Rev.* 2004;1:CD004347.
9. Welbury RR. A clinical study of microfilled composite resin for labial veneers. *Int J Paediatr Dent.* 1991; 1(1):9-15.
10. Nattress BR, Youngson CC, Patterson CJ, Martin DM, Ralph JP. An in vitro assessment of tooth preparation for porcelain veneer restorations. *J Dent.* 1995;23(3): 165-170.
11. Zhang F, Guido H, Razzoog M. Double-layer porcelain veneers: effect of layering on resulting veneer color. *J Prosthet Dent.* 2000;84(4):425-431.
12. Van Meerbeek B, Munck JD, Yoshida Y, et al. Buonocore memorial lecture. Adhesion to enamel and dentin: current status and future challenges. *Oper Dent.* 2003; 28(3):215-235.
13. Murray PE, Smith AJ, Garcia-Godoy F, Lumley PJ. Comparison of operative procedure variables on pulpal viability in an ex vivo model. *Int Endod J.* 2008;41(5): 389-400.
14. Granell-Ruiz M, Fons-Fonte A, Labaig-Rueda C, Martinez-Gonzalez A, Roman-Rodriguez JL, Sola-Ruiz MF. A clinical longitudinal study 323 porcelain laminate veneers. Period of study from 3 to 11 years. *Med Oral Patol Oral Cir Bucal.* 2010;15(3):e531-e537.
15. Stappert CJ, Ozden U, Gerds T, Strub JR. Longevity and failure load of ceramic veneers with different preparation designs after exposure to masticatory simulation. *J Prosthet Dent.* 2005;94(5):132-139.
16. Magne PD. Additive contour of porcelain veneers: a key element in enamel preservation, adhesion, and esthetics for aging dentition. *J Adhes Dent.* 1999;1(1): 81-92.
17. Ericson S, Hedegard BS, Wennstrom A. Roentgenographic study of vital abutment teeth. *J Prosthet Dent.* 1966;16:981-987.
18. Kerschbaum T, Voss R. Practical trial of crown and inlay [in German]. *Dtsch Zahnarztl Z.* 1981;36:243-249.
19. Calamia JR. Clinical evaluation of etched porcelain veneers. *Am J Dent.* 1989;2(1):9-15.
20. Walls AW, Steele JG, Wassell RW. Crowns and other extra-coronal restorations: porcelain laminate veneers. *Br Dent J.* 2002;193(2):73-82.
21. Borges GA, Agarwal P, Miranzi BA, Platt JA, Valentino TA, Santos PH. Influence of different ceramics on resin cement Knoop hardness number. *Oper Dent.* 2008; 33(6):622-628.

Manufacturers

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Approaches to managing asymptomatic enamel and dentin cracks

Samer S. Alassaad, DDS

Asymptomatic enamel and dentin cracks can pose a risk for multiple pathological and undesired consequences if intervention is postponed. This article reviews asymptomatic enamel and dentin cracks, and presents current management approaches utilized by a sample of general dentists. Becoming familiar with all forms of asymptomatic enamel and dentin cracks is crucial to adopting a proactive approach of prevention, early diagnosis, and intervention to control the potentially detrimental effects of these cracks on the dentition.

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An incomplete tooth fracture, also known as a tooth *crack*, is a fracture without visible separation of the segments along the plane of the fracture.[1] Incomplete tooth fractures can be subtle and difficult to diagnose, especially when asymptomatic. However, they pose a risk for multiple pathological and undesired consequences that can eventually render the tooth unsavable.

The pathological consequences can range from caries to pulpal and periodontal involvement to complete tooth fracture.[2-8] Walker et al demonstrated that enamel cracks provide caries-producing bacteria access to the dentin-enamel junction, thus leading to caries progression inside the tooth without any externally visible evidence.[2] Abou-Rass suggested that asymptomatic crack lines are precursors to the symptomatic *cracked tooth syndrome*.[3] Krell & Rivera found a 9.7% incidence of cracks in all teeth referred to an endodontic practice within a 6-year period, excluding cusp fracture, vertical root fracture, and split teeth.[5] In their study, 20% of the cases diagnosed as cracked teeth with reversible pulpitis and treated with full crown restorations nevertheless progressed to irreversible pulpitis or to necrotic pulp within 6 months.[5] Pitts & Natkin described periodontal involvement associated with tooth cracks as bone loss produced by chronic inflammation along the fracture line, causing a narrow isolated pocket and radiographically linear bone loss along the root surface.[6] In a study of cusp fracture in restored posterior teeth, Bader et al concluded that incomplete fractures are a predictor of complete cusp fractures, which have been found to have an incidence of 69.9/1000 persons each year.[7,9] Finally, Braly & Maxwell concluded that fractures are the third most common cause of tooth loss behind caries and periodontal disease.[10]

Based on the evidence that incomplete tooth fractures can lead to major complications, and taking into consideration that the incidence of fractures increases with age, a more proactive approach, in which asymptomatic cracks are addressed early—before major complications occur—should be considered. This is especially relevant with the current aging population.[7,10,11]

However, there has been less agreement among dentists on which teeth are at risk of fracture, and which teeth require intervention for the prevention of fracture.[12] A recent literature review concluded that there is no universally accepted restorative protocol to treat cracked tooth syndrome.[13] Consequently, this author conducted a literature review of managing asymptomatic enamel and dentin cracks, and conducted a survey using clinical images during multiple presentations to determine how general dentists currently manage these cracks in their general practice.

Materials and methods

The author gave presentations on managing incomplete tooth fractures to 4 different groups of dental professionals in Northern California between March 2012 and March 2013. A total of 71 dental professionals—54 general dentists, 15 dental specialists, and 2 dental hygienists—attended these presentations. At the beginning of the presentations, clinical photographs of asymptomatic enamel and dentin cracks in posterior teeth were projected on a screen to ensure uniformity in the referenced cases. Enamel and dentin cracks were classified according to their direction (vertical or oblique)

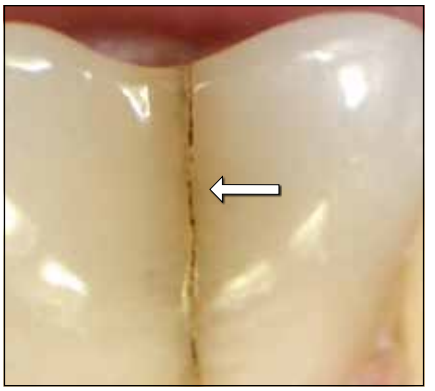

Fig. 1. Mandibular left first molar, lingual view. Stained enamel crack detectable by an explorer.

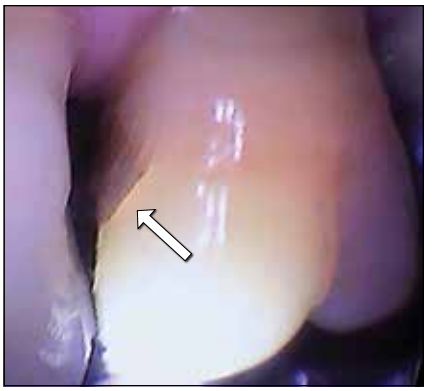

Fig. 2. Maxillary left first molar, buccal view. Oblique enamel crack originating from the corner of restoration and detectable by transillumination.

Fig. 3. Mandibular left second molar, buccal view. *Left.* Unstained vertical enamel crack. *Right.* The crack accepts methylene blue dye.

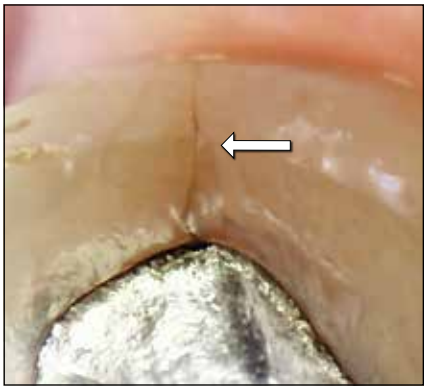

Fig. 4. Mandibular right second molar, occlusal view. Stained vertical enamel crack undetectable by an explorer.

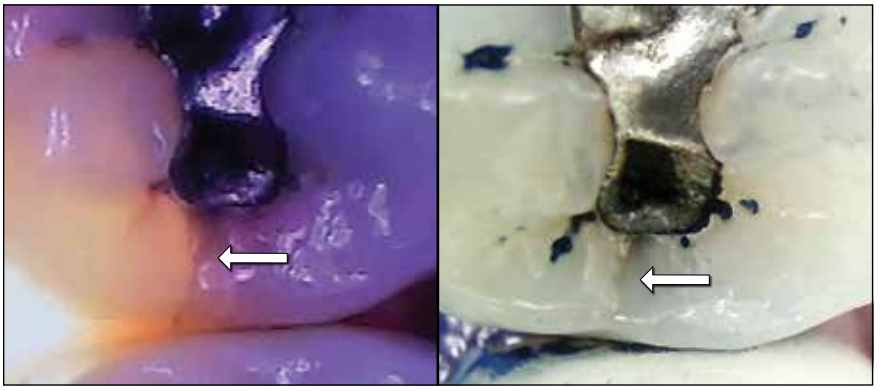

Fig. 5. Maxillary right second molar, occlusal view. *Left.* Unstained vertical enamel crack detectable by transillumination. *Right.* The crack does not accept methylene blue dye.

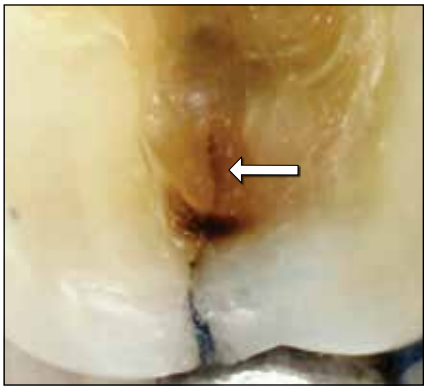

Fig. 6. Mandibular right first molar, occlusal view. Stained vertical dentin crack.

and the presence of stain. Enamel cracks were additionally classified based on their detection by tactile examination with an explorer, transillumination, and staining with methylene blue dye. All attendees were then asked to answer multiple choice questions regarding the approaches they utilize in their practice to manage the projected asymptomatic enamel and dentin cracks, such as when and what they recommend for intervention. To preserve anonymity, participants were asked not to write their names on their answer sheets. Some of the dental specialists who attended the presentations limited their practice to their specialty and did not directly treat asymptomatic cracks. Seven dental specialists and 2 dental hygienists did not complete the survey. Therefore, only responses from general dentists were included in the results. Of the 54 responses from general dentists, 3 were excluded as their answers were incomplete; descriptive statistics of responses from the remaining 51 general dentists were then computed.

Survey results

The majority of the general dentists (73%) would recommend to their patients the removal of intracoronal restorations in order to explore the extension of asymptomatic vertical enamel cracks in posterior teeth when these cracks were both stained and detectable by an explorer, even though the restorations were not compromised and the teeth had no evidence of decay (Fig. 1). This was followed by 62% of the general dentists who would recommend to their patients the removal of intracoronal restorations to explore for oblique enamel cracks originating from the corner of restorations and detectable by transillumination (Fig. 2). Twenty-nine percent would recommend removal for vertical enamel cracks that were unstained but accepting methylene blue dye (Fig. 3); 25% would recommend removal for vertical enamel cracks that were stained but undetectable by an explorer (Fig. 4); and 12% of the general dentists surveyed would recommend the removal of intracoronal restorations to explore for unstained vertical enamel cracks that were detectable by transillumination but were not accepting methylene blue dye (Fig. 5). Twenty percent of participants would not recommend the removal of intracoronal restorations to explore the extension of asymptomatic enamel cracks.

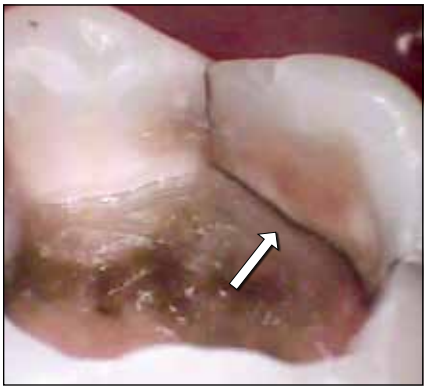

Fig. 7. Mandibular right first molar, occlusal view. Stained oblique dentin crack.

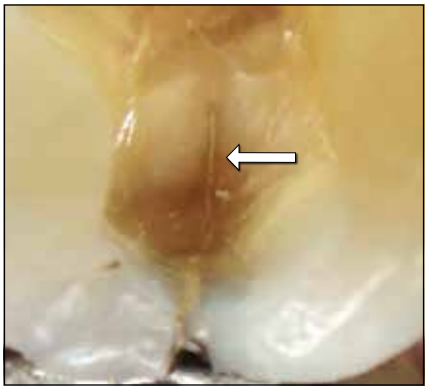

Fig. 8. Maxillary left first molar, occlusal view. Unstained vertical dentin crack.

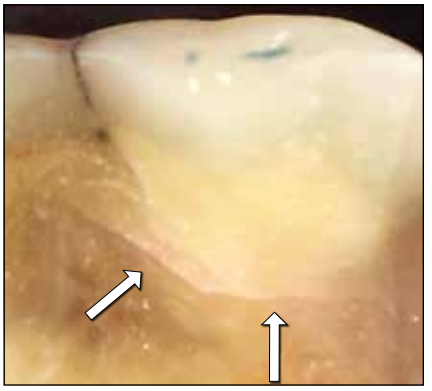

Fig. 9. Mandibular right first molar, occlusal view. Unstained oblique dentin crack.

With regard to asymptomatic dentin cracks, 86% of general dentists in the sample would recommend treatment for stained vertical dentin cracks (Fig. 6) and 84% would recommend it for stained oblique dentin cracks in posterior teeth (Fig. 7), followed by 65% for both unstained vertical dentin cracks and for unstained oblique dentin cracks (Fig. 8 and 9). Eight percent of the general dentists surveyed would not recommend any treatment for asymptomatic dentin cracks.

Participants were then asked to rank their treatment approaches (restorative and/or occlusal) for asymptomatic vertical and oblique dentin cracks noted after restoration removal, regardless of the relationship between isthmus width and cusp-to-cusp distance. Participants were able to give the same ranking to multiple treatment approaches if they would recommend them simultaneously, such as occlusal treatments in conjunction with a restorative approach. The treatments of choice (TOC) for the 47 general dentists who treat asymptomatic dentin cracks are presented in the Table. The majority of participants were in favor of occlusal coverage restorations, such as full crown and onlays. Protective occlusal hard bite plates and occlusal adjustments of opposing and cracked teeth were the primary TOC when used in conjunction with restorative treatments.

Table. The treatment of choice (TOC) for asymptomatic vertical and oblique dentin cracks chosen by general dentist survey participants (n = 47).

	TOC for vertical dentin cracks (%)	TOC for oblique dentin cracks (%)
Full crown restoration	51	40
Indirect occlusal coverage restoration (such as porcelain onlay restoration)	19	32
Direct bonded composite intracoronal restoration	17	13
Direct occlusal coverage restoration (such as bonded composite onlay restoration)	4	2
Protective occlusal hard bite plate[a]	21	23
Occlusal adjustment of opposing tooth[a]	17	17
Occlusal adjustment of cracked tooth[a]	13	19

[a]Occlusal treatments were mainly chosen as the TOC in conjunction with restorative treatments. As a result, the responses do not add up to 100%.

Literature review

Diagnosis of asymptomatic cracks

Asymptomatic enamel and dentin cracks can be very subtle and difficult to diagnose. They can be evaluated by transillumination, staining with dyes such as methylene blue dye, and tactile examination with a sharp explorer.[14-16] Visual examination can also be enhanced by magnification with tools such as magnifying loupes, intraoral photography, and microscopes.[14,15,17]

Management of asymptomatic enamel cracks

The traditional classifications of cracks placed less emphasis on the possibility of underlying pathologies of enamel cracks.[17] Fortunately, more modern approaches are taking asymptomatic enamel cracks into consideration.

Clark et al classified asymptomatic enamel cracks based on the risk of underlying pathologies such as dentin cracks, decay, and severely undermined enamel that allow microleakage.[17] According to their analysis, cracks with wedge-shaped enamel ditching and cracks that either detour from or do not follow anatomic grooves have a moderate risk of underlying pathology.[17] Diagonal cracks, cracks that house debris, and cracks with a brown, gray, or white corresponding "halo" have a high risk of underlying pathology.[17] The greater the risk, the more strongly it is recommended to remove the restoration for further evaluation, followed by treatment of any underlying pathology as needed, even if the tooth is asymptomatic.[17]

In their study of cusp fracture in restored posterior teeth, Bader et al concluded that external fracture lines that are detectable with an explorer should be considered strong indicators of elevated risk of complete fracture.[7] However, it has been emphasized that even dramatic enamel cracks may not necessarily indicate the presence of any underlying pathology.[8,17]

Ratcliff et al classified posterior enamel cracks based on the presence of stain and restorations, and suggested that stained cracks are most likely to be treated due to their appearance.[11] The authors also concluded that equilibrating the occlusion for maximum intercuspation and eliminating excursive interferences may prevent the propagation of asymptomatic cracks.[11]

Walker et al also suggested that stained cracks should be considered as permeable or permeated by cariogenic bacteria, and that cracks displaying a shadow under transillumination indicate the presence of caries.[2] Intervention is recommended for these cracks to block bacterial invasion and stop the progression of caries.[2]

Management of asymptomatic dentin cracks

Taking a more proactive approach of assessing enamel cracks based on the possibility of underlying pathologies—such as dentin cracks—brings another dilemma to practitioners regarding what to do when an asymptomatic dentin crack is discovered.

Dentin cracks should be considered structural cracks and therefore protection from occlusal forces to minimize fracture propagation is indicated.[3,8,17] However, Clark et al speculated that intracoronal restorations and occlusal adjustments might be proven insufficient to stop structural breakdown associated with cracks, and that occlusal coverage is mandatory.[17] Replacing an asymptomatic cracked cusp with a restoration when 1 cusp is involved has been recommended.[8] When more than 1 cusp is involved or there are asymptomatic vertical cracks, placing a full crown restoration is recommended.[3,8]

Currently the literature—including clinical trials—focuses mainly on the treatment of symptomatic cracks. However, the principles used to treat symptomatic cracks can be applied to the treatment of asymptomatic teeth predisposed to cracking; this includes the treatment of asymptomatic cracks to limit their progression and thus prevent any subsequent undesired consequences.[4]

Occlusal adjustment has been recommended as an initial treatment for cracked tooth syndrome.[4] Although there is no universally accepted restorative protocol in the treatment of symptomatic cracks, it is generally agreed that the aim of restorative therapy is to immobilize the segments of the tooth that tend to move during loading.[13] Opdam et al found that direct composite restorations maintained the pulp vitality of >90% of cracked, painful teeth, resulting in a complete elimination of pain in 75% of the affected teeth over a 7-year period.[18] The authors also had more success with cuspal coverage than without.[18] Modern approaches advocate reinforcing resin-based restorations with leno-weave ultra high modulus polyethylene ribbon fibers in order to bridge cracks and strengthen teeth against fractures.[19,20] Crown restorations were successful in maintaining pulp vitality in 80% of cracked teeth diagnosed with reversible pulpitis over a 6-year period.[5] Additionally, altering traditional crown preparations (such as beveling fractured cusps), using bases and build-ups under crown restorations, and placing margins more apically were suggested to minimize external forces and prevent the propagation of fractures underneath crown restorations.[21] Additional clinical trials comparing these direct and indirect treatment options are required to determine the best treatment for the various forms of incomplete tooth fractures.[18,22]

Prevention of cracks

Whenever possible, the proactive prevention of tooth cracks is the optimal treatment choice. This requires a deep understanding of the etiology of tooth cracks in an effort to control the contributing factors to their formation. The etiology of teeth cracks is complex and multifactorial; Lynch & McConnell proposed 10 factors that contribute to cracked teeth, citing 17 examples.[23] The most commonly emphasized etiologic factors are the loss of dentin support due to relatively large intracoronal restorations, and traumatic occlusal forces, especially when accompanied by excursive interferences.[4,7,11]

The susceptibility of teeth to fracture has been measured in terms of isthmus width in relation to cusp-to-cusp distance.[24] If teeth have been weakened due to wide cavity preparations, they need to be stabilized via indirect restorations, such as full cuspal coverage or bonded inlays.[25] However, the isthmus width is not the only factor that needs to be considered. The depth of the restoration along the isthmus width can be a more accurate measurement of the lack of dentin support.[7]

Recent research has shown that the prevalence of cusp fractures in amalgam restored teeth was found not to be significantly different than resin composite restored teeth.[26] These new findings may further shift the emphasis away from treatment plans that are based mainly on the choice of restoration materials, as these may result in designs that weaken the teeth. Conservative cavity preparations that preserve tooth structure without connecting multiple occlusal preparations have been advocated.[17] Additionally, rounded internal line angles have been recommended over sharp line angles to avoid stress concentrations.[23]

Nonrestorative approaches have also been recommended to prevent cracks. Occlusal adjustment of nonfunctional cusps of teeth predisposed to cracking—such as teeth with excessive cuspal wear, heavy wear facets, worn restorations, or posterior malocclusion—has been recommended, especially when the patient has a history of cracked tooth syndrome.[4] Although occlusal guards have not been directly linked to preventing cracks and limiting their progression, they are considered useful protectors of teeth against the damage caused by bruxism. Thus they are an option worthy of consideration in patients with a history of symptomatic cracks and evidence of bruxism.[27]

Summary

The majority of the general dentists who completed this survey would recommend intervention for some forms of asymptomatic enamel cracks, but were more proactive when it came to treating asymptomatic dentin cracks, emphasizing restorations that provide occlusal coverage. Becoming familiar with the existence of all forms of asymptomatic enamel and dentin cracks, the modern methods of

their diagnosis, and the levels of risk of underlying pathologies is crucial to adopting a proactive approach of prevention, early diagnosis, and intervention before major complications occur. In the wake of limited available knowledge, and until more evidence validates the necessity and the different modes of intervention for all forms of asymptomatic cracks, clinicians must rely on their clinical experience in weighing the benefits and risks of observation vs intervention, and then guide their patients to make informed decisions.

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References

1. Luebke RG. Vertical crown-root fractures in posterior teeth. *Dent Clinic North Am.* 1984;28(4):883-894.
2. Walker BN, Makinson OF, Peters MC. Enamel cracks. The role of enamel lamellae in caries initiation. *Aust Dent J.* 1998;43(2):110-116.
3. Abou-Rass M. Crack lines: the precursors of tooth fractures - their diagnosis and treatment. *Quintessence Int Dent Dig.* 1983;14(4):437-447.
4. Agar JR, Weller RN. Occlusal adjustment for initial treatment and prevention of the cracked tooth syndrome. *J Prosthet Dent.* 1988;60(2):145-147.
5. Krell KV, Rivera EM. A six year evaluation of cracked teeth diagnosed with reversible pulpitis: treatment and prognosis. *J Endod.* 2007;33(12):1405-1407.
6. Pitts DL, Natkin E. Diagnosis and treatment of vertical root fractures. *J Endod.* 1983;9(8):338-346.
7. Bader JD, Shugars DA, Martin JA. Risk indicators for posterior tooth fracture. *J Am Dent Assoc.* 2004; 135(7):883-892.
8. Ailor JE Jr. Managing incomplete tooth fractures. *J Am Dent Assoc.* 2000;131(8):1168-1174.
9. Bader JD, Martin JA, Shugars DA. Incidence rates for complete cusp fracture. *Community Dent Oral Epidemiol.* 2001;29(5):346-353.
10. Braly BV, Maxwell EH. Potential for tooth fracture in restorative dentistry. *J Prosthet Dent.* 1981;45(4):411-414.
11. Ratcliff S, Becker IM, Quinn L. Type and incidence of cracks in posterior teeth. *J Prosthet Dent.* 2001;86(2): 168-172.
12. Bader J, Shugars D, Roberson T. Using crowns to prevent tooth fracture. *Community Dent Oral Epidemiol.* 1996;24(1):47-51.
13. Banerji S, Mehta SB, Millar BJ. Cracked tooth syndrome. Part 2: restorative options for the management of cracked tooth syndrome. *Br Dent J.* 2010;208(11): 503-514.
14. American Association of Endodontists. Endodontics: Colleagues for Excellence [newsletter]. *The Obvious and the Obscure: Steps for Crack Detection and Confirmation.* Available at: http://www.aae.org/uploadedFiles/Publications_and_Research/Endodontics_Colleagues_for_Excellence_Newsletter/LTFBonus Material_C.pdf. Accessed August 19, 2014.
15. Alassaad SS. Incomplete cusp fractures: early diagnosis and communication with patients using fiber-optic transillumination and intraoral photography. *Gen Dent.* 2011;59(2):132-135.
16. Wright HM Jr, Loushine RJ, Weller RN, Kimbrough WF, Waller J, Pashley DH. Identification of resected root-end dentinal cracks: a comparative study of transillumination and dyes. *J Endod.* 2004;30(10):712-715.
17. Clark DJ, Sheets CG, Paquette JM. Definitive diagnosis of early enamel and dentin cracks based on microscopic evaluation. *J Esthet Restor Dent.* 2003; 15(7):391-401; discussion 401.
18. Opdam NJ, Roeters JJ, Loomans BA, Bronkhorst EM. Seven-year clinical evaluation of painful cracked teeth restored with a direct composite restoration. *J Endod.* 2008;34(7):808-811.
19. Belli S, Erdemir A, Ozcopur M, Eskitascioglu G. The effect of fibre insertion on fracture resistance of root filled molar teeth with MOD preparations restored with composite. *Int Endod J.* 2005;38(2):73-80.
20. Belli S, Cobankara FK, Eraslan O, Eskitascioglu G, Karbhari V. The effect of fiber insertion on fracture resistance of endodontically treated molars with MOD cavity and reattached fractured lingual cusps. *J Biomed Mater Res B Appl Biomater.* 2006;79(1): 35-41.
21. Casciari BJ. Altered preparation design for cracked teeth. *J Am Dent Assoc.* 1999;130(4):571-572.
22. Wright EF, Bartoloni JA. Diagnosing, managing, and preventing cracked tooth syndrome. *Gen Dent.* 2012; 60(5):e302-e307.
23. Lynch CD, McConnell RJ. The cracked tooth syndrome. *J Can Dent Assoc.* 2002;68(8):470-475.
24. Cavel WT, Kelsey WP, Blankenau RJ. An in vivo study of cuspal fracture. *J Prosthet Dent.* 1985;53(1):38-42.
25. Geurtsen W, Schwarze T, Gunay H. Diagnosis, therapy, and prevention of the cracked tooth syndrome. *Quintessence Int.* 2003;34(6):409-417.
26. Wahl MJ, Schmitt MM, Overton DA, Gordon MK. Prevalence of cusp fractures in teeth restored with amalgam and with resin-based composite. *J Am Dent Assoc.* 2004;135(8):1127-1132; quiz 1164-1165.
27. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M. Principles for the management of bruxism. *J Oral Rehabil.* 2008;35(7):509-523.

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Exercise No. 358 Fissurotomy (Micro) Dentistry Subject Code 259

The 15 questions for this exercise are based on the article, *Approaches to managing asymptomatic enamel and dentin cracks,* on pages 58-62. This exercise was developed by Daniel S. Geare, DMD, in association with the *General Dentistry* Self-Instruction committee.

Reading the article and successfully completing this exercise will enable you to understand:

- the nature of symptomatic and asymptomatic cracks;
- the causes of symptomatic and asymptomatic cracks; and
- treatment options for cracked teeth.

1. Pathological consequences of cracked teeth include all of the following except one. Which is the exception?
   A. caries
   B. periodontal involvement
   C. pulpal pathology
   D. temporomandibular dysfunction

2. Tooth fractures are the _____ most common cause of tooth loss.
   A. fifth
   B. fourth
   C. third
   D. second

3. There is a universally agreed upon protocol for treating cracked teeth. This is true for both symptomatic and asymptomatic cracks.
   A. Both statements are true.
   B. The first statement is true; the second is false.
   C. The first statement is false; the second is true.
   D. Both statements are false.

4. Enamel and dentin cracks are classified according to the
   A. severity of symptoms.
   B. location in the mouth.
   C. direction of the crack.
   D. progression over time.

5. _____ percent of dentists studied recommended removing fillings as part of cracked tooth therapy.
   A. Seventy-three
   B. Seventy-five
   C. Eighty-eight
   D. Ninety-two

6. The technique used for detecting vertical enamel cracks was
   A. methylene blue dye.
   B. bite stick.
   C. methyl red dye.
   D. water refraction.

7. Asymptomatic cracks can be evaluated by all of the following except one. Which is the exception?
   A. transillumination
   B. chewing ice
   C. staining with dyes
   D. microscopy

8. A high-risk crack includes
   A. a brown halo.
   B. wedge-shaped ditching.
   C. intersecting lines.
   D. veering off anatomic grooves.

9. External fracture lines that are detectable with an explorer are an indicator of high-risk cracks. This is because dramatic enamel cracks are strong indicators of pathology.
   A. Both statements are true.
   B. The first statement is true; the second is false.
   C. The first statement is false; the second is true.
   D. Both statements are false.

10. Occlusal coverage is indicated in all of the following cases except one. Which is the exception?
    A. One cusp is cracked.
    B. Two or more cusps are cracked.
    C. There are enamel and dentin cracks.
    D. There are enamel cracks only.

11. Initial treatment of cracked tooth syndrome includes
    A. filling the cracked area with composite.
    B. root canal therapy.
    C. occlusal adjustment.
    D. a full coverage crown.

12. Direct composite restorations were found to eliminate cracked tooth symptoms in _____% of cases.
    A. 90
    B. 80
    C. 77
    D. 75

13. Techniques in crown prep design that minimize external forces include all of the following except one. Which is the exception?
    A. apical crown margins
    B. beveling cusp tips
    C. internal pulp caps
    D. minimal wall reduction

14. One of the most common etiologic factors in teeth cracks is
    A. traumatic occlusal forces.
    B. aging amalgam restorations.
    C. the age of the individual.
    D. recurrent decay.

15. For oblique dentin cracks, the most recommended course of treatment was
    A. indirect occlusal coverage.
    B. direct bonded composite.
    C. occlusal adjustment.
    D. full crown restoration.

Answer form is on the inside back cover. Answers for this exercise must be received by October 31, 2015.

Perspective of cardiologists on the continuation or discontinuation of antiplatelet therapy before dental treatment: a questionnaire-based study

Ruchi Banthia, MDS ▪ Pallavi Singh ▪ Priyank Banthia, MDS ▪ Rajbhan Singh, MBBS, DMd ▪ Santosh Gupta ▪ Sapna Raje

Antiplatelet and anticoagulant agents have been extensively researched and developed as potential therapies in the prevention and management of arterial and venous thrombi. These medications are associated with an increase in bleeding time and risk of intraoperative and postoperative hemorrhage in the dental office. There is some controversy regarding whether these agents should be temporarily discontinued before dental procedures. In order to gain insight into this controversy, a survey of 50 cardiologists was conducted regarding suggested guidelines for dentists in the management of patients who are taking anticoagulant medication.

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Key words: antiplatelet therapy, bleeding risk, thromboembolic events, dental treatment

Platelets provide the initial hemostatic plug at the site of vascular injury. They are also involved in pathological processes and serve as an important contributor to arterial thrombosis possibly leading to myocardial infarction and cerebrovascular stroke.[1] Various antiplatelet and anticoagulant agents are used for the prevention and management of arterial thrombi.[2] The most common of these are low-dose aspirin (75-300 mg daily), clopidogrel, dipyridamole, and warfarin.[3,4] Antiplatelet therapy is associated with the inhibition of platelet aggregation. When platelets are inhibited, it takes longer for primary hemostasis to occur, hence bleeding time is prolonged, leading to an increased risk of intraoperative and postoperative hemorrhage during dental procedures.[3,5,6] Dental practitioners are encountering more and more cardiac patients taking antiplatelet medications in their routine practice. A decision needs to be made whether to temporarily discontinue antiplatelet therapy before performing any dental procedure on a patient with this medical history. The dilemma lies in the fact that although these medications increase the risk of hemorrhage during dental procedures, temporarily stopping them can put the patients at risk of thromboembolic events, such as stroke.[6] Therefore, a medical opinion and consent is mandatory for the management of cardiac patients in the dental office, and the risk of occurrence of thromboembolic event must be weighed against the risk of hemorrhage. Previously, discontinuation of antiplatelet therapy for either 3 or 7 days was recommended prior to dental procedures to avoid excessive bleeding, but with increasing concern over the thromboembolic risk, this is no longer recommended.[7] There is a great deal of confusion and controversy among dental surgeons regarding this issue. This study sought to obtain evidence-based results by conducting a survey on 50 cardiologists with the goal of helping to resolve the controversy.

Materials and methods

A structured questionnaire consisting of 7 questions was prepared by 2 of the study's coauthors (R. Banthia and P. Banthia). Verbal consent was obtained over the telephone from 72 cardiologists and the questionnaires were distributed by 2 different coauthors (P. Singh and R. Singh). The physicians were asked to respond in 2-3 days. The first 50 responses received were accepted for use in the study. The data was collected and the responses analyzed (Table).

Discussion

Antiplatelet therapy agents (also known as *blood thinners)* are used mainly for the inhibition of platelet activation or aggregation. They are effective in arterial circulation where anticoagulants have little effect.[7] Aspirin irreversibly acetylates cyclooxygenase, inhibiting the production of thromboxane A_2, resulting in decreased platelet aggregation.[8] Clopidogrel selectively inhibits ADP-induced platelet aggregation. Dipyridamole is an adenosine reuptake inhibitor and phosphodiesterase inhibitor with antiplatelet and vasodilating activity.[9]

Most of the cardiologists surveyed use either aspirin as monotherapy, or aspirin and clopidogrel as dual therapy. Aspirin and clopidogrel have synergistic antiplatelet effects that block complementary pathways in a platelet aggregation cascade.[10,11]

Dipyridamole is another antiplatelet therapeutic agent. Oral antiplatelet regimens vary in different institutions, but the recommendations of the *American College of Chest Physicians* in 2006 were that aspirin (75-162 mg) should be prescribed and continued indefinitely for all patients with stable coronary artery disease (CAD), and clopidogrel in combination with aspirin is advised for all stable CAD patients with a risk profile that indicates a high likelihood of developing acute myocardial infarction.[12]

The effect on primary hemostasis is minimal when antiplatelet agents are used as monotherapy in patients, with no additional risk factors for impaired clotting.[13] The risk of bleeding may increase in combination therapy cases.[14] While aspirin can double the bleeding time, this may still be in normal range. Only 20%-25% of patients on aspirin were reported to have abnormal bleeding times. Burger et al observed that patients on an aspirin regimen have, on average, an increased (1.5-fold) risk of intraoperative hemorrhagic risk, without an increase in surgical morbidity and mortality.[15] Clopidogrel, being a more potent antiplatelet agent, can cause a 1.5 to 3-fold increase in bleeding times.[16,17]

Table. Survey results

Questions	Responses			
1. Which antiplatelet agent do you most frequently prescribe?	Ninety percent of the doctors prescribed either aspirin as a monotherapy, or aspirin and clopidrogel as a dual therapy. The remaining 10% used other antiplatelet agents such as dipyramidole.			
2. Do you recommend stopping antiplatelet therapy before dental procedures?	A. Before minor dental procedures (such as scaling and extractions)? Eighty percent of the doctors were in favor of not stopping the antiplatelet therapy before minor dental procedures. Twenty percent recommended the discontinuation of antiplatelet therapy.	B. Before surgical procedures? All the doctors were in favor of stopping antiplatelet therapy before surgical procedures.	C. If 'yes', why? Discontinuation of antiplatelet therapy was recommended to avoid the risk of postoperative and intraoperative bleeding and risk of haemorrhage.	D. If 'yes', for how many days? Sixty percent of the doctors were in favor of stopping antiplatelet therapy 5 days prior to the dental procedure. Thirty percent recommended the discontinuation of antiplatelet therapy 7 days prior to the procedure. Ten percent recommended discontinuation starting 3 days prior, and continuing 2 days postoperatively.
3. On which criteria did you base your decision to continue or discontinue the antiplatelet therapy?	Fifty percent of the doctors based their decision on both clinical experience and evidence-based research. Twenty-five percent of the doctors based their decision solely on evidence-based research, and 25% based their decisions predominantly on their clinical experience.			
4. When 'written medical consent' is required by a dental practitioner, what protocol do you follow?	A. What investigations do you recommend? All of the doctors recommend bleeding time, clotting time, ECG, routine investigations, and blood sugar level tests. Ten percent of the doctors request a chest X-ray and an international normalized ratio (INR) blood test, as well.	B. Do you advise an INR? Ten percent of the doctors advised INR routinely; 90% advised it only when the patient is on an oral anticoagulant, such as low molecular weight heparin or warfarin.	C. Do you have printed consent forms in your clinic? Eighty percent of the doctors did not have printed consent forms.	
5. Have you ever been consulted for a bleeding episode—in a patient on antiplatelet therapy—by a dental office?	Five percent of the doctors had been consulted for the management of 1 or 2 bleeding episodes.			
6. How many episodes of thrombo-embolic events have been reported to you in cases where discontinuation of antiplatelet therapy was recommended?	All of the doctors encountered 1 or 2 cases of thromboembolic events after discontinuation of antiplatelet therapy.			
7. What is your final recommendation?	All of the doctors recommended discontinuation of the antiplatelet therapy before surgical procedures. Eighty percent recommended continuation of antiplatelet therapy before minor dental procedures, while 20% advised discontinuation of these agents before minor procedures.			

