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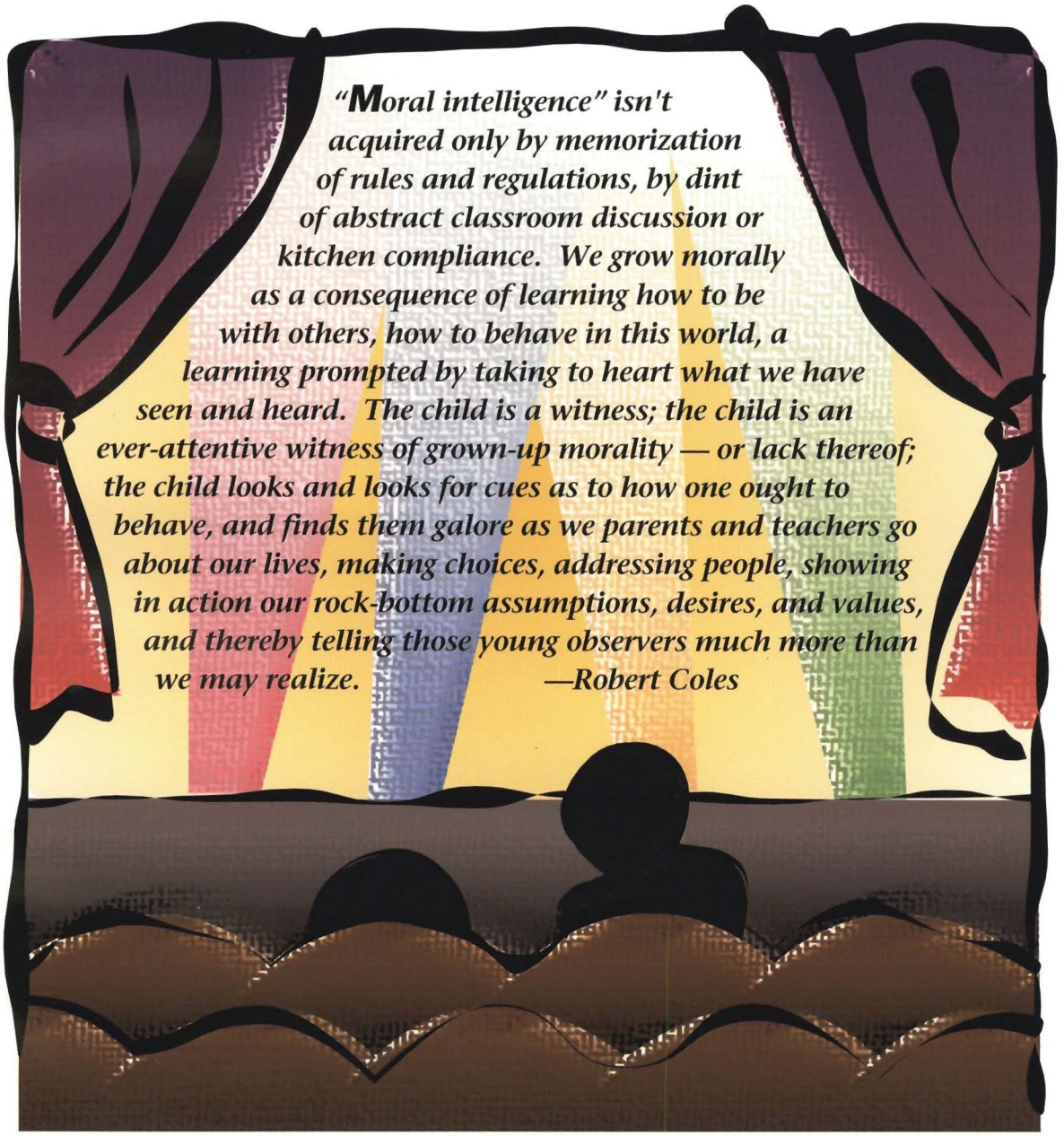
JANUARY—FEBRUARY 1997



THOU HAST BEEN...
A MAN THAT FORTUNE'S BUFFETS AND REWARDS
HAS TAKEN WITH EQUAL THANKS ...GIVE ME THAT MAN
THAT IS NOT PASSION'S SLAVE, AND I WILL WEAR HIM
IN MY HEART'S CORE, AYE, IN MY HEART OF HEARTS
AS I DO THEE...

—Hamlet to his friend Horatio

Handicaps • STEREOTYPE • Sexism
Children in Crisis

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JANUARY-FEBRUARY 1997 VOLUME 64 NUMBER 1

 USPS #279-480. Issued bimonthly—in January-February, March-April, May-June, July-August, September-October, and November-December—John Hancock Center, 875 North Michigan Avenue, Ste 4040, Chicago, IL 60611-1901, (312) 943-1244. Periodicals postage paid at Chicago, IL and additional mailing office. Subscription prices: within U.S.A., individuals $100.00 per volume, institution $120.00, single copies $35.00; Foreign (including Canada and Mexico) individuals $120.00 per volume, institution $150.00, single copies $40.00. Foreign subscriptions are sent airmail. Ninety dollars ($90.00) of the full membership dues are allocated to the Journal. **Subscriptions to the Journal cannot be sold to anyone eligible for membership in ASDC.** Member—American Association of Dental Editors.

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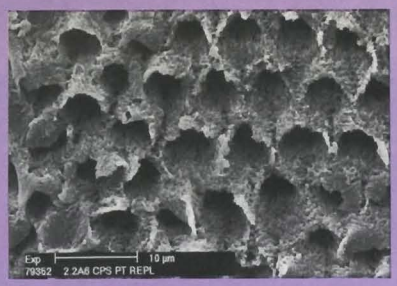
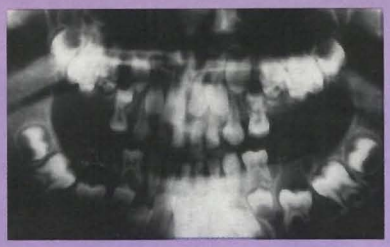
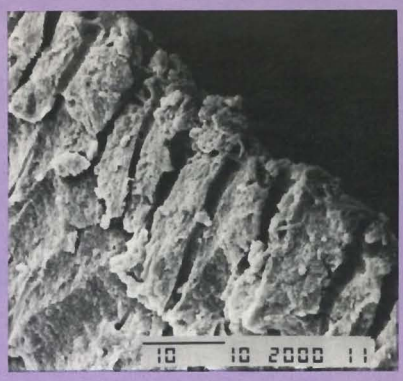
The child is a witness; the child is an ever-attentive witness of grown-up morality.
Art and design by Sharlene Nowak-Stellmach

CLINIC







BEHAVIOR

EARLY CHILDHOOD CARIES

ECONOMICS

REPORT

Calendar

1997

ASDC Seminar, ADA, Chicago, IL, May 10.
ASDC Annual Meeting, Registry Resort, Naples, FL, October 22-26.

1998

ASDC Annual Meeting, Hyatt Regency Beaver Creek, Avon, CO, October 14-18.

CLINIC

Comparison of in vivo and in vitro demineralized dentin with phosphoric and maleic acid

Maria Crysanti Cagidiaco, MD, DDS
Marco Ferrari, MD, DDS
Carel Leon Davidson, PhD

The prime mechanism of modern bonding to dentin is currently believed to be based on micromechanical interlocking between acid etched dentin and the resin bonding-agent.[1,2] The interlocking mechanism is based on a process known as hybridization by which complete removal of the smear layer and concurrent demineralization of the dentin surface layer occur, followed by resin interdiffusion into the microporosities of the exposed dentinal collagen matrix.[3]

For this, the morphology of the collagen network of superficially demineralized dentin is of paramount importance in establishing the quality of the bond with resin composites by way of hybridization.[1,4,5]

The type of conditioner and the condition of the dentin with respect to humidity and outward flow of liquid from the tubules might affect the "etching pattern".[6,7]

The purpose of the present study was to investigate whether differences can be observed between dentin that had been demineralized with either an inorganic or an organic acid, under clinical as well as under laboratory conditions.

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MATERIALS AND METHODS

In vitro samples

Sixteen anterior teeth were extracted for periodontal reasons. They were stored at room temperature in distilled water for no longer than three weeks. Semispherical Class V cavities were prepared with a diameter of 3 mm and a maximal depth of 2 mm. They were cut under water spray with a diamond point bur at high speed. The cavities were placed at the cementoenamel junction with half the cavity margin located in enamel and half in cementum. The samples were randomized in two groups of eight.

The cavities of Group 1 were etched with a viscous gel of 36 percent phosphoric acid (De Trey Etch, Dentsplay, Konstanz, Germany) for 15 s and then washed for 20 s. The samples were kept wet. Before being fixated in a 10 percent buffered formaldehyde solution for 12 hrs, the samples were sectioned in the middle of the cavity along their long axis by cutting a groove through the lingual surface and then fracturing them with a sharp chisel and mallet.

The samples of Group 2 were handled as in Group 1 with the exception that they were etched with a low-viscous, aqueous solution of 10 percent maleic acid (3M, St. Paul, USA).