Eighty percent of the cardiologists surveyed were not in favor of discontinuing antiplatelet therapy before minor dental procedures. However, all of the doctors surveyed were in favor of stopping antiplatelet therapy for surgical procedures. This discontinuation was recommended to avoid the risk of postoperative and intraoperative bleeding.

Ardekian et al investigated the effect of aspirin on the hemorrhagic risk in patients undergoing dental extractions.[18] None of the patients reported any episode of uncontrolled bleeding immediately after the procedure or in the following week.[18] Intraoperative bleeding was managed either by suturing, gauze packs, and/or use of tranexamic acid in local packing.[18]

The pharmacological actions of clopidogrel and dipyridamole suggest that patients taking these medications will be at no greater risk of excessive bleeding than those taking aspirin. While clopidogrel has shown an increased bleeding time compared to aspirin, it has proved to be clinically more potent; and it has been established that there is no risk of

excessive bleeding when using clopidogrel during dental procedures.[15-19] If a patient is on a dual therapy of aspirin and clopidogrel, it is recommended that the dental procedure be performed in a hospital setting, in order to more proactively manage any severe bleeding episode, as such patients are at a higher risk of hemorrhage. Patients with underlying hepatic, renal, or bone marrow disorders often have disease-related bleeding disorders. Bleeding risk also increases with age and with heavy alcohol consumption.[4]

Sixty percent of the cardiologists surveyed were in favor of stopping antiplatelet therapy 5 days prior to the dental procedure. Thirty percent recommended the discontinuation of antiplatelet therapy 7 days prior to the procedures. Ten percent recommended discontinuation starting 3 days prior, continuing 2 days postoperative.

All patients on antiplatelet therapy may have drug-induced alterations of platelets, the degree of which varies from person to person. Aspirin irreversibly inhibits platelet aggregation within 1 hour of ingestion, and clopidogrel does so within 2 hours. This inhibition lasts for the lifetime of platelets, approximately 7-10 days. This effect is only overcome by the production of new platelets.[20] Complete recovery of platelet aggregation may occur in 50% of cases by Day 3, and in 80% of cases by Day 4 postoperative.[21] Ferrari et al concluded that when aspirin (either 75 or 300 mg) was stopped in healthy patients after 2 weeks of therapy, all bleeding times returned to normal after 6 days.[22] The action of dipyridamole is reversible and ceases about 24 hours after the drug is discontinued.[16]

Only 5% of the medical practitioners surveyed had been consulted for the management of 1 or 2 bleeding episodes. Lockhart et al suggested that postoperative bleeding is considered significant if the bleeding continues beyond 12 hours, causes the patient to call or return to the dental office or emergency department, results in the development of a large hematoma or ecchymosis within oral soft tissues, or requires a blood transfusion.[23]

Life-threatening bleeding after dental surgery is rare.[24] According to Matocha, the incidence of postextraction hemorrhagic complications is 0.2%-2.3%.[25] McGaul reported a case of sublingual hematoma in mandibular anterior teeth following periodontal flap surgery in a patient on long-term antiplatelet therapy which resolved on its own.[26] Thomason et al reported severe bleeding following a gingivectomy in a patient taking 150 mg aspirin qd, which was resolved by platelet transfusion.[27]

Napenas et al found no differences in hemorrhages following dental treatments such as extractions, periodontal surgery, subgingival scaling, and root planing between patients receiving single or dual antiplatelet therapy.[28] In a study by Partridge et al, the amount of blood loss was found to be similar in patients on antiplatelet therapy and healthy patients (controls) during dentoalveolar surgery.[29] The Antiplatelet Trialists' Collaboration concluded that long-term antiplatelet therapy caused reductions in mortality, relative risk of myocardial attack, and cerebrovascular incidents; with only a mild (0.12%) increase of spontaneous hemorrhage risk.[30]

All the doctors surveyed who had been asked for consent before dental therapy on their patients with antiplatelet regimens recommended bleeding time, clotting time, ECG, routine investigations, blood sugar level, and blood pressure tests. Only 10% of the doctors advised chest X-rays, and only 10% advised an International Normalized Ratio (INR) in order to know the status of coagulation. An INR is advised for all patients on warfarin or heparin therapy.[31]

Although there is no suitable test to assess the increased risk of bleeding in patients taking antiplatelet therapy, platelet function is normally assessed using the cutaneous bleeding time test. In a normal healthy patient, bleeding time ranges from 2 to 10 minutes.[32] Prothrombin time (PT) and partial thromboplastin time have been used to evaluate anticoagulant levels. The INR was introduced in 1983 by the World Health Organization Committee on Biological Standards, who defined the INR as the ratio of the patient's PT to a control PT, raised to the power of the International Sensitivity Index (ISI).[33]

The INR is a more reliable and sensitive test for determining the level of anticoagulation as it depends on both the patient's blood and the sensitivity of the assigned ISI value. The PT alone would not be an accurate gauge in the evaluation of a patient's anticoagulant status. A patient with a normal coagulation profile would have an INR of 1. It is recommended that a patient undergoing invasive treatment should have a PT 1.5-2.0 times the normal value (INR = 1.5-2.5 when the ISI is 1).[33] In patients on antiplatelet therapy, the recommended INR is 2.0-3.0 for most procedures. This range of INR (2.0-3.0, average 2.5) minimizes the risk of both hemorrhage and thromboembolic events.[34] Nevertheless, minor surgical dental procedures can safely be performed with an INR between 2 and 4, while being aware that local measures may be needed to control bleeding.[6] In patients on warfarin and heparin, the INR should be checked within 24 hours prior to the procedure.[31]

A correlation between bleeding time test results and the rate of surgical bleeding complications has not been established.[35] Shalom & Wong concluded that cutaneous bleeding tests should not be used to estimate the hemorrhagic risk in patients on anticoagulant therapy.[36] Of the doctors surveyed, all reported 1 or 2 cases of thromboembolic events after discontinuation of antiplatelet therapy.

Thromboembolic events following the cessation of antiplatelet medications have also been reported in the literature. One retrospective analysis study reported that out of 475 patients admitted with myocardial infarction, 11 (2.3%) had discontinued aspirin within 15 days prior to the attack.[37] Nine patients discontinued aspirin due to a planned procedure, 1 of which was a dental procedure.[37] Another study by the same author reported that 5% of the patients who were admitted for acute coronary syndrome had admitted they had stopped using oral anticoagulant agents, and the authors concluded that a rebound effect occurs after an interruption of oral antiplatelet medication.[38] In a study by Ferrari et al, the mean delay time between aspirin withdrawal and an acute coronary event was 10 ± 1.9 days (range 4-17 days); and 13 of the patients (25.5%) who discontinued their aspirin medication did so prior to dental treatment.[22] Maulaz et al reported the mean interval between treatment disruption and cerebral infarction was 9.5 ± 7 days.[39] Kovich & Otley estimated that the risk of thromboembolic events associated with the withdrawal of aspirin 3-14 days days prior to cutaneous surgery was approximately 0.005%.[40]

Management of patients on antiplatelet therapy in a dental office

According to Scully & Wolff, oral surgical procedures must be done at the beginning of the day, as it allows more time to deal with any bleeding episode.[41] Procedures should also be performed early in the week so that prompt management of any delayed bleeding can be done.

Local anaesthetic containing a vasoconstrictor should be administered. Field blocks are contraindicated. If no alternative exists, local anaesthetic should be administered cautiously with repeated aspiration.[42,43]

Atraumatic and careful manipulation of tissues is recommended. Bleeding should be stopped by local measures, such as use of pressure packs for 15-30 minutes, packing of sockets with absorbable hemostatic dressings (oxidised cellulose, haemocollagen, or resorbable gelatin sponge), and suturing.[42,43]

The use of aspirin leads to increased bleeding time. If it increases to >20 minutes and surgery has to be performed as an emergency procedure, 1-desamino-8-D-arginine vasopressin can be used to shorten the bleeding time.[44] This involves the enhancement of Von Willebrand's factor which in turn acts as a platelet aggregant. It can be used at a dose of 0.3 μg/kg of body weight—not exceeding 20-24 μg—or as a nasal spray. This should be administered under a physician's guidance as this can cause drug-induced thrombosis in older subjects.[8]

Paracetamol is the painkiller drug of choice for patients on antiplatelet therapy. Nonsteroidal anti-inflammatory drugs (NSAIDs) are avoided, as they carry the potential risk of increasing bleeding time by having a reversible effect on platelet aggregation and function.[4] To ensure the absence of any antiplatelet effect, NSAIDs should be discontinued 5 half-lives before the procedure.[45]

Scully & Cawson developed the following list of instructions to be given to patients for the management of a clot in the postoperative period.[46]

- Rest until the local anesthetic wears off and the clot forms (2-3 hours).
- Avoid rinsing the mouth for 24 hours.
- Do not suck forcefully or disturb the socket with the tongue or any foreign objects.
- Avoid hot liquids and hard foods for the first day.
- Avoid chewing on the affected side until it is clear that a stable clot has formed.
- Apply pressure over the socket using a folded clean handkerchief or gauze pad for 20 minutes if bleeding continues or restarts. If bleeding does not stop, consultation with the dentist is advised.[46]

Patients with the following medical problems taking antiplatelet medications should not be treated in primary care without medical advice or should be referred to a hospital-based dental clinic: liver impairment and/or alcoholism; renal failure; hemostasis disorders; and patients currently receiving cytotoxic medication or dual antiplatelet therapy.[42,43,47]

A consensus opinion from *American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons,* and *American Dental Association* recommended continuing aspirin and clopidogrel therapy for minor dental surgical procedures in patients who have coronary artery stents, or delaying the treatment until the prescribed regimen is completed.[48] When >3 teeth need to be extracted, multiple visits are required. Scaling and gingival surgery should initially be restricted to a limited area.[49]

When a definite increase in intraoperative bleeding is anticipated, or when surgical hemostasis could possibly be difficult, aspirin can be replaced for a 10-day period by a shorter-acting NSAID regimen, interrupted the day before surgery. Postoperative antiplatelet therapy should be resumed immediately after surgery (first 6 hours).[49]

Medical consent is mandatory in cardiac patients taking antiplatelet therapy in order to know the exact medical condition of the patient and to prevent any unwanted sequelae. Properly structured consent forms should be mandatory. Eighty percent of the cardiologists surveyed did not have printed consent forms.

Conclusion

The Hippocratic oath states, "First do no harm." This must be considered immediately when deciding whether or not to temporarily discontinue antiplatelet therapy before dental treatment. Careful appraisal of the type of procedure, vascular status of the patient, and chances of complications—along with a risk-benefit analysis—should be undertaken in consultation with a cardiologist to ensure the complete well-being of the patient. The dental surgeon and physician should exercise judgment based on their skill, experience, and the facilities at their disposal so as to provide the best postoperative care to the patient. Fifty percent of the doctors surveyed in this study based their decisions on whether to temporarily discontinue antiplatelet therapy before a dental procedure on both clinical experience and evidence-based research; 25% percent based their decisions solely on evidence-based research; and the remaining 25% based their decisions predominantly on their clinical experience.

Clinical experience is a critical tool in the successful management of patients, but in today's world of ethical and lawful guidelines, clinical decisions need also to be based on evidence-based research.

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References

1. Bristol Laboratories Ltd. *Dispersible Aspirin Tablets BP 75mg* [product description]. Available at: http://www.mhra.gov.uk/home/groups/l-unit1/documents/websiteresources/con2023488.pdf. Accessed August 20, 2014.
2. Owens CD, Belkin M. Thrombosis and coagulation: operative management of the anticoagulated patient. *Surg Clin North Am.* 2005;85(6):1179-1189.
3. University of Dundee. *Surgical Management of the Primary Care Dental Patient on Antiplatelet Medication.* Available at: http://www.app.dundee.ac.uk/tuith/Static/info/antiplatelet.pdf. Accessed August 20, 2014.
4. Dogne JM, de Leval X, Benoit P, Delarge J, Masereel B, David JL. Recent advances in antiplatelet agents. *Curr Med Chem.* 2002;9(5):577-589.
5. Pototski M, Amenabar JM. Dental management of patients receiving anticoagulation or antiplatelet treatment. *J Oral Sci.* 2007;49(4):253-258.
6. Pharmacist's Letter. *Managing Anticoagulant and Antiplatelet Drugs Before Dental Procedures.* Available at: http://drkney.com/pdfs/dentist_anticoag_0511.pdf. Accessed August 20, 2014.
7. Jaya Kumar A, Kumari MM, Arora N, Haritha A. Is antiplatelet therapy interruption a real clinical issue? Its

implications in dentistry and particularly in periodontics. *J Ind Soc Periodontol.* 2009;13(3):121-125.
8. Merritt JC, Bhatt DL. The efficacy and safety of perioperative antiplatelet therapy. *J Thromb Thrombolysis.* 2002;13(2):97-103.
9. Sweetman SC, ed. *Martindale: The Complete Drug Reference.* 34th ed. London: Pharmaceutical Press; 2013.
10. Harder S, Klinkhardt U, Alvarez JM. Avoidance of bleeding during surgery in patients receiving anticoagulant and/or antiplatelet therapy. Pharmacokinetic and pharmacodynamic considerations. *Clin Pharmacokinet.* 2004;43(14):963-981.
11. Alam M, Goldberg LH. Serious adverse vascular events associated with perioperative interruption of antiplatelet and anticoagulant therapy. *Dermatol Surg.* 2002; 28(11):992-998; discussion 998.
12. Collet JP, Montalescot G. Premature withdrawal and alternative therapies to dual oral antiplatelet therapy. *Eur Heart J.* 2006;8:G46–G52.
13. Mielke CH Jr. Aspirin prolongation of the template bleeding time: influence of venostasis and direction of incision. *Blood.* 1982;60(5):1139-1142.
14. Lecompte T, Hardy JF. Antiplatelet agents and perioperative bleeding. *Can J Anaesth.* 2006;53(6 Suppl): S103-S112.
15. Burger W, Chemnitius JM, Kneissl GD, Rucker G. Low-dose aspirin for secondary cardiovascular prevention - cardiovascular risks after its perioperative withdrawal versus bleeding risks with its continuation - review and meta-analysis. *J Intern Med.* 2005;257(5):399-414.
16. Lenz TL, Hilleman DE. Aggrenox: a fixed-dose combination of aspirin and dipyridamole. *Ann Pharmacother.* 2000;34(11):1283-1290.
17. Wahl MJ. Myths of dental surgery in patients receiving anticoagulant therapy. *J Am Dent Assoc.* 2000;131(1): 77-81.
18. Ardekian L, Gasper R, Peled M, Brener B, Laufer D. Does low-dose aspirin therapy complicate oral surgical procedures? *J Am Dent Assoc.* 2000;131(3):331-335.
19. Little JW, Miller CS, Henry RG, McIntosh BA. Antithrombotic agents: implications in dentistry. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002;93(5): 544-551.
20. Schafer AI. Effects of nonsteroidal antiinflammatory drugs on platelet function and systemic hemostasis. *J Clin Pharmacol.* 1995;35(3):209-219.
21. Cahill RA, McGreat GT, Crowe BH, et al. Duration of increased bleeding tendency after cessation of aspirin therapy. *J Am Coll Surg.* 2005;200(4): 564-573, quiz A59-A61.
22. Ferrari E, Benhamou M, Cerboni P, Marcel B. Coronary syndromes following aspirin withdrawal: a special risk for late stent thrombosis. *J Am Coll Cardiol.* 2005; 45(3):456-459.
23. Lockhart PB, Gibson J, Pond SH, Leitch J. Dental management considerations for the patient with an acquired coagulopathy. Part 1: coagulopathies from systemic disease. *Br Dent J.* 2003;195(8):439-445.
24. Jeske AH, Suchko GD, ADA Council on Scientific Affairs and Division of Science. Lack of a scientific basis for routine discontinuation of oral anticoagulation therapy before dental treatment. *J Am Dental Assoc.* 2003; 134(11):1492-1497.
25. Matocha DL. Postsurgical complications. *Emerg Med Clin North Am.* 2000;18(3):549-564.
26. McGaul T. Postoperative bleeding caused by aspirin. *J Dent.* 1978;6(3):207-209.
27. Thomason JM, Seymour RA, Murphy P, Brigham KM, Jones P. Aspirin-induced post-gingivectomy haemorrhage: a timely reminder. *J Clin Periodontol.* 1999; 24(2):136-138.
28. Napenas JJ, Hong CH, Brennan MT, Furney SL, Fox PC, Lockhart PB. The frequency of bleeding complications after invasive dental treatment in patients receiving single and dual anti-platelet therapy. *J Am Dent Assoc.* 2009;140(6):690-695.
29. Partridge CG, Campbell JH, Alvarado F. The effect of platelet-altering medications on bleeding from minor oral surgery procedures. *J Oral Maxillofac Surg.* 2008; 66(1):93-97.
30. Antiplatelet Trialists' Collaboration. Collaborative overview of randomized trials of antiplatelet therapy—I: prevention of death, myocardial infarction and stroke by prolonged antiplatelet therapy in various categories of patients. *Br Med J.* 1994;308(6921):81-106.
31. University of Dundee. *Surgical Management of the Primary Care Dental Patient on Warfarin.* Available at: http://www.app.dundee.ac.uk/tuith/Static/info/warfarin.pdf. Accessed August 20, 2014.
32. Guyton AC, Hall JE. Hemostasis and blood coagulation. In. *Guyton and Hall Textbook of Medical Physiology.* 11th ed. New York: Saunders Elsevier; 2006: 457-468.
33. Hirsh J, Poller L. The international normalised ratio. A guide to understanding and correcting its problems. *Arch Intern Med.* 1994;154(3):282-288.
34. Hirsh J, Dalen J, Anderson DR, et al. Oral anticoagulants: mechanism of action, clinical effectiveness, and optimal therapeutic range. *Chest.* 2001;119(1 Suppl): 8S-21S.
35. Samama CM et al. Antiplatelet agents in the perioperative period: expert recommendations of the French Society of Anesthesiology and Intensive Care (SFAR) 2001–summary statement. *Can J Anaesth.* 2002; 49(6):S26-S35.
36. Shalom A, Wong L. Outcome of aspirin use during excision of cutaneous lesions. *Ann Plast Surg.* 2003; 50(3):296-298.
37. Collet JP, Himbert D, Steg PG. Myocardial infarction after aspirin cessation in stable coronary artery disease patients. *Int J Cardiol.* 2000;76(2-3):257-258.
38. Collet JP, Montalescot G, Blanchet B, et al. Impact of prior use or recent withdrawal of oral antiplatelet agents on acute coronary syndromes. *Circulation.* 2004;110(16):2361-2367.
39. Maulaz AB, Bezerra DC, Michel P, Bogousslavsky J. Effect of discontinuing aspirin therapy on the risk of brain ischemic stroke. *Arch Neurol.* 2005;62(8):1217-1220.
40. Kovich O, Otley CC. Thrombotic complications related to discontinuation of warfarin and aspirin therapy perioperatively for cutaneous operation. *J Am Acad Dermatol.* 2003;48(2):233-237.
41. Scully C, Wolff A. Oral surgery in patients on anticoagulant therapy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002;94(1):57-64.
42. Lockhart PB, Gibson J, Pond SH, Leitch J. Dental management considerations for the patient with an acquired coagulopathy. Part 1: coagulopathies from systemic disease. *Br Dent J.* 2003;195:439-445.
43. Pototski M, Amenabar JM. Dental management of patients receiving anticoagulation or antiplatelet treatment. *J Oral Sci.* 2007;49(4):253-258.
44. Little JW, Miller CS, Henry RG, McIntosh BA. Antithrombotic agents: implications in dentistry. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002;93(5): 544-551.
45. Douketis JD, Berger PB, Dunn AS, et al. The perioperative management of antithrombotic therapy: *American College of Chest Physicians Evidence-Based Clinical Practice Guidelines* (8th Edition). *Chest.* 2008;133(6 Suppl):299S-339S.
46. Scully C, Cawson RA. *Medical Problems in Dentistry.* 4th ed. Oxford: Wright Butterworth-Heinemann; 1998: 49-100.
47. Daniel NG, Goulet J, Bergeron M, Paquin R and Landry PE. Antiplatelet drugs: is there a surgical risk? *J Can Dent Assoc.* 2002:68(11):683-687.
48. Grines CL, Bonow RO, Casey DE, Jr, et al. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: a science advisory from the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association, with representation from the American College of Physicians. *J Am Dent Assoc.* 2007;138(5):652-655.
49. Brennan MT, Wynn RL, Miller CS. Aspirin and bleeding in dentistry: an update and recommendations. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2007;104(3):316-323.

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06/01/2010 to 05/31/2018

Exercise No. 359 Pharmacotherapeutics Subject Code 016

The 15 questions for this exercise are based on the article, *Perspective of cardiologists on the continuation or discontinuation of antiplatelet therapy before dental treatment: a questionnaire-based study,* on pages 64-68. This exercise was developed by Merlin Ohmer, DDS, FAGD, in association with the *General Dentistry* Self-Instruction committee.

Reading the article and successfully completing this exercise will enable you to understand:
- the clinical implications of antiplatelet therapy;
- when and how to consult the patient's cardiologist; and
- the effects of antiplatelet therapy on dental patients.

1. Platelets are involved in all of the following except one. Which is the exception?
   A. myocardial infarction
   B. hemostasis
   C. stroke
   D. phagocytosis

2. Anticoagulants are used to
   A. prevent hemorrhage.
   B. increase healing.
   C. prevent swelling.
   D. prevent thrombi.

3. Drugs used in antiplatelet therapy include all of the following except one. Which is the exception?
   A. aprotinin
   B. aspirin
   C. warfarin
   D. clopidogrel

4. There is little consequence to stopping anticoagulant therapy. Rarely is a physician consultation necessary.
   A. Both statements are true.
   B. The first statement is true; the second is false.
   C. The first statement is false; the second is true.
   D. Both statements are false.

5. What percentage of cardiologists surveyed recommend stopping antiplatelet therapy prior to dental extraction?
   A. 20
   B. 40
   C. 60
   D. 80

6. If discontinuation of antiplatelet therapy is recommended, the majority of cardiologists surveyed recommend discontinuing exactly _____ days prior to surgery.
   A. 2
   B. 3
   C. 5
   D. 7

7. INRs must be obtained on anticoagulant patients. An ECG is also recommended.
   A. Both statements are true.
   B. The first statement is true; the second is false.
   C. The first statement is false; the second is true.
   D. Both statements are false.

8. Aspirin functions by
   A. reversibly affecting cyclooxygenase.
   B. irreversibly affecting acetic acid production.
   C. causing increased platelet aggregation.
   D. inhibiting thromboxane production.

9. Dipyridamole acts
   A. by causing vasoconstriction.
   B. as a phosphotriesterase accelerator.
   C. as an adenosine reuptake inhibitor.
   D. in ADP depletion.

10. Aspirin is reported to increase bleeding time by _____%.
    A. 50
    B. 100
    C. 150
    D. 200

11. What percentage of patients on aspirin therapy have an abnormal bleeding time?
    A. 20-25
    B. 30-35
    C. 40-45
    D. 50-55

12. A study by Ardekian et al indicated a ____% reported incidence of bleeding problems after dental extraction.
    A. 0
    B. 10
    C. 15
    D. 20

13. Aspirin's effect on platelets is seen within ____ hour(s) following ingestion.
    A. 1
    B. 2
    C. 3
    D. 4

14. The average platelet lifespan is ____ days.
    A. 3-5
    B. 7-10
    C. 12-15
    D. 18-21

15. The incidence of prolonged bleeding complications postextraction in patients on antiplatelet therapy is _____%.
    A. 0
    B. 0.2-2.3
    C. 2.5-4.7
    D. 4.9-7.1

Answer form is on the inside back cover. Answers for this exercise must be received by October 31, 2015.

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Mepivacaine: a closer look at its properties and current utility

William G. Brockmann, DDS, PhD

The use of mepivacaine in dentistry has remained strong since its introduction in the 1960s. It has retained its place as a valuable local anesthetic, either as a primary agent or as an alternative to lidocaine or articaine. Mepivacaine is commonly used in medically compromised patients—for whom elevations in blood pressure or heart rate are not advisable—in a formulation with a vasoconstrictor, or in pediatric populations in a formulation without a vasoconstrictor. Pharmacologically, these are the 2 groups most susceptible to side effects and toxicity, thus mepivacaine is commonly indicated. Most often the decision to use mepivacaine is based on its vasoconstrictor effect or lack thereof (depending on the formulation). However, the pharmacokinetics of mepivacaine are not well understood or assumed to be similar to that of other local anesthetics. It is important to understand the unique pharmacologic characteristics of mepivacaine in order to minimize the potential for inadvertent toxicity.

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Currently used local anesthetics and their formulations each have individual characteristics that allow them to claim clinically useful niches to validate their continued market presence. While the introduction of articaine—in 1982 in Canada and in 2000 in the US—has gained much of the market share in North America, lidocaine and mepivacaine have remained commonly used agents.[1] In Canada, where articaine is the most used agent, a survey of general dentists showed that from 1993 to 2007, mepivacaine usage declined but remained the third most used agent.[2] Mepivacaine's niche has been considered by many dentists to be the "safer" lidocaine alternative, to be used in elderly or cardiovascular disease patients, because it not only comes without a vasoconstrictor, but it is available with levonordefrin, which is assumed to have less vasopressor potency and 25% of the direct beta effects on the heart.[3] It is now known, however, that levonordefrin acts similarly to norepinephrine, as it elevates not only systolic blood pressure, but diastolic and mean arterial pressures as well. Used at 5 times the concentration of epinephrine, it possesses relatively the same or more potential for cardiac stimulation, especially elevations in blood pressure.[4] Mepivacaine is also commonly used—in a formulation without levonordefrin—for children, as it is available without a vasoconstrictor for shorter postoperative duration, whereas articaine and lidocaine have longer durations and must have vasoconstrictors for their efficacy. Many general and pediatric practices use mepivacaine formulations exclusively with these characterizations in mind. Whereas the 2 different vasoconstrictors used in local anesthetics bear many similarities to each other with similar clinical limitations, it is worthwhile to look at mepivacaine by itself.

Rather than being a lidocaine substitute, mepivacaine possesses distinct pharmacokinetic characteristics of its own that are important for the clinician to understand. This article reviews the unique pharmacology of mepivacaine and the potential clinical implications involved with its use.

History and chemistry: pipecholyl xylidines

In the years immediately after the development of the xylidine derivative, lidocaine, there followed a series of chemical syntheses based upon the same successful 2,6-xylidine-amide structure, with emphasis on increasing the duration of action. In 1957, af Ekenstam et al synthesized (along with many other structures with anesthetic properties) mepivacaine and bupivacaine, both very similar in structure and with longer durations than lidocaine.[5] Each preserved the 2,6-xylidine group on the aromatic ring, but each had a shortened intermediate chain, and replaced the terminal tertiary amine with a less basic methyl-piperidine ring. Homologous local anesthetics that share the mepivacaine structure, such as bupivacaine and ropivacaine, are referred to as *pipecholyl xylidines* due to the presence of this pipecholyl acid moiety (Fig. 1). Bupivacaine differs from mepivacaine only by substitution of the methyl group on the piperidine ring by a

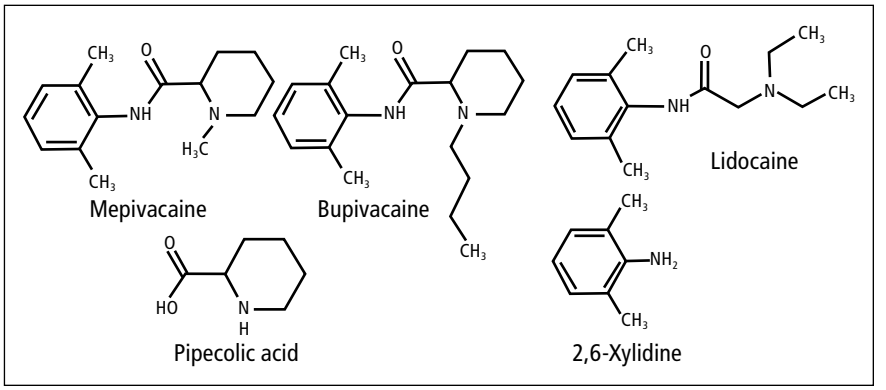

Fig. 1. Pipecholyl xylidines compared to lidocaine.

Table. Physicochemical properties and relative in vitro conduction blocking potency of the intermediate-acting amide local anesthetics.[15]

Anesthetic	MW base (g/mol)	pKa (36°C)	Hydrophobicity[a] (mol/L)	Lipid solubility (distribution coefficient $Q_{7.4}$)[b]	Relative conduction blocking potency (in vitro, isolated nerve fibers)
Lidocaine	234	7.8	304 (366)	43 (110)	2.0
Prilocaine	220	8.0	129	25	1.8
Mepivacaine	246	7.7	90 (130)	21 (42)	1.5

[a]Octanol/buffer partition coefficients for unprotonated species only: 25°C. Values in parentheses: 36°C.

[b]$Q_{7.4}$ = (total drug/ml octanol)/(total drug/ml buffer) at pH 7.4. Includes ionized and unionized partition coefficients.

Margins of error not shown.

more lipid soluble butyl (C_4H_9) group. It was not until the late 1970s, however, that bupivacaine was approved for clinical use as a nerve blocker in dentistry.[6-8]

Mepivacaine, being less toxic than bupivacaine, was immediately evaluated for clinical use in various types of regional and dental anesthesia and was identified as having a faster onset and longer duration than lidocaine without a vasoconstrictor.[9-12] Approved by the FDA in 1960, it was subsequently studied in many dental trials, where it rapidly established its dual utility in dentistry when used in a formulation without a vasoconstrictor or in a formulation with 1:20,000 levonordefrin (Neo-Cobefrin, Novocol Pharmaceutical of Canada, Inc.) which markedly increased its duration.[13,14]

Lipid solubility and potency

Mepivacaine, lidocaine, and prilocaine are considered to be "intermediate" in terms of their lipid solubility and anesthetic potencies (Table).[15] The correlation of lipid solubility, and the ability to penetrate membranes with increased affinity for hydrophobic binding sites are the main determinants of anesthetic potency.[16] The lipid solubility and hydrophobicity (tendency to be readily soluble in nonpolar solvents but only sparingly in water) is lowest in mepivacaine compared to the other local anesthetics.

Hydrophobicity correlates with in vitro methods that measure conduction blocking (anesthetic) potency on isolated nerve fibers, such as rabbit vagus and sciatic. These methods showed mepivacaine to have a correspondingly lower anesthetic potency than lidocaine or prilocaine under in vitro conditions.[17] However, these measurements were derived under controlled conditions and do not directly reflect in vivo anesthetic potency. They also do not correctly reflect the clinical end-points of mepivacaine use, where success is measured by an adequate conduction block, and the physiologic variables that affect tissue concentration, diffusion, and ionization are more complex. Animal studies with live nerve blocks have shown the potency of mepivacaine to be equivalent or greater, with a faster onset time, and longer duration of anesthesia than lidocaine and prilocaine.[18] Mepivacaine, lidocaine, and prilocaine have all been shown in numerous clinical studies to have similar intermediate anesthetic efficacy, regardless of differences in their lipid solubility.[19-21]

Effectiveness in infected tissues

The ionization constant, or pKa, of mepivacaine is also the lowest of the intermediate agents (articaine and lidocaine have the same pKa). At 36°C, the pKa's of the intermediate-acting agents are all close enough to make onset times roughly equivalent under normal physiologic conditions.[22] Estimates of the effects of acidic conditions such as those found in infected tissue—using quantitative structure-activity relationship modeling (QSAR) calculations—have shown the relative anesthetic potency of lidocaine drops 72.8% from a normalized 100 (at pH 7.4) to 27.2 (at pH 6.5).[22] In comparison, the relative anesthestic potency of prilocaine decreases by 66.5%, and mepivacaine decreases by 64.7%. Theoretically, mepivacaine would lose less potency when injected into areas of reduced pH and this may be considered an advantage. However, realistically, this is a rare occurrence if a regional block is available.

Hypersensitivity and cross reactivity

For patients who are known to be allergic to sulphites, the availability of mepivacaine without vasoconstrictor and sodium metabisulphite is a highly valuable clinical asset, since articaine and lidocaine must have vasoconstrictors added for their efficacy. Allergy to mepivacaine itself, as well as any of the amide local anesthetics is very rare, but does occur.[23] Most case reports of mepivacaine allergic reactions prior to the mid-1980s were related to the methylparaben used as antimicrobials in the cartridges; these were subsequently removed by FDA mandate.[24]

A large 2009 study that analyzed the French Pharmacovigilance database over a 12-year period (1995-2006) found 16 cases of allergic reactions to amide local anesthetics, of which 11 were immediate Type I reactions occurring within 1 hour with severe symptoms.[25] Of the Type 1 reactions, 6 were due to lidocaine, 2 to mepivacaine, 2 to articaine, and 1 to bupivacaine. Of the other 5 reactions, which were delayed-type skin reactions, 4 were due to lidocaine and 1 to mepivicaine.[25]

Cross-reactivity among the amides, previously thought to be rare, was found in 6 cases (38%) and all were between lidocaine and mepivacaine. In patients with a true lidocaine or mepivacaine allergy, neither lidocaine nor mepivacaine should be used as a substitute due to very possible cross-allergenicity.[25]

A 2006 report in Spain published a similar case of a patient with confirmed lidocaine and mepivacaine allergies who was not allergic to bupivacaine, despite its structural similarity.[26] No cross-allergenicity to articaine was found in either study. The findings in these and other studies suggest that articaine may have rare cross-allergenicity with other members of the amide class, including delayed-type reactions.[27,28]

Systemic absorption

At therapeutic concentrations, mepivacaine has an intrinsic vasodilating activity intermediate between lidocaine and prilocaine.[29] In various arterial blood flow studies, lidocaine markedly increased blood flow whereas mepivacaine and prilocaine tended to either maintain or decrease peripheral blood flow.[30-32] This would appear to be an additional safety factor, by keeping systemic levels low. However, in maxillary infiltration studies conducted by Goebel et al, serum levels of the mepivacaine formulation without vasoconstrictors were always higher and more persistent than lidocaine.[33,34] Complimentary studies by Goebel et al comparing lidocaine and mepivacaine with their respective vasoconstrictors, epinephrine and levonordefrin, also showed that mepivacaine produced significantly higher and more variable serum levels than lidocaine at all time intervals.[34,35] Medical studies comparing lidocaine and mepivacaine in regional blocks also indicate that mepivacaine, even in the vasoconstrictor formulation, produce higher and more sustained systemic blood levels on the order of 35% to 38%.[36,37]

The studies by Goebel et al showed that mepivacaine displays mean peak levels at about 30 minutes, with or without a vasoconstrictor, while lidocaine with epinephrine produces peak levels earlier, at approximately 15 minutes.[33-35] Since the duration of mepivacaine without a vasoconstrictor is about 30 minutes, reinjection at that time occurs at peaking serum levels. In addition, at all later time intervals, differences in mepivacaine serum levels are not significantly different whether or not vasoconstrictor is used. Serum levels at time intervals beyond 30 minutes in maxillary infiltration studies are essentially the same regardless of whether or not the vasoconstrictor formulation was used.[38] The mean peak (Cmax) percentage difference between plain 2% mepivacaine and 2% mepivacaine with levonordefrin was only 8%, while the peak difference between lidocaine with and without epinephrine was 29%.[32] Systemic levels of mepivacaine decrease less when compared to lidocaine in the presence of a vasoconstrictor. A formulation with 3% mepivacaine (no vasoconstrictor) produces the highest systemic levels of local anesthetic, which are 1.5 times higher than 2% mepivacaine with levonordefrin, and thus has the most potential for chronic accumulation.[35]

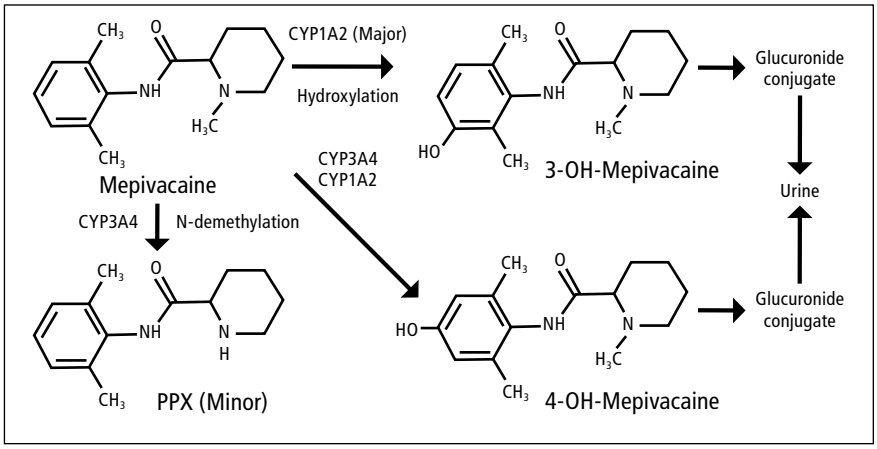

Fig. 2. Mepivacaine metabolism.