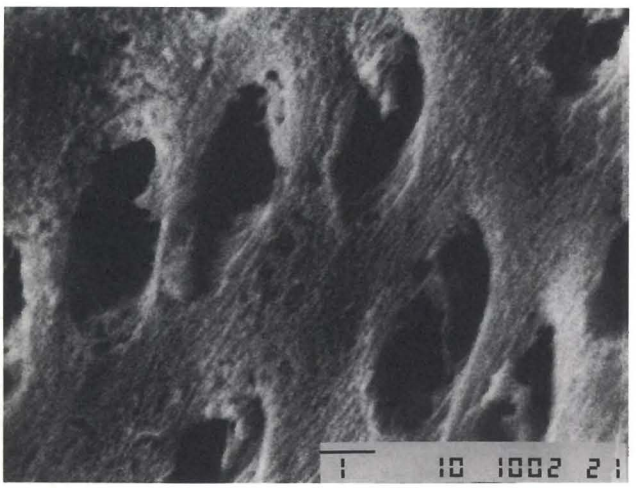

Figure 1. Dentin surface after etching with 36 percent phosphoric acid for fifteen seconds in vitro. *The tubule orifices appeared enlarged because of the loss of peritubular dentin. The collagen network can be observed (SEM 10000x).*

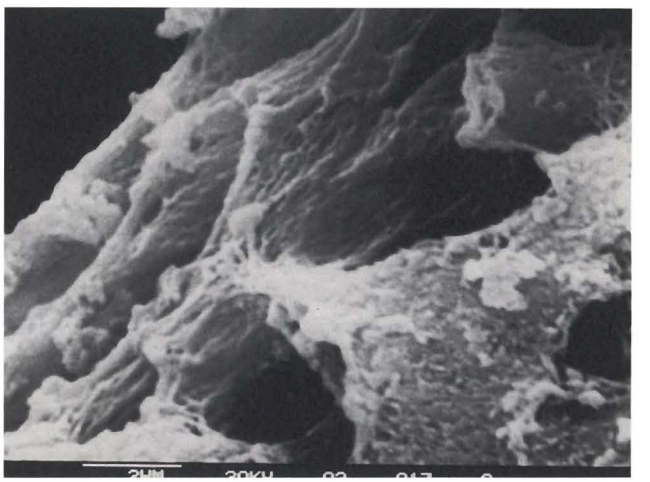

Figure 2. Dentin surface etched with 36 percent phosphoric acid in vitro. *Many intratubular and intertubular collagen fibers are exposed (SEM 10000x).*

In vivo samples

Eight periodontally involved anterior teeth, already scheduled for extraction were selected. Before the experimental procedures, the informed consent of the patient was obtained. The vitality of the teeth was confirmed by electrical and thermal vitality tests, before the experiment began. The sample teeth were divided in two groups of four samples each, for the different conditioning procedures. Rubber dam was applied before cavity preparation. Class V cavities were prepared, using local anesthesia (with 1:100,000 adrenaline), in a way similar to that followed in the *in vitro* study.

The cavity walls in Group 3 were conditioned as in Group 1, and those of Group 4 as in Group 2. Keeping the rubber dam in place, the sample teeth were gently extracted. This procedure was possible because of their mobility. After extraction, the sample teeth were treated in a way similar to that followed for the *in vitro* samples.

All the fractured samples were critical-point dried in a Balzer CPD Device (Balzer Ltd., London, England), mounted in aluminum stubs and conductively coated with gold in a sputtering device, before being examined with a Scanning Electron Microscope (Cambridge Stereoscan 250, Cambridge Co., London, England) along their entire fractured surfaces. Micrographs were taken at intervals to illustrate representative aspects.

RESULTS

Acid-etching in Group l produced significant changes in the dentin surface, enlarging the tubule orifices approximately to 3μm in diameter, due to the loss of peritubular dentin (Figure 1).

The porosity of intertubular dentin after demineralization within the tubules was evident. These changes could be seen clearly at high magnification, where many exposed collagen fibers on the surface could be distinguished (Figure 2). Direct observation of the cavity walls showed collagen fibrils also within the demineralized tubule shafts at the lateral walls (Figure 3). When the specimens were observed in cross-section, collagen fibers in the etched tubules were observed (Figure 4). Also a different appearance and orientation of the collagen fibers lining the tubules could be detected (Figure 5). The depth of demineralization of the perpendicularly cut tubules at the cavity floor, was not uniform and ranged between 5 and 10 μm, if the total of all of the specimens was taken into account.

High magnification of Group 1 samples revealed a surface layer of collapsed collagen fibers. Many small pores were observed among collagen fibers and seemed to be covered and connected by an amorphous surface coating (Figure 6).

The collagen fibers in the intertubular dentin had a random orientation. In group 2 the intertubular dentin was examined at various magnifications, and the surface

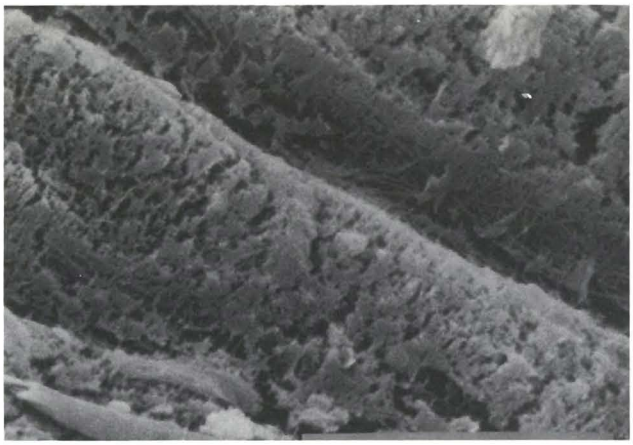

Figure 3. Lateral wall of a Class V cavity etched with 36 percent phosphoric acid in vitro. *Along the etched shafts it is possible to detect exposed collagen fibers (SEM 10000x).*

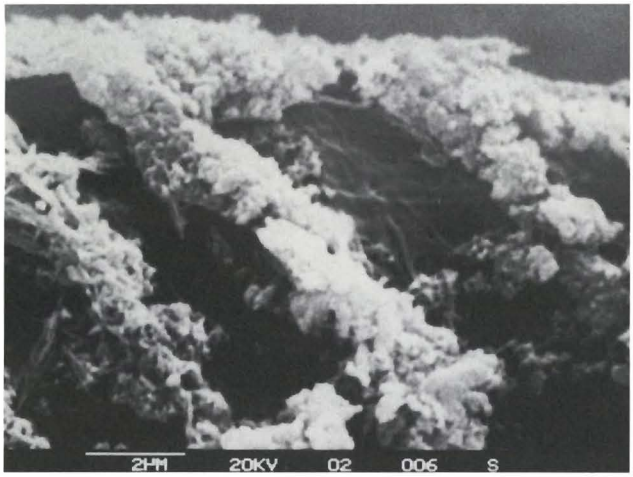

Figure 4. Cross-section of dentin etched in vitro *with phosphoric acid. The subsurface details show collagen fibers in the etched tubules (SEM 10000x).*

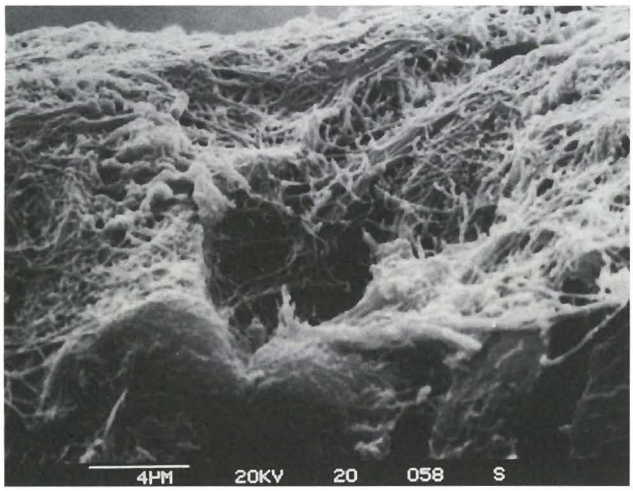

Figure 5. A well-developed collagen network of the bulk dentin is exposed after etching with 36 percent phosphoric acid in vitro. *Variable orientations of the collagen fibers can be seen (SEM 5000x).*

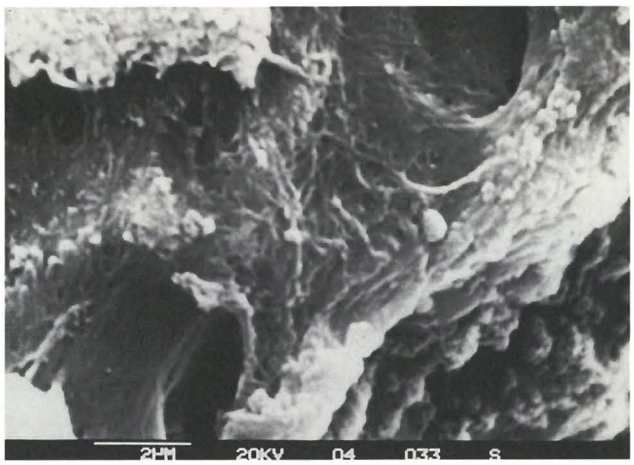

Figure 6. Phosphoric acid exposes small pores among collagen fibers, in in vitro *sample (SEM 10000x).*

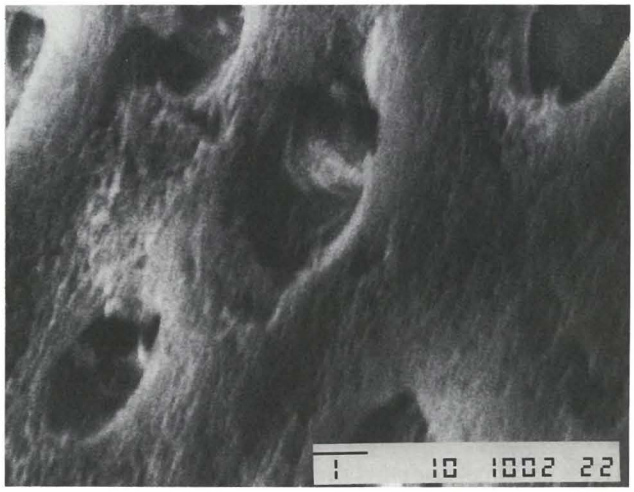

Figure 7. Etching treatment with 10 percent maleic acid in vitro *leaves a similar morphology to that observed on samples in Figures 1-6 (SEM 10000x).*