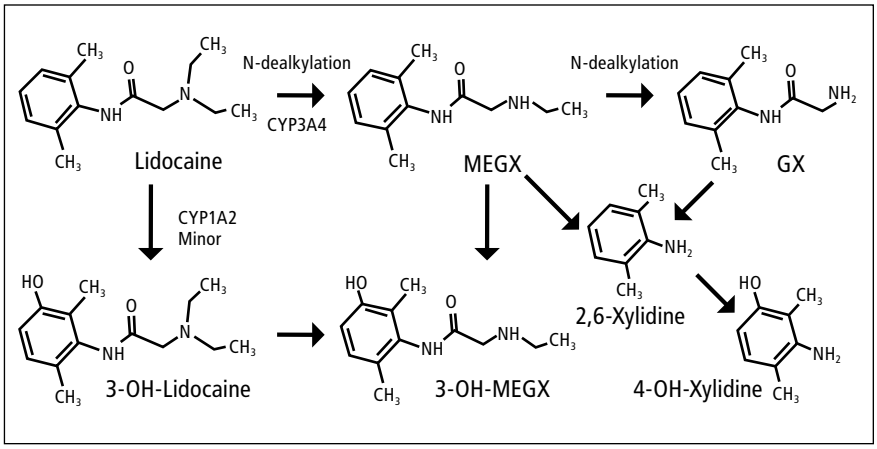

Fig. 3. Lidocaine metabolism.

Metabolism and disposition compared to other local anesthetics

Mepivacaine, like the other amide local anesthetics, undergoes extensive hepatic biotransformation with <5% urinary excretion of the unchanged drug. Metabolism is primarily through hydroxylation of the parent compound to inactive 3-OH-mepivacaine and 4-OH-mepivacaine by CYP1A2 (Fig. 2). CYP1A2 is constitutive, and CYP1A1 is the inducible isoform which also participates in metabolism. CYP3A also contributes to the formation of 4-OH-mepivacaine.[39] All these hydroxyl (OH) metabolites are excreted as glucuronide conjugates and comprise >50% of the total mepivacaine dose. Urinary sampling shows the 3-OH-mepivacaine to be the major metabolite earlier in the process, with the 4-hydroxy compound produced later in smaller amounts, indicating the predominance of CYP1A metabolism.[40] Demethylation by CYP3A produces pipecoloxylidide, a minor metabolite accounting for about 1% of the urinary output.

Lidocaine metabolism involves similiar enzymatic activities, but CYP3A4 plays the larger role in dealkylation to the major metabolite monoethylglycinexylidide (MEGX), an active metabolite that still retains central nervous system

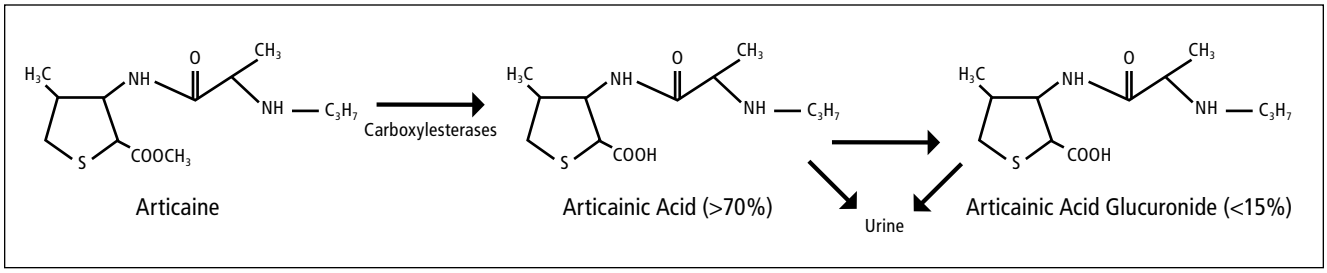

Fig. 4. Articaine metabolism.

(CNS) toxicity. CYP1A contributes to forming MEGX in addition to generating 3-OH-lidocaine which is converted to 3-OH-MEGX.[41] These are all eventually eliminated as glucuronide conjugates. MEGX is metabolized to inactive glycinexylidide (GX) and excreted for the most part renally.[42] The remaining MEGX and GX is further broken down to 2,6-xylidine metabolites when the xylidine ring is removed by hydrolytic reactions involving hepatic carboxylesterases or amidases (Fig. 3).[43]

Prilocaine metabolism occurs in the liver and kidney by carboxylesterases and CYP3A4 that generate o-toluidine. CYP2E1 produces hydroxylated toluidine metabolites that represent more than 40% of the urinary metabolites of prilocaine. O-toluidine and its hydroxylated variants can oxidize hemoglobin to methemoglobin, a critical dose-limiting restriction for prilocaine use.[44] Drug interactions with prilocaine mainly involve inducers of CYP3A4 (carbamazepine, phenytoin, rifampin), or other drugs that contribute to methemoglobinemia, such as benzocaine, nitrates, or acetaminophen.[45]

Articaine has the simplest and most rapid metabolism of the amides due to its carboxyl group ester linkage. Articaine is metabolized rapidly into articainic acid by plasma carboxylesterases with a plasma half-life of 20 minutes.[46] Forty percent to 70% is excreted as articainic acid, and 4% to 15% as articainic acid glucuoronide (Fig. 4). Almost all major P450 cytochromes participate in the remaining metabolism of articaine, but only 10% of the total dose is metabolized by cytochromes, making articaine relatively resistant to pharmacokinetic drug interactions.[47]

Lidocaine clearance is reduced metabolically by inhibitors of CYP3A4, such as cimetidine, erythromycin, and azole antifungals. Antidepressants and benzodiazepines that heavily utilize CYP3A4 have also been associated with serious lidocaine toxicities, including sertraline (Zoloft), escitalopram (Lexapro), desipramine (Norpramin), and flurazepam (Dalmane).[48] Because of the high hepatic extraction ratio of lidocaine, drugs that reduce hepatic blood flow also reduce its metabolism significantly. Beta-blockers that decrease cardiac output, especially propranolol, decrease lidocaine elimination by decreasing hepatic blood flow.[49] Propranolol also prolongs mepivacaine blood levels, but mepivacaine is not as dependent on hepatic blood flow since it has a lower hepatic clearance (extraction ratio) of 0.51 compared to 0.72 for lidocaine.[50-52]

Pharmacokinetic drug interactions with mepivacaine are more likely to involve inhibitors of CYP1A2. Potent inhibition of CYP1A2 occurs with selective serotonin receptor inhibitors fluvoxamine (Luvox) and fluoxetine (Prozac) and moderate inhibition with paroxetine (Paxil) and sertraline (Zoloft).[52] Other strong inhibitors of CYP1A2 and drugs known to interfere with mepivacaine metabolism include caffeine, grapefruit juice, fluoroquinolone antibiotics, and verapamil (Calan), mexiletine (Mexitil), and zileuton (Zyflo).[53] Lastly, significant hepatic CYP1A2 levels are not present in infancy (<1 year), and may not be fully functional before the age of 3 years, which prolongs metabolism of mepivacaine in infancy, whereas fetal CYP3A7 and immature CYP3A are present at birth.[54,55]

The main difference in pharmacokinetic safety levels between local anesthetics is the *total clearance* rate (Cl_{tot}). Local anesthetic duration of activity is determined by vascular redistribution from the local site and is only indirectly and weakly correlated with final elimination of the drug from the body. Total clearance (expressed in volume/time) is the removal of a drug from a volume of plasma in a given unit of time as the sum of all clearances by all the various elimination mechanisms, such as renal and hepatic. Of the currently used intermediate-acting agents, mepivacaine has the slowest total clearance. In comparison, the total body clearance of lidocaine is 3 times higher than that of mepivacaine (0.95-1.1 l/min vs 0.45 l/min).[36] Medical studies examining systemic levels of mepivacaine after caudal or regional blocks have clinically demonstrated the drug's relatively long persistence.[56,57] The typically slower total clearance of mepivacaine is a factor that can lead to potential accumulation in cases of repetitive dosing over time or excessive doses, especially when children are involved.[58-60] In neonates, the lidocaine total plasma clearance normalized to body weight is not significantly different than adults because more lidocaine is excreted renally and unchanged.[61] The neonatal capacity for aromatic hydroxylation, however, is very limited, thus the total plasma clearance of mepivacaine is <50% the adult clearance rate, coupled with a hepatic clearance that is approximately 25% that of adults.[62] Concomittant drug interaction from CYP1A inhibition would also contribute to prolonged or toxic blood levels. Prilocaine has a notably fast total clearance—almost 2.5 times that of lidocaine—which not only indicates high hepatic extraction but also extrahepatic metabolism.[63,64] This hydrolysis of the amide bond occurs very efficiently, considering only 5% of prilocaine is excreted

unchanged. Articaine has the fastest total body clearance of all amide local anesthetics, ranging from 3.9 l/min for intraoral injections to 8.9 l/min for IV injections.[46]

In dentistry, local anesthetic toxicity occurs more frequently in children, most often with mepivacaine.[65] Plain mepivacaine is favored and useful in pediatric dentistry for its shorter duration of activity, but plain mepivicaine leads to higher systemic levels which are subject to a slow total clearance rate. Even with levonordefrin, serum levels are not decreased as they are with lidocaine and a vasoconstrictor. Mepivacaine—especially 3% mepivacaine without a vasoconstrictor—has been implicated in most reported fatalities due to excessive dosing.[66,67] Factors that contribute include practitioners not understanding fully the implications of injecting additional anesthetic in low body weight children, basing the dosage on the number of carpules rather than the patient's weight, and in many instances not understanding the synergistic effects of concommitant opioids or CNS depressants in pediatric sedation protocols. For example, opioids such as meperidine can decrease convulsant thresholds by producing respiratory acidosis and elevating arterial carbon dioxide, which increase the CNS toxicity of local anesthetics.[68] All these factors can contribute to toxicity due to mepivacaine's inherent characteristic of maintaining prolonged and relatively high serum levels even when used with a vasoconstrictor.

Conclusion

Mepivacaine is an efficacious and useful intermediate-acting local anesthetic for use in dentistry as it can be used when required and without a vasoconstrictor. The presence of a vasoconstrictor increases the duration of action but has little effect on systemic blood levels. The slower total clearance of mepivacaine makes it more susceptible to the various mechanisms that can lead to chronic toxicity, such as the lack of fully functional enzymes in infants, inadvertant excessive dosing of 3% mepivacaine, or repeated dosing in conjunction with long appointments, renal failure, or drug inhibition of CYP1A (in elderly patients). Children <5 years of age are most susceptable to overdose and are also often given sedation that can increase toxicity. Weight-based dosing in children should be used as a matter of course, and the maximum recommended dose should never be exceeded, especially when using 3% mepivacaine. It has been recommended by many other authors that, since the 3% formulation is potentially 1.5 times more toxic than the 2% formulation, mepivacaine use in children be restricted to smaller volumes, and that its use be limited to supraperiosteal injections whenever possible.[60]

Author information

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References

1. Pogrel MA. Permanent nerve damage from inferior alveolar nerve blocks—an update to include articaine. *J Calif Dent Assoc.* 2007;35(4):271-273.
2. Gaffen AS, Haas DA. Survey of local anesthetic use by Ontario dentists. *J Can Dent Assoc.* 2009;75(9):649.
3. James P. Cassidy, James C. Phero, William H. Grau. Epinephrine: systemic effects and varying concentrations in local anesthesia. *Anesth Prog.* 1986;33(6):289-297.
4. Robertson VJ, Taylor SE, Gage TW. Quantitative and qualitative analysis of the pressor effects of levonordefrin. *J Cardiovasc Pharmacol.* 1984;6(5):929-935.
5. af Ekenstam, Egner B, Pettersson G. N-alkyl pyrrolidine and N-alkylpiperidine carboxylic acid amides. *Acta Chem Scand.* 1957;11:1183-1190.
6. Henn F, Brattsand R. Some pharmacological and toxicological properties of a new long-acting local analgesic, Lac-43 (marcaine), in comparison with mepivacaine and tetracaine. *Acta Anaesthiol Scand Suppl.* 1966;21:9-30.
7. Lund P, Cwik J, Vallesteros F. Bupivacaine—a new long-acting local anesthetic agent. A preliminary clinical and laboratory report. *Anesth Analg.* 1970;49(1):103-114.
8. Laskin JL, Wallace WR, DeLeo B. Use of bupivacaine hydrochloride in oral surgery - a clinical study. *J Oral Surg.* 1977;35(1):25-29.
9. Dhuner KG, Egner B, Ekenstam BA, Oljelundo O, Ulfendahl LR. Trials with carbocaine; a new local anaesthetic drug. *Brit J Anaesth.* 1956;28(11):503-506.
10. Dhuner K, Oljelund O, Aagesen G. Carbocain-d, 1-N-methyl-pipecolic acid 2, 6-dimethylanilide; a new local anesthetic agent. *Acta Chir Scand.* 1957;112(5):350-358.
11. Luduena FP, Hoppe JO, Coulston F, Drobeck HP. The pharmacology and toxicology of mepivacaine, a new local anesthetic. *Toxicol Appl Pharmacol.* 1960;2:295-315.
12. Goulding CE Jr, Ozdil T, Powell WF. Carbocaine: clinical appraisal of 132 cases. *Anesth Analg.* 1961;40:523-527.
13. Ross N, Dobbs EC. A preliminary study on carbocaine. *J Am Dent Soc Anesthesiol.* 1960;7(9):4-5.
14. Lock F, Vernino D, Sadove M. Mepivacaine HCl (carbocaine): a preliminary clinical study. *J Oral Surg Anesth Hosp Dent Serv.* 1961;19:16-20.
15. Stritchartz GR, Sanchez V, Arthur GR, Chafetz R, Martin D. Fundamental properties of local anesthetics. II. Measured octanol buffer partition coefficients and pKa values of clinically used drugs. *Anesth Analg.* 1990;71(2):158-170.
16. Ritchie JM, Greengard P. On the mode of action of local anesthetics. *Annu Rev Pharmacol.* 1966;6:405-430.
17. Wildsmith JAW, Gissen AJ, Gregus J, Covino BJ. Differential nerve blocking activity of amino-ester local anaesthetics. *Br J Anaesth.* 1985;57(6):612-620.
18. Pateromichelakis S, Prokopiou AA. Local anaesthesia efficacy: discrepancies between in vitro and in vivo studies. *Acta Anaesthesiol Scand.* 1988;32(8):672-675.
19. Petersen JK, Luck H, Kristensen F, Mikkelsen L. A comparison of four commonly used local analgesics. *Int J Oral Surg.* 1977;6(1):51-59.
20. Hinkley SA, Reader A, Beck M, Meyers WJ. An evaluation of 4% prilocaine with 1:200,000 epinephrine and 2% mepivacaine with 1:20,000 levonordefrin compared with 2% lidocaine with:100,000 epinephrine for inferior alveolar nerve block. *Anesth Prog.* 1991;38(3):84-89.
21. Cohen HP, Cha BY, Spangberg LS. Endodontic anesthesia in mandibular molars: a clinical study. *J Endod.* 1993;19(7):370-373.
22. Canos-Rius N, Martin-Biosca Y, Sagrado S, Villanueva-Camanas RM, Medina-Hernandez MJ. Estimation of the effect of the acidosis and alkalosis on the anesthetic potency of local anesthetics by biopartitioning micellar chromatography and micellar electrokinetic chromatography. *Eur J Med Chem.* 2005;40(2):215-223.
23. Speca SJ, Boynes SG, Cuddy MA. Allergic reactions to local anesthetic formulations. *Dent Clin North Am.* 2010;54(4):655-664.
24. Luebke NH, Walker JA. Discussion of sensitivity to preservatives in anesthetics. *J Am Dent Assoc.* 1978;97(4):656-657.
25. Fuzier R, Lapeyre-Mestre M, Mertes PM, et al. Immediate-and delayed-type allergic reactions to amide local anesthetics: clinical features and skin testing. *Pharmacoepidemiol Drug Saf.* 2009;18(7):595-601.
26. Gonzalez-Delgado P, Anton R, Soriano V, Zapater P, Niveiro E. Cross-reactivity among amide-type local anesthetics in a case of allergy to mepivacaine. *J Investig Allergol Clin Immunol.* 2006;16(5):311-313.
27. Bircher AJ, Messmer SL, Surber C, Rufli T. Delayed-type hypersensitivity to subcutaneous lidocaine with tolerance to articaine: confirmation by in vivo and in vitro tests. *Contact Dermatitis.* 1996;34(6):387-389.
28. Duque S, Fernandez L. Delayed-type hypersensitivity to amide local anesthetics. *Allergol Immunopathol (Madr).* 2004;32(4):233-234.
29. Blair MR. Cardiovascular pharmacology of local anaesthetics. *Brit J Anaesth.* 1975;47(Suppl):247-252.
30. Lindorf HH. Investigation of the vascular effect of newer local anesthetics and vasoconstrictors. *Oral Surg Oral Med Oral Pathol.* 1979;48(4):292-297.
31. Willatts DG, Reynolds F. Comparison of the vasoactivity of amide and ester local anaesthetics. An intradermal study. *Br J Anaesth.* 1985;57(10):1006-1011.
32. Lofstrom JB. 1991 Labat Lecture. The effect of local anesthetics on the peripheral vasculature. *Reg Anesth.* 1992;17(1):1-11.
33. Goebel WM, Allen G, Randall F. The effect of commercial vasoconstrictor preparations on the circulating venous serum level of mepivacaine and lidocaine. *J Oral Med.* 1980;35(4):91-96.

34. Goebel W, Allen G, Randall F. Circulating serum levels of mepivacaine after dental injection. *Anesth Prog.* 1978;25(2):52-56.
35. Goebel WM, Allen G, Randall F. Comparative circulating serum levels of mepivacaine with levo-nordefrin and lidocaine with epinephrien [sic]. *Anesth Prog.* 1979;26(4):93-97.
36. Simon MAM, Vree TB, Gielen MJM, Booij LHJ, Lagerwerf AJ. Similar motor block effects and disposition kinetics between lidocaine and (±)mepivacaine in patients undergoing axillary brachial plexus block during day case surgery. *The Scientific World.* 2002;2: 1306-1319.
37. Takasaki M. Blood concentrations of lidocaine, mepivacaine and bupivacaine during caudal analgesia in children. *Acta Anaesthesiol Scand.* 1984;28(2):211-214.
38. Goebel WM, Allen G, Randall F. The effect of commercial vasoconstrictor preparations on the circulating venous serum level of mepivacaine and lidocaine. *J Oral Med.* 1980;35(4):91-96.
39. Oda Y, Furuichi K, Tanaka K, et al. Metabolism of a new local anesthetic, ropivacaine, by human hepatic cytochrome P450. *Anesthesiology.* 1995;82(1):214-220.
40. Meffin PJ, Thomas J. The relative rates of formation of the phenolic metabolites of mepivacaine in man. *Xenobiotica.* 1973;3(10):625-632.
41. Orlando R, Piccoli P, De Martin S, Padrini R, Floreani M, Palatini P. Cytochrome P450 1A2 is a major determinant of lidocaine metabolism in vivo: effects of liver function. *Clin Pharmacol Ther.* 2004;75(1):80-88.
42. Imaoka S, Enomoto K, Oda Y, et al. Lidocaine metabolism by human cytochrome P-450s purified from hepatic microsomes: comparison of those with rat hepatic cytochrome P-450s. *J Pharmacol Exper Ther.* 1990;255(3):1385-1391.
43. Alexson SEH, Diczfalusy M, Halldin M, Swedmark S. Involvement of liver carboxylesterases in the in vitro metabolism of lidocaine. *Drug Metab Dispos.* 2002; 30(6):643-647.
44. Higuchi R, Fukami T, Nakajima M, Yokoi T. Prilocaine- and lidocaine-induced methemoglobinemia is caused by human carboxylesterase-, CYP2E1-, and CYP3A4-mediated metabolic activation. *Drug Metab Dispos.* 2013;41(6):1220-1230.
45. Centers for Diease Control and Prevention. Prilocaine-induced methemoglobinemia—Wisconsin, 1993. *MMWR Morb Mortal Wkly Rep.* 1994;43(35):655-657.
46. Oertel R, Rahn R, Kirch W. Clinical pharmacokinetics of articaine. *Clin Pharmacokinet.* 1997;33(6):417-425.
47. US Food and Drug Administration Center for Drug Evaluation and Research. *Approval Package for Application Number 20-971.* Available at: http://www.accessdata.fda.gov/drugsatfda_docs/nda/2000/020971_septocaine_biopharmr.pdf. Accessed August 13, 2014.
48. Klein JA, Kassarjdian N. Lidocaine toxicity with tumescent liposuction. A case report of probable drug interactions. *Dermatol Surg.* 1997;23(12):1169-1174.
49. Stenson RE, Constantino RT, Harrison DC. Interrelationships of hepatic blood flow, cardiac output, and blood levels of lidocaine in man. *Circulation.* 1971; 43(2):205-211.
50. Popescu SM, Nechifor M, Baniceru M, Croitoru O, Popescu F. Effect of propranolol on mepivacaine serum concentrations in dental practice. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2008;105(4):e19-e23.
51. Dillane D, Finucane BT. Local anesthetic systemic toxicity. *Can J Anaesth.* 2010;57(4):368-380.
52. von Moltke LL, Greenblatt DJ, Duan SX, et al. Phenacetin O-deethylation by human liver microsomes in vitro: inhibition by chemical probes, SSRI antidepressants, nefazodone and venlafaxine. *Psychopharmacology (Berl).* 1996;128(4):398-407.
53. Faber MS, Jetter A, Fuhr U. Assessment of CYP1A2 activity in clinical practice: why, how, and when? *Basic Clin Pharmacol Toxicol.* 2005;97(3):125-134.
54. Tateishi T, Nakura H, Asoh M, et al. A comparison of hepatic cytochrome p450 protein expression between infancy and postinfancy. *Life Sci.* 1997;61(26):2567-2574.
55. Tanaka E. In vivo age-related changes in hepatic drug-oxidizing capacity in humans. *J Clin Pharm Ther.* 1998; 23(4):247-255.
56. Moore DC, Bridenbaugh LD, Bagdi PA, Bridenbaugh PO. Accumulation of mepivacaine hydrochloride during caudal block. *Anesthesiology.* 1968;29(3):585-588.
57. Tucker GT, Moore DC, Bridenbaugh PO, Bridenbaugh LD, Thompson GE. Systemic absorption of mepivacaine in commonly used regional block procedures. *Anesthesiology.* 1972;37(3):277-287.
58. Moore DC, Bridenbaugh LD, Bagdi PA, Bridenbaugh PO. Accumulation of mepivacaine hydrochloride during caudal block. *Anesthesiology.* 1968;29(3):585-588.
59. Morishima HO, Daniel SS, Finster M, Poppers PJ, James LS. Transmission of mepivacaine hydrochloride (carbocaine) across the human placenta. *Anesthesiology.* 1966;27(2):147-154.
60. Chin KL, Yagiela JA, Quinn CL, Henderson KR, Duperon DF. Serum mepivacaine concentrations after intraoral injection in young children. *J Calif Dent Assoc.* 2003; 31(10):757-764.
61. Mihaly G, Moore RG, Thomas J, Triggs EJ, Thomas D, Shanks C. The pharmacokinetics and metabolism of the anilide local anaesthetics in neonates. I. Lignocaine. *Eur J Clin Pharmacol.* 1978;13(2):143-152.
62. Moore RG, Thomas J, Triggs EJ, Thomas DB, Burnard ED, Shanks CA. The pharmacokinetics and metabolism of the anilide local anaesthetics in neonates. III. Mepivacaine. *Eur J Clin Pharmacol.* 1978;14(3):203-212.
63. Yagiela JA. Local anesthetics. *Anesth Prog.* 1991; 38(4-5):128-141.
64. Arthur GR, Scott DH, Boyes RN, Scott DB. Pharmacokinetic and clinical pharmacological studies with mepivacaine and prilocaine. *Br J Anaesth.* 1979;51(6): 481-485.
65. Moore PA, Hersh EV. Local anesthetics: pharmacology and toxicity. *Dent Clin North Am.* 2010;54(4):587-599.
66. Hersh EV, Helpin ML, Evans OB. Local anesthetic mortality: report of case. *ASDC J Dent Child.* 1991;58(6): 489-491.
67. Moore PA. Preventing local anesthesia toxicity. *J Am Dent Assoc.* 1992;123(9):60-64.
68. Moore PA, Goodson JM. Risk appraisal of narcotic sedation for children. *Anesth Prog.* 1985;32(4):129-139.

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Exercise No. 360 Anesthesia and Pain Control Subject Code 132

The 15 questions for this exercise are based on the article, *Mepivacaine: a closer look at its properties and current utility,* on pages 70-75. This exercise was developed by Anthony S. Carroccia, DDS, MAGD, ABGD, in association with the *General Dentistry* Self-Instruction committee.

Reading the article and successfully completing this exercise will enable you to:
- identify mepivacaine's properties;
- recognize potential clinical implications; and
- differentiate between mepivacaine and other local anesthetics.

1. Mepivacaine was first synthesized in which year?
   A. 1957
   B. 1982
   C. 1993
   D. 2007

2. All of the following anesthetics are classified as intermediate for lipid solubility and anesthetic potencies. Which is the exception?
   A. mepivacaine
   B. articaine
   C. lidocaine
   D. prilocaine

3. All of the following local anesthetics are considered pipecholyl xylidines except one. Which is the exception?
   A. mepivacaine
   B. bupivacaine
   C. ropivacaine
   D. lidocaine

4. Bupivacaine differs from mepivacaine by the substitution of a(n)
   A. aromatic ring.
   B. tertiary amine.
   C. ethyl group.
   D. butyl group.

5. Mepivacaine loses _____% of relative anesthetic potency in infected tissues.
   A. 27.2
   B. 64.7
   C. 66.5
   D. 72.8

6. Mepivacaine has the largest increase in the cardiotoxic dose. This is because it is correlated to relative potency.
   A. Both statements are true.
   B. The first statement is true; the second is false.
   C. The first statement is false; the second is true.
   D. Both statements are false.

7. Cross-reactivity among the amide anesthetics, though rare, was found in _____ cases, all between lidocaine and mepivacaine.
   A. 6
   B. 8
   C. 16
   D. 38

8. Thirty years ago, case reports of allergic reactions to mepivacaine were due to which agent?
   A. metabisulphite
   B. methylparaben
   C. vasodilator
   D. vasoconstrictor

9. Which of the following local anesthetics demonstrated a marked increase in arterial blood flow?
   A. cocaine
   B. lidocaine
   C. mepivacaine
   D. prilocaine

10. Which local anesthetic has the most potential for chronic accumulation?
    A. 2% lidocaine
    B. 2% lidocaine with epinephrine
    C. 2% mepivacaine with levonordefrin
    D. 3% mepivacaine

11. Prilocaine interactions with all of the following drugs may contribute to methemoglobinemia except one. Which is the exception?
    A. carbamazepine
    B. glyburide
    C. phenytoin
    D. acetaminophen

12. Inhibition of mepivacaine metabolism can be accomplished by all of the following drugs except one. Which is the exception?
    A. Lithobid
    B. Luvox
    C. Paxil
    D. Prozac

13. All of the following are factors that contribute to pediatric local anesthetic fatalities except one. Which is the exception?
    A. injecting additional anesthetics
    B. number of carpules used
    C. failing to understand synergism of opioids and/or depressants
    D. dosing by weight

14. Among amide anesthetics, mepivacaine has the slowest total body clearance. Articaine has the fastest total body clearance.
    A. Both statements are true.
    B. The first statement is true; the second is false.
    C. The first statement is false; the second is true.
    D. Both statements are false.

15. Which cytochrome has the major role when metabolizing mepivacaine?
    A. CYP1A1
    B. CYP1A2
    C. CYP3A
    D. CYP3A4

Answer form is on the inside back cover. Answers for this exercise must be received by October 31, 2015.

A direct approach for fabrication of a provisional restoration immediately after tooth preparation for custom cast dowel and core

Kunwarjeet Singh, BDS, MDS ▪ Nidhi Gupta, BDS, MDS

When a tooth is fractured at the gingival level due to accidental trauma and there is insufficient coronal tooth structure to retain a crown, a custom cast dowel and core often is necessary, followed by the fabrication of a definitive crown. It is very difficult to fabricate the provisional restoration for a tooth until the custom cast dowel and core are cemented permanently. This article describes a direct procedure for fabricating a provisional restoration (with a prefabricated temporary titanium post and an acrylic resin denture tooth) immediately after the tooth has been prepared for a custom cast dowel and core. This technique produces good clinical results while being less time-consuming than an indirect approach.

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Key words: provisional restoration, temporary titanium post, acrylic resin tooth, custom cast dowel and core

The importance of provisional restorations in fixed prosthodontics has been well-documented in the literature.[1-3] A variety of techniques are available—from a single unit to a complete arch provisional fixed prosthesis—depending on the patient's needs and the clinical situation.[1-3]

One of the greater challenges when restoring a tooth that has been fractured due to trauma is making the provisional restoration esthetically pleasing. A particular challenge for dentists involves a tooth that is fractured at the gingival level that has already been prepared for a custom cast dowel and core, despite the fact there is little intact supragingival tooth structure for crown retention (Fig. 1).[4] Only 1 technique has been described in the literature for making a provisional restoration while the custom cast dowel and core is being fabricated. This technique involves fitting an orthodontic wire into the prepared canal and fabricating a provisional crown with autopolymerizing resin, using the direct technique.[3]

This article describes a simple direct chairside technique for fabricating a provisional restoration by using a temporary titanium post and an acrylic tooth. This should accomplish excellent esthetic results due to the acrylic tooth's well-polished, glazed, labial surface. An additional benefit to this technique is that the dentist can use the temporary titanium post later (after sterilization) for the fabrication of a provisional crown in another patient.

Materials and methods

This technique requires an acrylic lateral incisor tooth and a temporary titanium post; both must be the proper size (Fig. 2). The acrylic tooth (both the lingual fossa and cingulum) must be trimmed from the palatal side in order to resemble a laminate veneer. To maintain the polished glazed appearance, dentists should be careful to avoid trimming the labial surface of the acrylic tooth. The fit and extension of the post should be evaluated to ensure it fits passively into the prepared canal and extends sufficiently into the oral cavity to retain the provisional restoration.

The tooth (including the canal and surrounding soft tissues) should be lubricated by placing petroleum jelly on a small piece of cotton rolled on paper points. Next, the temporary post (with the required oral extension) should be placed in the canal. The autopolymerizing tooth-colored acrylic resin monomer and polymer can be mixed in a dappen dish. The fitting surface

Fig. 1. Lateral view of a fractured left maxillary lateral incisor.

Fig. 2. A temporary titanium post and acrylic tooth.

Fig. 3. Labial view of a provisional restoration with temporary titanium post.

Fig. 4. Palatal view of the provisional restoration.

Fig. 5. A partially seated provisional restoration.

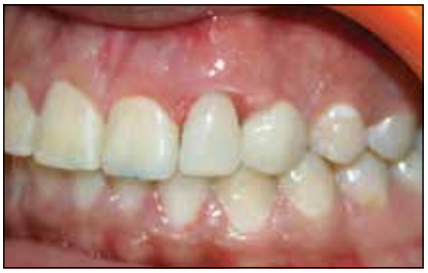

Fig. 6. A provisional restoration after cementation with temporary luting cement.

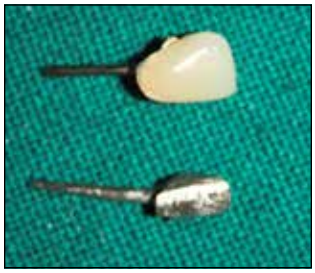

Fig. 7. A provisional restoration after removal.

Fig. 8. A custom cast dowel and core placed using permanent cement.

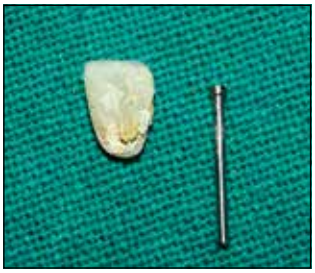

Fig. 9. A temporary titanium post after separation from the acrylic crown.

Fig. 10. A partially seated provisional restoration.

Fig. 11. A provisional restoration after cementation with temporary luting agent.

Fig. 12. A definitive restoration placed.

of the trimmed acrylic tooth should be wetted with the monomer, and some acrylic resin should be placed on the fitting surface of the acrylic tooth. Then the tooth should be placed over the temporary post extending into the oral cavity and held in the proper position while applying acrylic resin from the palatal side with the help of a cement carrier and Hollenbeck carver. Make sure that that the acrylic resin on the palatal and proximal surfaces blends well with the acrylic tooth veneer.

The resin should still be "rubbery" 2-3 minutes after application, at this point the entire assembly should be removed. Polymerization should be monitored carefully. If the resin is allowed to become rigid, it might lock into the undercuts within the post preparation and between adjacent teeth. Removing the assembly at this later point would be both time-consuming and risk the restorability of the tooth.

To hasten the polymerization of the resin, place the assembly in warm water. The temporary post must not be disturbed while the resin is soft. Once the resin becomes hard, it should be finished, polished, and cleaned (Fig. 3 and 4). After evaluation (Fig. 5), the tooth should be cemented with a zinc oxide noneugenol cement. An example of the tooth after cementation is shown in Figure 6.

After the permanent cementation of the custom cast dowel and core (Fig. 7 and 8), the temporary titanium post should be separated carefully from the acrylic crown by using carbide burs (Fig. 9). The same acrylic crown can be used again by trimming it from the palatal side. Stabilize the crown properly on the labial side of the custom cast dowel and core, and adapt the tooth-colored autopolymerizing resin from the palatal and proximal side after applying the petroleum jelly over the core. Next, the provisional crown (Fig. 10) should be finished, polished, and cemented with a temporary cement (Fig. 11). Figure 12 shows a definitive crown with a well-established emergence profile maintained by the provisional restoration.

Discussion

Placing a custom cast dowel and core is 1 of the more common approaches for the functional and esthetic rehabilitation of a traumatized tooth that has been fractured at the gingival level, with inadequate tooth structure for crown rentention. One of the biggest challenges a dentist faces is providing immediate esthetic rehabilitation after making the tooth preparation.

Several laboratory and clinical techniques for fabricating provisional restorations have been described, including indirect, direct, and indirect-direct techniques for both single and multiple unit fixed restorations.[5-7] The indirect technique is used widely in clinical practice; however, this approach requires additional time and laboratory support. As a result, some dentists prefer the direct technique, which involves a less time-consuming procedure for fabricating provisional fixed partial dentures chairside.

Only 1 technique has been described in the literature for fabricating a provisional restoration immediately after a tooth is prepared for custom dowel and core. This technique involves fitting an orthodontic wire into the prepared canal and fabricating the provisional restoration by applying autopolymerizing resin around the wire that is extended into the oral cavity. However, the fabrication of a provisional crown is more time-consuming and offers inferior esthetics compared to the technique described in this article. Based on the authors' experience, fabricating a provisional crown using a temporary titanium post and a prefabricated acrylic denture tooth is less time-consuming with better esthetics, greater patient acceptance, and less irritation of surrounding tissues, while allowing dentists to maintain a proper emergence profile until the definitive restoration is fabricated.

Conclusion

This article describes a direct approach for fabricating a provisional restoration immediately after tooth preparation for a custom cast dowel and core using a temporary titanium post and an acrylic denture tooth. This procedure achieves functional and esthetic provisional restorations with minimal adjustments and optimal esthetics.

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References

1. Federick DR. The provisional fixed partial denture. *J Prosthet Dent.* 1975;34(5):520-526.
2. Hunter RN. Construction of accurate acrylic resin provisional restorations. *J Prosthet Dent.* 1983;50(4):520-521.
3. Rosenstiel SF, Land MF, Fujimoto J. *Contemporary Fixed Prosthodontics.* 4th ed. St. Louis: Mosby; 2000.
4. Shillingburg HT, Hobo S, Whitsett LD, Brackett SE. *Fundamentals of Fixed Prosthodontics.* 3rd ed. Chicago: Quintessence Publishing Co.; 1997:250.
5. Zinner ID, Trachtenberg DI, Miller RD. Provisional restorations in fixed partial prosthodontics. *Dent Clin North Am.* 1989;33(3):355-377.
6. Psichogios PC, Monaco EJ. Expedient direct approach for esthetic and functional provisional restorations. *J Prosthet Dent.* 2003;89(3):319-322.
7. Bennani V. Fabrication of indirect-direct provisional fixed partial denture. *J Prosthet Dent.* 2000;84(3):364-365.

Self-Instruction

Exercise No. 340

November/December 2013, p. 66

1. D	2. C	3. B	4. C
5. B	6. B	7. A	8. B
9. D	10. B	11. C	12. C
13. C	14. C	15. D	

Exercise No. 341

November/December 2013, p. 72

1. A	2. B	3. D	4. A
5. B	6. A	7. C	8. D
9. A	10. C	11. A	12. A
13. C	14. C	15. D	

Exercise No. 342

November/December 2013, p. 79

1. A	2. C	3. B	4. B
5. C	6. B	7. D	8. A
9. D	10. D	11. B	12. A
13. D	14. C	15. C	

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10 A B C D	10 A B C D	10 A B C D
11 A B C D	11 A B C D	11 A B C D
12 A B C D	12 A B C D	12 A B C D
13 A B C D	13 A B C D	13 A B C D
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The efficacy of 2 different doses of dexamethasone to control postoperative swelling, trismus, and pain after third molar extractions

Cicero Newton Lemos Felicio Agostinho, MSc ▪ Vanessa Camila da Silva, PhD ▪ Etevaldo Matos Maia Filho, PhD
Maria Luiza Cruz, PhD ▪ Eider Guimaraes Bastos, PhD

This article addresses the effect of 2 different concentrations (4 and 12 mg) of dexamethasone to control pain, swelling, and trismus after third molar surgery. A clinical study was conducted with 27 male and female patients, all presenting with bilaterally displaced mandibular third molars. The treatment protocol required the surgical removal of each tooth in 2 separate operations. The patients were given a preoperative dose of dexamethasone—4 mg for one surgery, 12 mg for the other. The choice of which side would be operated on first and which dose of dexamethasone would be taken was performed randomly, under double-blind conditions. The trismus was assessed by measuring the interincisal distance. Pain intensity was measured both by the amount of painkillers (acetaminophen 750 mg) taken postsurgery and by the Visual Analogue Pain Scale. Data were collected 1 hour preoperative, then at 24 and 48 hours postoperative. A statistical analysis (student's t, Wilcoxon, and Friedman tests) of the results showed no significant differences ($\alpha = 0.05$) between the analyzed variables for the 2 different doses of dexamethasone (4 and 12 mg).

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The pharmacological control of inflammatory processes after third molar surgeries has been a source of concern for oral and maxillofacial surgeons. This has led to an increased use of corticosteroids, nonsteroidal anti-inflammatories, and other painkillers to control the most involved sequelae in these surgeries—pain, swelling, and trismus—in order to provide maximum relief postsurgery.[1-4] Perhaps the greatest factor in the postsurgical sequelae is the inflammatory process initiated by the surgical act.[5,6]

A great source of the fear and aversion experienced by some patients concerning oral surgery is the postoperative pain associated with these procedures. Thus any preoperative technique that is able to prevent or decrease pain postsurgery is strongly recommended. Numerous procedures have been used preoperatively to assure postsurgical comfort, such as the use of corticosteroids, antihistamines, and other painkillers, as well as the application of ice to the affected area.[7]

Some characteristics are peculiar to the inflammatory pattern, such as swelling, leucocytosis, an increase in vascular permeability, and hyperalgesia with its subsequent alteration of blood flow. The association of these inflammatory factors culminates with plasma extravasations, an increase in the chemical mediators of inflammatory response, and increased peripheral nervous pressure; all of which promote pain.[7] It is important to stress that this sequelae is not observed immediately after the surgical procedure. It is gradually triggered, hitting its peak at approximately 48 hours postsurgery.[8]

Glucocorticosteroids are known as the most powerful anti-inflammatory available for clinical practice and are classified as pleiotropic hormones. They are responsible for the prevention (or suppression) of the inflammatory process and other immunologically mediated processes. According to Boumpas et al, these hormones must be administrated within certain pharmacological patterns in order to achieve ideal dosage.[9] According to Kingery et al, the role corticosteroids play on the postoperative process is still controversial.[10] This is because it is not well known whether such compounds act more on decreasing postsurgery swelling or actually function as a painkiller.[10]

Glucocorticosteroids, such as dexamethasone, stop the inflammatory cascade at its initial steps.[8] The administration of these steroids before third molar surgery is commonly done preoperatively in a single dose. However, the authors of this study have not found a protocol in the literature that clearly defines patient selection, optimal surgery time, which corticosteroid to use, and the best administration route for this type of drug.[8]

The present study is a comparative evaluation of the effects of 2 different doses of dexamethasone (4 and 12 mg) on controlling postoperative symptoms of pain after surgical extraction of displaced third molars.

Materials and methods

This study was approved by the Ethics Committee of the Federal University of Maranhao-UFMA, Sao Luis, Brazil (Protocol No. 23115 006060/2009-87). The patients consent included the option to quit the research at any time, with no consequences to their treatment. Thirty-four patients were initially selected. Of these, 1 became pregnant after the first surgery, 2 did not show up at their postoperative appointments, and 4 did not return after the first surgery. The final study group included 27 male and female patients, with an age range of 15 to 41 years.

This study was a randomized, paired, and double-blinded, longitudinal clinical trial. The patients' indicated treatment was for the removal of bilateral misplaced mandibular third molars, all in similar positions, without any manifestation of a local or systemic disorder that could counterindicate the surgery or choice of drug treatment.

Exclusion criteria were any surgeries that lasted >1hour and/or presented any type of complications during and after the operation, patients who used postoperative painkillers other than acetaminophen,

patients whose 2 surgical times varied more than 5 minutes, and patients who did not show for the 2 postoperative evaluations (at 24 and 48 hours).

Surgical procedure

The order in which the selected patients were submitted to the surgical procedure was randomized. For each patient, the left mandibular third molar was extracted first, then the right mandibular third molar was extracted 15 days later. After each extraction, the patients were examined 2 times (at 24 and 48 hours) for pain, swelling, and trismus.

Dexamethasone was administered 1 hour before surgery; the doses were randomized and different for each extraction—for example, if the patient was given 4 mg before the first surgery, he/she would be given 12 mg before the second surgery.

Before the first surgery, certain areas of the patient's left side of the face—mandibular angle, tragus, external eye corner, nose wing, buccal commissure, and soft pogonion—were marked by a dermographic pen. Taking the mandibular angle as a reference and using a silk thread (Ethicon, Inc.), the linear distances from the mandibular angle to the other points were measured. These distances were registered in the patient's clinical file, and used as a reference for swelling. The superior-inferior intercisal distance (ID) was also measured with a paquimeter as a reference for trismus. All preoperative swelling and trismus measurements were done in triplicate and noted on the patient's clinical record.

The presurgical treatment started with an intraoral antiseptic 0.12% chlorhexidine digluconate mouthwash (Periogard, Colgate-Palmolive Company). The inferior alveolar nerve was anesthesized on the lingual and buccal sides of the tooth with 2% mepivacaine and adrenalin, 1:100,000 (Mepiadre 100, DFL Industrio e Comercio). An incision was made with a scalpel to expose the surgical site and mucoperiosteal detachment, then an ostectomy and odontosection was performed with surgical spherical burs (No. 4, FGM Produtos Odontologicos) and surgical conical burs (No. 702, FGM Produtos Odontologicos). The sites were irrigated with 0.9% sterile saline solution. Bone regularization, alveolar ridge cleaning, and suture with 4-0 silk thread (Ethicon, Inc.) were performed postextraction.

The patients received postsurgical instruction: semiliquid foods for the first 48 hours, bed rest, and avoidance of ice packs so as not to interfere with the postoperative swelling assessments. Each patient was medicated immediately for postsurgical pain with an oral administration of acetaminophen (750 mg) (Cetafrim, Luper Industria Farmaceutica Ltda). Acetaminophen was given to each patient with instructions to take 1 pill every 6 hours *only if pain was present.* Each patient then informed the researcher at the 24- and 48-hour postoperative evaluations how many pills he/she had taken. Pain was also measured using the Visual Analog Pain Scale (VAS) as indicated by the patient within the same 2 intervals.

To assess postoperative swelling, new measurements of the facial points were done at both 24 and 48 hours. The same was done for the trismus assessments. Suture removal was performed at postoperative Day 7. The surgery, data collection, and suture removal were all performed by the same researcher.

To avoid the patients knowing the amount of the administrated dose of dexamethasone, and to maintain the double-blind conditions of the research, both doses of the drug (4 and 12 mg) were manipulated so that all the pills were the same size and color, with no listed specifications.

Postoperative evaluations

Each patient returned for 24- and 48-hour postoperative evaluations. The clinical measurements of maximum interincisal opening and facial contour, for the evaluation of trismus and swelling, respectively, were noted in the patient's file.

The mouth opening and facial points that were measured at 24 and 48 hours postoperative were substracted from the preoperative measurements with the same dexamethasone dosage. To evaluate whether there were differences in trismus and swelling on postoperative Days 1 and 2, the measurements taken at the 48-hour evaluation were substracted from the measurements taken at the 24-hour evaulation. The collected data was analyzed using SPSS Statistics Package (version 17.0, SPSS, Inc.). The tests performed were t-test, Wilcoxon, and Friedman ($\alpha = 0.05$).

Results

The study population consisted of 27 patients: 17 women (63%) and 10 men (37%), ages 15-41 (21.7 ± 6.37). Patients treated with 4 mg of dexamethasone had a mean surgical time to remove the impacted teeth of 24.2 minutes; the patients who received a 12 mg dose had a mean surgical time of 25.6 min.

The most prevalent position for the displaced mandiubular third molars was vertical (51.9%), followed by mesioangular (33.3%), horizontal (11.1%), and distoangular (3.7%). Using the Pell and Gregory system, almost half of the sample was Class III (48.1%) with more than half in the C position (51.9%), followed by Class II (40.7%) with approximately 33.3% in the B position, and Class 1 (11.1%) with 14.8% in the A position.[11]

Trismus

The ID for the preoperative dose of 4 mg dexamethasone ranged from 39 to 59 mm (49.48 ± 5.06). The ID at 24 hours postoperative ranged from 19 to 58 mm (39.48 ± 10.96); the ID at 48 hours postoperative ranged from 20 to 55 mm (38.92 ± 10.71).

For the class of patients who received the 12 mg dexamethasone dose, the preoperative ID ranged from 39 to 58 mm (48.40 ± 4.87); at postoperative 24 hours, the ID ranged from 15 to 50 mm (38.77 ± 9.65); and the ID at postoperative 48 hours ranged from 14 to 56 mm (37.92 ± 11.25). For the patients who received a 4 mg dose, the preoperative ID was the same as the patients who took a 12 mg dose; the ID at 24 hours postoperative ranged from 19 to 58 mm (39.48 ± 10.96), and the ID at 48 hours postoperative ranged from 20 to 55 mm (38.92 ± 10.71).

Swelling

The paired t-test analysis of the relationship between facial swelling—at 1-hour preoperative and 24- and 48-hour postoperative—and the doses of dexamethasone (4 and 12 mg) showed no significant difference between the 2 doses of dexamethasone at either postoperative time ($P = 0.68$ at 24 hours, $P = 0.87$ at 48 hours) (Table).

A significant increase on the calculated means between pre- and postoperative times was observed between the 2 doses.

Table. Mean, standard deviation (SD) and significance (*P* value) of the facial measurements of swelling in relation to the surgical timepoints (preoperative and 24/48 hours postoperative) and the doses of dexamethasone (4 and 12 mg).

Variables		Time	4 mg	12 mg	*P* value
Trismus		24 hours postoperative and 1 hour preoperative	10.00 (8.83)	9.62 (7.54)	0.689
		48 hours postoperative and 1 hour preoperative	10.55 (9.30)	10.48 (8.94)	0.875
		24 hours postoperative and 48 hours postoperative	0.55 (4.45)	0.85 (5.92)	0.922
Swelling	Angle – tragus	24 hours postoperative and 1 hour preoperative	0.25 (0.81)	0.40 (0.97)	0.621
		48 hours postoperative and 1 hour preoperative	0.48 (0.89)	0.88 (2.18)	0.538
		24 hours postoperative and 48 hours postoperative	0.22 (0.80)	0.48 (1.84)	0.497
	Angle – external eye corner	24 hours postoperative and 1 hour preoperative	0.74 (1.37)	1.11 (1.76)	0.447
		48 hours postoperative and 1 hour preoperative	2.22 (2.65)	2.03 (2.29)	0.985
		24 hours postoperative and 48 hours postoperative	1.48 (2.50)	0.92 (1.81)	0.360
	Angle – nose wing	24 hours postoperative and 1 hour preoperative	1.51 (1.96)	2.55 (3.47)	0.301
		48 hours postoperative and 1 hour preoperative	3.92 (3.66)	4.29 (3.96)	0.708
		24 hours postoperative and 48 hours postoperative	2.40 (3.54)	1.74 (3.07)	0.455
	Angle – buccal commissure	24 hours postoperative and 1 hour preoperative	2.48 (2.77)	3.03 (3.86)	0.613
		48 hours postoperative and 1 hour preoperative	5.11 (4.48)	4.40 (4.77)	0.414
		24 hours postoperative and 48 hours postoperative	2.62 (4.03)	1.37 (3.16)	0.268
	Angle – soft pogonion	24 hours postoperative and 1 hour preoperative	2.37 (2.96)	2.11 (2.92)	0.523
		48 hours postoperative and 1 hour preoperative	3.88 (3.71)	3.59 (4.43)	0.737
		24 hours postoperative and 48 hours postoperative	1.51 (3.30)	1.48 (2.57)	0.881

Pain

Patients in the 4 mg dexamethasone group used a total of 67 tablets of acetaminophen (750 mg) during the first 48 hours after surgery, while the 12 mg dexamethasone group consumed 75 acetaminophen (750 mg) tablets. According to the Wilcoxon test, there was no significant difference between the amount of acetaminophen tablets used for the 2 different doses at 24 hours or 48 hours postsurgery ($P = 0.59$ for both times). According to the Friedman test, there was no difference between the 2 doses of dexamethasone and the level of pain at postoperative 24 and 48 hours ($P = 0.636$).

Discussion

It was observed in both the 4 mg and the 12 mg groups that the sensitivity to pain decreased during the first week postsurgery, as shown in both the VAS and the quantity of acetaminophen ingested postoperatively.[12-16] In a study presented by Laureano Filho et al, the authors obtained similar results when comparing 4 mg and 8 mg preoperative doses of dexamethasone.[17] Dionne et al administered a 4 mg dose of dexamethasone to 33 patients both 12 hours before surgery and also shortly after, while 28 patients received a placebo.[18]

The present study analyzed the degree of inflammation via samples of prostaglandin E2 (PGE2) and thromboxane B2 (TxB2), which were collected during the surgery at the site of the operation. Dexamethasone was found to significantly decrease the levels of PGE2 and TxB2, but had a minimal effect in decreasing pain 24 hours postsurgery. When comparing the 2 different measures of pain used in this study (VAS and painkiller tablet comsumption), it was observed that there was no difference between patients in the 4 and 12 mg dexamethasone dosage groups, suggesting a similar analgesic effect of dexamethasone for both doses.

A study by Esen et al evaluated 20 patients with bilateral displaced third molars and recorded the number of painkillers taken postsurgery after a corticosteroid (methylprednisolone 125 mg) was administered 1 hour before the first surgery vs the number of painkillers taken postsurgery after a placebo was administered 1 hour before the second surgery.[16] The results showed that patients who received the methylprednisolone before the surgery used 42% fewer analgesics within the first 24 hours postsurgery than patients who received the placebo.[16] These results indicate the efficacy of corticosteroids in controlling pain in the first hours postsurgery. However, in another study of displaced third molar surgery, Grossi et al found no significant difference in the amount of drugs taken postsurgery when they compared patients who received 4 mg dexamethasone versus patients who received 8 mg preoperatively.[8]

The postoperative analgesic used in this study was acetaminophen, as it is considered a safe drug that does not change coagulation time, platelet aggregation, or neutrophilic defense.[19] No other drug was allowed until the closing of the postoperative phase of the study.

As reported in other studies, the authors of the present study observed that the preoperative administration of 4 mg of dexamethasone significantly decreased the swelling by postoperative Day 2.[8,19] There was no difference in swelling when compared to the 12 mg dose. This would suggest that both doses presented the same effect in relation to swelling. In another lower third molar surgery study, Paiva compared the effects of preoperative doses of dexamethasone (4 and 8 mg).[20] The results showed a reduction on the swelling of the buccal comissure jaw angles of patients who were treated with the 8 mg dosage.[20] This is one of the primary regions to present swelling in lower third molar surgery. This may be due to the fact that the area requires extensive manipulation of the surrounding tissues and also because the surgery involves cutting through part of the masseter region that includes the buccinator muscle.

In a study by Lago-Mendez et al, the authors correlated postoperative pain to the degree of surgical difficulty in 139 patients.[21] The pain was found to be—through a VAS evaluation—more intensive in postoperative Day 1, decreasing gradually until the sutures were removed on postoperative Day 7. The average time period of the surgical procedures was 36.8 ± 22.8 minutes. Patients who had longer surgical procedures reported greater pain intensity on postoperative Days 0 (day of surgery), 5, and 6.

A study by Al-Khateeb & Nussair also demonstrated that the length of the surgical procedure can be directly correlated to the degree of difficulty in such surgical extractions, and consequently, can also be related to the intensity of the swelling presented by the patient postsurgery.[22] In order to mitigate any possible connection between longer surgical times and differing doses of dexamethasone, the present study recommended a selective inclusion of patients who had no more than 5 minutes difference between the 2 surgeries, thus averaging similar operative times between doses of 4 mg (24.22 min) and 12 mg (24.66 min), with no difference in the variable time.

According to Peterson et al, the mesioangular position of the mandibular third molar is the most prevalent, affecting approximately 45% of the teeth.[23] In the present study, the authors found that the vertical position had the highest prevalence (51.9%) of the teeth removed. These data also agree with Aguiar et al who found a higher prevalence of vertical impacted teeth.[12] Regarding the level of retention, slightly less than half of the sample in the present study was Class III (48.1%).[11] This data corroborates those obtained by Aguiar et al.[12]

In a similar study of 23 patients undergoing lower and upper third molar surgery, it was observed that 56.5% of the patients presented trismus on postoperative Day 2.[5] The results confirmed the high incidence of this kind of sequelae in third molar surgeries. These results were further corroborated by Oliveira et al, who found that trismus and other complications—such as paresthesia and alveolitis—are directly correlated to the degree of difficulty of the surgery, whether or not the surgery involved an osteotomy, and whether the procedure required a prolonged surgical time.[24]

In this study, the authors found that the positions of the lower third molars did not affect the analysis of the trismus variable between the 2 different doses of dexamethasone. This would suggest that the different doses presented similar effects, and have no impact regarding limited mouth opening postoperatively. Flores et al investigated the use of anti-inflammatory medication and/or antibiotics in third molar surgery.[5] The authors reported that such medicines effectively reduced the incidence of trismus, but were not able to completely avoid it. In the same study, it was also reported that no significant relationship between the use of such medicines and the level of surgical trauma could be found.[5]

Gbotolorun et al reported that the main factor directly associated with the difficulty of removing mandibular third molars was radiological variables.[25] The authors of that study also found that clinical variables (such as age and body mass index) exerted some influence on the degree of surgical difficulty.[25] According to their results, patients of advanced age and high body mass index were submitted to longer, more complicated surgeries.[25]

It is well known that cortisol found in the body changes its concentration over the course of a day, presenting a peak serum level in the early morning.[26] If the daily production of cortisol by the human body averages 20 mg/day—with peaks of 300 mg reached during stressful situations such as trauma and infectious processes—this limit has to be taken into account if a patient is administered a maximum dose of glucocorticoid.[27] There have been studies of patients taking methylprednisolone (125 mg, equivalent to 625 mg of cortisol) with no noted side effects.[28] Other studies in which doses of 12 mg of dexamethasone (equivalent to 320 mg of cortisol) were administered reported no postsurgical complications and no pharmacological side effects.[19,20]

The relationship between the use of glucocorticoids and the development of infections is controversial, as it depends on the dose and duration of treatment. Cornia & Anawalt reported that the dose and duration of supplemental steroids used traditionally may produce adverse reactions such as infections, and delay the healing process.[26] The relationship between the use of glucocorticoids and possible side effects depends on the intensity and duration of the therapy.[6,26,27]

It has been reported that corticosteroid therapy, whether in single large doses or short-term therapies, can cause side effects.[6,27] In therapies in which a glucocorticoid was administered for >1week, some signs of steroid toxicity have been reported, including hyperglycemia, myopathy, growth suppression, peptic ulcer disease, effects on the central nervous system, increased susceptibility to infection, and signs of suppression of the hypothalamic-pituitary-adrenal (HPA) axis.[6,27]

The effects of corticosteroids on the HPA axis were first investigated by Williamson et al.[6] In their study, an 8 mg dose of dexamethasone was administered to patients undergoing third molar extraction. A significant difference was found between the patient's preoperative values of 11-deoxycortisol (cortisol) and the values measured on postoperative Day 3. However, no significant difference was found when comparing the preoperative values with the values measured on postoperative Day 7. These results seem to indicate that the integrity of the HPA axis is completely restored 7 days postsurgery.[6] Therefore, it can be assumed that the preoperative administration of a single dose of corticosteroids—to decrease the length of HPA axis suppression postsurgery—is a relatively safe and effective procedure.

Summary

In pharmacology, the terms *clinical efficacy* or *clinical maximum* are both defined as the plateau region in a concentration-against-effect intensity graph.[29] This plateau may be determined primarily by the receptor-effector effect of the drug and its properties.[30] The results of the present study suggest that the therapeutic doses of 4 and 12 mg dexamethasone have shown similar effects regarding the evaluated variables—swelling, pain, and trismus. The results suggest that this is due to the fact that the investigated drug has a desired therapeutic effect at either dose.

The data also suggests that the 12 mg dose of dexamethasone and the 4 mg dose presented the same therapeutic effect on the evaluated variables at both 24 and 48 hours postsurgery. Thus, it can be concluded that both doses are effective and there is no need to use the larger dose to ensure successful postoperative management of these variables.

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References

1. Bamgbose BO, Akinwande JA, Adeyemo WL, Ladeinde AL, Arotiba GT, Ogunlewe MO. Effects of co-administered dexamethasone and diclofenac potassium on pain, swelling and trismus following third molar surgery. *Head Face Med.* 2005;1:11.
2. Bamgbose BO, Akinwande JA, Adeyemo WL, Ladeinde AL, Arotiba GT, Ogunlewe MO. Prospective, randomized, open-label, pilot clinical trial comparing the effects of dexamethasone coadministered with diclofenac potassium or acetaminophen and diclofenac potassium monotherapy after third-molar extraction in adults. *Curr Ther Res Clin Exp.* 2006;67(4):229-240.
3. Tiigimae-Saar J, Leibur E, Tamme T. The effect of prednisolone on reduction of complaints after impacted third molar removal. *Stomatologia.* 2010;12(1):17-22.
4. Ustun Y, Erdogan O, Esen E, Karsli ED. Comparison of the effects of 2 doses of methylprednisolone on pain, swelling, and trismus after third molar surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2003; 96(5):535-539.
5. Flores JA, Machado E, Machado P, Flores FW, Mezomo MB. Avaliacao da prevalencia de trismo em pacientes submetidos a exodontia de terceiros molares. *RGO.* 2007:55:17-22.
6. Williamson LW, Lorson EL, Osbon DB. Hypothalamic-pituitary-adrenal suppression after short-term dexamethasone therapy for oral surgical procedures. *J Oral Surg.* 1980;38(1):20-28.
7. Bodnar J. Corticosteroids and oral surgery. *Anesth Prog.* 2001;48(4):130-132.
8. Grossi GB, Maiorana C, Garramone RA, et al. Effect of submucosal injection of dexamethasone on postoperative discomfort after third molar surgery: a prospective study. *J Oral Maxillofac Surg.* 2007;65(11): 2218-2226.
9. Boumpas DT, Chrousos GP, Wilder RL, Cupps TR, Balow JE. Glucocorticoid therapy for immune-mediated diseases: basic and clinical correlates. *Ann Intern Med.* 1993;119(12):1198-1208.
10. Kingery WS, Castellote JM, Maze M. Methylprednisolone prevents the development of autotomy and neuropathic swelling in rats, but has no effect on nociceptive thresholds. *Pain.* 1999;80(3):555-566.
11. Pell GJ, Gregory GT. Impacted third molars: classification and modified technique for removal. *Dent Digest.* 1933;39:338.
12. Aguiar ASW, Oliveira ACX, Martins PC, Freire ROM. Avaliacao do grau de abertura bucal e dor pos-operatoria apos a remocao de terceiros molares inferiores retidos. *Rev Cir Traumatol Buco-Maxilo-fac.* 2005;5(3):57-64.
13. Cerqueira PR, Vasconcelos BC, Bessa-Nogueira RV. Comparative study of the effect of a tube drain in impacted lower third molar surgery. *J Oral Maxillofac Surg.* 2004;62(1):57-61.
14. Saska S, Scartezini GR, Souza RF, Hochuli-Vieira E, Filho AP, Gabrielli MAC. Cloridrato de tramadol/paracetamol no controle da dor pos-operatoria em cirurgias de terceiros molares inclusos. *Rev Cir Traumatol Buco-Maxilo-fac.* 2009;9(4):99-106.
15. Scott J, Huskinsson EC. Graphic representation of pain. *Pain.* 1976;2(2):175-184.
16. Esen E, Tasar F, Akban O. Determination of the anti-inflammatory effects of methylprednisolone on the sequelae of third molar surgery. *J Oral Maxillofac Surg.* 1999;57(10):1201-1206; discussion 1206-1208.
17. Laureano Filho JR, Maurette PE, Allais M, Cotinho M, Fernandes C. Clinical comparative study of the effectiveness of two dosages of dexamethasone to control postoperative swelling, trismus and pain after the surgical extraction of mandibular impacted third molars. *Med Oral Patol Oral Cir Bucal.* 2008;13(2):129-132.
18. Dionne RA, Gordon SM, Rowan J, Kent A, Brahim JS. Dexamethasone suppresses peripheral prostanoid levels without analgesia in a clinical model of acute inflammation. *J Oral Maxillofac Surg.* 2003;61(9):997-1003.
19. Graziani F, D'Aiuto F, Arduino PG, Tonelli M, Gabriele M. Perioperative dexamethasone reduces post-surgical sequelae of wisdom tooth removal. A split-mouth randomized double-masked clinical trial. *Int J Oral Maxillofac Surg.* 2006;35(3):241-246.
20. Paiva FM. Estudo clinico comparativo entre duas dosagens de dexametasona no controle do edema e trismo apos cirurgia de terceiros molares inferiores inclusos Dissertacao de Mestrado em ciencias da Saude (UFMA). 2004:8-79.
21. Lago-Mendez L, Diniz-Freitas M, Sena-Rivera C, Gude-Sampedro F, Gandara-Rey JM, Garcia-Garcia A. Relationships between surgical difficulty and postoperative pain in lower third molar extractions. *J Oral Maxillofac Surg.* 2007;65(5):979-983.
22. Al-Khateeb TH, Nussair Y. Effects of the proteolytic enzyme serrapeptase on swelling, pain and trismus after surgical extraction of mandibular third molars. *Int J Oral Maxillofac Surg.* 2008;37(30):264-268.
23. Peterson LJ, Ellis EJ III, Hupp JR, Tucker MR, eds. *Cirurgia Oral e Maxilofacial Contemporanea.* Rio de Janeiro: Koogan; 2000.
24. Oliveira LB, Schmidt DB, Assis AF, Gabrielli MAC, Hochuli-Vieira E, Filho VAP. Avaliacao dos acidentes e complicacoes associados a exodontia dos terceiros molares. *Rev Cir Traumatol Buco-Maxilo-fac.* 2006;6(2):51-56.
25. Gbotolorun OM, Arotiba GT, Ladeinde AL. Assessment of factors associated with surgical difficulty in impacted mandibular third molar extraction. *J Oral Maxillofac Surg.* 2007;65(10):1977-1983.
26. Cornia PB, Anawalt BD. *Rational use of Perioperative Corticosteroid Supplementation in Patients at Risk for Acute Adrenal Insufficiency.* Available at: http://www.turner-white.com/pdf/hp_oct03_acute.pdf. Accessed August 28, 2014.
27. Kim K, Brar P, Jakubowisk J, Kaltman S, Lopez E. The use of corticosteroids and nonsteroidal antiinflammatory medication for the management of pain and inflammation after third molar surgery: a review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2009;107(5):630-640.
28. Beirne RO, Hollander B. The effect of methylprednisolone on pain, trismus, and swelling after removal of third molars. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1986;61(2):134-138.
29. Wilkinson GR. Farmacocinetica: dinamica da absorcao, da distribuicao e da eliminacao dos farmacos. In: Brunton LL, ed. *Goodman & Gillman. As Bases Farmacologicas da Terapeutica.* 10th ed. Rio de Janeiro: McGraw-Hill; 2005:3-25.
30. Zeitler DL. Prophylactic antibiotics for third molar surgery: a dissenting opinion. *J Oral Maxillofac Surg.* 1995;53(1):61-64.

Manufacturers

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852.2811.9662, www.spss.com

Clinical evaluation of silorane-based and dimethacrylate-based resin composites: 1-year follow-up

M. Jacinta M.C. Santos, DDS, MSc, PhD ▪ Abraham Kunnilathu, BDS, CRCA ▪ Shawn Steele, DDS ▪ Gildo C. Santos Jr., DDS, MSc, PhD

This study sought to evaluate the 1-year clinical performance of silorane-based and dimethacrylate-based Class II resin composite restorations using 2 bonding strategies. Eighty-two restorations were placed in 32 patients (median age 37 years) by a single operator. Only Class II restorations were included. Each patient received 1-2 pairs of resin composite restorations with both restoration materials. Between-group comparisons were made using an adjusted chi-square test and an adjusted McNemar's chi-square test to analyze the intrasystem data ($\alpha = 0.05$). Both systems demonstrated acceptable clinical performance after 1 year.

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The increased popularity of resin composites is due in part to their esthetics, low cost, and acceptable clinical performance.[1-3] The longevity of resin composite restorations has been attributed to several factors. Clinical variables (such as restoration type, size, and location), the technique employed, operator experience, and behavioral aspects related to patients—such as caries risk, age, occlusion, and hygiene—are considered relevant to the long-term success of composite restorations.[1,3] Studies have reported that fractures and recurrent caries are the most common reasons for failure.[1,2,4,5] Dimethacrylate-based resin composites (DBCs) are known to experience polymerization shrinkage and shrinkage stress (generated during setting).[6,7] These well-documented phenomena can compromise marginal adaptation, resulting in microleakage, postoperative sensitivity, marginal staining, recurrent caries, and inevitably restoration failure.[7-9] To overcome these problems, research has focused on modifying the composition of the restorations (such as altering the amount and size of filler) and using high molecular weight monomers.[10,11] The fundamental problem of polymerization shrinkage remains related to the radical polymerization of the methacrylate-based resin composites (MBCs). The predominant base monomers in dental resin composites have been dimethacrylates—such as bisphenol A-glycidyl methacrylate (Bis-GMA), tetraethylene glycol dimethacrylate (TEGDMA), and urethane dimethacrylate (UDMA).[12] By converting dimethacrylate-based monomer molecules into a polymer network, van der Waals spaces are exchanged for shorter covalent bindings, resulting in a tighter arrangement of molecules, which leads to a reduction in material volume.[6] Recently, a new type of resin composite that is silorane-based was introduced commercially. Siloranes contain a siloxane backbone with 4 attached oxirane rings that open to form a polymer chain.[10] The oxirane ring opening results in volumetric expansion that offsets the shrinkage that results when converting monomers into polymers. In vitro studies have reported that silorane-based composites (SBCs) and standard DBC composites have similar mechanical properties.[10,11,13] However, the SBC polymerization shrinkage is approximately 50% that of the current resin-based composites; a longer gelation time with a reduced shrinkage strain has been reported as well.[11,14,15] These factors translate to a different polymerization rheology, allowing the material to better compensate for stresses during polymerization and improving marginal adaptation.[15]

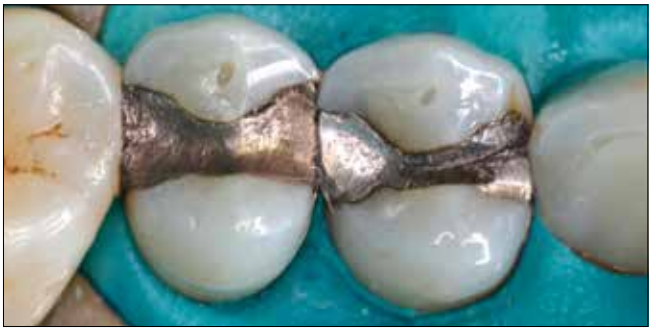

Fig. 1. An example of 2 deficient mesiocclusal distal amalgam restorations.

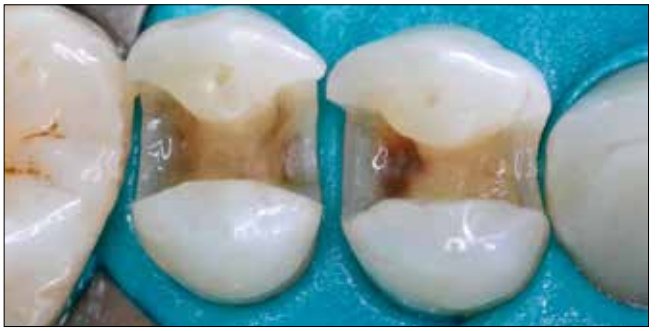

Fig. 2. Cavity preparations on teeth No. 15 (left) and No. 14 (right).

Table 1. Dimethacrylate-based (DBC) and silorane-based (SBC) resin composite systems with restoration specifications, according to the manufacturer's instructions.

Material	Composition	Adhesive	Procedure
DBC: Filtek Supreme (Lot No. N142167)	Resin: Bisphenol A-glycidyl methacrylate (Bis-GMA), urethane dimethacrylate (UDMA), tetraethylene glycol dimethacrylate (TEGDMA), ethoxylated bisphenol A glycol dimethacrylate (Bis-EMA), polyethylene glycol dimethacrylate (PEGDMA); filler: 72.5% weight, volume 55.6% zirconia/silica; etchant: 35% weight phosphoric acid; adhesive: Bis-GMA, hydroxyethyl methacrylate (HEMA), dimethacrylates, ethanol, water, silica nanofiller, copolymer of methacrylic acid (10%), photoinitiator, stabilizers	Adper Single Bond Plus (Lot: N162108)	Etch enamel/dentin with Scotchbond Etchant for 15 seconds, rinse with water for 10 seconds, gently air dry and immediately use applicator to apply 2 coats of adhesive, air dry for 5 seconds to evaporate solvents, photocure for 10 seconds, apply composite in 2 mm increments, and photocure for 20 seconds.
SBC: Filtek LS (Lot: N280238)	Resin: polysilorane; filler: 53% volume; 73% weight quartz; adhesive: primer (Bis-GMA, HEMA, phosphorylated methacrylates, Vitrebond co-polymerer, water, ethanol, silica filler, initiators, stabilizers), bond (hydrophobic dimethacrylate, phosphorilated methacryltes, TEGDMA, silica filler, initiators, stabilizers)	Silorane system adhesive (Lot: N144629)	Apply self-etch primer for 15 seconds, air dry for 5 seconds to evaporate solvents, photocure for 10 seconds, apply bonding agent, air spread, and photocure for 10 seconds, apply composite in 2 mm increments and photocure for 40 seconds.

SBCs require a dedicated adhesive system for bonding to dentin and other DBC materials. The silorane-dedicated adhesive system is available in a self-etch version which, according to the manufacturer, was created to fulfill the criterion of simplification; that is, using the reduced number of operative steps found in self-etch adhesive systems.[16] Although 3-step etch-and-rinse adhesive systems are considered the gold standard, 2-step self-etch adhesives have reported satisfactory results.[17,18] In that context, this study sought to evaluate the clinical performance of silorane-based and dimethacrylate-based resin composite restorations at 1 year, in accordance with the *United States Public Health Systems* (USPHS) criteria.[19] The null hypothesis tested was that there would be no difference between the 2 composite restorative systems in terms of clinical performance.

Materials and methods

This study selected 32 patients, ranging in age from 21 to 53 years (median age 37 years), who required at least 1 pair of Class II restorations (Fig. 1). Before the start of treatment, patients were informed of the research methodology, risks, and benefits, as well as their right to withdraw from the research at any time. Written informed consent was obtained.

The following were exclusion criteria: high caries risk (presence of incipient lesions, plaque, and/or xerostomia), generalized periodontal disease, a removable or fixed orthodontic appliance, signs of bruxism or clenching, more than 1 unit absent from the posterior region, poor oral hygiene, and pregnancy.

Preoperative bitewing radiographs were taken of the teeth that were to be restored. Vitality was tested with carbon dioxide snow (-26.2°C). A total of 82 Class II restorations were placed in pairs randomly, using 2 composite systems. Randomization was determined by selecting the restorative system (via a coin toss), then using the selected system on the tooth with the lowest number. The other system was then placed on the remaining tooth.

Clinical procedures

All cavities were prepared under local anesthesia and rubber dam isolation (Hygienic Dental Dam, Coltene/Whaledent, Inc.) using water-cooled high-speed carbide burs (No. 330 and No. 245, Brasseler USA). Carious tissues were removed using low-speed carbide round burs (No. 1-3, Brasseler USA). After caries removal, the undercuts were covered with a resin-modified glass ionomer (Ketac Nano, 3M ESPE). In cases with deeper areas, a calcium hydroxide lining (Kerr Life, Kerr Dental) was used. The enamel margins at the proximal box were finished using hand instruments to remove unsupported enamel and to bevel the cavosurface margins of the gingival and proximal walls (Fig. 2). All cavities were restored with 1 of the 2 resin composite/bonding agents: DBC (Filtek Supreme/Single Bond, 3M ESPE) and SBC (Filtek LS Low Shrink Posterior Restorative System/LS Adhesive Self-Etch Bond, 3M ESPE), according to the manufacturers' instructions (Table 1) (Fig. 3). A sectional metal matrix, fixed with an elastic ring (Garrison Dental), was installed to restore the proximal contact and marginal ridge. The resin composites were placed incrementally and photocured for 20 seconds by a light-emitting diode lamp (Elipar FreeLight 2, 3M ESPE) with a power density of 660 mW/cm^2. Increments of resin composite (maximum thickness of 2 mm) were placed and photocured. Occlusal anatomy was developed using hand instruments before the composite was cured at the occlusal surface. After polymerization, the margins were inspected carefully for overhangs, which were removed with a No. 12 scalpel. Occlusal adjustments were performed with diamond finishing burs (No. H133F and H134F, Brasseler USA) under cooled water. The occlusal surfaces were then polished carefully with rubber tips (PoGo Points, DENTSPLY Caulk) and embrasures were refined with fine disks (Sof-Lex Finishing and Polishing System Kit, 3M ESPE).