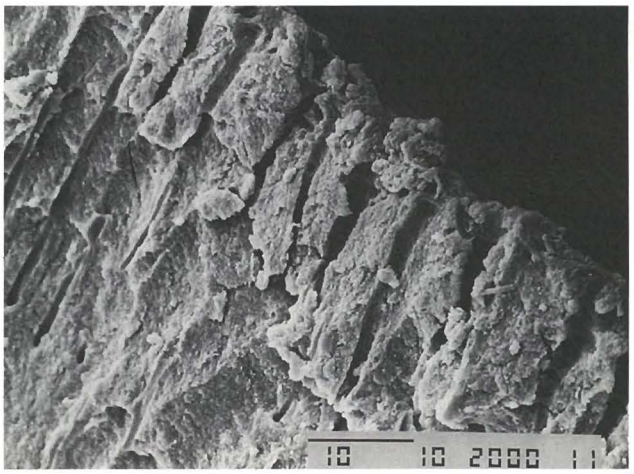

Figure 8. The depth of demineralization in the tubules of this sample, after etching in vitro *with maleic acid, is about 10 µm (SEM 2000x).*

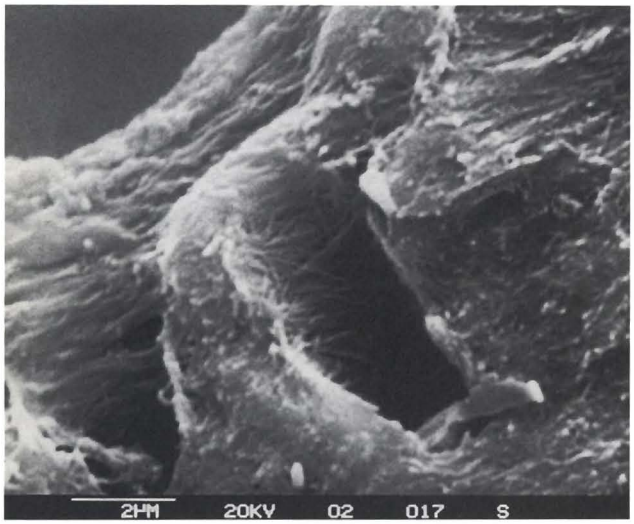

Figure 9. Dentin sample in vivo *etched with 36 percent phosphoric acid. Intratubular collagen is visible (SEM 10000x).*

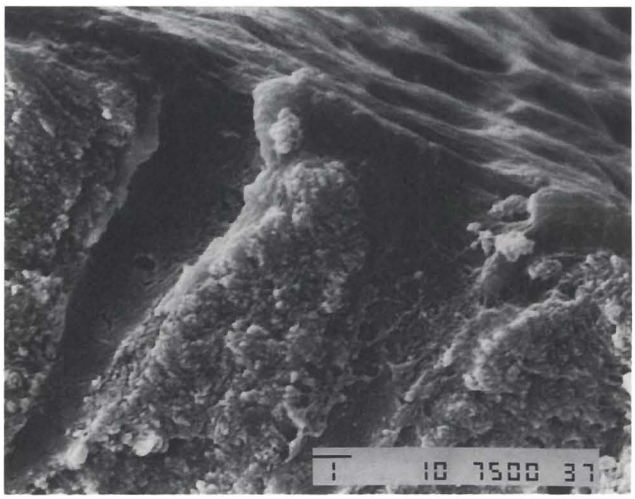

Figure 10. Another sample in vivo *etched with 10 percent maleic acid. The intertubular dentin is similar to that conditioned with phosphoric acid (SEM 7500x).*

appeared to be similar in porosity to the samples of Group 1 (Figure 7).

The depth of demineralization in the tubules was between 3 and 10 µm (Figure 8), and a sharp demarcation within the open tubules between the more apical peritubular dentin and collagen fibers was noted.

In groups 3 and 4 the SEM pictures of the *in vivo* samples were completely in agreement with those of the *in vitro* samples. In fact, the *in vivo* samples presented similar differences of depth of demineralization and in the appearance of the intertubular dentin layers for both etching agents (Figures 9-10). When observed along the fractured surfaces, the Group 4 samples exhibited a continuous amorphous surface layer of approximately 0.5-1.0 µm thickness, composed of collagen fibers (Figure 10). The collagen fibers were randomly distributed on the dentin surface (Figure 11). The collagen network at the surface of the conditioned dentin was evident (Figure 12).

The surface layer of collapsed, demineralized collagen fibers appeared, however, to be intimately associated with the dentin surface. The continuity between subsurface collagen fibers and the surface layer was visible in all the *in vivo* samples (Figures 9-12).

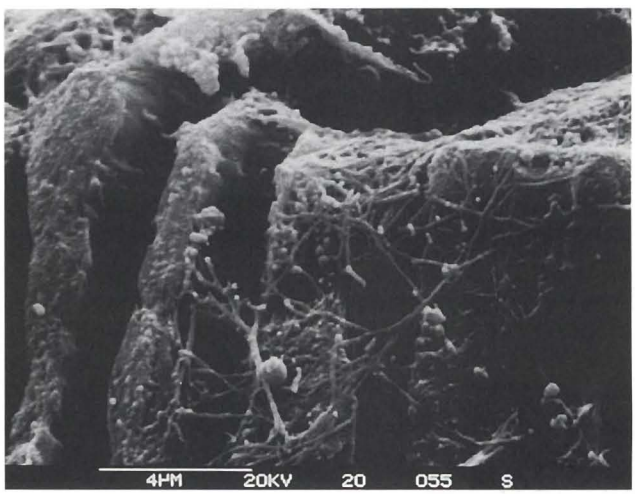

Figure 11. Dentin after in vivo *etching with phosphoric acid. The collagen fibers are randomly disposed on the dentin surface (SEM 7500x).*

DISCUSSION

Both ways of acidic substrate conditioning used in this investigation created a collapsed network of collagen fibers. When the mineral component of the dentin matrix is removed by acid, the residual collagen phase loses its rigidity and will collapse. As a result, the fibers will come together, appearing in some cases to be fused.[8] For this reason, the critical-point drying technique is extremely important in order to be certain about the exposure of the dentin collagen layer; otherwise the demineralized layer will collapse into a dense mass.[9] Furthermore, high

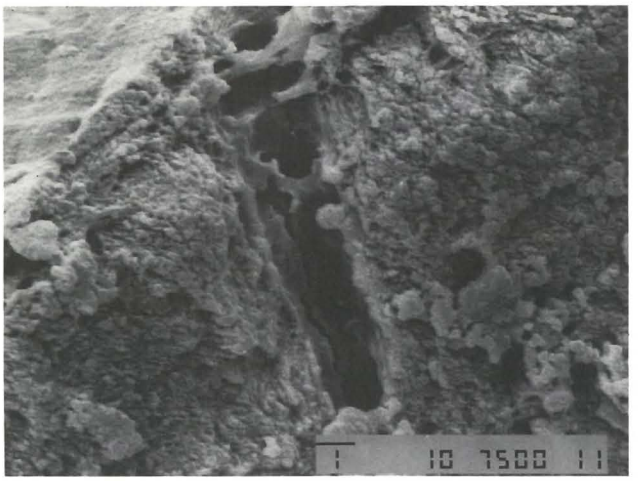

Figure 12. After in vivo *etching with maleic acid the collagen layer is evident and the peritubular dentin is dissolved, leaving the collagen fibers exposed (SEM 7500x).*

magnification microscopy is essential to a clear detection of the morphology of the extremely small pores, throughout the dentinal collagen network.[3] Once collapsed, the collagen network can be reexpanded with a hydrophilic primer that penetrates into the spaces between the collagen fibers. This collagen bulk can more easily be impregnated by the primer, if the dentin is kept wet.[7] Moreover, it has been shown that desiccation of etched dentin with compressed air can increase the collapse of the collagen network.[10] Diffusion of resin into the tissue, altered morphologically during conditioning, should ideally extend up to and include the unaltered dentin.[6] The diffusion of monomers and the penetration of bonding resin into the demineralized surface can form the hybrid layer and modify its physical properties, creating a gradient of elastic moduli at the bonded interface, allowing flexibility and thus protecting the bond from stresses and disruption.[8,11]

Several recent studies drew attention to dentin morphology and, in particular, to the tubular orientation in relation to bond strength and the marginal seal of restorations.[6,12-16] Because of that and based on our observations of a collagen-fiber-network in the shafts and intertubular dentin, it can be speculated that also in those areas of cavity walls presenting tubules not sectioned perpendicularly (as in the case at the cervical margins), the micromechanical interlocking can still be formed between the collagen layer and the resin monomer.

This study demonstrated a similar morphology of collagen network for *in vitro* and *in vivo* prepared samples, which is in agreement with the literature.[10,17,18] The *in vitro* model can be used, therefore, to study clinical conditions. What matters is the nature and the concentration of the conditioners, when depth of demineralization is involved.[7,19]

CONCLUSIONS

- ☐ Both of the tested etching agents are similarly effective in completely removing the smear layer and demineralization of the dentin, leaving a porous collagen network layer.
- ☐ Conditioning *in vitro* or *in vivo* produce a similar micromorphology of the dentin.
- ☐ The 36 percent phosphoric acid and 10 percent maleic acid were similarly effective in demineralizing dentin.

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Repair of class I resin-composite restoration

Theodore P. Croll, DDS

Using wear-resistant resin composites that have the best physical characteristics, dentists can reasonably expect a Class I resin-composite restoration to last ten years or more, before requiring additional clinical intervention. Differences in respective coefficients of thermal expansion, however, among resin-composite, dentin and enamel; normal wear from occlusion and mastication; and the difficulties of existing in the moist, microflora-laden, acid pH environment of the mouth, all work to break down even the best of restorative resin systems and their bonding mechanisms. The same influences also limit the life of silver amalgam and other types of restorations.

A chief advantage of bonded resin-composite Class I restorations is that defective areas can often be repaired. Such treatment can be an inexpensive, simple alternative to complete replacement of the restoration and also permits conservation of tooth structure. This article pictures and describes a step-by-step method of repairing a defective marginal area of a bonded Class I resin-composite restoration.

TECHNIQUE

Ten years and three months after placement of a bonded resin-composite occlusal Class I restoration in a mandibular permanent first molar, a teenaged patient required repair of a defective margin (Figure 1).

Probing with an explorer revealed carious substance at the resin-enamel interface. After application of benzocaine topical anesthetic gel around the gingival margins of the tooth, the patient claimed no discomfort from placement of the rubber dam retainer. Local anesthetic injections, therefore, were not used. The defective resin and carious substance were removed, and the resin-composite restoration was repaired in the following manner:

1. A small, slow-speed inverted cone bur was used to remove defective resin and debride the carious substance (Figure 2). This bur, by its design, cut a mechanically interlocking retention form in the enamel and resin that made up the walls of the new cavity preparation.
2. Care was taken to remove all carious substance (Figure 3). Preparation depth in this case was just short of dentin exposure.
3. After removal of the carious substance, a large round diamond bur was used to roughen the surface of the residual resin-composite and peripheral enamel (Figure 4).
4. Forty-percent phosphoric acid was applied, and gently agitated with a ball burnisher for about 20 seconds (Figure 5).
5. After complete rinsing and drying of the occlusal surface, the residual resin material, adjacent enamel and enamel within the preparation all had a dull, frosty appearance, indicating proper etching (Figure 6).

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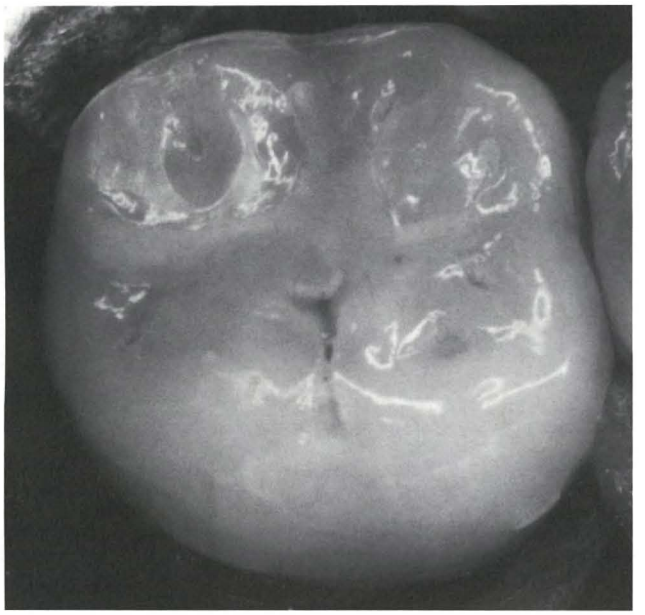

Figure 1. This ten-year-old occlusal Class I resin-composite restoration has caries associated with a marginal defect.

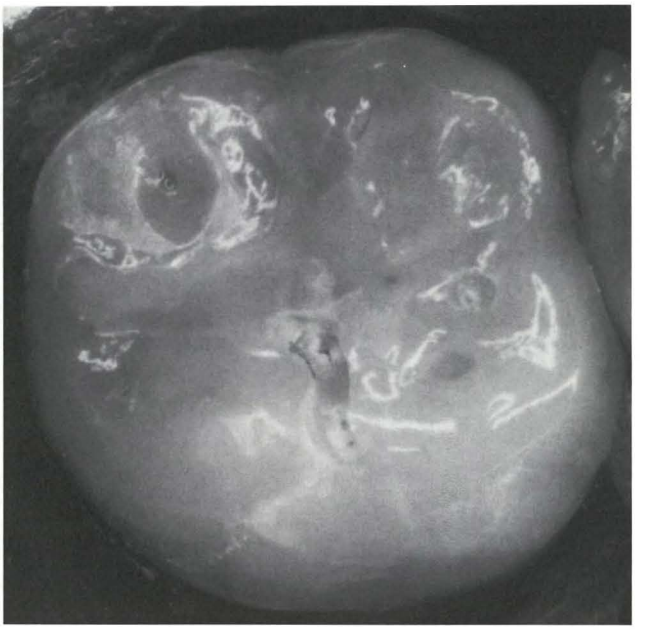

Figure 3. Preparation of "outline-form" reveals small, deeper areas of carious infection.

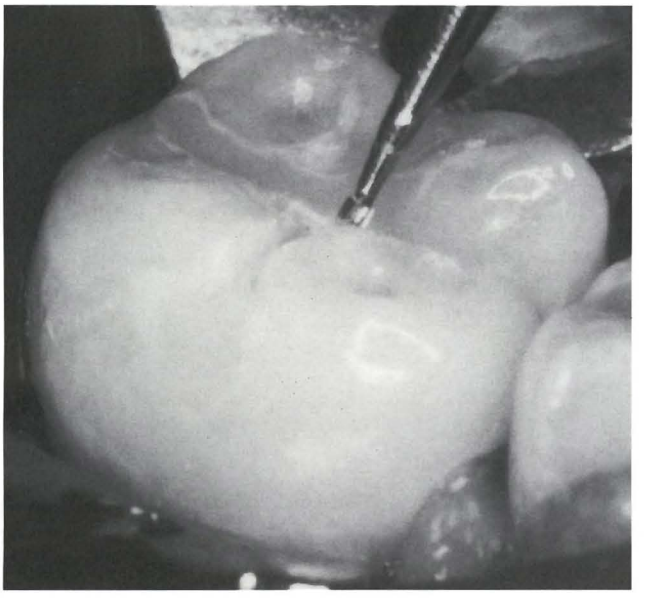

Figure 2. A small inverted cone bur is used to remove defective resin and carious substance.

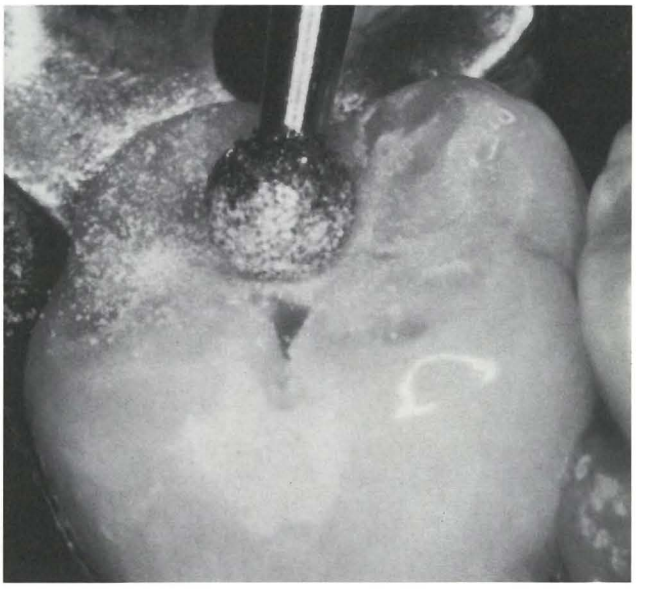

Figure 4. A large, slow-speed, diamond bur roughens the residual resin-composite surface and adjacent enamel.

6. A thin layer of liquid resin bonding agent was painted over the entire occlusal surface and on the walls and floor of the new cavity preparation (Figure 7). This layer of liquid resin was not polymerized immediately; it was left to saturate the enamel surface for about ten seconds.

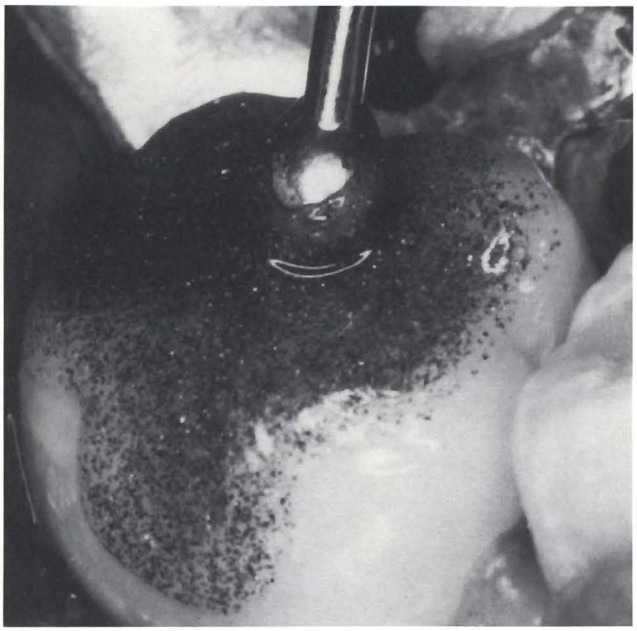

Figure 5. Forty percent phosphoric acid is applied for twenty-thirty seconds.