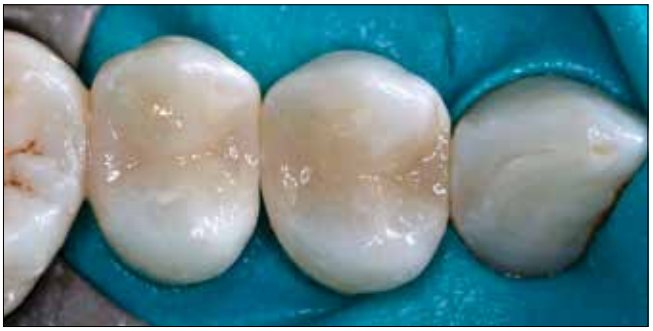

Fig. 3. The teeth in Figure 2, after placement of dimethacrylate-based resin composite on tooth No. 15 (left) and silorane-based resin composite on tooth No. 14 (right).

Fig. 4. The composite restorations at the 1-year follow-up examination.

Table 2. Modified United States Public Health Systems criteria for the clinical evaluation of silorane- and dimethacrylate-based resin composites used in this study.[19]

	Alpha	Bravo	Charlie
Postoperative sensitivity	No postoperative sensitivity	Postoperative sensitivity	N/A
Secondary caries	No evidence of caries contiguous with the margin of the restoration	Caries evident contiguous with the margin of the restoration	N/A
Marginal discoloration	No discoloration on the margin between the restoration and the tooth structure	Discoloration on the margin between the restoration and the tooth structure. <50% of cavosurface margin affected by stain (usually localized)	More than 50% of cavosurface margin affected by stain
Marginal adaptation	No visible evidence of ditching along the margin; no explore catch at the margins, or there is a catch in 1 direction	Visible evidence of ditching along the margin; explorer catches; no dentin or base visible	Dentin or base is exposed along the margin; restoration is mobile, fractured or missing
Color match	No mismatch in color, shade, and translucency between restoration and adjacent tooth structure	Mismatch between restoration and tooth structure within the normal range of color, shade and translucency	Mismatch between restoration and tooth structure outside the normal range of color, shade and translucency
Anatomic form	Restoration continuous with existing anatomic form	Restoration discontinuous with existing anatomic form	
Surface roughness	Smooth surface	Slightly rough or pitted; can be refinished	Rough; cannot be refinished

Evaluation procedures

One week following placement, the restorations were assessed using only mirrors and probes, in accordance with the modified USPHS criteria, by 2 independent investigators calibrated in the use of the system (Table 2).[19] The investigators did not participate in the clinical procedures and neither the patients nor the evaluators knew which restorative system was used on the selected teeth. When disagreement occurred during the evaluation process, the evaluators had to reach a consensus before the patient could be dismissed. The same procedures performed at the baseline were performed again 1 year post-treatment (Fig. 4). Between-group comparisons were made using an adjusted chi-square test and an adjusted McNemar's chi-square test to analyze the intrasystem data ($\alpha = 0.05$).

Results

Table 3 summarizes the results of the *Alpha* ratings for both resin composites at baseline and at 1-year recalls, according to the USPHS criteria.[19]

Recall rate

At the 1-year recall, 29 patients—including 76 of the 82 restorations (92.68%)—were evaluated. Thirty-eight composite restorations from each system were assessed by the evaluators.

Marginal discoloration/marginal adaptation

Marginal discoloration was detected in some restorations from both groups. At the 1-year evaluation, the percentage of Alpha scores were 76.3% for the SBC samples, compared to 92.1% for the DBC samples. The marginal discoloration was considered clinically acceptable by USPHS *Bravo* criteria, and no *Charlie* scores were rated for any of the resin-based restorative materials.[19] Concerning marginal adaptation,

Table 3. Alpha scores (%) for resin composite systems used in this study.

	Baseline (n = 82)		1-year follow-up (n = 76)	
	SBC	DBC	SBC	DBC
Postoperative sensitivity	100.0	89.5	100.0	100.0
Secondary caries	100.0	100.0	100.0	100.0
Marginal discoloration	100.0	100.0	76.3	89.5
Marginal adaptation	100.0	100.0	78.9	92.1
Color match	84.2	89.5	84.2	89.5
Anatomic form	100.0	100.0	94.7	97.4
Surface roughness	97.4	100.0	97.4	100.0

Abbreviations: DBC, dimethacrylate-based composite; SBC, silorane-based composite.

both restorative systems recorded some enamel defects, with 78.9% of SBC and 92.1% of DBC samples receiving Alpha scores; however, no significant differences were found between both materials ($P = 0.09$).

Surface texture

In terms of surface texture, there was no significant difference between the 2 restorative systems ($P > 0.05$). All of the DBC restorations demonstrated ideal surface texture, compared to 97.4% of the SBC restorations.

Postoperative sensitivity/ Secondary caries

Four (10.52%) of the DBC restorations demonstrated postoperative sensitivity at the baseline; however, no postoperative sensitivity was reported for either composite at the 1-year recall. None of the restorations had recurrent caries after 1 year of clinical service.

Anatomic form

All restorations at the 1-year recall were continuous with existing anatomic form.

Color match

In terms of the color match between the restoration and the tooth, 84.2% of the SBC restorations were rated Alpha, compared to 89.5% of the DBC restorations; this difference was not statistically significant ($P > 0.05$). The color match was assessed at baseline and did not change after 1 year.

Clinical success rate

At the 1-year recall, all composite restorations showed acceptable clinical performance. The chi-square test revealed no significant difference between SBC and DBC for all aspects evaluated at baseline and at the 1-year recall ($P > 0.05$). The McNemar statistical test was used to draw a comparison between baseline and 1 year for each resin composite system and no significant differences were found.

Discussion

The null hypothesis that there would be no difference in the clinical performance between the 2 resin composite systems was confirmed. At 1 year, no significant difference was found between the SBC and DBC for any of the aspects evaluated at different recall appointments ($P > 0.05$). The main reported disadvantage of DBC is its inherent degree of polymerization shrinkage during the setting reaction, when the monomers link to form polymer chains, inevitably leading to volume shrinkage.[6] Although high filler content can reduce polymerization contraction, current resin composites shrink in volume by 2.6%-7.1%.[20]

Conversely, the SBC system has been reported as a low shrinkage composite, with a volumetric shrinkage of approximately 1% (approximately 50% of the DBC system).[11,14,15] However, the clinical relevance of this difference in polymerization shrinkage could not be upheld in this study at the 1-year evaluation. Both composite materials presented satisfactory results with no significant difference between them.

To increase the validity of this clinical trial and allow for future comparisons with similar studies, care was taken to reduce bias. A randomized allocation and fully blinded investigation was employed using 2 calibrated evaluators, with 1 experienced dentist placing all of the restorations. A paired allocation design was employed and a maximum of 2 pairs of restorations was allowed per patient to reduce the effects of clustering.[21]

In the present study, 76 restorations were evaluated at the 1-year recall. The sample size was comparable to previous studies in which 1 operator performed all of the restorative treatments, rather than using a multicenter approach.[22-25] Six restorations (7.3%) were not examined at the 1-year recall, as 3 patients could not participate.

The 1-year recall was used as an initial evaluation of the marginal adaptation, since polymerization shrinkage develops at an early stage during restoration placement.[26] It is the authors' intention to evaluate the restorations for a longer period to assess the medium-term durability of these restorations. Comparing the baseline data with those obtained at the 1-year evaluation revealed an increase in Bravo ratings for marginal discoloration, the most common problem reported in clinical trials involving resin composite restorations.[22-27] This problem is possibly caused by several factors and may be due to excess composite material left behind after finishing and polishing, gaps as a result of polymerization shrinkage, a deficient adaptation around the margins, and/or the adhesive interface failing over time.[3,12,22-27] In most cases, marginal discoloration of a restoration does not indicate a need for immediate replacement, but should be interpreted as a clinical sign of possible failure in the future.[3]

In the present study, 2 restorative systems and their respective adhesives were employed. Filtek Silorane was designed for posterior restorations; it requires its own dedicated adhesive system, to which hydrophobic bifunctional monomers are added to match the hydrophobic silorane resin.[10,13] The manufacturer elected to use a self-etch adhesive version with this restorative system for simplification.[16] However, the literature has shown increased marginal discoloration when a

self-etch adhesive system is employed, due to its weak etching ability at the enamel margins.[22,27,28] While the DBC system (used in conjunction with an etch-and-rinse adhesive) showed slightly better results, this difference was not statistically significant ($P > 0.05$). The SBC restoration showed slightly poorer marginal adaptation, although this difference was not significant at the 1-year evaluation.

The results obtained in the present study are in agreement with a 2011 study that evaluated SBC restorations for a 2-year period.[29] However, 2 other clinical studies have reported a significantly worse marginal adaptation for the SBC compared to a DBC for the same timeframe.[22,26]

Color matching remained the same after 1 year. Although exceptional esthetic results are not considered essential for posterior restorations, both composites showed acceptable results. Previous studies have reported on the high color stability of SBC.[30,31]

Although postoperative sensitivity has been considered as another clinical complication of posterior composite restorations, it was not an issue in the present study. After cavity preparation, all deep areas and undercuts were covered with a resin-modified glass ionomer to protect the deep dentin. In the present study, the SBC restorations demonstrated no postoperative sensitivity, while 4 DBC restorations exhibited postoperative sensitivity at the baseline; this problem was not present at the 1-year recall.

Conclusion

It can be concluded that both the SBC and DBC systems offered satisfactory results at the 1-year evaluation. The null hypothesis was accepted, since no significant differences were noticed between the 2 resin composite systems. As a result, the advantage of using a low shrinkage resin composite system could not be validated at the 1-year evaluation. Additional recall visits are planned to verify the clinical performance of these resin-based materials over time.

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References

1. Demarco FF, Correa MB, Cenci MS, Moraes RR, Opdam NJ. Longevity of posterior composite restorations: not only a matter of materials. *Dent Mater.* 2012;28(1):87-101.
2. Da Rosa Rodolpho PA, Donassollo TA, Cenci MS, et al. 22-Year clinical evaluation of the performance of two posterior composites with different filler characteristics. *Dent Mater.* 2011;27(10):955-963.
3. Ferracane JL. Resin-based composite performance: are there some things we can't predict? *Dent Mater.* 2012;29(1):51-58.
4. Nordbo H, Leirskar J, von der Fehr FR. Saucer-shaped cavity preparations for posterior approximal resin composite restorations: observations up to 10 years. *Quintessence Int.* 1998;29(1):5-11.
5. Bernardo M, Luis H, Martin MD, Leroux BG, Rue T, Leitao J, DeRouen TA. Survival and reasons for failure of amalgam versus composite posterior restorations placed in a randomized clinical trial. *J Am Dent Assoc.* 2007;138(6):775-783.
6. Peutzfeldt A. Resin composites in dentistry: the monomer systems. *Eur J Oral Sci.* 1997;105(2):97-116.
7. Davidson CL, Feilzer AJ. Polymerization shrinkage and polymerization shrinkage stress in polymer-based restoratives. *J Dent.* 1997;25(6):435-440.
8. Lutz F, Krejci I, Barbakow F. Quality and durability of marginal adaptation in bonded composite restorations. *Dent Mater.* 1991;7(2):107-113.
9. Boaro LC, Goncalves F, Guimaraes TC, Ferracane JL, Versluis A, Braga RR. Polymerization stress, shrinkage and elastic modulus of current low-shrinkage restorative composites. *Dent Mater.* 2010;26(12):1144-1150.
10. Weinmann W, Thalacker C, Guggenberger R. Siloranes in dental composites. *Dent Mater.* 2005;21(1):68-74.
11. Palin W, Fleming G, Nathwani H, Burke F, Randall R. In vitro cuspal deflection and microleakage of maxillary premolars restored with novel low-shrink dental composites. *Dent Mater.* 2005;21(4):324-335.
12. Ferracane JL. Resin composite—state of the art. *Dent Mater.* 2011;27(1):29-38.
13. Ilie N, Hickel R. Silorane-based dental composite: Behavior and abilities. *Dent Mater J.* 2006;25(3):445-454.
14. Lien W, Vandewalle K. Physical properties of a new silorane-based restorative system. *Dent Mater.* 2010;26(4):337-344.
15. Ilie N, Jelen E, Clementino-Luedemann T, Hickel R. Low-shrinkage composite for dental application. *Dent Mater J.* 2007;26(2):149-155.
16. 3M ESPE. *Filtek LS Low Shrink Posterior Restorative System.* Available at: http://multimedia.3m.com/mws/mediawebserver?mwsId=SSSSSufSevTsZxtUNY_9MYt1evUqevTSevTSevTSeSSSSSS--. Accessed September 17, 2014.
17. Zander-Grande C, Loguercio AD, Stanislawczuk R, Martins GC, Gomes OM, Reis A. The effect of 6-month water storage on the bond strength of self-etch adhesives bonded to dentin. *Am J Dent.* 2011;24(4):239-244.
18. Peumans M, Kanumilli P, De Munck J, Van Landuyt K, Lambrechts P, Van Meerbeek B. Clinical effectiveness of contemporary adhesives: a systematic review of current clinical trials. *Dent Mater.* 2005;21(9):864-881.
19. Ryge G. Clinical criteria. *Int Dent J.* 1980;30(4):347-358.
20. Feilzer AJ, De Gee AJ, Davidson CL. Curing contraction of composites and glass-ionomer cements. *J Prosthet Dent.* 1988;59(3):297-300.
21. Hickel R, Roulet JF, Bayne S, et al. Recommendations for conducting controlled clinical studies of dental restorative materials. *Clin Oral Investig.* 2007;11(1):5-33.
22. Baracco B, Perdigao J, Cabrera E, Giráldez I, Ceballos L. Clinical evaluation of a low-shrinkage composite in posterior restorations: one-year results. *Oper Dent.* 2012;37(2):117-129.
23. Celik C, Arhun N, Yamanel K. Clinical evaluation of resin-based composites in posterior restorations: 12-month results. *Eur J Dent.* 2010;4(1):57-65.
24. Ermis RB, Kam O, Celik EU, Temel UB. Clinical evaluation of a two-step etch&rinse and a two-step self-etch adhesive system in Class II restorations: two-year results. *Oper Dent.* 2009;34(6):656-663.
25. Arhun N, Celik C, Yamanel K. Clinical evaluation of resin-based composites in posterior restorations: two-year results. *Oper Dent.* 2010;35(4):397-404.
26. Schmidt M, Kirkevang LL, Horsted-Bindslev P, Poulsen S. Marginal adaptation of a low-shrinkage silorane-based composite: 1-year randomized clinical trial. *Clin Oral Investig.* 2011;15(2):291-295.
27. Perdigao J, Dutra-Correa M, Castilhos N, et al. One-year clinical performance of self-etch adhesives in posterior restorations. *Am J Dent.* 2007;20(2):125-133.
28. Deliperi S, Bardwell DN, Wegley C. Restoration interface microleakage using one total-etch and three self-etch adhesives. *Oper Dent.* 2007;32(2):179-184.
29. Burke FJ, Crisp RJ, James A, et al. Two year clinical evaluation of a low-shrink resin composite material in UK general dental practices. *Dent Mater.* 2011;27(7):622-630.
30. Furuse AY, Gordon K, Rodrigues FP, Silikas N, Watts DC. Colour-stability and gloss-retention of silorane and dimethacrylate composites with accelerated aging. *J Dent.* 2008;36(11):945-952.
31. Perez MM, Ghinea R, Ugarte-Alvan LI, Pulgar R, Paravina RD. Color and translucency in silorane-based resin composite compared to universal and nanofilled composites. *J Dent.* 2010;38(Suppl)2:e110-e116.

Manufacturers

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Coltene/Whaledent, Inc., Cuyahoga Falls, OH
330.916.8800, www.coltene.com

DENTSPLY Caulk, Milford, DE
800.532.2855, www.caulk.com

Garrison Dental, Spring Lake, MI
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Kerr Dental, Orange, CA
800.537.7123, www.kerrdental.com

3M ESPE, St. Paul, MN
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Cytotoxicity of dentin bonding agents

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This study sought to evaluate the cytotoxicity of 5 dentin bonding agents (Admira Bond, Adper Single Bond Plus, Clearfil SE Bond, Clearfil S3 Bond, and Heliobond) by XTT assay using human gingival fibroblast cells. Samples of dentin bonding agents were prepared on a black 96-well microplate, and the cytotoxicity of each bonding material was measured every 24 hours for 7 days, then on Days 14, 21, and 28. One-way ANOVA and Bonferroni post hoc test were used for statistical analyses.

All 5 materials were evaluated as severely cytotoxic ($P < 0.001$) on the first day, with cell viabilities ranging from 6% to 24%. All the bonding agents showed severe cytotoxicity with viability results <10%. With the exception of Adper Single Bond Plus, toxicity continued to Day 28 for all the compounds. The utmost care must be considered during the clinical utilization of dentin bonding agents to keep them within the area of restoration and prevent their contact with adjacent tissues.

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Dentin bonding agents are designed to improve bonding strength between the resin and tooth structure, increase the retention of restorations, reduce microleakage across the dentin-resin interface, and scatter occlusal stress.[1] Like many dental materials, dental adhesives may make direct contact with soft and/or hard tissues for a prolonged period of time; during this time, they may exert residues that can affect the health and structure of adjacent tissues, thus it is necessary to determine the cytotoxicity of these materials to identify and exclude any individual components that may adversely interact with adjacent cellular structures.[2]

Before using a material in clinical conditions, it is necessary to evaluate the material's biocompatibility.[3] Among the in vitro tests that have been utilized to evaluate the biological effects of dental adhesives, cell culture tests generally have provided rapid, sensitive, inexpensive, convenient, and repeatable methods of material screening and ranking; in addition, these tests can be carefully controlled.[4-7] Cell culture tests that investigate toxicity in a simplified manner that minimizes the effect of confounding variables usually are the first step toward evaluating biocompatibility.[8] This method uses a range of cell types to quantitatively investigate the cytotoxicity of numerous dental materials and present biological endpoints.[9,10]

XTT (tetrazolium salts reduction) cell proliferation assay has been used as a non-radioactive, colorimetric assay system for measuring the drug sensitivity of tumor cell lines. After its reduction by metabolic active cells, the color of tetrazolium salt changes from yellow to bright orange.[11,12] Since the formazan produced by XTT reduction is soluble and can be quantified directly using an enzyme-linked immunosorbent assay (ELISA) reader, it can be used in real-time assays. Due to its many advantages—such as being more economical in time and cost and having lower detectable limits—an XTT assay is preferred over the cytotoxicity tests based on the release of ^{51}Cr from the cells.[13]

Previous cell culture studies have stated that the components of resin restoratives may elicit significant toxicity when they make direct contact with fibroblasts.[14] This in vitro study sought to investigate the cytotoxicity of 5 commercially available dentin bonding agents using a cell culture technique on human gingival fibroblast (HGF) cells by XTT assay.

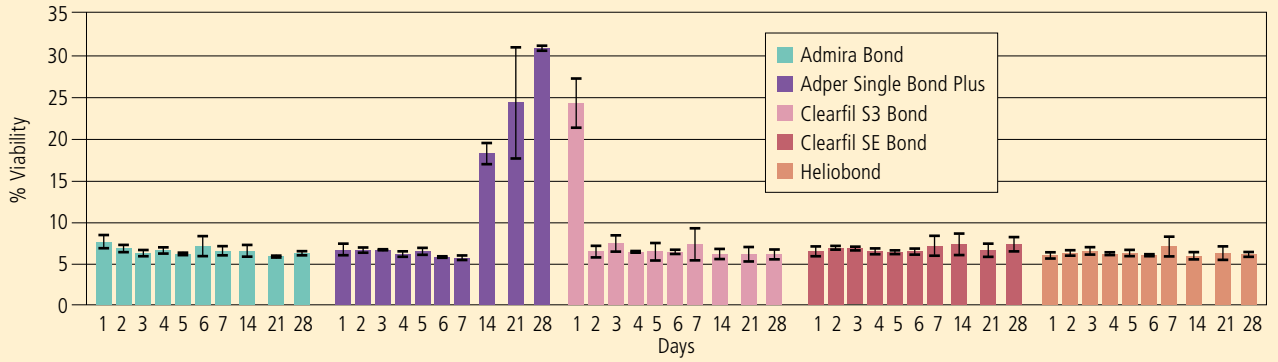
Table 1. Adhesive materials used in the study.

Name	Composition	Lot No.
Admira Bond	Ormocers, bisphenol A glycidyl methacrylate (Bis-GMA), hydroxyethyl methacrylate (HEMA), brain heart infusion (BHI) agar, acetone, organic acids	45309
Adper Single Bond Plus	Bis-GMA, HEMA, dimethacrylates, ethanol, water, photoinitiator, polyacrylic, polyitaconic acid	51202
Clearfil S3 Bond	10-methacryloyloxydecyl dihydrogen phosphate (MDP), Bis-GMA, HEMA, hydrophobic dimetacrylate, ethyl alcohol, camphorquinone, silanated colloidal silica	41117
Clearfil SE Bond	MDP, Bis-GMA, HEMA, hydrophobic dimethacrylate	41491
Heliobond	Bis-GMA, triethylene	K00250

Materials and methods

Five dental adhesives were examined: Admira Bond (Voco America, Inc.), Adper Single Bond Plus (3M ESPE), Clearfil S3 Bond (Kuraray America, Inc.), Clearfil SE Bond (Kuraray America, Inc.), and Heliobond (Ivoclar Vivadent, Inc.) (Table 1). The adhesives were photocured with a halogen light curing unit (Optilux 501, Kerr Dental) per manufacturer recommendations. Each dental adhesive sample was prepared in a 96-well black microplate under laminar air flow. A total volume of 10 µl of adhesive material was pipetted into 10 wells. The ratio of sample surface area to solution volume was adjusted to approximately 4.32 cm^2/ml, which is within the 0.5-6 cm^2/ml range recommended by the *International Organization for Standardization*.[15] The polymerization period for the adhesive materials was accomplished per manufacturers' instructions.

Chart. XXT assay results showing percent cell viability for the 5 dentin bonding agents used in the study. Baseline began at 100% for all agents.



Samples were topped with 200 µl of Dulbecco's modified eagle medium (DMEM)—containing 10% fetal bovine serum, 100 U/ml of penicillin, and 100 µg/ml of streptomycin—to obtain medium extracts of adhesive materials. Plates were incubated in a highly humid atmosphere (5% CO_2 at 37°C) for 24 hours. Next, the medium was removed and the samples were rinsed with 500 µl of phosphate-buffered balanced salt solution and topped again with 200 µL DMEM. Removed medium extracts (approximately 2000 µl) were filtered and set aside in sterile Eppendorf tubes at -20°C until cytotoxicity could be tested. This procedure was repeated every 24 hours for 7 days and again on Days 14, 21, and 28.

Table 2. Percentage and standard deviation (SD) survival rates of human gingival fibroblast cells during each measurement period.

Day	Admira Bond	Adper Single Bond Plus	Clearfil S3 Bond	Clearfil SE Bond	Heliobond
Baseline	100	100	100	100	100
1	7.76 (0.86)	6.60 (0.81)	24.06 (3.01)	6.58 (0.72)	6.00 (0.52)
2	6.88 (0.51)	6.46 (0.44)	6.36 (0.81)	6.91 (0.40)	6.35 (0.47)
3	6.36 (0.55)	6.56 (0.20)	7.48 (1.05)	6.79 (0.41)	6.62 (0.60)
4	6.47 (0.52)	6.07 (0.41)	6.40 (0.28)	6.49 (0.50)	6.15 (0.31)
5	6.03 (0.28)	6.29 (0.59)	6.43 (1.16)	6.52 (0.40)	6.18 (0.55)
6	7.03 (1.39)	5.71 (0.27)	6.40 (0.40)	6.50 (0.55)	6.00 (0.29)
7	6.50 (0.72)	5.59 (0.45)	7.34 (2.08)	7.12 (1.37)	7.09 (1.35)
14	6.51 (0.84)	18.25 (1.43)	6.13 (0.77)	7.40 (1.45)	5.95 (0.59)
21	5.89 (0.17)	24.23 (6.72)	6.15 (0.95)	6.62 (0.91)	6.26 (1.00)
28	6.24 (0.41)	30.78 (0.40)	6.12 (0.72)	7.42 (1.07)	6.07 (0.44)

Cell culture

This study was approved by the ethics committee of Ege University, Turkey (No. 10-6/13), and the 3 patients who volunteered to donate signed informed consents. To obtain HGF cells, 3 biopsies were performed on healthy gingival tissues of 3 patients who were undergoing surgical treatment for impacted third molars. Each biopsy sample was placed into the DMEM culture medium during transportation to the laboratory. Biopsy samples were washed in DMEM and placed in tissue culture flasks using the explant culture method. HGF cells were grown in the humid atmosphere described previously using the supplemented medium. The medium was changed every 3 days, and cells were passaged when confluent with 0.05% trypsin in 0.02% ethylenediaminetetraacetic acid.

Cytotoxicity tests

Cytotoxicity was analyzed with an XTT cell proliferation reagent. HGF cells (1 x 105) were placed onto each well of a 96-well plate and incubated for 24 hours. At that time, culture medium was aspirated and 100 µl extracts of adhesive materials were pipetted onto the HGF cells. All procedures were performed in triplicate. At 24, 48, 72, and 96 hours, formazan formation was quantified spectrophotometrically at 450 nm with a microplate reader. Cell viability was calculated according to the following formula using optical density (OD):

% cell viability = (OD ratio of test group/OD ratio of control group) x 100.

Cell viability was scored using a method described by Sjogren et al.[16] Using this approach, material with cell viability >90% was deemed noncytotoxic, materials with 60%-90% cell viability were considered slightly cytotoxic, 30%-59% cell viability was regarded as moderately cytotoxic, and materials with <30% cell viability were considered severely cytotoxic.[16]

Table 3. Statistical analysis of the test materials at different measurement periods.

Comparison	Day	Difference	95% CI
Admira Bond vs Adper Single Bond Plus	14	11.74	7.411 to 16.06
	21	18.34	14.01 to 22.66
	28	24.54	20.22 to 28.87
Admira Bond vs Clearfil S3 Bond	1	16.30	11.97 to 20.63
Adper Single Bond Plus vs Clearfil S3 Bond	1	17.46	13.13 to 21.79
	14	-12.11	-16.44 to -7.787
	21	-18.08	-22.41 to -13.75
	28	-24.66	-28.99 to -20.34
Adper Single Bond Plus vs Clearfil SE Bond	14	-10.84	-15.17 to -6.517
	21	-17.61	-21.94 to -13.28
	28	-23.36	-27.69 to -19.04
Adper Single Bond Plus vs Heliobond	14	-12.30	-16.63 to -7.974
	21	-17.97	-22.30 to -13.65
	28	-24.72	-29.04 to -20.39
Clearfil SE Bond vs Heliobond	Not significant		
Admira Bond vs Clearfil SE Bond	Not significant		
Admira Bond vs Heliobond	Not significant		

For those comparisons with significant differences, $P < 0.001$ according to Bonferroni post hoc test.

Statistical analysis

Statistical analysis of the results was performed via 1-way ANOVA and Bonferroni post hoc test using analytic software (GraphPad Prism 5.01 for Windows, GraphPad Software, Inc.); $P < 0.05$ was accepted as statistically significant.

Results

The XTT results of cell viability are presented in the Chart. On the first day of the experiment, all adhesive materials showed severe cytotoxicity ($P < 0.001$) and cell viability of <30%. From Day 22 to Day 28, the cell viability of Admira Bond, Clearfil SE Bond, Clearfil S3 Bond, and Heliobond remained <30% (Table 2).

Adper Single Bond Plus had a different viability curve compared to the other adhesives. Although severe cytotoxicity was detected during the first 7 days, the cytotoxicity of this compound started to decrease at Day 14, while cell viability increased to 20% ($P < 0.001$). At Days 21 and 28, cytotoxicity continued to decrease while viability increased to 24% and 31%, respectively, which showed that aging had a positive effect on the material's cytotoxicity.

There was a significant difference between the potential cytotoxicity of the materials tested. The effects of Clearfil S3 Bond were significantly different on the first day compared to the other materials. However there was no significant difference when Clearfil SE Bond was compared to Heliobond or Admira Bond, or when Admira Bond was compared to Heliobond (Table 3).

Discussion

Conservative dental intervention is intended to preserve pulp vitality and prevent possible adverse effects caused by restorative biomaterials used in dental clinical practice. Whenever deep cavities are treated with dentine adhesive systems, their components (such as monomers, acids, or solvents) may pass through the dentinal tubules before and after adhesive curing—particularly when the dentin permeability is high—which may lead to cytotoxic effects.[17,18] A 2012 study by Kierklo et al reported that both Adper Single Bond 2 and Heliobond had cytotoxic effects on HGF cells.[19] This finding was confirmed in the present study, as the materials containing hydroxyethyl methacrylate, bisphenol A glycidyl methacrylate, and acetone showed severe cytotoxicity on primary HGF cells.

It is important to test the biocompatibility of materials that make contact with normal tissues to determine the host-to-graft acceptance. Assays that measure cytotoxicity are critical for testing materials to be used on human tissues. Increased public concern regarding the use of animals in evaluating dental materials has made in vitro testing more acceptable ethically. However, preparing test materials for experiments may alter the cytotoxicity of a material significantly.[20] Every effort was made to simulate in vitro conditions in the laboratory due to the importance of the proper preparation of materials to test target cells.

The literature describes cell culture tests that use various cell types to establish the damage caused by dental materials; however, primary fibroblast cell cultures have not been used frequently in such experiments.[21-23] The present study used HGF cells as the cell culture. The study model used followed previous studies that have recommended HGF cells for testing the cytotoxicity of dental materials.[24-26]

A 1994 study by Al-Nazhan & Spangberg used HGF and mouse fibroblast (L-929) cells and found that the Golgi apparati of the L-929 cells were identified less frequently than those in the HGF cells.[27] In addition, the organelles of the L-929 cells were destroyed almost completely after only 4 hours exposure to the test materials (polymers). The authors concluded that HGF cells were more resistant to environmental impact and appeared to be more suitable for testing various materials.[27]

According to the literature, continuous cell lines with homogeneous cells are suitable for establishing a concentration-dependent response curve in short-term experiments.[2,28] Tests involving primary gingival fibroblasts attempt to simulate clinical assays by establishing the impact of materials on cell cultures in long-term trials.[2,28] Primary gingival fibroblast cultures are more closely related to the original tissue and therefore much easier to identify.[29,30] In addition, cultures with primary cells generally are recommended for long-term experiments as they provide heterogeneous cells with physiological states of aging and offer

a metabolic state relative to their original tissue that is nearly identical.[2,29,30] Previous in vitro experiments that utilized primary gingival fibroblast cultures simulated in vivo conditions efficiently.[29,30] For these reasons, primary gingival fibroblast cultures have been preferred to continuous cell lines.

As dental adhesive technology develops, selecting bonding agents to provide the attachment between the surfaces of dental structures and restorative materials becomes more important. These bonding materials are considered to be technique-sensitive agents; in all cases, manufacturers' recommendations should be followed meticulously. While these bonding materials may perform efficiently enough, they also may have destructive effects on oral soft tissues. Oral cells can be exposed to polymers either directly via contact with gingival tissue, or indirectly when materials released by the polymers migrate toward the pulpal tissue or surrounding tissues.

The results of this study supported the findings of previous in vitro investigations that used the MTT assay to evaluate the cytotoxicity of resinous components.[31,32] In addition, the extracts obtained from the Adper Single Bond Plus demonstrated cytotoxic effects, even though aging helped to improve cell viability. These results are in keeping with the 2009 study by Porto et al, who reported that Adper Single Bond Plus affected macrophages in vitro.[33]

Conclusion

The findings of the present study showed that all of the bonding agents tested generated severe cytotoxicity on HGF cells at 1 or more points. Based on these results, dentists must take care to keep dentin bonding agents within the area where they are needed and prevent their contact with adjacent tissues, especially in cases with deep cavities.

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References

1. Schmalz G. Concepts in biocompatibility testing of dental restorative materials. *Clin Oral Investig.* 1997; 1(4):154-162.
2. Szep S, Kunkel A, Ronge K, Heidemann D. Cytotoxicity of modern dentin adhesives—in vitro testing on gingival fibroblasts. *J Biomed Mater Res.* 2002;63(1):53-60.
3. Braden M, Wright PS, Parker S. Soft lining materials—a review. *Eur J Prosthodont Restor Dent.* 1995;3(4): 163-174.
4. Huang FM, Chang YC. Cytotoxicity of dentine-bonding agents on human pulp cells in vitro. *Int Endod J.* 2002; 35(11):905-909.
5. Lin NJ, Bailey LO, Becker ML, Washburn NR, Henderson LA. Macrophage response to methacrylate conversion using a gradient approach. *Acta Biomater.* 2007; 3(2):163-173.
6. Nocca G, De Palma F, Minucci A, et al. Alterations of energy metabolism and glutathione levels of HL-60 cells induced by methacrylates present in composite resins. *J Dent.* 2007;35(3):187-194.
7. Browne RM. The in vitro assessment of the cytotoxicity of dental materials—does it have a role? *Int Endod J.* 1988;21(2):50-58.
8. Brown D. Resilient soft liners and tissue conditioners. *Br Dent J.* 1988;164(11):357-360.
9. Schweikl H, Schmalz G. Toxicity parameters for cytotoxicity testing of dental materials in two different mammalian cell lines. *Eur J Oral Sci.* 1996;104(3):292-299.
10. Thonemann B, Schmalz G, Hiller KA, Schweikl H. Responses of L929 mouse fibroblasts, primary and immortalized bovine dental papilla-derived cell lines to dental resin components. *Dent Mater.* 2002;18(4): 318-323.
11. Roehm NW, Rodgers GH, Hatfield SM, Glasebrook AL. An improved colorimetric assay for cell proliferation and viability utilizing the tetrazolium salt XTT. *J Immunol Methods.* 1991;142(2):257-265.
12. Scudiero DA, Shoemaker RH, Paull KD, et al. Evaluation of a soluble tetrazolium/formazan assay for cell growth and drug sensitivity in culture using human and other tumor cell lines. *Cancer Res.* 1998;48(17): 4827-4833.
13. Bean TA, Zhuang WC, Tong PY, Eick JD, Chappelow CC, Yourtee DM. Comparison of tetrazolium colorimetric and 51Cr release assays for cytotoxicity determination of dental biomaterials. *Dent Mater.* 1995;11(5):327-331.
14. Vallittu PK, Ruyter IE, Buykuilmaz S. Effect of polymerization temperature and time on the residual monomer content of denture base polymers. *Eur J Oral Sci.* 1998;106(1):588-593.
15. Bouillaguet S, Shaw L, Gonzalez L, Wataha JC, Krejci I. Long-term cytotoxicity of resin-based dental restorative materials. *J Oral Rehabil.* 2002;29(1):7-13.
16. Sjogren G, Sletten G, Dahl JE. Cytotoxicity of dental alloys, metals, and ceramics assessed by millipore filter, agar overlay, and MTT tests. *J Prosthet Dent.* 2000; 84(2):229-236.
17. Vajrabhaya LO, Pasasuk A, Harnirattisai C. Cytotoxicity evaluation of single component dentin bonding agents. *Oper Dent.* 2003;28(4):440-444.
18. Geurtsen W, Spahl W, Muller K, Leyhausen G. Aqueous extracts from dentin adhesives contain cytotoxic chemicals. *J Biomed Mater Res.* 1999;48(6):772-777.
19. Kierklo A, Pawinska M, Tokajuk G, Poplawska B, Bielawska A. Cytotoxicity evaluation of three light-cured dentin adhesive materials on human gingival fibroblasts, ex vivo. *Adv Med Sci.* 2012;57(2):1-6.
20. Spangberg L, Pascon EA. The importance of material preparation for the expression of cytotoxicity during in vitro evaluation of biomaterials. *J Endod.* 1988;14(5): 247-250.
21. Franz A, Konig F, Lucas T, Watts DC, Schedle A. Cytotoxic effects of dental bonding substances as a function of degree of conversion. *Dent Mater.* 2009;25(2): 232-239.
22. de Almeida LC, Soares DG, Gallinari MO, de Souza Costa CA, Dos Santos PH, Briso AL. Color alteration, hydrogen peroxide diffusion, and cytotoxicity caused by in-office bleaching protocols. *Clin Oral Investig.* 2014. [Epub ahead of print].
23. Khedmat S, Dehghan S, Hadjati J, Masoumi F, Nekoofar MH, Dummer PM. In vitro cytotoxicity of four calcium silicate-based endodontic cements on human monocytes, a colorimetric MTT assay. *Restor Dent Endod.* 2014;39(3):149-154.
24. Heidemann D, Lampert F. Cell culture of human gingival epithelium [in German]. *Dtsch Zahnarztl Z.* 1978; 33(1):29-32.
25. Heidemann D, Lampert F. Human gingiva in cell culture [in German]. *Dtsch Zahnarztl Z.* 1980;35 (3):430-433.
26. Heidemann D, Lampert F. The testing of dental filling materials in cell cultures [in German]. *Dtsch Zahnarztl Z.* 1980;35(2):349-353.
27. Al-Nazhan S, Spangberg L. Morphological cell changes due to chemical toxicity of a dental material: an electron microscopic study on human periodontal ligament fibroblasts and L929 cells. *J Endod.* 1990;16(3): 129-134.
28. Heidemann D, Geurtsen W. Biological compatibility of Socketol tested on 2 different cell culture systems [in German]. *Dtsch Zahnarztl Z.* 1981;36(10):667-672.
29. Caughman WF, Caughman GB, Dominy WT, Schuster GS. Glass ionomer and composite resin cements: effects on oral cells. *J Prosthet Dent.* 1990;63(5):513-521.
30. Lehmann F, Leyhausen G, Geurtsen W. Cytotoxic alterations in different fibroblast cultures caused by matrix monomers [abstract No. 929]. *J Dent Res.* 1993;72:219.
31. Bouillaguet S. Biological risks of resin-based materials to the dentin-pulp complex. *Crit Rev Oral Biol Med.* 2004;15(1):47-60.
32. Cetinguc A, Olmez S, Vural N. HEMA diffusion from dentin bonding agents in young and old primary molars in vitro. *Dent Mater.* 2007;23(3):302-307.
33. Porto IC, Andrade AK, Guenes GM, Ribeiro AI, Braz R, Castro CM. In vitro potential cytotoxicity of an adhesive system to alveolar macrophages. *Braz Dent J.* 2009;20(3):195-200.