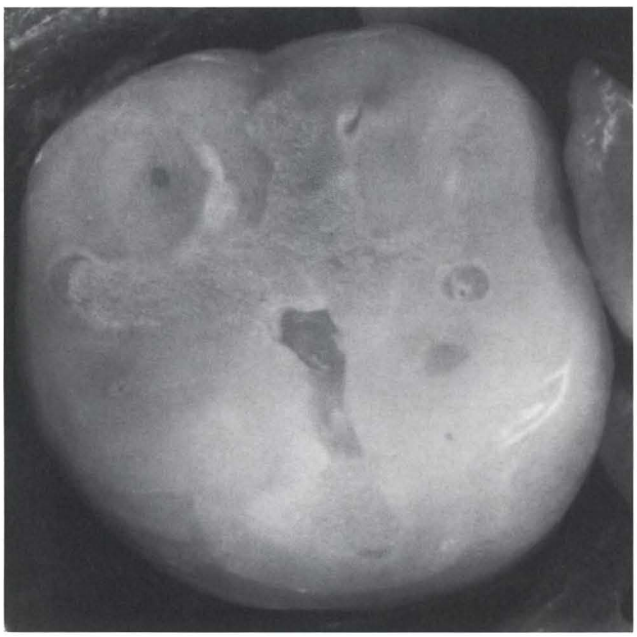

Figure 6. The completed preparation and properly etched surface is shown, ready for renewed restoration.

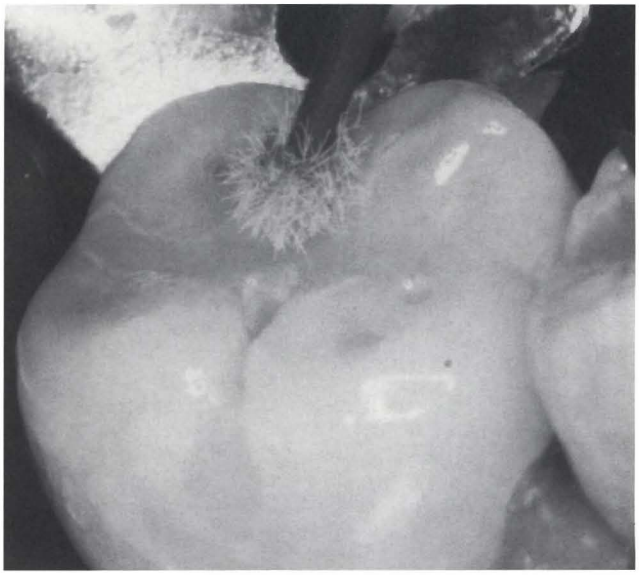

Figure 7. Clear resin bonding liquid is applied in a thin layer, across the occlusal surface and within the new preparation.

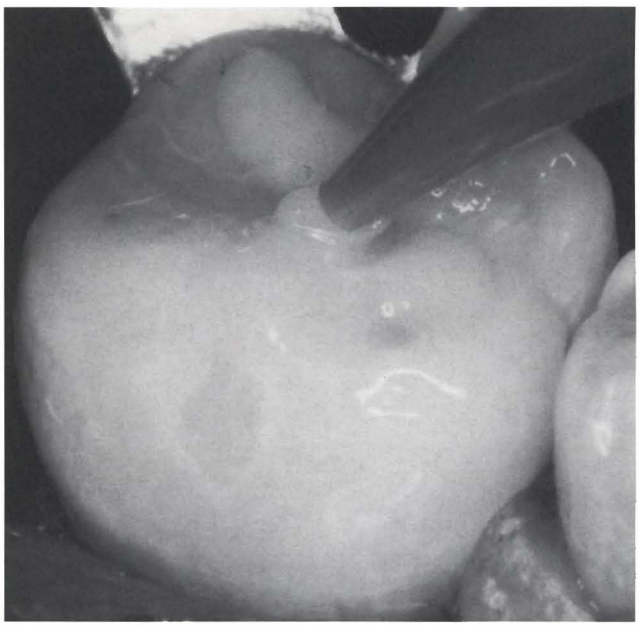

Figure 8. "Posterior" resin-composite restorative material is syringed into the preparation, with a bit of excess overflowing the cavosurface margins.

7. Before application of the light beam, wear resistant resin-composite restorative material was syringe-injected into the preparation (Figure 8). A small extra portion of resin-composite overflowed, as excess, upon the occlusal surface (Figure 8).

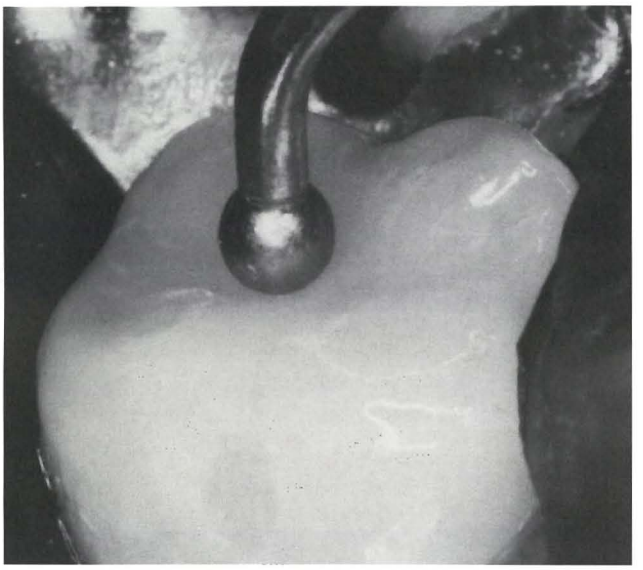

Figure 9. A ball burnisher is used to compress the filled resin into the repair preparation, and to spread it in a thin layer, over the residual restorative material and adjacent enamel.

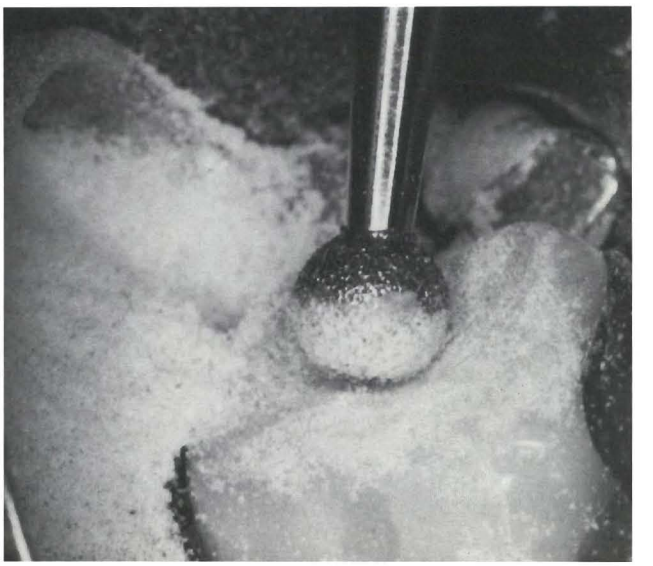

Figure 11. Using stones, burs or disks, anatomical form is sculpted. Diamond burs at slow speed are ideal for this step.

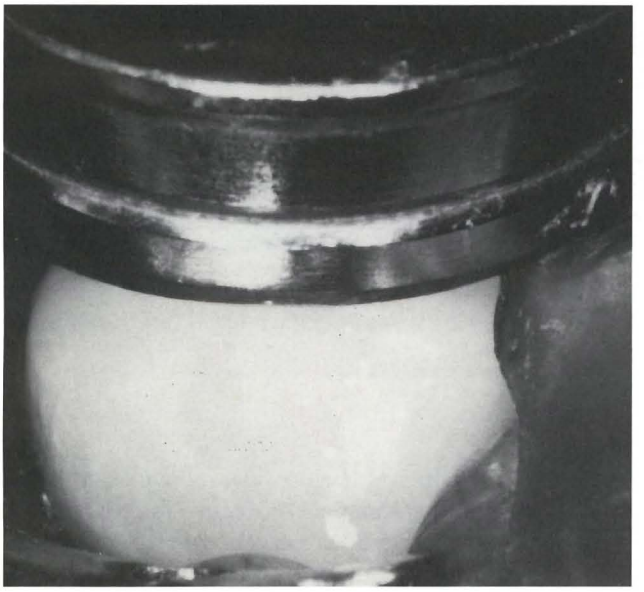

Figure 10. The visible light beam is applied for forty seconds.

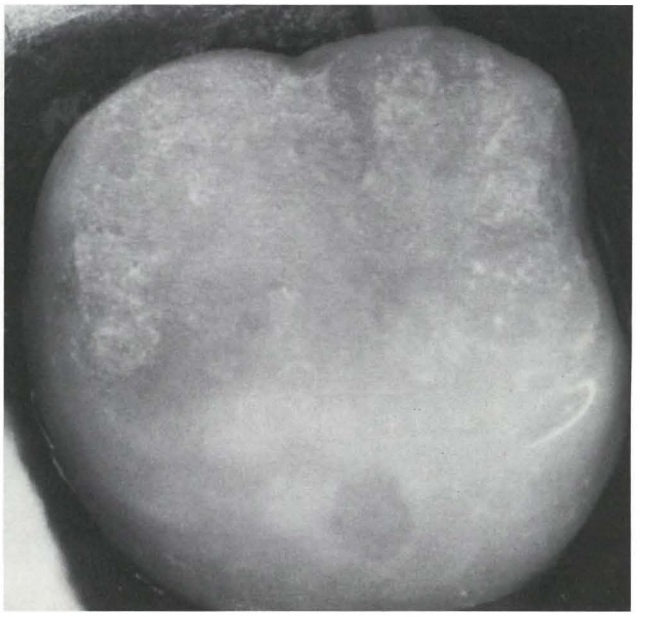

Figure 12. After re-etching, rinsing and drying, the repaired surface is ready for final resin sealant application.

8. Using a large ball burnisher, the resin-composite was compressed into place, with care to avoid incorporation of air bubbles. Excess material was spread over the residual restorative material and adjacent enamel margins to serve as a new, reinforced bonded sealant (Figure 9). (Because the resin-composite layer was less than 2mm in thickness, it was applied and polymerized in one portion. In cases of deeper preparations, several

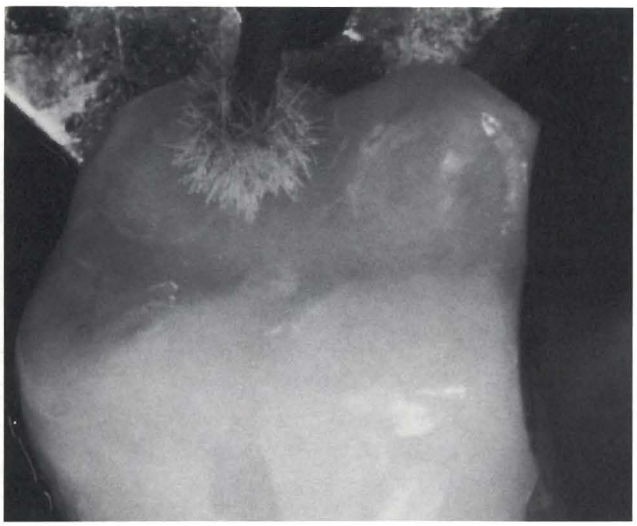

Figure 13. Clear resin sealant is applied and after a ten second wait, light polymerized.

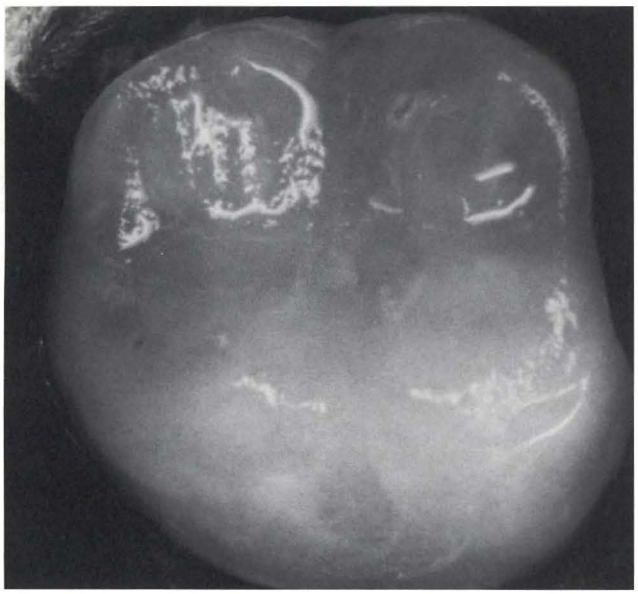

Figure 14. Occlusal surface before evaluation of occlusion.

layers should be applied, each polymerized separately, to minimize the effects of resin shrinkage related to polymerization.)

9. The visible light beam was applied for sixty seconds (Figure 10).
10. A large slow-speed diamond bur was then used to sculpt ideal occlusal form and develop acceptable occlusal contacts (Figure 11). (Some practitioners may desire at this time to remove the rubber dam and evaluate occlusal contacts. I find the markings from articulating paper easier to remove, however, after final resin sealing is completed). After re-applying 40 percent phosphoric acid to the resin and enamel surfaces, followed by complete water rinsing and drying (Figure 12), clear resin sealant should be painted over the entire occlusal surface. After the liquid resin saturates the surface for ten to fifteen seconds, the light beam is applied for thirty more seconds (Figures 13 and 14).
11. After removal of the rubber dam, occlusal contacts in all mandibular functional positions, are

Figure 15. After inspecting opposing teeth for sharp, pointed cusps, and rounding them, articulating paper markings confirm acceptable occlusal contacts.

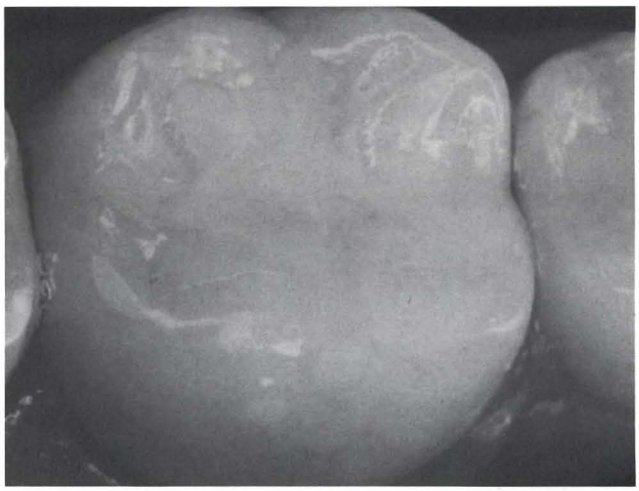

Figure 16. The molar is shown two years after the repair, and twelve years after placement of the original Class I resin-composite restoration.

evaluated. Opposing cusps that are excessively pointed, should be rounded with disks or stones, to eliminate "jack-hammer-like" concentration of forces on the resin surface.[1] Occlusal prematurities, designated by articulating paper markings on the resin, are adjusted as indicated (Figure 15). If much adjustment needs to be done, additional re-sealing of the surface should follow.

12. This Class I resin-composite restoration is shown twelve years after initial placement, and two years after the above described repair (Figure 16).

DISCUSSION

Overall strengths of repaired areas in resin-composite restorations are not as great as those of the original restorative material.[2-7] Clinical experiences of more than ten years using this procedure reveal, however, that repaired surfaces do not detach, fracture, or otherwise breakdown even after the repair is completed. The author has not yet re-repaired any such restoration.

To enhance the resin-to-resin bond, all of the residual restorative surface should be roughened with the diamond bur. This treatment exposes fresh resin that has not been contaminated by exposure to the oral environment. In addition, when the liquid bonding agent is applied to the dried, etched surface, a 10-second delay permits the resin to saturate and optimizes length of resin tags into interprismatic regions.[8] Perhaps this enhances retention and helps to eliminate microleakage.

The final application of sealant is important. Not only are contraction gaps eliminated and margins truly sealed, but the resin-composite surface that had just been damaged by the finishing process, is resurfaced with fresh resin.[9] It has been shown that final surface sealing of such restorations also significantly enhances wear resistance.[10]

Even though there exists a chemical union in the resin-to-resin bond, and the liquid resin sealant micromechanically bonds to the etched enamel, there is no reason not to take advantage of mechanical interlocking retention form in the repair preparation. The three methods of retention augment each other, and the amount of tooth structure sacrificed by using an inverted cone bur is minimal. In addition, perhaps such retention form helps to minimize the effects of polymerization shrinkage of the filled resin. It is conceivable that forces of shrinkage are absorbed by surrounding resin and enamel walls, and dissipated by using such a cavity design. This potentiality should be investigated *in vitro*.

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ROLE OF FLUORIDE TOOTHPASTES IN CARIES DECLINE

The results of the present study suggest that the role of fluoride toothpastes in the caries decline in industrialized countries may have been overrated, or that the difference in fluoride exposure between sexes was too small to effect a detectable change in per cent DMFT gender difference. Further studies are needed because, whatever the answer may be, it will strongly influence the choice of caries preventive strategies for the future.

Haugejorden, Ola: Using the DMF gender difference to assess the "major" role of fluoride toothpastes in the caries decline in industrialized countries: a meta-analysis. Community Dent Oral Epidemiol, 24:369–375, December 1996.

Fluoride uptake by proximal surfaces from professionally applied fluorides: An in vitro study

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Milton Houpt, DDS, PhD
Zia Shey, DMD, MS

Professionally applied topical fluorides are used widely as a supplement to systemic fluoridation. These were first introduced as a liquid, which was swabbed onto isolated and dried teeth. Many clinical studies established the effectiveness of this technique in reducing dental caries.[1-5] Subsequently fluoride gels were developed and these were applied in Styrofoam trays to teeth which were not isolated. In order to determine the effectiveness of fluoride gels an enamel biopsy technique was developed to assess fluoride uptake by enamel.[6-9] Many enamel fluoride uptake studies were performed on prepared enamel surfaces or on prepared blocks of enamel harvested from the buccal or lingual surfaces of human or bovine teeth.[3,5,10-13] Some studies were performed using the buccal or lingual surfaces of recently exfoliated teeth or of teeth extracted for orthodontic purposes.[1,14,15] Other investigators used root surfaces to evaluate the fluoride uptake.[16] There were studies performed to evaluate the morphological changes of enamel surfaces with the application of fluoride, and in those studies the SEM microscope was used.[1,2,11,17,18] There has been little investigation, however, of fluoride uptake by proximal surfaces of teeth. This in vitro study was performed to determine fluoride uptake of enamel on the interproximal surfaces of posterior teeth, following topical application of various types of fluoride.

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Dr. Houpt is Professor and Chairman, Department of Pediatric Dentistry; Dr. Shey is Professor, Department of Pediatric Dentistry and Associate Dean for Student Affairs and Graduate Dental Education, University of Medicine & Dentistry of New Jersey, New Jersey Dental School.

METHODS AND MATERIALS

Preparation of the samples

Twelve extracted human premolar and forty-eight molar teeth were collected and stored in a dilute solution of hydrogen peroxide solution. Small pieces of tape (Curity Wet Proof, Colgate Palmolive Co., NY) had two circular windows or holes (diameter of 2.0 mm) punched out 1 mm apart. These were placed carefully on the dried mesial surface of each tooth at the contact area. The punched out pieces of tape were saved, and one was used to cover one of the holes in the tape. The edges of the tape and the covered hole were then sealed with a nail varnish (Cutex quick color nail paint). The exposed hole became the fluoride treated experimental site and the sealed hole represented the control site. The teeth were randomly divided into four groups with fifteen teeth in each group and mounted in wax blocks with each block containing five teeth.