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Surgical crown lengthening: a periodontal and restorative interdisciplinary approach

Simran R. Parwani, MDS ▪ Rajkumar N. Parwani, MDS

Surgical crown lengthening helps to provide an adequate retention form for proper tooth preparation, thus enabling dentists to create esthetically pleasing and healthy restorations. Long-term stability requires accurate diagnosis and development of a comprehensive treatment plan in each case. This sequence of events stresses the importance of communication between the restorative dentist and the periodontist. This article presents 2 cases that involve surgical crown lengthening (including mucoperiosteal flap and ostectomy) for the restoration of teeth.

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Key words: periodontist, restorative dentist, surgical crown lengthening

Surgical crown lengthening procedures are intended to provide an adequate retention form, while allowing dentists to properly prepare the tooth, make impressions, place restorative margins on sound tooth structure, and adjust gingival levels for esthetics.[1-3] These lengthening procedures may include the removal of only soft tissue or both soft tissue and alveolar bone. When there is adequate attached gingiva and ≥3 mm of tissue coronal to the alveolar bone crest, soft tissue reduction alone is indicated. This reduction may be accomplished by either a gingivectomy or using the mucoperiosteal flap technique. When there is inadequate attached gingiva and <3 mm of soft tissue, a mucoperiosteal flap procedure and bone recontouring is required. In cases of caries or tooth fracture, surgery should provide at least 4 mm of clearance from the apical extent of the caries or fracture to the bone crest to ensure that restorative margins are placed on sound tooth structure with an adequate retention form.[4]

In cases involving anterior teeth, dentists should keep in mind that patient awareness and expectations have increased and that an outcome with less-than-optimal esthetics would not be acceptable.[5] An essential goal of treatment is long-term stability; in such cases, the integrity of the dentogingival junction must be respected, and restorations and the periodontium must be in harmony. A predictable, successful outcome can only be expected if a complete and accurate diagnosis is obtained and used to generate an appropriate treatment plan.[6]

When assessing a patient, it is important to determine the patient's chief complaint which could include pain, swelling, impaired function, unsatisfactory esthetics, or any combination thereof. Next, the medical status of the patient must be reviewed and vital signs recorded. This step will determine the patient's suitability for dental treatment and identify any special precautions that must be taken (for example, premedication for the prevention of bacterial endocarditis).[7] Medications such as phenytoin, cyclosporine, and calcium-channel blockers have the potential to adversely affect gingival health and esthetics.[8] It has been reported that poorly controlled diabetes and smoking predispose patients to periodontal disease and adversely affect their response to treatment.[9-11] Anticoagulant therapy, including low-dose aspirin, often must be modified to ensure adequate hemostasis during and after surgical procedures.[12,13] While there is some controversy over the absolute necessity of discontinuing aspirin for 1 week prior to surgery, prudent clinical judgment and appropriate medical consultation (when needed) are in the best interests of the patient.[14,15]

Following a thorough review of the patient's medical status, a clinical examination is conducted. This examination should begin with extraoral conditions, giving attention to facial symmetry and height, lip length and thickness, profile, and smile line. Generally, the interpupillary line is the reference point for assessing facial symmetry.[16] Face height usually is analyzed by dividing the face into thirds. The upper third often varies, depending upon the patient's hairstyle. The middle and lower thirds of the face are more involved in dental esthetic considerations. The midface is measured from the glabella (the most prominent point of the forehead between the eyebrows) to the subnasale, the point directly below the nose. The lower third of the face is measured from the subnasale to the soft tissue menton (the lower border of the chin). When measured in repose, the length of the middle third of the face should equal that of the lower third.[17] Lip length is measured from the subnasale to the lower border of the upper lip and tends to increase with age. The average lip length is 20-22 mm for young (20-30 years) women and 22-24 mm for young men.[17] In repose, young women display approximately 3-4 mm of the maxillary central incisors, while young men display approximately 2 mm less.[17] With advancing age, as a consequence of dropping (reduction of muscular tonicity) of the upper and lower lips, there is a decrease in exposure of the maxillary incisors and an increase in exposure of the mandibular incisors.[18] The smile line should be observed in a variety of situations, including rest, speech, smiling, and laughter. With a normal full smile, the upper lip should rest at the level of the midfacial gingival margins of the maxillary anterior teeth; the lower lip should rest at the incisal edges of the maxillary anterior teeth; the incisal edges of the maxillary anterior teeth should be parallel to the curvature of the lower lip.[17] When any significant discrepancies exist in ≥1 extraoral parameters, it may be unrealistic to expect intraoral procedures alone to provide a satisfactory result. In these cases, it may be necessary to consider orthognathic and/or plastic surgical procedures, or to modify patient expectations.[16,17]

A thorough intraoral examination must be conducted, combining clinical and radiographic observations. The condition

Fig. 1. Preoperative clinical view with a short clinical crown length (case No. 1).

Fig. 2. The patient in case No. 1 after an incision was made for crown lengthening.

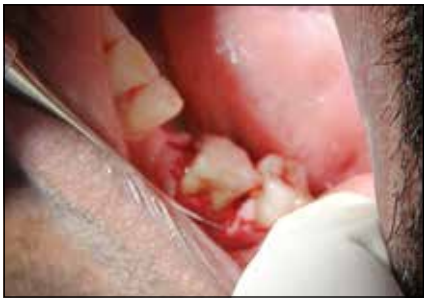

Fig. 3. A postostectomy view of the patient in case No. 1.

Fig. 4. The patient in case No. 1 after crown preparation is performed.

Fig. 5. The patient in case No. 1 after placement of a provisional restoration.

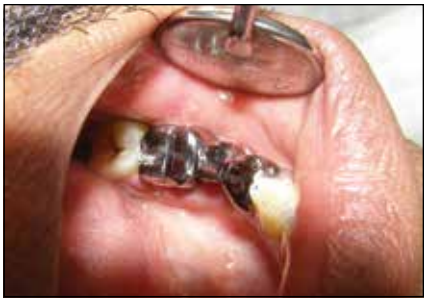

Fig. 6. The final restoration is placed (case No. 1).

and dimensions of the teeth should be determined, checking for caries, fractures, and pulpal pathoses. The height of the anatomic crown is measured from the cementoenamel junction (CEJ) to the incisal edge, while the height of the clinical crown is measured from the gingival margin to the incisal edge. Comparing these 2 measurements will determine whether short clinical crowns are a result of incisal wear or the coronal position of the gingival margin.

If excessive incisal wear has occurred, then parafunctional habits must be investigated and dealt with appropriately. Once the cause of the incisal wear has been identified and controlled, restorative procedures can be planned to replace lost tooth structure. Coronal position of the gingival margin in relation to the CEJ may be the result of delayed passive eruption or gingival enlargement. Delayed passive eruption occurs when the dentogingival junction fails to migrate apically to the vicinity of the CEJ after the tooth has erupted into occlusion. Width of keratinized gingiva may be excessive or within normal range, and the alveolar crest may be at the CEJ or 1-2 mm apical.[19] Most often, gingival enlargement is due to inflammation caused by dental plaque, but it is also associated with such medications as phenytoin, cyclosporine, and calcium channel blockers, and with pathological conditions such as hereditary gingival fibromatosis.[20] The width and thickness of keratinized gingiva must be measured, as should probing depths, clinical attachment levels, and the level of the alveolar crest with respect to the CEJ. Interproximal bone levels can be estimated using radiographs taken parallel to the long axes of the teeth. Facial and lingual bone levels can be determined through sounding with local anesthesia (often in conjunction with other procedures which require local anesthesia, such as scaling and root planing, or at the beginning of a surgical procedure). Alveolar bone thickness and any hard or soft tissue irregularities should be recorded as well. The condition of the teeth must be evaluated carefully and any necessary restorative treatment noted.[6]

Excessive gingival display

For most patients, the lower edge of the upper lip assumes a *gum wing* profile which limits the amount of gingiva that is exposed when a person smiles. Patients with a high lip line expose a zone of gingival tissue and may often express concern about their "gummy smile." The form and position of the lips during speech and smiling cannot be changed easily. However, if it is deemed necessary, the dentist can modify/control the form of the teeth and interdental papillae, as well as the position of the gingival margins and the incisal edges of the teeth. Thus, it is possible to improve dentofacial esthetics for these patients by using a combination of periodontal and restorative treatment measures.

If excessive gingival exposure is due to insufficient length of the clinical crowns, a crown lengthening procedure is indicated to reduce the amount of gingiva exposed, which in turn will alter the shape and form of the anterior teeth favorably.

For young adults with an intact periodontium, the gingival margin normally resides approximately 1 mm coronal to

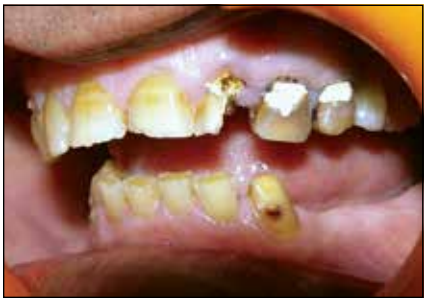

Fig. 7. A preoperative clinical view with defective restorative margins (case No. 2).

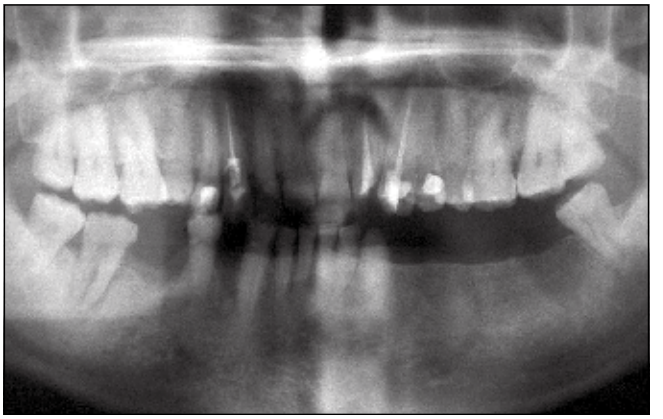

Fig. 8. A preoperative orthopantomograph of the patient in case No. 2.

the CEJ. However, some patients may have a free gingiva height >1 mm, resulting in a disproportionate gingival crown. If a patient's complaint concerns "small front teeth" and the periodontium is a thin biotype, a full exposure of anatomical crown can be accomplished by a gingivectomy or gingivoplasty. If the periodontium is a thick biotype with a bony ledge at the osseous crest, an apically positioned flap procedure is the optimal choice, as it allows for osseous recontouring. Subjects with normal occlusal relationships and incisal guidance are good candidates for this treatment. For patients in this category, the incisal line of the anterior teeth should remain unaltered, but the clinical crowns can be made longer by surgically exposing the root structure and locating the cervical margins of the restorations apical to the CEJ.

Exposure of sound tooth structure

Crown lengthening procedures may be required to solve such problems as inadequate amount of tooth structure for proper restorative therapy, subgingival location of fracture lines, and subgingival location of carious lesions. Techniques used to accomplish crown lengthening in such cases include an apically positioned flap procedure or a forced tooth eruption (with or without fiberotomy).[21] This article presents 2 case reports in which crown lengthening was performed with a mucoperiosteal flap and ostectomy.

Case report No. 1

A 32-year-old man presented to the Dental College and Research Centre, Indore, India, with a chief complaint that the crown on a posterior tooth had become loose 3-4 days earlier. The crown was short, so the patient's prosthodontist referred him to the hospital for crown lengthening surgery, as there was a strong possibility that the onlay may once again become loose at a future date. The patient was in excellent general health with no known allergies, was not on any medications, and denied using tobacco. Clinical examination revealed a short clinical crown on tooth No. 19 (Fig. 1). After a week of thorough full-mouth oral prophylaxis, surgical crown lengthening was performed on the tooth. Treatment consisted of a mucoperiosteal flap reflection, an ostectomy (to provide harmony with the adjacent teeth), and a 3 mm clearance between the final restoration margin and the alveolar crest (Fig. 2 and 3). Crown preparation for this tooth was performed at the same appointment. The restoration margins were kept supragingival as it was a posterior tooth (Fig. 4). Impressions were made for a provisional crown. The surgical wound area was irrigated and the flaps were replaced with interrupted sutures. Postoperative instructions were given. The next day, a provisional restoration was placed on tooth No. 19 (Fig. 5). With the patient's consent, a bridge restoration (with support from teeth No. 21 and No. 19; tooth No. 20 was missing) was prepared. A noneugenol periodontal pack was placed.

The pack and sutures were removed after 8 days. After allowing 1.5 months for the gingival margins to heal and stabilize, the final restoration (a white metal bridge) was placed, resulting in a remarkable improvement in the patient's periodontal health (Fig. 6). Gingival health, comfort, and optimum esthetics were achieved and maintained. Weekly follow-up visits were scheduled for the first month and biweekly visits scheduled for the next 6 months, to ensure that meticulous plaque control was maintained and optimum healing had occurred. Subsequent recall visits at 3-month intervals over the next year revealed that the patient continued to practice highly effective plaque control and was very pleased with the results.

Case report No. 2

A 52-year-old man presented to the Restorative Department of the Dental College and Research Centre, Indore, India, with defective restoration margins in his 2 left anterior teeth, for which he had received endodontic treatment 8 years earlier. The patient stated that at that time his dentist had advised him that he should get the crowns of the endodontically treated teeth placed after the treatment. However, due to personal problems, the patient could not do so. Clinical and radiographic examinations (Fig. 7 and 8) revealed extensive mutilation on the distal aspect of tooth No. 10, and a carious tooth No. 11; both teeth had received endodontic treatment previously. Tooth

No. 10 also had a large periapical lesion. The prosthodontist determined that crown lengthening (via flap and ostectomy for new restorative margins) was necessary, as the apical extent of the old cement restorations were subgingival.

To establish sound margins for the new restorations, it was necessary to extend the preparation apically in close proximity to the alveolar crest. A diagnostic wax-up was performed to establish the desired end results. Using this wax-up, a surgical guide was fabricated to facilitate the crown lengthening procedure, allow the surgeon to identify accurately the future location of the restoration margins, and ensure at least 3 mm of clearance between the margins and the crest of alveolar bone. Scaling and root planing were performed to reduce inflammation and allow for healing, and oral hygiene instructions were given. Surgical crown lengthening was performed in the region of teeth No. 9-12 under local anesthesia. The mucoperiosteal flap reflection from the buccal aspect revealed alveolar bone at the level of the CEJ. To ensure the maintenance of interdental papillae, neither palatal nor interdental soft tissue was reflected. Osseous recontouring was performed on the buccal aspects of teeth No. 10 and 11 so that the alveolar crest would be located 3 mm apical to the CEJ (Fig. 9 and 10). Using a tunneling technique, ostectomy and osteoplasty were performed in the interdental areas. After the surgical wound was irrigated, interrupted sutures were placed (Fig. 11). A noneugenol periodontal pack was placed and postoperative instructions were given. The pack and sutures were removed after 8 days, allowing the dentogingival junction to heal with the gingival margin at the level of the CEJ. Endodontic treatment was repeated for both teeth, as the temporary cement had dissolved partially and root canals were reinfected. A fiber post and core using glass ionomer cement (Miracle Mix, GC America, Inc.) was fabricated for tooth No. 10 (Fig. 12). A follow-up 2 months after the crown lengthening procedure revealed a stable, esthetic result.

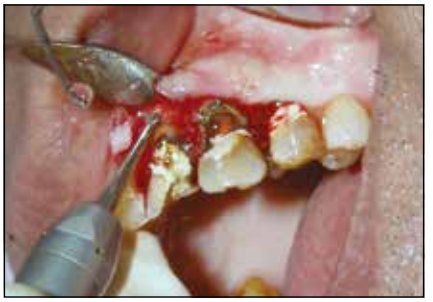

Fig. 9. An ostectomy is performed on the patient in case No. 2.

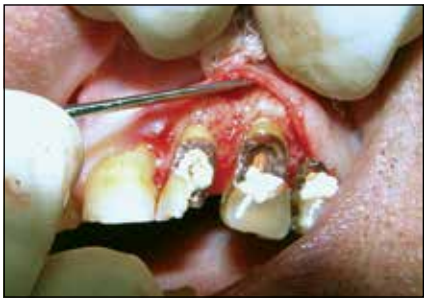

Fig. 10. The patient in case No. 2, postostectomy.

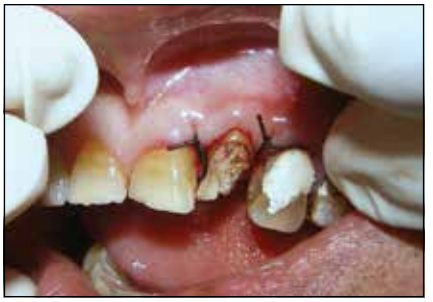

Fig. 11. Interrupted sutures are placed on the patient in case No. 2.

Fig. 12. The patient in case No. 2, after post and core placement.

Discussion

Contemporary dental treatment must result in true oral health while incorporating comfort, function, and esthetics. The key to a successful outcome with long-term stability is the establishment of an accurate diagnosis and subsequent development of a comprehensive treatment plan. The integrity of the dentogingival junction must be observed by ensuring adequate biological width. Harmony must exist between soft and hard tissues and between the periodontium of adjacent teeth.[6]

During preparation for full coverage restorations, margin placement should be guided by the position of the CEJ; as a result, interproximal margins (particularly on anterior teeth) should be more coronal than buccal and lingual margins. This approach will help ensure adequate biological width and the maintenance of healthy, intact interproximal papillae. When periodontal surgical procedures are performed in anterior areas, it is necessary to defer placement of final full coverage restorations for approximately 6 months to stabilize gingival margin levels. For patients with particularly thin buccal alveolar bone and gingiva, it may be prudent to monitor the maturation of the healing tissue for a longer period of time. For patients with relatively thick buccal alveolar bone and gingiva, it may be reasonable to place final restorations within 6 months following periodontal surgery. Effective daily plaque control and periodic recall are essential to maintain long-term stability. By following these guidelines, the clinician can promote a stable, comfortable, and functional periodontium and provide the patient with an optimal esthetic result.[6]

The sequence of events in crown lengthening highlights the importance of communication between a restorative dentist and the periodontist. There should be a minimum of 3 mm distance between the restorative margin and alveolar crest.[22] In a case of recurrent decay beyond the crown margins, the apical extent of caries (as well as maintenance of the biologic width) acts as a guide in establishing the position of the new crown margins. Planned crown margins should be placed on sound tooth structure at least 1 mm apical to the most apical extent of caries.[22]

Patients with defective restorative margins who have undergone prior endodontic therapy have been treated successfully

by a combination of orthodontic extrusion for the involved tooth (approximately 1 mm/week for 4 weeks) and surgical crown lengthening involving a mucoperiosteal flap reflection with ostectomy. Orthodontic extrusion is particularly useful in cases where interdental papillae in the vicinity of the involved teeth have lost their height.

Both of the cases presented here were treated by surgical crown lengthening with mucoperiosteal flap and ostectomy in adjunct to endodontic and prosthodontic treatments. Postoperative follow-ups were uneventful.

Conclusion

Surgical crown lengthening with mucoperiosteal flap and ostectomy in collaboration with restorative treatment helps to provide harmony of a restoration with the adjacent teeth and periodontium by maintaining at least 3 mm of clearance between the margin of the restoration and the alveolar crest. The success of the 2 cases presented here emphasizes the importance of an interdisciplinary approach between a restorative dentist and periodontist in diagnosis, treatment, and postoperative maintenance of such cases.

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References

1. Kois JC, Vakay RT. Relationship of the periodontium to impression procedures. *Compend Contin Educ Dent.* 2000;21(8):684-686.
2. Minsk L. Esthetic crown lengthening. *Compend Contin Educ Dent.* 2001;22(7):562-564.
3. Studer S, Zellweger U, Scharer P. The aesthetic guidelines of the mucogingival complex for fixed prosthodontics. *Pract Periodontics Aesthetic Dent.* 1996;8(4): 333-341.
4. Melnick PR. Preparation of the periodontium for restorative dentistry. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Carranza's Clinical Periodontology,* 10th ed. St. Louis: Saunders; 2006: 1039-1049.
5. Morley J. The role of cosmetic dentistry in restoring a youthful appearance. *J Am Dent Assoc.* 1999;130(8): 1166-1172.
6. Jorgensen MG, Nowzari H. Aesthetic crown lengthening. *Periodontol 2000.* 2001;27:45-58.
7. Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *JAMA.* 1997;277(22):1794-1801.
8. Meraw SJ, Sheridan PJ. Medically induced gingival hyperplasia. *Mayo Clin Proc.* 1998;73(12):1196-1199.
9. Grossi SG, Skrepcinski FB, DeCaro T, Zambon JJ, Cummins D, Genco RJ. Response to periodontal therapy in diabetics and smokers. *J Periodontol.* 1996;67(10 Suppl):1094-1102.
10. Preber H, Bergstrom J. Effect of cigarette smoking on periodontal healing following surgical therapy. *J Clin Periodontol.* 1990;17(5):324-328.
11. Trombelli L, Scabbia A. Healing response of gingival recession defects following guided tissue regeneration procedures in smokers and non-smokers. *J Clin Periodontol.* 1997;24(8):529-533.
12. Schafer AI. Effects of nonsteroidal anti-inflammatory therapy on platelets. *Am J Med.* 1999;106(5B):25S–36S.
13. Speechley JA, Rugman FP. Some problems with anticoagulants in dental surgery. *Dent Update.* 1992;19(5): 204-206.
14. Alexander RE. Eleven myths of dentoalveolar surgery. *J Am Dent Assoc.* 1998;129(9):1271-1279.
15. Thomason JM, Seymour RA, Murphy P, Brigham KM, Jones P. Aspirin-induced post-gingivectomy haemorrhage: a timely reminder. *J Clin Periodontol.* 1997; 24(2):136-138.
16. Allen EP. Surgical crown lengthening for function and esthetics. *Dent Clin North Am.* 1993;37(2):163-179.
17. Robbins JW. Differential diagnosis and treatment of excessive gingival display. *Pract Periodontics Aesthet Dent.* 1999;11(2):265-272.
18. Vig RG, Brundo GC. The kinetics of anterior tooth display. *J Prosthet Dent.* 1978;39(5):502-504.
19. Coslet JG, Vanarsdall R, Weisgold A. Diagnosis and classification of delayed passive eruption of the dentogingival junction in the adult. *Alpha Omegan.* 1977; 70(3):24-28.
20. Evian CI, Cutler SA, Rosenberg ES, Shah RK. Altered passive eruption: the undiagnosed entity. *J Am Dent Assoc.* 1993;124(10):107-110.
21. Wennstrom JL, Pini Prato GP. Mucogingival therapy—periodontal plastic surgery. In: Lindhe J, Karring T, Lang NP, eds. *Clinical Periodontology and Implant Dentistry.* 4th ed. New Delhi, India: Jaypee Brothers Medical Publishers (P) Ltd.; 2003:576-649.
22. Silvestri L. PERIODONTICS*: Crown Lengthening Revisited - An Integral Part of the Reconstructive Treatment Plan. Periodontics 2001.* Available at: http://www.oralhealthgroup.com/news/periodontics-crown-lengthening-revisited--an-integral-part-of-the-reconstructive-treatment-plan/1000145060/?&er=NA. Accessed September 11, 2014.

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Surface roughness of different composite resins subject to in-office bleaching

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This study used atomic force microscopy to evaluate the effects of an in-office dental bleaching protocol on the surface roughness of 3 resins: microfilled, microhybrid, and nanofilled. Two disks of each resin were prepared and evaluated. Twenty-four areas (5 x 5 µm) were scanned using an atomic force microscope. Then, each disk was treated with 35% hydrogen peroxide activated by a light emitting diode. After bleaching, the disks were scanned again. Data from the 24 areas before and after bleaching were evaluated qualitatively using topographical and 3-dimensional images, as well as profile lines. Quantitatively, roughness data (mean roughness and root mean square) were statistically evaluated using standard ANOVA and Student-Newman-Keuls tests ($P < 0.05$). The bleaching procedure increased roughness for each resin analyzed. Based on the results, it was concluded that in-office bleaching with 35% hydrogen peroxide-based gel significantly increased the surface roughness of microfilled, microhybrid, and nanofilled resins.

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Key words: dental bleaching, hydrogen peroxide, resin composites, roughness, atomic force microscopy

Restoration technology has advanced since dental resin composites were first introduced. One important advance in recent years is the application of nanotechnology to dental composites.[1] Microfilled composites were introduced in the early 1980s.[1] These composites offer a high polish (which can be maintained over time) and excellent enamel-like translucency. For these reasons, they are indicated for restoring anterior teeth and cervical abfraction lesions. However, they should not be used in heavy stress-bearing areas as their physical properties typically are inferior to those of hybrid composites.[2] Microhybrid composites are stronger than most microfilled composites and therefore can be used in both anterior and posterior teeth.[3] Microhybrid composites also provide optimal mechanical and physical properties combined with good polishing properties.[2,3]

Nanocomposites have been developed to combine the advantages of hybrid and microfilled composites in a single restorative material.[4] Nanocomposites offer low shrinkage, which is attributed to their high filler volume. These nanocomposites have favorable mechanical properties that are equal to and may surpass those of hybrid materials.[1,4] They also demonstrate better surface quality, better polish and gloss, increased retention, and increased wear resistance.[4]

Dental bleaching is a common esthetic clinical procedure. In-office bleaching involves applying a bleaching agent (typically a gel containing 35%-38% hydrogen peroxide) on the tooth surface and allowing it to remain on the teeth for 30-45 minutes. The bleaching agent is activated chemically (or by using a visible light curing lamp) to enhance the process.[5] Anterior resin composite restorations are usually removed before the bleaching procedure. However, when the restorative material is placed in distal areas—such as the premolars and molars—where there is no esthetic compromise in terms of a change in resin color after bleaching, many dentists choose not to remove the restoration.[6]

Composites with greater surface roughness can lead to increased pigment accumulation and alter the rate of water absorption, as well as cause changes in the optical properties, negatively impacting the appearance of the restoration.[6] A rougher surface accumulates more bacteria than a smooth surface, which in turn reduces the free energy of the surface.[7,8] Different techniques are used to evaluate surface roughness, including profilometry, scanning electron microscopy, and atomic force microscopy (AFM).[6,9-13]

The present study used AFM to evaluate the effect of an in-office dental bleaching protocol (using 35% hydrogen peroxide) on the surface roughness of 3 resin types: microfilled, microhybrid, and nanofilled.

Materials and methods

Three different sample groups were prepared: a microfilled resin (Durafill VS, shade A2, Heraeus Kulzer), a microhybrid resin (Opallis, shade EA2, FGM Produtos

Table. Roughness (Ra) and root mean square (RMS) surface values and standard deviations (SD) (in nm) for the different resins before and after bleaching.

	Ra		RMS	
	Initial	Final	Initial	Final
Microfilled	26.2 (4.0)^{A}	36.4 (4.2)^{B}	19.2 (5.0)^{A}	28.7 (4.8)^{B}
Microhybrid	14.9 (3.7)^{A}	23.1 (4.2)^{B}	35.4 (5.8)^{A}	47.4 (5.6)^{B}
Nanofilled	23.7 (3.9)^{A}	31.5 (6.0)^{B}	29.6 (4.7)^{A}	40.2 (7.8)^{B}

Different letters indicate statistically significant values ($P < 0.05$).

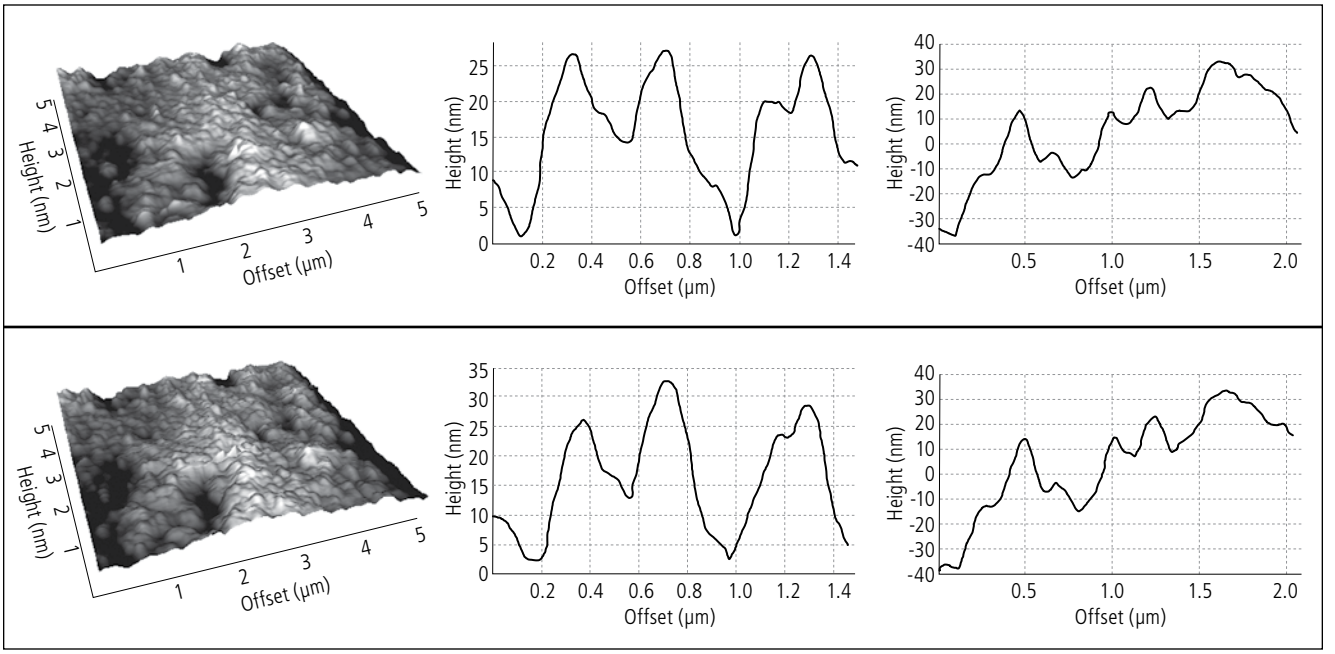

Fig. 1. *Top.* Three-dimensional images and line profiles for an area of the microfilled resin before bleaching. *Bottom.* The same area after bleaching treatment.

Odontologicos), and a nanofilled resin (Filtek Z350, shade A2, 3M ESPE). Disc-shaped standardized specimens were prepared (9 mm diameter x 2 mm thick) with the use of a Mylar bipartite matrix and stainless steel bolts for fixing. The resins were mixed and placed in the matrix with a spatula until each specimen was completely filled. At that point, each resin sample was covered with 0.05 mm polyester tape and compressed with a glass microscope slide to obtain a flat surface. Photocuring was performed in a single step by touching a light emitting diode (LED) tip (output 600 mW/cm^2) to the glass for 40 seconds. Two discs were evaluated in each group.

Samples were stored in neutral artificial saliva and placed in an oven at 37°C. After 24 hours, samples were washed with distilled water and polished with 1200 grit paper. The samples were washed again after polishing and stored in artificial saliva for 7 days.

Next, the samples were rinsed with distilled water, dried using a nitrogen flow, and analyzed by an AFM (JPK Instruments AG) in contact mode. The temperature was kept close to 26°C (relative humidity ranged from 45% to 55%). For all analyses, cantilevers (CSC17/Al BS, Micromasch USA) with a curvature radius <10 nm were used. These cantilevers had a nominal resonant frequency of 12 KHz and an elastic modulus of 0.15 N/m. All images were acquired at a resolution of 512 x 512 points. In each sample, areas of 50 x 50 µm were evaluated for an overall surface analysis. Then, 24 different areas (5 x 5 µm) were recorded for an initial evaluation.

The samples were submitted to bleaching, using a solution containing 3 standard droplets of hydrogen peroxide and 1 droplet of thickening agent. The mixture was homogenized and applied immediately on the surfaces of the standardized specimens; 1 minute after application, the mixture was photocured for 20 seconds by an LED (Radii, SDI (North America), Inc.). The LED intensity was kept constant (650 to 700 mW/cm^2) at a distance of 5-10 mm from the resin. After 3 minutes, the samples were photocured again using the same procedures. The bleaching solution was left on the resin for a total of 15 minutes. The procedure was repeated 6 times to simulate 2 sessions of 3 in-office procedures, the maximum protocol suggested by the manufacturer.

The 24 areas were scanned again postbleaching. The mean roughness value (Ra), the root mean squared (RMS) height of the surface topography roughness values, and the line profiles were obtained (IP software, JPK Instruments AG).

Data from the initial and final evaluations were evaluated qualitatively, using topographical and 3-dimensional (3D) images, as well as profile lines. Quantitatively, the roughness data was evaluated statistically using standard ANOVA and Student-Newman-Keuls tests ($P < 0.05$).

Results

The Ra and RMS values for the different resins are cited in the Table. An increase was observed in the roughness values for all 3 resins. Statistical analyses confirmed these findings. Figure 1 presents a representative 3D image and line profiles for an area on the microfilled resin disk. Comparing the initial and final line profiles, it was observed that the bleaching procedure resulted in nanometric changes on the surface. This behavior was observed also for the microhybrid and nanofilled resin disks (Fig. 2 and 3).

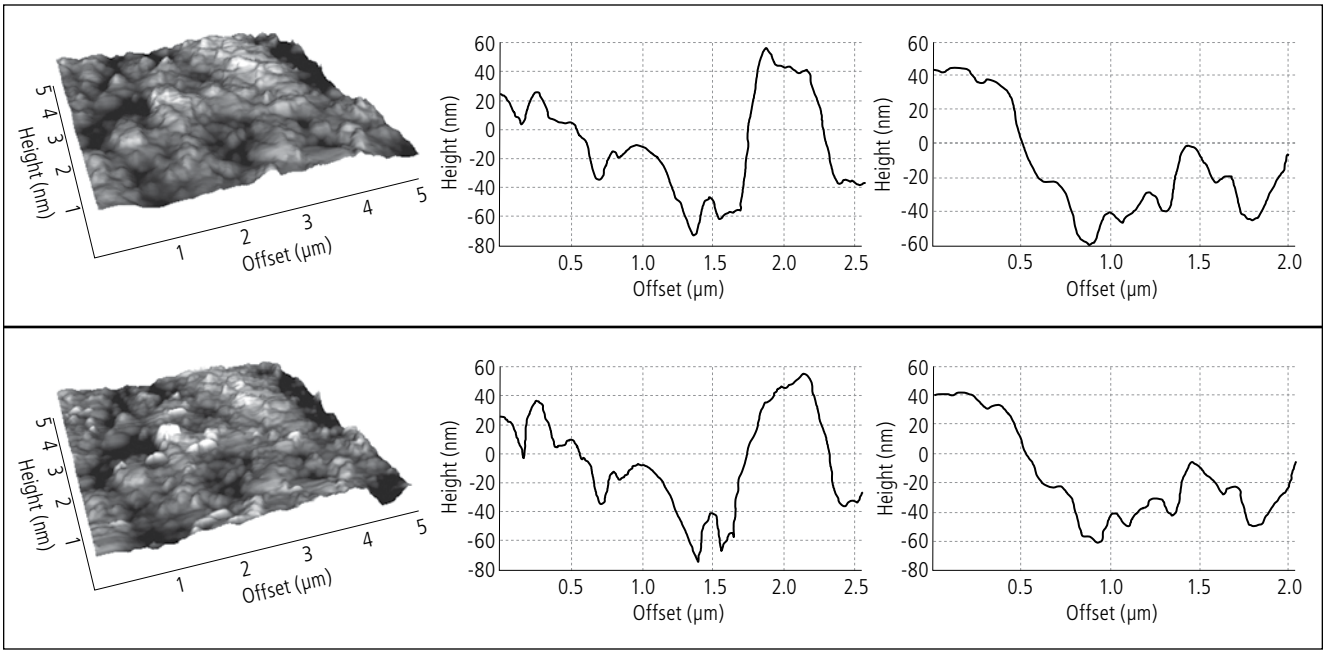

Fig. 2. *Top.* Three-dimensional images and line profiles for an area of the microhybrid resin before bleaching. *Bottom.* The same area after bleaching treatment.