Table 1 ☐ Mean fluoride concentration (ppm) in proximal enamel with various topical agents. (Fluoride uptake is represented by the difference between control and treated sites at each depth.)

	Treated			Control			Difference (Treated *minus* Control)			Statistically significant difference		
Treatment	Layer 1	Layer 2	Layer 3	Layer 1	Layer 2	Layer 3	Layer 1	Layer 2	Layer 3	Layer 1	Layer 2	Layer 3
Regular gel	2021 ± 1439	1100 ± 670	897 ± 449	870 ± 372	587 ± 511	559 ± 458	1150	512	338	.0003*	.0846	.1663
Thixotropic gel	2073 ± 1389	1301 ± 696	1737 ± 1652	1014 ± 619	785 ± 537	817 ± 756	1058	515	919	.0009*	.0830	.0001*
Foam	2039 ± 1916	1529 ± 2172	1024 ± 794	919 ± 687	775 ± 646	726 ± 516	1120	753	297	1.0004*	.0120*	.0221
Liquid	587 ± 249	363 ± 156	281 ± 184	559 ± 643	490 ± 714	400 ± 527	27	126	118	.9276	.6673	.6251

* Statistically significant at ANOVA p-value ≤ .05

Fluoride treatments

The teeth were treated for four minutes with one of the following commercially available office-applied topical fluoride preparations: 1.23 percent acidulated phosphofluoride (APF) gel, thixotropic (APF) gel, APF foam and 2 percent neutral sodium fluoride solution. The preparations were applied according to each manufacturer's instructions for four minutes. The teeth in the first group were treated with a thin ribbon of conventional gel placed in a foam lined tray (Oral-B, Redwood, CA) and held in place with constant finger pressure. The second group of teeth was treated with a thin ribbon of thixotropic gel placed in a foam lined tray held in place with intermittent finger pressure. The third group of teeth was treated with a thin ribbon of foam placed in a foam lined tray held in place with constant finger pressure. The fourth group of teeth was treated with 2 percent neutral sodium fluoride solution applied continually with cotton tip applicators. The teeth were then soaked for twenty-four hours at room temperature in an artificial saliva (metastable calcium phosphate) solution that was prepared from 1.5 mmol/L calcium chloride, 8.2 mmol/L sodium bicarbonate, 4.8 mmol/L sodium chloride, 137 mmol/L potassium chloride and 4 mmol/L potassium dihydrogen phosphate, adjusted to pH 7.0.[19] After soaking in artificial saliva for twenty-four hours, the teeth were removed from the wax blocks and the fluoride uptake of each surface was analyzed by enamel acid-etch biopsy.

Acid-etch biopsy technique

Each treated surface and each control surface were etched three different times to evaluate the penetration of the fluoride at different depths. 10 μL of 0.1N of perchloric acid was used with a micropipette (Corning, NY) three times for thirty seconds each time, to obtain three successive layers of etched enamel. Each 10 μL of perchloric acid etching solution was then absorbed with a round (8 mm diameter) filter paper (Whatman No. 3) and placed in a test tube containing 0.5 mL of deionized water for twenty minutes. A 0.5 mL aliquot of TISAB (Total Ionic Strength Adjustment Buffer, Orion Research, Cambridge, MA) and 10 μL of 0.5N sodium hydroxide was placed in the test tube to adjust the biopsy solution to a final pH of 5.2.

Fluoride analysis

A 0.25 mL aliquot of the biopsy solution was analyzed for fluoride, using a fluoride specific ion electrode (Model 94-09, Orion Research Inc., Cambridge, MA) and a reference electrode (Model 90-01, Orion Research Inc., Cambridge, MA), which were standardized, using fluoride solutions containing the same acid-TISAB contents as the standard solutions. The electrodes were immersed in a 0.01 ppm fluoride standard solution between use and operated at least once daily to ensure that they were functioning correctly. The electrodes were soaked in the biopsy specimens for ten minutes before a reading was taken on the Digital Ionalyser (Orion Research Inc., Cambridge, MA). The first two hours each day the electrodes were calibrated with a standard fluoride solution. In addition, the calcium in each layer was analyzed by atomic absorption spectroscopy. The depth of the successive biopsies was estimated, using the methods described in similar studies.[20-24]

Statistical analysis

A two-way analysis of variance (ANOVA) with treatment and tooth as factors was used to analyze the raw data. A Fisher's Protected LSD test was conducted to demonstrate significant differences between multiple means. Statistical significance was declared when the p-value of a two-tailed test was 0.05 or less.

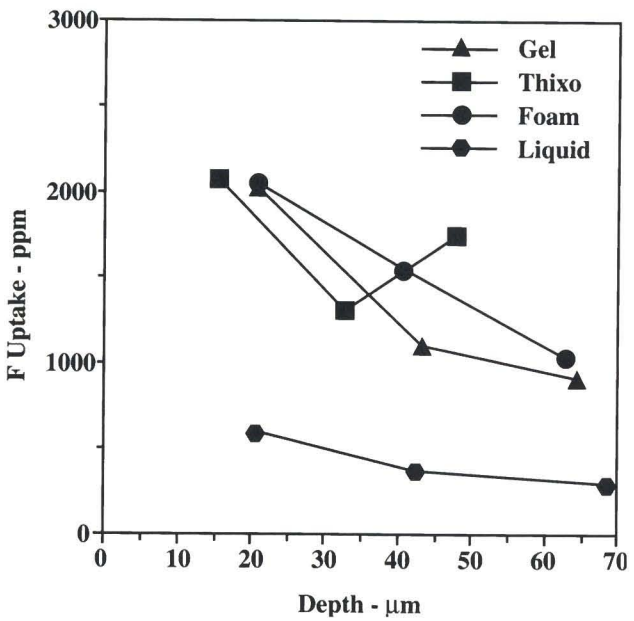

Figure. In vitro fluoride uptake by mesial enamel from topical fluoride preparations.

RESULTS

Table 1 and Figure illustrate the results of this study. No statistically significant differences were found in the fluoride contents in the control sites of each group.

When the experimental and control sites were compared, statistically significant differences were observed with all treatments, with the exception of the neutral fluoride solution (Fisher protected LSD test, p-value < 0.05), which produced little fluoride uptake. In the surface layer the differences in fluoride uptake of the control and experimental sites for both gels and foam were 1150, 1058 and 1120 ppm F; whereas, with the solution the difference was only 27 ppm F. There was a significantly higher fluoride uptake by the outermost layer when compared to the innermost layer. For example, the gel had a fluoride uptake of 2021 ppm for layer 1 and 897 ppm for layer 3. In general, there was a decrease in the mean fluoride uptake as the depth of the enamel layer increased (Figure). These results can be compared with those obtained by other investigators (Table 2) who obtained much greater fluoride uptake on surfaces that were exposed and not in contact. It was also demonstrated that teeth in contact do not allow as much fluoride uptake as buccal surfaces of teeth, which can be readily exposed to the topical fluoride agent.

Table 2 ☐ Comparison of similar studies.

Author	Sample (N)	F Preparation	PPM F-Layer 1
Aasenden et al, 1968	25 blocks, 8 teeth	APF	850-1100
Mellberg et al, 1973	unknown	NaF or NHSiF	563-6235
Wefel et al, 1979	20	APF gels	3964-6157
Retief et al, 1980	20	APF, Duraphat or Fluor Protector	711-3057
Dijkman et al, 1988	40	APF	1637
Wei et al, 1988	40	APF gel & foam	5579-6517
Wei et al, 1988	24	APF foam	1567-5920
Wei et al, 1988	40	APF gels	4246-4824
Whitford et al, 1995	46	APF gel & foam	6520-6834
This Study, 1996	60	APF gels & foam NaF liquid	2021-2073 587

MORE UPTAKE BY NONCONTACT SURFACES

DISCUSSION

This study has demonstrated use of a practical in vitro model to study fluoride uptake by enamel on interproximal surfaces of teeth. It has demonstrated significant uptake of fluoride from a four-minute application of commercially available acidulated phosphofluoride. Future studies should be performed with this model to investigate the effect of shortening the time of application of the topical fluoride. In addition, the effect of topical fluoride on artificial caries in the interproximal areas should be studied.

CONCLUSION

It is concluded that fluoride is deposited into the proximal surfaces of molar teeth in contact when these teeth are exposed to a four-minute topical application of acidulated phosphofluoride.

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CARIES PREVALENCE AND SALT FLUORIDATION

The comparison of the mean caries indices in 9-year-old children of Strasbourg between the period 1974–1985 and the period 1985–1991 showed that during the latter time interval, which corresponded to the introduction of salt fluoridation, the caries reduction was significantly more important. The mean DMFT and DMFS indices were respectively reduced by a factor of 3 and 2.5 when compared with the first time interval. The present study was therefore conducted in order to determine if this more important reduction could be related to the introduction of salt fluoridation.

The dft index was indeed significantly lower (reduction of 35.5 percent in the FS users. Such an observation, which was confirmed by the use of the Generalized Linear Model ($P=0.03$), was important since at the age of nine about 40 percent of the deciduous teeth were still present. However, the DMFT and DMFS although lower among the children who consumed FS, did not show any significant difference with those of the non-consumers. This absence of significance could be related 1) to the fact that the period of salt fluoridation was too short, 2) to the relatively small number of FS consumers examined in this study and/or 3) to the fact that at the age of nine, only 60 percent of the permanent teeth have erupted, some having been only exposed very briefly to the risk of caries.