Discussion

There may be times when it is necessary to replace a restorative resin composite for esthetic reasons. To the naked eye, resin composite restorations in posterior teeth may have an imperceptible postbleaching color, since there is less light in the region and consequently less visual acuity. Another situation to consider is the maintenance of bleaching, which is periodically indicated in patients who have previously been submitted to bleaching treatment, as tooth staining may reappear 1-4 years after the procedure.[14]

The present study used AFM to evaluate how an in-office dental bleaching protocol (utilizing 35% hydrogen peroxide—the maximum dose recommended by the manufacturer) affected the surface roughness of 3 resins. Previous studies have evaluated the effect of bleaching with 35% hydrogen peroxide on resin surfaces; however, to the best of the authors' knowledge, no study has used AFM scanning before and after a bleaching procedure using 35% hydrogen peroxide at the maximum dose on microfilled, microhybrid, and nanofilled resins.[6-8,15-18]

Topographical analysis of each surface was measured using Ra and RMS values. Ra corresponds to the arithmetic mean of the absolute values of the profile. As it is a mean value, it does not always provide a direct indication of the state of the surface. The profile analysis has the advantage of being used most frequently in research related to roughness, and thus allows comparisons to be made among different scientific findings.[15-18] RMS describes the root mean squared height of the surface topography in relation to the mean plane.[19]

In the present study, significant differences in the roughness were found before and after bleaching, regardless of the area analyzed. The overall topography did not change significantly; however, significant modifications were found in the nanometric scale. Profile lines of the same areas confirmed the nanometric modifications before and after dental bleaching, in keeping with previous studies.[15,17,18,20] Other studies did not discover such alterations, possibly because different methodologies were used; in addition, different products and formulations of bleaching substances may have had different effects on the surfaces.[7,8,16,21] Exposure time and peroxide concentration could affect resin surfaces as well.

The present study evaluated the effect of in-office bleaching on the roughness of different composite resins. Using different experimental models on surface areas before and after bleaching demonstrated that the procedure increased surface roughness by introducing nanometric changes on the surface.

Conclusion

In-office dental bleaching performed in office with 35% hydrogen peroxide-based gel—used at the maximum dose recommended by the manufacturer—increased the surface roughness of microfilled, microhybrid, and nanofilled resins. Additionally, since the methodology used the same area before and after treatment, problems related to area selection were reduced. It is possible to conclude that the resultant topographical alterations were caused exclusively by the action of the bleaching agent.

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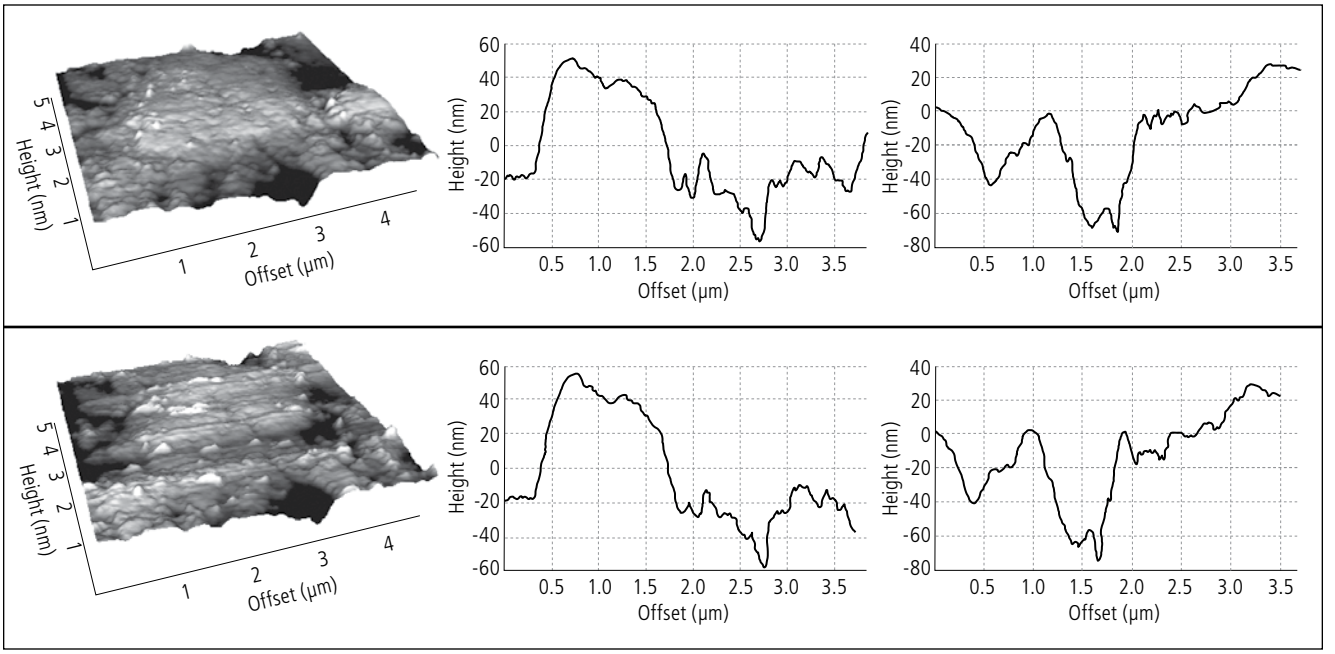

Fig. 3. *Top.* Three-dimensional images and line profiles for an area of the nanofilled resin before bleaching. *Bottom.* The same area after bleaching treatment.

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References

1. Beun S, Glorieux T, Devaux J, Vreven J, Leloup G. Characterization of nanofilled compared to universal and microfilled composites. *Dent Mater.* 2007;23(1):51-59.
2. Ferracane JL. Current trends in dental composites. *Crit Rev Oral Biol Med.* 1995;6(4):302-318.
3. Ritter AV. Direct resin-based composites: current recommendations for optimal clinical results. *Compend Contin Educ Dent.* 2005;26(7):481-482, 484-490.
4. Janus J, Fauxpoint G, Arntz Y, Pelletier H, Etienne O. Surface roughness and morphology of three nanocomposites after two different polishing treatments by a multitechnique approach. *Dent Mater.* 2010;26(5): 416-425.
5. da Costa J, Lubisich E, Ferracane J, Hilton T. Comparison of efficacy of an in-office whitening system used with and without a whitening priming agent. *J Esthet Restor Dent.* 2011;23(2):97-104.
6. Varanda E, do Prado M, Simao RA, Dias KR. Effect of in-office bleaching agents on the surface roughness and morphology of different dental composites: an AFM study. *Microsc Res Tech.* 2013;76(5):481-485.
7. Moraes RR, Morimon JL, Schneider LF, Correr Sobrinho L, Camacho GB, Bueno M. Carbamide peroxide bleaching agents: effects on surface roughness of enamel, composite and porcelain. *Clin Oral Invest.* 2006;10(1):23-28.
8. Silva MF, Davies RM, Stewart B, et al. Effect of whitening gels on the surface roughness of restorative materials in situ. *Dent Mater.* 2006;22(10):919-924.
9. Hegedus C, Bistey T, Flora-Nagy E, Keszthelyi, Jenei A. An atomic force microscopy study on the effect of bleaching agents on enamel surface. *J Dent.* 1999; 27(7):509-515.
10. Schemehorn B, Gonzalez-Cabezas C, Joiner A. A SEM evaluation of a 6% hydrogen peroxide tooth whitening gel on dental materials in vitro. *J Dent.* 2004; 32(Suppl 1):35-39.
11. Tholt de Vasconcellos B, Miranda-Junior WG, Prioli R, Thompson J, Oda M. Surface roughness in ceramics with different finishing techniques using atomic force microscope and profilometer. *Oper Dent.* 2006;31(4): 442-449.
12. de Andrade IC, Basting RT, Lima-Arsati YB, do Amaral FL, Rodrigues JA, Franca FM. Surface roughness evaluation and shade changes of a nanofilled resin composite after bleaching and immersion in staining solutions. *Am J Dent.* 2011;24(4):245-249.
13. Mourouzis P, Koulaouzidou EA, Helvatjoglu-Antoniades M. Effect of in-office bleaching agents on physical properties of dental composite resins. *Quintessence Int.* 2013;44(4):295-302.
14. American Dental Association. Home-use bleaching agents. Dental product spotlight. *J Am Dent Assoc.* 2001;132(8):1292-1293.
15. Turker SB, Biskin T. Effect of three bleaching agents on the surface properties of three different esthetic restorative materials. *J Prosthet Dent.* 2003;89(5):466-473.
16. Duschner H, Gotz H, White DJ, Kozak KM, Zoladz JR. Effects of hydrogen peroxide bleaching strip gels on dental restorative materials in vitro: surface microhardness and surface morphology. *J Clin Dent.* 2004; 15(4):105-111.
17. Basting RT, Fernandez Y, Fernandez C, Ambrosano GM, de Campos IT. Effects of a 10% carbamide peroxide bleaching agent on roughness and microhardness of packable composite resins. *J Esthet Dent.* 2005;17(4): 256-262.
18. Gurgan S, Yalcin F. The effect of 2 different bleaching regimens on the surface roughness and hardness of tooth-colored restorative materials. *Quintessence Int.* 2007;38(2):e83-e87.
19. Prado M, Gusman H, Gomes BP, Simao RA. Effect of disinfectant solutions on gutta-percha and resilon cones. *Microsc Res Tech.* 2012;75(6):791-795.
20. Bailey SJ, Swift EJ Jr. Effects of home bleaching products on composite resins. *Quintessence Int.* 1992; 23(7):489-494.
21. Langsten RE, Dunn WJ, Hartup GR, Murchison DF. Higher-concentration carbamide peroxide effects on surface roughness of composites. *J Esthet Restor Dent.* 2002;14(2):92-96.

Manufacturers

FGM Produtos Odontologicos, Joinville, SC, Brazil
55.47.3441.6100, www.fgm.ind.br

Heraeus Kulzer, South Bend, IN
800.435.1785, www.heraeus-kulzer-us.com

JPK Instruments AG, Pittsford, NY
585.249.7692, usa.jpk.com

Micromasch USA, Lady's Island, SC
866.776.8477, www.spmlips.com

SDI (North America), Inc., Bensenville, IL
800.228. 5166, www.sdi.com.au

3M ESPE, St. Paul, MN
888.364.3577, solutions.3m.com

An in vitro comparison of antimicrobial toothbrushes

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The purpose of this study was to determine the efficacy of toothbrushes that advertise self-disinfecting, antimicrobial properties due to the inclusion of silver nanoparticles or chlorhexidine in the bristles. Three different types of toothbrushes—silver nanoparticle, chlorhexidine-coated, and a control—were submerged in suspensions of *Streptococcus mutans* and *Candida albicans.* At designated times postinoculation, organisms were removed from the toothbrush heads, then serially diluted, plated, and incubated. The colony-forming units (CFUs) were counted and a mean percent reduction was determined for each organism group. With the *S. mutans* groups, the chlorhexidine-coated toothbrushes had significantly greater percent reduction in CFUs at all 3 time points compared to the control or silver nanoparticle toothbrushes. With the *C. albicans* groups, neither the chlorhexidine-coated nor the silver nanoparticle toothbrushes had a significant reduction in CFUs compared to the control. Neither of the antimicrobial toothbrushes delivered the advertised claim of a 99.9% reduction in CFUs with either microorganism. However, the inclusion of chlorhexidine in toothbrush bristles appeared to be the most promising of the methods tested for toothbrush self-disinfection.

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Recent advertisements for antimicrobial toothbrushes claim to remove up to 99.9% of residual bacteria from toothbrush heads that can cause caries, halitosis, colds, and flu. The elimination of toothbrush microbial contaminants could be beneficial to patients, as these same pathogenic organisms have been associated with a wide variety of systemic diseases.[1] Studies have linked significant bacteremias to routine toothbrushing, especially in patients with severe periodontitis.[2] Moreover, bacteria from the environment, including enterobacteria found in bathrooms, can embed themselves on toothbrush bristles.[3] Toothbrush decontamination could provide a means of preventing the inoculation or reintroduction of microorganisms from infected to noninfected tissues. A toothbrush that could help in controlling reinfection or cross-contamination following everyday toothbrushing would be a significant health benefit.

Many of the microorganisms that make up the oral flora have been linked to systemic ailments, which include circulatory, renal, pulmonary, and intestinal infections. Some authors have recommended disposable toothbrushes for patients that are taking immunosuppressants or who are undergoing transplant or other major surgeries, thereby avoiding any deleterious bacterial encounters.[4] The introduction of an antimicrobial toothbrush could provide a means to promote health and wellness, as any potential bacteremia could jeopardize patients with serious ailments such as diabetes or immunosuppressive diseases. In addition, the elimination of cariogenic bacteria left on a toothbrush could help improve the caries status of a patient. Kozai et al found viable *S. mutans* on toothbrushes several hours following use.[5]

Even after only 1 use, a new toothbrush harbors a complex array of microorganisms, such as staphylococci, fungi, and pseudomonas.[6] It is plausible that multiple strains of bacteria—which may include cariogenic and putative periodontal pathogens—could be introduced into the oral cavity with further use of the same brush, thus heightening the health risk in susceptible patients. A 2007 study found that air-drying of toothbrushes was an incomplete method for disposing of microorganisms.[6] In fact, it has been shown that in a moist environment, approximately half the original amount of tested viral contaminants flourished on a toothbrush after 7 days.[4] Herpes simplex viruses have also been shown to survive on toothbrushes at 1 week.[7] Al-Ahmad et al reported that microorganisms can penetrate the mucous membrane during routine toothbrushing of inflamed gingiva, enter the circulatory system, and then undermine the healing of dental implants or proceed to life-threatening endocarditis or pneumonia infections.[1] A 1989 dog study by Glass et al found that brushing with a self-contaminated toothbrush produced more gingival and oral lesions than using a sterile toothbrush, and these lesions showed delayed healing with the reintroduction of microorganisms.[8] Nascimento et al demonstrated that Candida species, such as *C. albicans,* can be transmitted via toothbrushes to immunocompromised individuals.[9] Sato et al found that rinsing with water alone was not an effective form of toothbrush care.[3]

A 1994 study by Glass & Jensen showed ultraviolet sanitation devices to be useful in reducing the number of bacteria and viruses on toothbrushes.[7] Other studies have sought to quantify the microbial reduction on toothbrushes by chlorhexidine treatments.[3,8,10,11] Nelson-Filho et al found a chlorhexidine rinse to be a very effective means of inhibiting biofilm formation on toothbrush bristles, while Aysegul et al and Sato et al produced similar results using a chlorhexidine spray.[3,10,11] Alternatively, Glass et al questioned whether antimicrobial rinses could successfully penetrate into the biofilm aggregates that cover toothbrushes in order to affect the entire microbial mass.[8]

Toothbrushes with inherent antimicrobial properties could prove valuable in preventing the establishment of these microbial colonies. Yokosuka et al studied the bacterial content on chlorhexidine-coated filaments of toothbrushes and determined that these brushes could reduce the bacterial load, with the antibacterial activity of the tip and base of the filament lasting for 8 and 20 days,

respectively.[12] A more recent study by Turner et al could not find any statistically significant difference between an experimental brush with a chlorhexidine coating and a control group.[13] Similarly, Efstratiou et al found that triclosan-coated toothbrushes failed to limit the bacterial contamination.[6]

Another method of toothbrush sanitation involves incorporating metallic nanoparticles into the bristles, thereby imparting an intrinsic means of controlling the oral biofilm that resides on the toothbrush following routine use. Silver and other metallic nanoparticles are now commonplace in medicine, particularly in molecular imaging, drug delivery, diagnosis and treatment of cardiovascular diseases, wound healing, and the development of materials and medical devices with antimicrobial properties.[14] Silver nanoparticles have been shown to act as an effective antimicrobial, though the exact mechanism remains unclear.[15] Studies have suggested that silver nanoparticles contribute to structural changes in bacterial membranes, which result in cell death.[15,16] According to Allaker, bacteria that makes contact with silver nanoparticles will absorb silver ions, which inhibit respiratory enzymes and help generate free radicals, subsequently damaging the cell membrane.[15] Felloni et al found that toothbrush heads coated with silver particles yielded antimicrobial effects, whereas Quirynen et al failed to note any antibacterial properties when toothbrush tufts were coated with zeolithic crystals of silver and zinc.[16,17] Al-Ahmad et al examined toothbrushes with silver particle heads and found them to contain even more *C. albicans* than the control group, which led them to suggest that any reduction in bacteria on silver-coated brushes may be due to the dehydration of the brush head.[1]

Such findings seem to contradict the claims of manufacturers who claim their toothbrushes have self-disinfection properties. Advertisements for a silver nanoparticle toothbrush (Mouth Watchers Superior Manual Toothbrush, Mouth Watchers) and a chlorhexidine-coated toothbrush (GUM Travel Toothbrush, Sunstar Americas, Inc.) both advertise that their products are an effective, low-cost, and simple means of toothbrush decontamination, which warranted study. The bristles of the Mouth Watchers Superior Manual Toothbrush are embedded with silver nanoparticles, which the manufacturer claims will eliminate 99.9% of bacteria in 6 hours.[18] The bristles of the GUM Travel Toothbrush are coated with chlorhexidine.[19] These toothbrushes claim to eliminate residual toothbrush microorganisms. Dental providers should be aware of toothbrushes that could prove useful in preventing reinfection and cross-contamination of oral microorganisms.

The purpose of this study was to determine the antimicrobial characteristics of 3 toothbrush types—silver nanoparticle, chlorhexidine-coated, and a control with no antimicrobial claims. The research was geared toward measuring the extent of residual microorganism contaminants that remain on each type of toothbrush after testing at specified time points utilizing 2 common intraoral microbes, *S. mutans* and *C. albicans.* The null hypothesis to be tested was that there would be no significant difference in percent reduction in CFUs based on brush type or microorganism over time.

Materials and methods

A total of 240 toothbrushes—80 GUM Summit toothbrushes (Sunstar Americas, Inc.) as the control group, 80 Mouth Watchers Superior Manual silver nanoparticle toothbrushes, and 80 GUM Travel chlorhexidine-coated toothbrushes—were included in this study. All brushes were sterilized in a low temperature hydrogen peroxide plasma sterilization system (Sterrad NX, Advanced Sterilization Products) before testing. The organisms tested, *S. mutans* ATCC 35668 and *C. albicans* ATCC 10231, were grown on trypticase soy agar with 5% sheep blood (BBL Trypticase Soy Agar (TSA II), Becton, Dickinson and Company). *S. mutans* plates were incubated at 35 ± 2°C for 24 hours in 5% CO_2. *C. albicans* plates were incubated at 35 ± 2°C for 48 hours in ambient air. The inoculation suspension of *S. mutans* was prepared by growing the organism overnight for 12 hours at 35 ± 2°C in trypticase soy broth (TSB) (Becton, Dickinson and Company) and then diluting the broth culture 1:10 with fresh TSB to achieve a final inoculation suspension of approximately 1.5×10^7 CFU/mL. The inoculation suspension of *C. albicans* was prepared by growing the organism overnight at 35 ± 2°C in TSB to achieve a final inoculation suspension of approximately 1.5×10^7 CFU/mL.

Microorganisms were placed on the toothbrushes by submerging them in the inoculation suspensions so that the heads were covered completely. Forty brushes of each brush type were each placed into an individual tube containing the *S. mutans* inoculum, where they remained for 3 minutes. The procedure was repeated with 40 of each brush type in the *C. albicans* inoculum. Following that, the toothbrushes were rinsed for <30 seconds with 40 ml of sterile water, then placed in a standing position (with the heads up) in sterile racks and held at room temperature.

The toothbrush types were then randomly subdivided into groups of 10 to be tested at 0, 8, 16, and 24 hours. At each time point, the toothbrushes of each group were placed in a standardized amount of sterile saline sufficient to cover the heads. Brushes were vortex-mixed for 15 seconds to remove organisms from the toothbrush head. The brushes were discarded after the vortex mixing. The saline was serially diluted and plated on TSA II. All plates were incubated at 35 ± 2°C. *S. mutans* plates were incubated for 48 hours in 5% CO_2 and *C. albicans* plates were incubated for 72 hours in ambient air. After incubation, CFUs on the plates were counted and the CFU/mL calculated.

The mean CFU/mL recovered for each test group at each time point was calculated and converted to log10 format to discern the percent reduction of microorganisms from baseline (0 hours). The CFU/mL at subsequent time points was compared to the first time point to determine the decrease in CFU/mL recovered over time. The rate of reduction for the organism groups was then compared. A 3-log10 reduction was determined to be equal to a 99.9% killing of bacteria. If these brushes were to indeed provide clinically effective disinfection, then a 99.9% reduction in microbes from baseline would be needed.[20] Groups were compared using a repeated measures ANOVA to examine the effects of brush type and bacteria on percent reduction in CFU over time ($\alpha = 0.05$).

Results

The repeated measures ANOVA found a significant difference based on brush type ($P = 0.022$) but not based on microorganism ($P = 0.091$); however, there were significant interactions over time ($P < 0.05$). The data was further analyzed with multiple 1-way ANOVAs per type of microorganism. A Bonferroni correction was applied because multiple comparisons were done between groups per microorganism ($\alpha = 0.008$). Significant differences were found between groups based on brush type or time per microorganism ($\alpha < 0.008$) (Table).

Table. Percent reduction in colony-forming units (CFUs) per microorganism and brush type over time.

		Percent reduction (standard deviation) in CFUs		
Microorganism	Brush type	8 hours	16 hours	24 hours
Streptococcus mutans	Control	88.6 (6.5)^{Bb}	96.3 (3.0)^{Ab}	95.4 (2.8)^{Ab}
	Chlorhexidine	98.8 (1.0)^{Aa}	99.8 (0.1)^{Aa}	99.3 (0.8)^{Aa}
	Silver nanoparticle	90.6 (6.3)^{Ab}	93.0 (2.6)^{Ac}	96.3 (2.3)^{Ab}
Candida albicans	Control	75.6 (25.9)^{Ba}	96.2 (5.4)^{Aa}	98.7 (1.7)^{Aab}
	Chlorhexidine	95.6 (3.9)^{Aa}	95.0 (7.7)^{Aa}	97.5 (1.2)^{Ab}
	Silver nanoparticle	72.1 (48.9)^{Aa}	97.4 (2.4)^{Aa}	99.5 (0.3)^{Aa}

Groups with the same superscript uppercase letter per row or superscript lowercase letter per column were not significantly different ($P > 0.008$).

With the *S. mutans* groups, at all 3 time points, the chlorhexidine-coated brush had significantly greater percent reduction in CFUs compared to both the control and silver nanoparticle brush groups. The percent reduction in CFUs for the control and silver nanoparticle brush groups were not significantly different from each other, except at 16 hours, when the percent reduction for the control brush was significantly greater ($P < 0.008$) than for the silver nanoparticle brush. The control brush also had a significantly greater percent reduction at times 16 and 24 hours than at 8 hours ($P < 0.008$). However, there were no significant differences between the time points of the chlorhexidine-coated and silver nanoparticle brushes.

With the *C. albicans* groups, no difference was found between the 3 brush types at 8 and 16 hours. At 24 hours, the silver nanoparticle brush had significantly greater reduction in CFUs compared to the chlorhexidine-coated brush, but neither were significantly different from the control group. All the brush groups had significantly greater reductions in CFUs at 16 and 24 hours than those recorded at 8 hours. Neither the chlorhexidine-coated nor the silver nanoparticle brush showed any individual statistical difference at any of the time points, as the 8-, 16-, and 24-hour samples showed similar reductions.

Discussion

This study sought to determine whether the purported claims of toothbrush self-disinfection were possible, and to see how time may play a role in this disinfection. This research did not quantify the actual benefit of any disinfection. However, other studies have shown at least a possible association between toothbrush disinfection and the maintenance of health in certain populations.[2,4,5] While the level of disinfection provided by a brush could change over time, this study did not try to factor in this variable, and should be the subject of future research.

In order for either the silver nanoparticle or chlorhexidine-coated brush manufacturers to make the claim of clinical disinfection, 99.9% of the microorganisms should have been eliminated by the 8-hour mark; this same level should have been maintained at 16- and 24-hours.[20] Conversely, if the control brush surpassed this 99.9% threshold, then a reduction in microbes could be seen as a normal factor of time, which could possibly eliminate the need for further toothbrush disinfection. Finally, the authors assumed that 8 hours would be a reasonable interval between brushings. The subsequent measurements at 16 and 24 hours were used to verify the prior reading and discover any trends.

After a review of the results, the null hypothesis was rejected. There were significant differences based on brush type and time after contamination, but the differences were specific to the type of microorganism. In the *S. mutans* groups at 8 hours, the percent reduction in CFUs of the chlorhexidine-coated brush was significantly greater than the other 2 brushes. This trend continued for this brush type at 16 and 24 hours, with reductions of 99.8% and 99.3%, respectively. Furthermore, the 99.8% reduction indicated that the chlorhexidine-coated brush was nearly clinically disinfected at 16 hours. The control brush showed significant improvement from 8 to 16 hours, which suggests that *S. mutans* is subject to natural reduction under normal circumstances with the passage of time, although not enough to achieve clinical disinfection without added measures. Any differences in disinfection over time were not apparent with the chlorhexidine-coated brush, which suggests that disinfection was maintained over time. At all time points, the silver nanoparticle brush performed worse than the chlorhexidine-coated brush, and it was statistically worse than the control brush at 16 hours. This indicates that the silver nanoparticle brush did not perform any better than the standard control toothbrush and, furthermore, that it does not provide the level of effective disinfection that its manufacturer claims.

Looking at all time points, the chlorhexidine-coated brush came close to the 99.9% disinfection threshold for *S. mutans,* and it was the superior brush when compared to the silver nanoparticle brush and the control. A 2009 study by Turner et al could find no significant difference between control and chlorhexidine-coated brushes.[13] However, the present study linked the chlorhexidine-coated brush to a notable improvement against *S. mutans* when compared to both the control and silver nanoparticle toothbrushes.

The *C. albicans* groups did not present any identifiable trends between the brush types at 8 and 16 hours, with all 3 types being statistically similar in terms of percent reduction. When the 24-hour brushes were sampled, the silver nanoparticle brush was statistically better than the chlorhexidine-coated brush—but neither were significantly different that the control brush. Neither the chlorhexidine-coated nor the silver nanoparticle brush showed any individual statistical difference at any of the time points, as the 8-, 16-, and 24-hour samples showed similar reductions. The control brush did display a significant increase in reduction at 16 and 24 hours compared to 8 hours, so perhaps the disinfection properties of the chlorhexidine-coated and the silver nanoparticle brush were more evident at the earlier 8-hour point. It also could be that the nature of the testing circumstances did not allow *C. albicans* to flourish on a brush after 16 hours, regardless of the disinfection properties of the toothbrush.

Silver nanoparticles have proven to be effective in disinfection; however, in this study they did not produce the 99.9% reduction promoted by the manufacturer—nor were they as effective against *S. mutans* as the chlorhexidine group.[18] These results are in keeping with previous studies in showing that silver nanoparticles were not effective in toothbrush disinfection, at least when *S. mutans* and *C. albicans* were sampled.[1,17]

Conclusion

The chlorhexidine-coated toothbrush demonstrated significantly greater reduction in CFUs compared to both the control and silver nanoparticle toothbrushes when subjected to *S. mutans.* With the *C. albicans* groups, no clear differences were evident between the brush types. The silver nanoparticle brush did not meet the stated effectiveness of disinfection with the microorganisms used in this study.

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Disclaimer

The views expressed in this study are those of the authors and do not reflect the official policy of the United States Air Force, the Department of Defense, or the United States government. The authors do not have any financial interest in the companies whose materials are discussed in this article.

References

1. Al-Ahmad A, Wiedmann-Al-Ahmad M, Deimling D, et al. An antimicrobial effect from silver-coated toothbrush heads. *Am J Dent.* 2010;23(5):251-254.
2. Sconyers JR, Crawford JJ, Moriarty JD. Relationship of bacteremia to toothbrushing in patients with periodontitis. *J Am Dent Assoc.* 1973;87(3):616-622.
3. Sato S, Pedrazzi V, Guimaraes Lara EH, Panzeri H, Ferreira de Albuquerque RF Jr, Ito IY. Antibacterial spray for toothbrush disinfection: an in vivo evaluation. *Quintessence Int.* 2005;36(10):812-816.
4. Glass RT, Lare MM. Toothbrush contamination: a potential health risk? *Quintessence Int.* 1986;17(1): 39-42.
5. Kozai K, Iwai T, Miura K. Residual contamination of toothbrushes by microorganisms. *ASDC J Dent Child.* 1989;56(3);201-204.
6. Efstratiou M, Papaioannou W, Nakou M, Ktenas E, Vrotsos IA, Panis V. Contamination of a toothbrush with antibacterial properties by oral microorganisms. *J Dent.* 2007;35(4):331-337.
7. Glass RT, Jensen HG. The effectiveness of a u-v toothbrush sanitizing device in reducing the number of bacteria, yeasts and viruses on toothbrushes. *J Okla Dent Assoc.* 1994;84(4):24-28.
8. Glass RT, Martin ME, Peters LJ. Transmission of disease in dogs by toothbrushing. *Quintessence Int.* 1989; 20(11):819-824.
9. Nascimento AP, Watanabe E, Ito IY. Toothbrush contamination by Candida spp. and efficacy of mouthrinse spray for their disinfection. *Mycopathologia.* 2010; 169(2):133-138.
10. Nelson-Filho P, Faria G, da Silva RA, Rossi MA, Ito IY. Evaluation of the contamination and disinfection methods of toothbrushes used by 24- to 48-month-old children. *J Dent Child (Chic).* 2006;73(3):152-158.
11. Aysegul O, Elgin IE, Gulcin A, Nedim S. The efficacy of chlorhexidine spray vs mouthwash in the microbial contamination of child toothbrushes. *J Dent Child (Chic).* 2007;74(3):177-181.
12. Yokosuka N, Tanaka T, Ebisudani K, Iwai T. Studies on bacterial contamination of chlorhexidine coated filaments of the toothbrush [in Japanese]. *Nihon Shishubyo Gakkai Kaishi.* 1989;31(3):960-969.
13. Turner LA, McCombs GB, Hynes WL, Tolle SL. A novel approach to controlling bacterial contamination on toothbrushes: chlorhexidine coating. *Int J Dent Hyg.* 2009;7(4):241-245.
14. Martínez-Gutierrez F, Thi EP, Silverman JM, et al. Antibacterial activity, inflammatory response, coagulation and cytotoxicity effects of silver nanoparticles. *Nanomedicine.* 2012;8(3):328-336.
15. Allaker RP. The use of nanoparticles to control oral biofilm formation. *J Dent Res.* 2010;89(11):1175-1186.
16. Felloni A, Brambilla E, Cagetti MG, Strohmenger L. Bacterial contamination of toothbrushes: a clinical study [in Italian]. *Il Dentista Mod.* 1999;17:47-51.
17. Quirynen M, De Soete M, Pauwels M, Gizani S, Van Meerbeek B, van Steenberghe D. Can toothpaste or a toothbrush with antibacterial tufts prevent toothbrush contamination? *J Periodontol.* 2003;74(3):312-322.
18. Mouth Watchers Innovative Dental Care. *Adult Superior Manual Toothbrush.* Available at: http://www.mouthwatchers.com/collections/all-products/products/adult-superior-manual-toothbrush. Accessed August 26, 2014.
19. Sunstar Americas, Inc. *GUM Travel Toothbrush, Twin Pack.* Available at: http://www.gumbrand.com/products/travel-twinpack. Accessed August 26, 2014.
20. Versalovic J. *Manual of Clinical Microbiology.* 10th ed. Washington, DC: ASM Press; 2011.

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Evaluation of the association between periodontal disease and diabetic retinopathy

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Periodontitis may act as a risk factor for diabetes and its various complications such as cardiorenal complications. The current literature is deficient regarding the association between periodontal disease and diabetic retinopathy (DR). This cross-sectional study of 100 type II diabetes patients sought to evaluate the association of periodontal disease with the occurrence and severity of DR. The results showed a definite association between periodontal disease status and the occurrence of DR, although a cause and effect relationship could not be established.

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The association between periodontal disease and diabetes has been explored in several studies over the years, and it is generally accepted that periodontal disease is more prevalent and more severe in diabetic patients than those without the disease.[1] Uncontrolled hyperglycemia in diabetic patients may predispose them to various complications. Most of these complications are vascular in nature. Macrovascular changes may lead to an increased risk of myocardial infarction and stroke as a result of atherosclerosis, while microvascular pathologies include retinopathy, end stage renal disease, neuropathies, poor wound healing, enhanced risk of infection, and periodontal disease.[2,3]

Diabetic retinopathy (DR) is a microvascular complication of diabetes and a leading cause of blindness. It occurs when diabetes damages the tiny blood vessels inside the retina, the light-sensitive tissue at the back of the eye. Hyperglycemia-induced intramural pericyte death and thickening of the basement membrane lead to incompetency of the vascular walls. These damages change the formation of the blood-retinal barrier and also make the retinal blood vessels more permeable.[4] Proinflammatory cytokines such as interleukin-6 (IL-6) have been shown to be involved in the pathogenesis of DR.[5] Periodontal disease has been associated with poorer metabolic control of hyperglycemia.[6] Advanced glycation end products (AGEs) formed as a result of hyperglycemia interact with various tissues. Blood vessel alterations secondary to AGE accumulation may contribute to the classic macrovascular and microvascular complications of *diabetes mellitus* (DM).[7] Severe periodontal disease is associated with elevated blood lipopolysaccharide levels as a result of periodontogenic bacteria, which induce higher levels of IL-6 and tumor necrosis factor-α. While there are various studies linking the occurrence and severity of various cardiorenal complications and periodontal disease in diabetic patients, the literature is deficient regarding the association of periodontal disease and DR.[8,9] This study is aimed at evaluating the association of periodontal disease with the occurrence and severity of DR.

Materials and methods

This cross-sectional study was conducted among 100 known type II DM patients who reported to different ophthalmologic and endocrinal clinics of Indore, India. The patients were divided into a test group (patients with DR) and a control group (diabetics without DR). The test group was further divided into nonproliferative and proliferative retinopathies, which reflect the initial and advanced stages of retinopathy, respectively. The diagnosis of retinopathy was established by an ophthalmologist based on clinical findings (such as blurring of vision and watering of eyes) and a funduscopic examination showing the engorgement of blood vessels and hemorrhagic spots (Fig. 1 and 2).

The test group consisted of type II DM patients (35-65 years), who were recently diagnosed with DR (≤3 months duration) and had at least 20 teeth present. The control group met the same parameters, except they had no DR. Informed consent was taken from all the patients prior to conducting the study.

Patients with type I DM, those suffering from other systemic diseases and diabetic comorbidities, patients who have undergone surgery for retinopathy, those with a history of periodontal treatment and antimicrobial drug usage in the past 6 months, patients with other causative factors of retinopathy, smokers, and patients using tobacco in chewable form were excluded from the study.

Demographic variables (such as age and sex), the duration of DM and DR, and biochemical markers (random blood glucose and glycated hemoglobin) were noted in all patients.

Oral and periodontal examinations

Greene & Vermillion's *Simplified Oral Hygiene Index* (OHI-S) was used to evaluate the oral hygiene status of the patients.[10] The OHI-S was calculated as a debris and calculus index on the buccal surfaces of teeth No. 11, 16, 26, and 31 and the lingual surfaces of teeth No. 36 and 46. The scores of debris and calculus were added to obtain the OHI-S of each patient. Bleeding on probing (BOP) was noted as *present* or *absent* at 4 sites (mesiobuccal, distobuccal, midbuccal, and midlingual) of each tooth. Probing pocket depth (PPD) and clinical attachment loss (CAL) were noted at the same 4 sites of the commonly known *Ramfjord's teeth* (teeth No. 11, 16, 24, 36, 41, and 44) with the help of a periodontal probe.[11] The numbers of missing and carious teeth were also noted.

All data were expressed as means. The unpaired t-test was applied to calculate the significance of the difference of means of various periodontal parameters

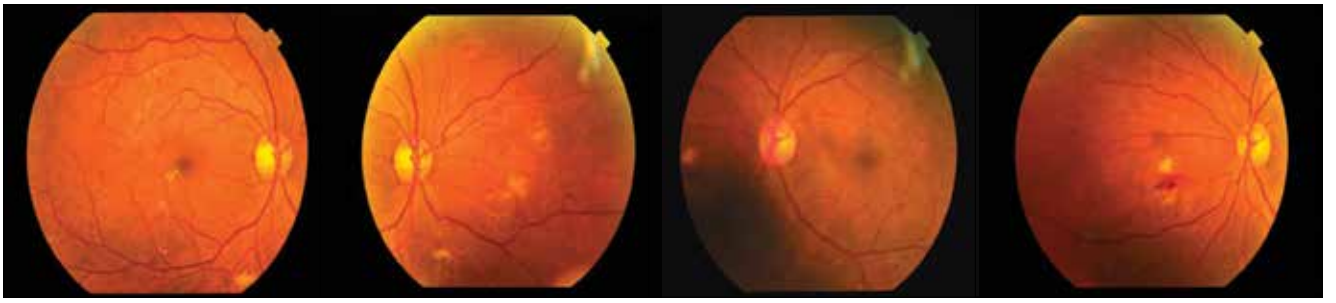

Fig. 1. Funduscopic pictures of retinopathy.

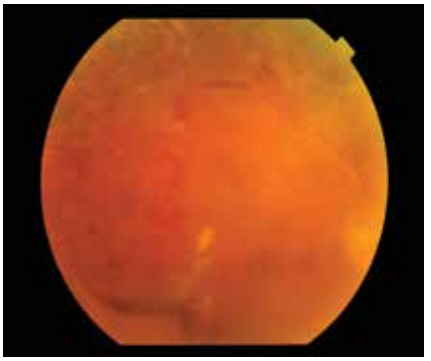

Fig. 2. Funduscopic picture of a healthy retina.

between the 2 groups. For all analyses, $P < 0.05$ was considered significant. A multiple logistic regression analysis was applied to evaluate the impact of individual factors, including age, numbers of missing and carious teeth, PPD, CAL, BOP and OHI-S scores. An adjusted odds ratio was calculated at a 95% confidence interval using the regression analysis.

Results

The study group consisted of 100 known type II DM patients, consisting of 73 men and 27 women with a mean age of 47.5. There were 50 patients in both the test and the control groups. There were no statistically significant differences between the 2 groups in terms of variables such as age, duration of diabetes, random blood sugar, glycated hemoglobin levels, and mean number of missing and carious teeth. The comparison of the periodontal parameters of the test and control group revealed no statistically significant difference in mean number of bleeding sites. The differences in mean OHI-S scores, mean PPD, and mean CAL between the test and control groups were found to be highly significant ($P = 0.00$) (Table 1).

Table 1. Comparison of clinical and biochemical parameters in diabetic patients with and without retinopathy using unpaired t-test.

Parameters	Group[a]	Mean (SD)	t-test	Significance (2-tailed)
Mean age (years)	1	48.55 (9.63)	1.014	0.313
	2	46.57 (9.90)	1.014	0.313
Mean duration of diabetes mellitus (years)	1	4.96 (3.80)	-0.137	0.892
	2	5.06 (3.54)	-0.137	0.891
Mean random blood sugar (mg%)	1	157.29 (14.56)	-1.229	0.222
	2	160.94 (15.09)	-1.228	0.222
Mean glycated hemoglobin (g%)	1	7.03 (1.46)	0.154	0.878
	2	7.15 (1.08)	0.155	0.878
Mean number of missing teeth	1	0.96 (1.41)	-0.153	0.878
	2	1.00 (1.12)	-0.154	0.878
Mean number of carious teeth	1	1.57 (1.24)	0.716	0.475
	2	1.39 (1.29)	0.716	0.476
Mean number of bleeding sites	1	107.78 (7.77)	1.665	0.099
	2	105.27 (7.34)	1.667	0.099
Mean probing pocket depth (mm)	1	2.56 (0.31)	4.975	0.000
	2	2.30 (0.20)	5.018	0.000
Mean clinical attachment loss (mm)	1	2.57 (0.36)	3.185	0.002
	2	2.38 (0.22)	3.213	0.002
Mean simplified oral hygiene index	1	2.78 (0.46)	4.067	0.000
	2	2.46 (0.29)	4.101	0.000

[a]Group 1, patients with retinopathy; Group 2, patients without retinopathy.
Abbreviation: SD, standard deviation.

A multiple regression analysis failed to show any significant effect of any individual parameter except PPD and OHI-S at a 95% confidence interval, however, the odds ratio was found to be <1 (Table 2).

There were no statistically significant differences between patients with nonproliferative and patients with proliferative retinopathies in terms of other variables (Table 3).

Discussion

This cross-sectional study was conducted on 100 known type II DM patients. All patients were under medication (oral hypoglycemic agents) for the management of DM. They were under regular medical supervision and none of the patients had altered their diabetic treatment in the year previous to the initiation of this study.

The mean duration of DM in both the test and control groups was similar (approximately 5 years). This mean

duration of DM was shorter compared to another study that evaluated the relationship between periodontal disease and DR. In a study conducted by Noma et al, the mean duration of diabetes was 14.3 ± 7.1 years.[12] Those authors found a significant relationship between the severity of DM and that of DR.[12]

No statistically significant difference in the mean number of missing teeth and number of carious teeth was found between the test and control groups. Similarly, no difference in the mean number of sites showing BOP was found. BOP represents an early sign of periodontal disease, and diabetic patients tend to show an increased incidence in and severity of this disease. Statistically significant differences between the means of PPD, CAL, and OHI-S scores between the test and control groups indicated poorer oral hygiene and periodontal health in the test group. Similar findings were reported by Noma et al, who concluded that the severity of periodontal disease significantly correlated with the severity of DR.[12] The authors' study also found a significant relationship between the duration of a patient's diabetes and the severity of the disease.[12] In the current study, the mean duration of diabetes was similar in both groups. As all other factors were found to be similar in the test and control groups between this study and the study by Noma et al, it can be inferred that in the presence of periodontal disease, complications of diabetes may occur early, and periodontal disease can place diabetic patients at an increased risk of developing ocular complications.[12]

In this study, diabetic subjects with retinopathy were found to have poorer oral hygiene and poorer periodontal status than diabetics without retinal changes, even though the mean glycated hemoglobin (Hb) values (an indicator of metabolic control) were found to be similar in both groups. Because the periodontal status reflects cumulative pathological change occurring over years, the HbA1c provides diabetic control information over 3-4 months, and the random blood sugar levels reflect a particular time assessment of diabetes control, it can be interpreted that a subject who presently has good metabolic control may have previously had poorer diabetic status. This makes it difficult to establish an unequivocal and precise association between the severity of risk and disease presentation.[13]

Table 2. Comparison of clinical and biochemical parameters in diabetic patients with either proliferative or nonproliferative retinopathy using unpaired t-test.

Parameters	Group[a]	Mean (SD)	t-test	Significance (2-tailed)
Mean age (years)	1	50.54 (10.03)	1.189	0.240
	2	47.41 (9.11)	1.178	0.245
Mean duration of diabetes mellitus (years)	1	5.62 (5.65)	0.497	0.622
	2	5.03 (2.76)	0.468	0.643
Mean random blood sugar (mg%)	1	160.04 (22.71)	0.556	0.580
	2	157.34 (11.77)	0.526	0.602
Mean glycated hemoglobin (g%)	1	7.03 (1.56)	0.303	0.763
	2	7.15 (1.34)	0.299	0.766
Mean number of missing teeth	1	0.88 (0.85)	-0.322	0.748
	2	1.00 (1.73)	-0.342	0.734
Mean number of carious teeth	1	1.42 (1.41)	-1.248	0.218
	2	1.86 (1.19)	-1.228	0.226
Mean number of sites	1	109.00 (5.79)	1.090	0.281
	2	106.69 (8.95)	1.134	0.263
Mean probing pocket depth (mm)	1	2.62 (0.36)	1.064	0.292
	2	2.53 (0.25)	1.028	0.310
Clinical attachment loss (mm)	1	2.61 (0.39)	0.837	-0.407
	2	2.53 (0.32)	0.821	0.416
Mean simplified oral hygiene index	1	2.85 (0.52)	0.851	0.399
	2	2.74 (0.40)	0.831	0.411

[a]Group 1, patients with proliferative retinopathy; Group 2, patients with non-proliferative retinopathy.
Abbreviation: SD, standard deviation.

The mean number of missing teeth in our study ranged from 0 to 7, with an average of 1 tooth. Loe observed in his review that the frequency of edentulousness increased with the duration of diabetes, ranging from 7% edentulous for patients who had diabetes for 5 years; 14% for patients whose duration was 10 years; and 75% for diabetic patients whose duration was 20 years.[1] The smaller number of missing teeth in the present study can be attributed to the shorter mean duration of the disease (approximately 5 years).[1]

Working with a sample of type I DM patients, Al-Shammari et al also concluded that the severity of the periodontal disease in a diabetic patient is associated with both the duration of the disease and the presence of DM complications.[14] In our study, there were no statistically significant differences found between proliferative and nonproliferative retinopathy patients in any of the parameters studied. No association was found between the severity of retinopathy and periodontal status. This might be attributed to the small sample size (27 patients with proliferative retinopathy and 24 patients with nonproliferative retinopathy).

In this study, a multiple regression analysis failed to show a significant effect of any individual parameter except PPD and OHI-S at a 95% confidence interval. Though both PPD and OHI-S were found to have a significant effect ($P < 0.05$), the odds ratio was found to be very small (<1). This statistical distortion can be attributed to the fact that the arithmetic mean can be greatly affected by extreme values and may not reflect an accurate picture. Diabetic type II patients with retinopathy were almost 5 times as likely to have periodontal disease than type II diabetics without retinopathy.[15] Type I DM patients with retinal changes had a greater loss of periodontal attachment than type I diabetics without retinal changes.[16]

Table 3. Multiple regression analysis.

Parameters	Standard error	Wald test	Significance	Exp (B) Regression coefficients
Mean age (years)	0.028	0.427	0.513	0.982
Mean duration of diabetes mellitus (years)	0.070	1.217	0.270	1.081
Mean random blood sugar (mg%)	0.018	1.874	0.171	1.025
Mean glycated hemoglobin (g%)	0.220	0.143	0.706	1.087
Mean number of missing teeth	0.215	1.091	0.296	0.799
Mean number of carious teeth	0.217	1.296	0.255	1.280
Mean number of sites	0.039	2.548	0.110	0.940
Mean probing pocket depth (mm)	1.745	9.891	0.002	0.004
Clinical attachment loss (mm)	1.409	0.869	0.351	3.721
Mean simplified oral hygiene index	0.793	4.856	0.028	0.174
Constant	6.961	5.536	0.019	1.297

As stated before, DR is a result of microvascular retinal changes. Hyperglycemia-induced, intramural pericyte death, and thickening of the basement membrane leads to incompetence of the vascular walls. Small blood vessels—such as those found in the eye—are especially vulnerable to poor blood glucose control. The over-accumulation of glucose and/or fructose in the blood tend to damage the tiny vessels in the retina.[17]

As the disease progresses, severe non-proliferative DR moves to the advanced proliferative stage. The lack of oxygen due to damaged blood vessels in the retina causes fragile new blood vessels to grow along the retina and in the clear, gel-like vitreous humor that fills the inside of the eye. Fibrovascular proliferation can also cause tractional retinal detachment. The new blood vessels may also grow into the angle of the anterior chamber of the eye, possibly causing neovascular glaucoma.[4]

There are various mechanisms associated with periodontal disease and both the development and severity of retinopathy. Periodontal disease creates a state of insulin resistance in the body leading to a worsening of metabolic control in DM, hence periodontal disease can act as a risk factor for the development of various complications of DM. Grossi et al suggested that the upregulation of proinflammatory cytokines and tumor necrosis factor-α due to periodontal disease may be a factor in insulin resistance and poor glycemic control.[18]

Periodontal disease leads to an elevated systemic inflammatory state manifested by raised levels of C-reactive protein, IL-6, and fibrinogen. These systemic inflammatory markers indicate insulin resistance.[19] IL-6 has also been implicated in the pathogenesis of retinopathy.[20] Periodontal disease can also increase the generation of relatively highly reactive oxygen species in both diabetics and healthy individuals.[21] This is one of the mechanisms of hyperglycemia-induced diabetic complications.[22] The reactive oxygen species promote severe tissue damage and cell death.

In their 1997 study, Murata et al concluded that vascular endothelial growth factor (VEGF) breaks the blood retinal barrier (BRB), leading to increased permeability of the retinal blood vessels and hence increased fragility.[23] Pradeep et al concluded that gingival crevicular fluid serum VEGF levels increased progressively with an increase in periodontal disease severity, and decreased after treatment of periodontal disease.[24] An increased VEGF secondary to periodontal disease can affect the BRB, leading to retinal pathology.

Periodontal disease can also lead to atherosclerosis, which may result in a lack of oxygen in the retina causing the proliferation of new fragile blood vessels. These can bleed and destroy the retina, cause retinal detachment, or neovascular glaucoma.[25,26]

The long-term complications of DM are mainly associated with prolonged hyperglycemia. There are various biochemical pathways that lead to vascular complications. These include increased polyol pathway flux (sorbitol/aldose reductase) and oxidative stress, increased formation of AGEs, accumulation of diacylglycerol, and activation of protein kinase C (PKC).

Moderate or poor glycemic control in DM patients raises PKC levels and activity, resulting in neutrophil priming. The increased inflammation and oxidative stress are consistent with an increased risk of DM complications, including periodontitis.[27]

Study limitations

The small sample size (100 patients) can be attributed to inclusion and exclusion criteria which were stringent in order to minimize confounding factors. Due to the limited sample size, it was not possible to obtain the statistical power needed to adequately evaluate the potential modulating role of periodontal disease on the severity of retinopathy. As this was a cross-sectional study, a larger sample size would have provided more authentic results.

Randomization was used in an attempt to eliminate bias, and controlled for various confounders, but in so doing, important variables may have been missed, such as the socioeconomic status of the patients, which might have acted as a confounding factor.

Antidiabetic medications may have affected finding a clear association, but it would have been unethical to deprive the patients of their DM treatment. Any previous (>6 months) successfully maintained periodontal therapy may have altered the results.

Conclusion

The results from this study show that periodontal disease may act as a risk factor for the early development of DR. This is a preliminary study, so it would be premature to suggest any consensus regarding the true association of periodontal disease with diabetic complications. Long-term longitudinal trials with a larger sample size are recommended to evaluate the strength of the association of these 2 disease entities.

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References

1. Loe H. Periodontal disease. The sixth complication of *diabetes mellitus. Diabetes Care.* 1993;16(1):329-334.
2. Lusis AJ. Atherosclerosis. *Nature.* 2000;407:233-241.
3. Doi T, Vlassara H, Kirstein M, Yamada Y, Striker GE, Striker LJ. Receptor-specific increase in extracellular matrix productions in mouse mesangial cells by advanced glycosylation end products is mediated via platelet-derived growth factor. *Proc Natl Acad Sci U S A.* 1992;89(7):2873-2877.
4. Pardianto G. Understanding diabetic retinopathy [in Indonesian]. *Mimbar Ilmiah Oftalmologi Indonesia.* 2005;2:65-66.
5. Funatsu H, Yamashita H, Shimizu E, Kojima R, Hori S. Relationship between vascular endothelial growth factor and interleukin-6 in diabetic retinopathy. *Retina.* 2001;21(5):469-477.
6. Shlossman M, Knowler WC, Pettitt DJ, Genco RJ. Type 2 *diabetes mellitus* and periodontal disease. *J Am Dent Assoc.* 1990;121(4):532-536.
7. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent *diabetes mellitus.* The Diabetes Control and Complications Trial Research Group. *N Engl J Med.* 1993;329(14):977-986.
8. Saremi A. Periodontal disease and mortality in type 2 diabetes. *Diabetes Care.* 2005;28(1):27-32.
9. Thorstensson H, Kuylenstierna J, Hugoson A. Medical status and complications in relation to periodontal disease experience in insulin-dependent diabetics. *J Clin Periodontol.* 1996;23(3 Pt 1):194-202.
10. Green JC, Vermillion JR. The simplified oral hygiene index. *J Am Dent Assoc.* 1964;68:7-13.
11. Ramfjord SP. Indices for prevalence and incidence of periodontal disease. *J Periodontol.* 1959;30:51-59.
12. Noma H, Sakamoto I, Mochizuki I, et al. Relationship between periodontal disease and diabetic retinopathy. *Diabetes Care.* 2004;27(2):615.
13. Preshow PM, De Silva N, Mc Cracken GI, et al. Compromised periodontal status in an urban Sri Lankan population with type 2 diabetes. *J Clin Periodontol.* 2010;37(2):165-171.
14. Al-Shammari KF, Al-Ansari JM, Moussa NM, Ben-Nakhi A, Al-Arouj M, Wang HL. Association of periodontal disease severity with diabetes duration and diabetic complications in patients with type 1 *diabetes mellitus. J Int Acad Periodontol.* 2006;8(4):109-114.
15. Schossman M, Knowler WC, Pettitt DJ, Genco RJ. Type II diabetes mellitus and periodontal disease. *J Am Dent Assoc.* 1990;121(4):532-536.
16. Glavind L, Lund B, Loe H: The relationship between periodontal state and diabetes duration, insulin dosage and retinal changes. *J Periodontol.* 1968;39(6):341-347.
17. Toke B, Hammes HP, Porta M. Clinical Presentations and Pathological Correlates of Retinopathy. In: Hammes HP, Porta M, eds. *Experimental Approaches to Diabetic Retinopathy. Front Diabetes.* Basel, Switzerland: Karger; 2010;20:1-19.
18. Grossi SG, Skrepcinski FB, De Caro T, Zambon JJ, Cummin D, Genco RJ. Response to periodontal therapy in diabetics and smokers. *J Periodontol.* 1996;67(10 Suppl):1094-1102.
19. Yki-Jarvinen H, Sammalkorpi K, Koivisto VA, Nikkila EA. Severity, duration, and mechanisms of insulin resistance during acute infections. *J Clin Endocrinol Metab.* 1989;69(2):317-323.
20. Funatsu H, Yamashita H, Shimizu E, Kojima R, Hori S. Relationship between vascular endothelial growth factor and interleukin-6 in diabetic retinopathy. *Retina.* 2001;21(5):469-477.
21. Al-Mubarak S, Ciancio S, Aljada A, Mohanty P, Ross C, Dandona P. Comparative evaluation of adjunctive oral irrigation in diabetics. *J Clin Periodontol.* 2002;29(4):295-300.
22. Stewart JE, Wager KE, Friedlander AH, Zadeh HH. The effect of periodontal treatment on glycemic control in patients with type 2 diabetes mellitus. *J Clin Periodontol.* 2001;28(4):306-310.
23. Murata T, Nagai R, Ishibashi T, Inomata H, Ikeda K, Horiuchi S. The relationship between accumulation of advanced glycation end products and expression of vascular endothelial growth factor in human diabetic retinas. *Diabetologia.* 1997;40(7):764-769.
24. Pradeep AR, Prapulla DV, Sharma A and Sujatha PB. Gingival crevicular fluid and serum vascular endothelial growth factor: their relationship in periodontal health, disease and after treatment. *Cytokine.* 2011;54(2):200-204.
25. Viswanath K, McGavin DD. Diabetic retinopathy: clinical findings and management. *Community Eye Health.* 2003;16(46):21-24.
26. Frank RN. Diabetic retinopathy. *N Engl J Med.* 2004;350(1):48-58.
27. Nassar H, Kantarci A, van Dyke TE. Diabetic periodontitis: a model for activated innate immunity and impaired resolution of inflammation. *Periodontol 2000.* 2007;43:233-243.

An association between temporomandibular disorder and gum chewing

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This single center, randomized, small study sought to investigate the prevalence and frequency of chewing gum consumption, and whether there is a relationship between these factors and the presence of symptoms associated with temporomandibular disorder (TMD).

Subjects were divided into 7 groups based on their parafunctional oral habits. Of these, subjects who chewed gum were divided into 5 subgroups (A-E) based on their gum chewing habits. Group A chewed gum <1 hour/day (n = 12), Group B chewed gum 1-2 hours/day (n = 11), Group C chewed gum 3 hours/day (n = 6), and Group D chewed gum >3 hours at a time (n = 8); the frequency of gum chewing in Groups A-D was once a week. Group E subjects chewed gum 1-3 times/week for at least 1 hour each occurrence (n = 2). Sixty-three percent of the subjects in Group D reported TMD symptoms of arthralgia and myofascial pain. Thirty-three percent of the subjects in Group C showed symptoms of arthralgia. Eighty-three percent of the subjects in Group A and 27% in Group B reported myofascial pain. All subjects in Group E reported masseter hypertrophy. The remaining 2 groups were Group F, subjects that didn't chew gum but had other parafunctional oral habits (n = 2), and Group G, subjects who didn't have parafunctional oral habits (n = 12).

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Temporomandibular disorder (TMD) is a significant public health problem affecting approximately 5% to 12% of the population.[1] TMD is the second most common musculoskeletal condition (after chronic lower back pain) resulting in pain and disability.[1] Pain-related TMD can impact an individual's daily activities and quality of life. Overall, the annual cost of TMD management, not including imaging, has doubled in the last decade to $4 billion.[1]

For over 30 years, TMD has been classified as a group of disorders characterized by the presence of peri-auricular and masticatory muscle pain, the presence of arthralgia (joint pain) and noises during mandibular opening and closing movements, and deviations or restrictions in range of motion.[2] There are a number of factors that contribute to TMD development, including parafunctional oral habits, malocclusions, stress, and trauma, although the presence of these factors does not necessarily lead to the development of TMD disorders.[3,4] When these factors occur with a certain frequency, duration, and intensity that exceeds the body's physiological tolerance, the result may be tissue damage.[5] This damage tends to begin in structures that are less tolerant to physiological change, such as teeth, muscles, and joints.[6]

During normal oral function, there is a predominance of vertical masticatory cycles, with tooth contact occurring only in the final stages of mastication.[3] The presence of parafunctional oral activity greatly increases the frequency and intensity of masticatory forces, which may cause tooth wear facets, especially in cases of bruxism (grinding/gnashing/clenching of teeth).[7] The presence of tooth wear leads to a predominance of larger and more irregular horizontal and cyclic masticatory movements. The excess wear on the anterior teeth wear leads to a progressive loss of anterior guidance and increased friction forces, which further accelerate tooth wear.[3] This loss of anterior guidance creates a vicious cycle, resulting in posterior overload that activates the action of the masseteric muscles, which further increases the friction between the tooth surfaces.[7] Therefore, in excursive movements and in the absence of a guidance to disocclude, the centric cusps make contact on both the working (side toward which the mandible moves in a lateral movement) and the nonworking sides of the jaw, while the noncentric cusps make contact only on the working side. This causes the centric cusps to make contact twice as much as noncentric cusps, thus generating an inversion of the upward, U-shaped curvature of the maxillary and mandibular occlusal planes in the coronal plane (defined as the *Wilson Curve*). In this way, the interference on the nonworking side causes the temporomandibular joint (TMJ) to become a class I lever system, resulting in higher TMJ overload, causing ligament sprain and increased intra-articular space, which may lead to an immediate Bennet movement (the lateral translation of the head of the mandible on the working side during a lateral excursion) before starting the progressive movement. This is not a dental problem, but a joint problem that involves the jaw. However, the consequences of this movement will lead to occlusal teeth surfaces with parafunctional facets.[7]

Previous studies have established a positive relationship between TMD and parafunctional oral habits such as lip and tongue suction, bruxism, oral breathing without obstructive cause, nail and pencil biting, jaw "play," and chewing gum.[4,8-14]

Given how the major properties of food, including hardness, size, volume, and elasticity, affect mastication, the present study—in agreement with several other studies—defined a gum chewing habit as *intense* when it is maintained >3 hours at a time.[11-15] Intense gum chewing is strongly associated with the presence of joint noises and pain at the TMJ level, muscle soreness or pain at rest (especially in the masseter), and muscle hypertrophy. This is especially true in subjects with a prior history of TMD.[4]

The existence of oral parafunctional habits in individuals *without* TMD suggests that these habitual movements do not always result in TMD, but can act as risk factors. The current study sought to determine a relationship between the prevalence and frequency of chewing gum and signs and symptoms associated with TMD.

Table 1. Temporomandibular disorder (TMD) diagnosis in study population (n = 38).

TMD diagnosis	Frequency	Percent
Myofascial pain	Yes 15	39.5
	No 23	60.5
Arthralgia	Yes 25	65.8
	No 13	34.2
Masseter hypertrophy	Yes 13	34.2
	No 25	65.8
Headaches	Yes 6	15.8
	No 32	84.2

Table 2. Correlations between TMD symptoms and chewing gum.

TMD diagnosis	Groups (%)				
	A	B	C	D	E
Arthralgia			33 ($P = 0.043$)	63 ($P = 0.047$)	
Myofascial pain	83 ($P = 0.025$)	27 ($P = 0.047$)		63 ($P = 0.038$)	
Masseter hypertrophy					100 ($P = 0.045$)

Variables were analyzed by chi-square tests with a significance level of 5%.
Groups: A, subjects who chewed gum <1 hour/day (n = 12); B, subjects who chewed gum 1-2 hours/day (n = 11); C, subjects who chewed gum 3 hours/day (n = 6); D, subjects who chewed gum >3 hours/day (n = 5); E, subjects who chewed gum 1-3 times/week (n = 2).

Materials and methods

This study was a single center, randomized trial in students (n = 50) at the University of Lisbon, Portugal. The sample was composed of 43 females and 7 males with an age range of 19-27 years.

Exclusion criteria were orthodontic treatment, craniofacial anomalies, degenerative TMJ, and non-rehabilitation of edentulous spaces. Inclusion criteria included a signature of informed consent and permanent dentition only.

Subjects were divided into 7 groups who were then given a questionnaire concerning their parafunctional oral habits. Based on this questionnaire, 5 groups (A-E) were created based on the subjects' chewing gum habits as indicated on this questionnaire. Group A chewed gum <1 hour/day (n = 12), Group B chewed gum 1-2 hours/day (n = 11), Group C chewed gum 3 hours/day (n = 6), Group D chewed gum >3 hours/day (n = 8)—the frequency of gum chewing in Groups A-D was once a week. Group E subjects chewed gum 1-3 times/week for at least 1 hour each occurrence (n = 2). The remaining 2 groups were Group F, subjects that didn't chew gum but had other parafunctional oral habits (n = 2); and Group G, subjects that didn't have parafunctional oral habits (n = 12).

The questionnaire concerning parafunctional oral habits was based on the *Oral Behavior Checklist* found in the *Diagnostic Criteria for Temporomandibular Disorders (2014).*[16] Primary outcomes before and after each observation period were recorded using the Numeric Rating Scale (NRS), in which patients determine their perception of pain on a scale of 1 to 10, 10 being the most severe.[17] The subjects were told to maintain their habits for 6 months. For Groups A-E, a specific type of gum (Trident, Cadbury) was provided by the clinician. The data were collected by a calibrated operator.

Results

Clinical examination

The diagnoses of each subject were made according to the research diagnostic criteria for TMD. This included examination for muscle pain, joint pain, jaw limitation (vertical and side excursions), joint sounds, masseter hypertrophy (a benign increase in size), and headaches (Table 1).[16]

Statistical analysis

Variables were analyzed by chi-square tests with a significance level of 5%. Correlations between TMD symptoms and gum chewing are listed in Table 2.

Thirty-three percent of the subjects in Group C and 63% of the subjects in Group D reported arthralgia ($P = 0.043$ and 0.047, respectively). Eighty-three percent, 27%, and 63% of the subjects in Groups A, B, and D reported myofascial pain ($P = 0.025$, $P = 0.047$, and $P = 0.001$, respectively). All of the subjects in Group E reported masseter hypertrophy ($P = 0.045$). Of the subjects in Group F, there was no consistent relationship between the presence of parafunctional oral habits and the presence of TMD ($P = 0.3$). None of the subjects in Group G reported any signs and symptoms of TMD.

The most common oral habit in this study was gum chewing, followed by tongue placement between teeth and jaw play, which are nonphysiologic, mandibular positioning habits. The results showed that the subjects that chewed gum for more hours—regardless of the frequency of the habit—had more signs/symptoms of TMD.

Discussion

The most important function of the stomatognathic system is chewing, defined as a voluntary and rhythmic activity of the masticatory muscles during the process of opening and closing of the jaw.[18,19] The command for this activity is carried out by the cerebral cortex which receives the information sent by the motor nerves concerning the speed, time of action, and muscle contraction/relaxation, allowing for the adaptation of muscle and joint structures to the food type.[20] Protective reflexes are constantly present, protecting against contacts that are damaging to the system. The presence of isotonic muscle contraction allows adequate blood flow to oxygenate the tissues and eliminate the metabolic products accumulated at the cellular level.[3]

In the literature, a number of investigations have proposed oral parafunctional habits as a possible cause of TMD, and its prevalence and association with signs and symptoms of dysfunction have been published.[3] Studies conducted in adults have shown a positive correlation between

parafunction and muscle and joint pain.[12] Parafunctional oral habits seem to present more in adolescents than adults, and in females more than males. Thus, there is speculation that parafunctional activities can contribute to a higher prevalence of TMD symptoms in females and adolescents.[11] There was no statistical significance between gender and the presence of TMD in the present study, which could be due to the small number of male subjects (n = 7).

The literature also mentions that chewing gum interferes with the normal mastication pattern. It involves an increased mastication frequency due to a greater number of masticatory cycles and an increase in muscle intensity.[21] Since chewing gum does not fragment during mastication, the masticatory muscle workload is greater and more sustainable than is the case with "fragmentable" foods. The masticatory cycles are also shorter, resulting in a lowered recognition from the muscle and joint structures of the existing force loads, which in turn creates a lower adaptation to these forces; this results in a lower tolerance of the masticatory system to the forces.[22,23] There is also a predominance of unilateral mastication, which translates into an overload of the working side jaw muscles, resulting in muscle fatigue and hypertrophy. Unilateral mastication is also responsible for a lower proprioception (sense of place and strength of effort by the surrounding structures) and consequently a greater imbalance in muscle contraction.[15]

This study was conducted on 50 university students between the ages of 19 and 27 in order to investigate the prevalence and inter-relationship of several parafunctional habits and their contribution to the presence of various signs and symptoms of TMD. Although this study was limited by a small study sample size and restricted age range, the results concur with those found by Wincour et al and Gavish et al.[10,11] Further research is needed with larger subject populations exhibiting greater ranges of both the frequency and intensity of oral parafunctional habits in order to make more and better comparisons between groups. Specifically, a larger study is needed that includes subjects whose habit is chewing gum >3 hours at a time *and* more than once a week, to confirm whether the frequency of this habit exacerbates the symptoms of TMD found in intensive chewing.

The results from this study showed that it is the *intensity* of the habit—as reflected by hours of chewing per day—that was the most significant indicator of risk to the stomatognathic system. There was no consistent relationship between the *frequency* of chewing gum more than once and up to 3 times per week and the signs and symptoms of TMD pain.

There was a correlation between chewing gum more than once per week and masseter hypertrophy. This is in line with findings by Sari & Sonmez and Vanderas & Manetas.[4,5]

Bruxism

The literature recommends that bruxism and TMJ pain be managed as separate problems.[23] It is important to understand the concept of a nonlinear relationship to avoid oversimplification of diagnosis and management. Svensson et al, Huang et al, and Kalamir et al have stated that while it seems evident that there are associations between TMJ pain and bruxism, care must be taken to not create a simplistic cause and effect relationship.[23-25] In a 2010 systematic review by Manfredini & Lobbezoo, this concept of cause and effect was corroborated, and the authors concluded that it was not possible to discuss the relationship between specific TMD signs and symptoms and different bruxism-related motor activities—namely clenching and grinding—due to the very low levels of specificity in the reviewed studies.[26]

Conclusion

Patients often consult general dentists concerning TMD symptoms, especially those associated with pain. The *Diagnostic Criteria for Temporomandibular Disorders (2014)* is presently one of the best techniques for diagnosis of TMD.[16] This evidence-based criteria will assist clinicians when assessing patients, while facilitating communication in consultations, referrals, and prognoses.

In this study, chewing gum more than once per week was related to masseter hypertrophy. The presence of TMD symptoms, including arthralgia and myofascial pain, were related to chewing gum for >3 hours at a time (Group D). The intensity of the oral parafunction—as defined in this study as chewing gum >3 hours at a time—was determined to be the most important factor in the propensity toward symptoms of TMD pain.

Further standardized, randomized, and externally validated studies are needed to determine the specificity of the present parameters.

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References

1. National Institutes of Health. National Institute of Dental and Craniofacial Research. *TMJD and Orofacial Pain Program*. Available at: http://www.nidcr.nih.gov/Research/DER/IntegrativeBiologyAndInfectiousDiseases/TMJD.htm. Accessed June 2, 2011.
2. Feteih RM. Signs and symptoms of temporomandibular disorders and oral parafunctions in urban Saudi Arabian adolescents: a research report. *Head Face Med.* 2006;2:25.
3. Okeson JP. *Management of Temporomandibular Disorders and Occlusion.* 6th ed. Available at: http://www.eugenol.com/attachments/0011/5436/Temporomandibular_Disorders_and_Occlusion.pdf. Accessed June 2, 2014.
4. Sari S, Sonmez H. Investigation of the relationship between oral parafunctions and temporomandibular joint dysfunction in Turkish children with mixed and permanent dentition. *J Oral Rehab.* 2002;29(1):108-112.
5. Vanderas AP, Manetas KJ. Relationship between malocclusion and bruxism in children and adolescents: a review. *Pediatr Dent.* 1995;17(1):7-12.
6. Carlson CR, Reid KI, Curran SI, et al. Psychological and physiological parameters of masticatory muscle pain. *Pain.* 1998;76(3):297-307.
7. Alonso AA, Albertini JS, Becheli AH. Oclusion y Diagnostico en Rehabilitacion Oral. 3rd ed. Anadir:2004.
8. Widmalm SE, Christiansen RL, Gunn SM. Oral parafunctions as temporomandibular disorder risk factors in children. *Cranio.* 1995;13(4):242-246.
9. Glaros AG, Tabacchi KN, Glass EG. Effect of parafunctional clenching on TMD pain. *J Orofac Pain.* 1998; 12(2):145-152.
10. Wincour E, Littner D, Adams I, Gavish A. Oral habits and their association with signs and symptoms of temporomandibular disorders in adolescents: a gender comparison. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2006;102(4):482-487.
11. Gavish A, Halachmi M, Winocur E, Gazit E. Oral habits and their association with signs and symptoms of temporomandibular disorders in adolescent girls. *J Oral Rehabil.* 2000;27(1):22-32.

12. Buzinelli RV, Berzin F. Electromyographic analysis of fatigue in temporalis and masseter muscles during continuous chewing. *J Oral Rehabil.* 2001;28(12): 1165-1167.
13. Farella M, Bakke M, Michelotti A, Martina R. Effects of prolonged gum chewing on pain and fatigue in human jaw muscles. *Eur J Oral Sci.* 2001;109(2):81-85.
14. Mioche L, Bourdiol P, Martin JF, Noel Y. Variations in human masseter and temporalis muscle activity related to food texture during free and side-imposed mastication. *Arch Oral Biol.* 1999;44(12):1005-1012.
15. Cortese SG, Biondi AM. Relacion de disfunciones y habitos parafuncionales orales com trastornos temporomandibulares en ninos y adolescentes. *Arch Argent Pediatr.* 2009;107(2):134-138.
16. International RDC-TMD Consortium. *Diagnostic Criteria for Temporomandibular Disorders (2014).* Available at: http://www.rdc-tmdinternational.org/TMDAssessmentDiagnosis/DCTMD.aspx. Accessed June 3, 2014.
17. United States Department of Veterinary Affairs. *Numeric Pain Rating Scale.* Available at: http://www.va.gov/PAINMANAGEMENT/docs/PainNRS.pdf. Accessed June 3, 2014.
18. Sakamoto K, Nakata H, Kakigi R. The effect of mastication on human cognitive processing: a study using event-related potentials. *Clin Neurophysiol.* 2009; 120(1):41-50.
19. Agrawal KR, Luca OW, Prinz JG, Bruce IC. Mechanical properties of foods responsible for resisting food breakdown in the human mouth. *Arch Oral Biol.* 1997; 42(1):1-9.
20. Throckmorton GS, Buschang BH, Havasaki H, Phelan T. The effects of chewing rates on mandibular kinematics. *J Oral Rehabil.* 2001;28(4):328-334.
21. Yoshida T, Ishikawa H, Yoshida N, Hisanaga Y Analysis of masseter muscle oxygenation and mandibular movement during experimental gum chewing with different hardness. *Acta Odontol Scand.* 2009;67(2):112-121.
22. Tzakis MG, Karlsson S, Carlsson GE. Effects of intense chewing on some parameters of masticatory function. *J Prosthet Dent.* 1992;67(3):405-409.
23. Svensson P, Jadidi F, Arima T, Baad-Hansen L, Sessle BJ. Relationships between craniofacial pain and bruxism. *J Oral Rehabil.* 2008;35(7):524-547.
24. Huang GJ, LeResche L, Critchlow CW, Martin MD, Drangsholt MT. Risk factors for diagnostic subgroups of painful temporomandibular disorders (TMD). *J Dent Res.* 2002;81(4):284-288.
25. Kalamir A, Pollard H, Vitiello AL, Bonello R. TMD and the problem of bruxism. A review. *J Bodywork Movement Therapies.* 2007;11(3):183-193.
26. Manfredini D, Lobbezoo F. Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2010;109(6):e26-e50.

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