Fabien V. *et al*: Caries prevalence and salt fluoridation among 9-year-old schoolchildren in Strasbourg, France. Community Dent Oral Epidemiol, 24:408–411, December 1996.

Enamel and dentin bond strength and bonding mechanism to dentin of Gluma CPS to primary teeth

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In contrast to the large number of research reports on the efficacy of resin adhesives to dentin of permanent teeth, very few have addressed resin bonding to primary dentin.[1-9]

Bonding to dentin has been difficult due to several factors, among them the use of hydrophobic materials; the hydrophilic nature of dentin (containing approximately 20 percent water by weight); the achievement of pulpal biocompatibility; the development of a sufficiently high bond strength to overcome the forces of shrinkage by polymerization generated by light-cured resin-based materials; and until recently, a poor understanding of the presence and nature of the smear layer.[10]

One method to improve resin composite adhesion to dentin is through resin infiltration of the dentin (hybridization).[11] The primer resins diffuse into the outer few micrometers of the dentin, making it more porous by acidic conditioning.

The newer bonding agents are hydrophilic and capable of forming a hybridization zone between resin and dentin. The bond strength of some of these hydrophilic primers and bonding agents or the formation of the hybrid zone in primary teeth, has not been reported.

This study evaluated the shear bond strength to enamel and dentin, and the mechanism for bonding to dentin of Gluma CPS bonding system when applied to primary tooth enamel and dentin.

MATERIALS AND METHODS

The materials used were Gluma CPS bonding system (Bayer AG, Leverkusen, Germany; batch 036470) combined with the hybrid-type composite resin Pekafill (Bayer AG, Leverkusen, Germany, batch 025113). The conditioning liquid of the Gluma CPS system consists of 20 percent phosphoric acid gel and 5 percent $SiCO_2$. The Gluma Primer is an aqueous solution of Glutaral and HEMA, and the Gluma Sealer consists of polyfunctional methacrylic esters.

Shear bond strength to enamel and dentin

For shear bond testing, twenty primary molars were used. They were stored in 1 percent chloramine solution for a maximum of three months after extraction. They were embedded in a slow setting epoxy resin in cylindrical rubber molds. Buccal surfaces were ground flat to expose peripheral dentin (ten teeth) or enamel (ten teeth) by grinding on wet SiC paper, the final grit was 600.

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The conditioning gel was applied for thirty seconds, rinsed and gently dabbed with a paper tissue (wet technique). Gluma Primer was applied for thirty seconds and thoroughly dried with compressed air. Gluma Sealer was applied and light-cured for twenty seconds (Translux CL, Kulzer, Germany). A cylindrical split Teflon mold (diameter: 3.5 mm, height: 1 mm) was filled with Pekafill hybrid composite in one increment, covered with a transparent matrix strip and light-cured for sixty seconds. Five enamel and five dentin specimens were immersed in deionized water at 37°C for twenty-four hours. The other ten specimens were subjected to a thermocycling procedure (2000 cycles between 5° and 55°C, dwell time 15 sec.). Maximal storage time of the specimens in water before start of thermocycling was two hours, minimal storage fifteen minutes. After water storage or thermocycling, the bonded cylinders were loaded until failure using a universal testing machine (Zwick, Type 1474, Eiseingen, Germany) in the shear mode at a crosshead speed of 1 mm/minute. Shear bond strength was calculated as the quotient from fracture load and the bonded area. The data were analyzed with a two-way ANOVA and Student-Newman-Keuls test. Failure types resulting from debonding of the resin cylinders from enamel and dentin were determined with a stereomicroscope at ×60.

Marginal adaptation in dentin cavities

The buccal surfaces of ten primary molars were ground flat on wet SiC papers (final grit 600) to expose peripheral dentin in which cylindrical buttjoint cavities (diameter: 3 mm, depth: 1-1.5 mm) were prepared with a cylindrical diamond bur. The cavities were treated with Gluma CPS bonding system and filled with Pekafill as described above. Excess of the restorative resin was removed by gentle grinding on wet SiC paper (grit 600) until the entire cavity margin was exposed for microscopic inspection. Five specimens were stored for fifteen minutes after light-curing and the other five for twenty-four hours in deionized water at 37°C before removal of the filling excess. The cavity margins were inspected at ×500. When a gap was identified, its maximum width (MGW) was measured with an ocular screw micrometer.

In order to improve the microscopic identification of the resin-impregnated hybrid layer, the fillings and their peripheral dentin surfaces were etched with a 20 percent phosphoric acid solution for five to ten seconds, washed and air-dried. The hybrid layer thicknesses (HLT) were then measured with the ocular screw micrometer at four locations 90° apart coronally, approximally, and apically on the five fifteen-minute specimens. The microscopic inspection of each specimen was performed within three minutes to avoid dehydration artifacts. Wilcoxon Ram Sum Test was used to evaluate the results.

Scanning electron microscopic examination (SEM) of enamel and dentin surfaces

The effect of surface conditioning with the Gluma CPS gel was also investigated on enamel, either pumiced or ground on 600 grit SiC paper; and on dentin prepared with SiC paper (600 grit). Following thirty seconds of conditioning time, the acid gel was rinsed off for fifteen seconds and the surfaces were dried with compressed air.

Buccal enamel and dentin surfaces of three additional teeth were prepared and conditioned as above. In order to assess the wettability and surface detail reproduction of the etched sites, Gluma Primer and Sealer were applied according to the manufacturer's instructions and coated with an approximately 2 mm thick layer of Pekafill, which finally was light-cured for sixty seconds. The specimens were then immersed in 6 M hydrochloric acid to dissolve the teeth. The remaining resin "impressions" were washed with 10 percent sodium hypochlorite to remove adhering organic tooth tissue, and rinsed thoroughly in deionized water before mounting for Au-sputtering and SEM inspection.

RESULTS

Shear bond strength

The results are displayed in Table 1. Statistical analysis by two-way ANOVA showed significant differences for the first main factor, enamel/dentin ($p = 0.001$) and no significant differences for the second main factor, with and without thermocycling ($p = 0.576$). When inspected with the stereomicroscope, all fractured surfaces were characterized as cohesive failure patterns in resin located close to the coupling interface.

The SEM illustrations (Figures 1 and 2) show representative fracture patterns at the enamel and dentin sides, respectively. The surfaces had been washed in 10 percent citric acid and 10 percent sodium hypochlorite before Au-sputtering in order to differentiate between adhesive failure and cohesive failure in resin close to the interface. In Figure 1, no enamel etching patterns are shown, which means that the failure site is in the resin. Figure 2 shows resin tags broken at their bases. The

Table 1 ☐ Shear bond strength (MPa) to enamel and dentin after 24 hours' water storage or 2000 thermocycles (n = 5).

	24 h/H_2O	2000 thermocycles
Enamel	18.7 ± 1.8[a]	18.8 ± 1.5[a]
Dentin	14.9 ± 2.6[b]	13.7 ± 1.2[b]

Figures with the same letter are not statistically significantly different ($P > 0.05$)

Table 2 ☐ Maximal gap width (μm).

Group/Specimen	1	2	3	4	5
15 minutes	0	3	0	0	0
24 hours	0	1	0	0	0

No statistically significant difference between the groups (Wilcoxon Ram Sum Test, $p = 0.882$).

intertubular substrate is considered resin or resin-impregnated collagen, since the acid-hypochlorite treatment had not generated the typical appearance of etched and deproteinized dentin.

Marginal adaptation in dentin cavities

Table 2 summarizes the maximum widths for marginal gaps found when inspected after fifteen minutes and twenty-four hours. In each group, four of the five cavity margins were gap free. Nonparametric statistical analysis showed that the marginal performance of the two groups was not significantly different (Wilcoxon Ram Sum Test, $p= 0.882$).

The thicknesses of the hybrid layer were not significantly different, when tested statistically by location around the cavity margins. Each specimen was characterized, therefore, by a mean HLT and a standard deviation (n=4). The average HLT from the five specimen means was 11.7 μm and the average standard deviation was 1.1 μm.

Figure 3 depicts an SEM view of the coronal dentin cavity margin. There is a clear differentiation between the dentin, the hybrid layer, the adhesive resin layer, and the composite resin. The hybrid layer is approximately 10 μm wide.

Enamel and dentin surface evaluation

The etch patterns produced on the abraded enamel surface (Figure 4) were uniform and deeper as compared to the sample that was pumiced (Figure 5). This was also confirmed in the resin replicas (Figures 6 and 7).

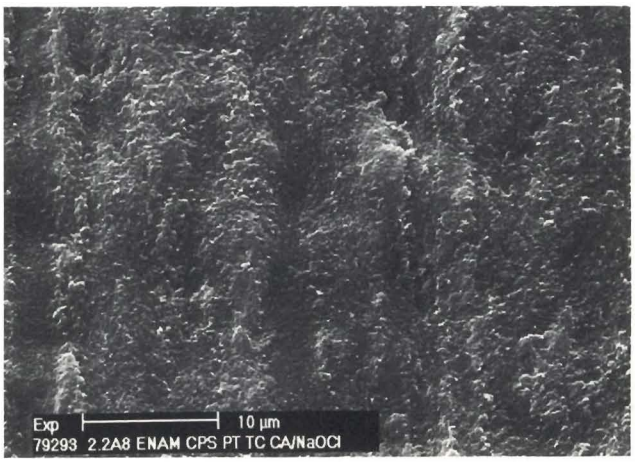

Figure 1. Resin cohesive failure close to the etched enamel surface (Photo #79293).

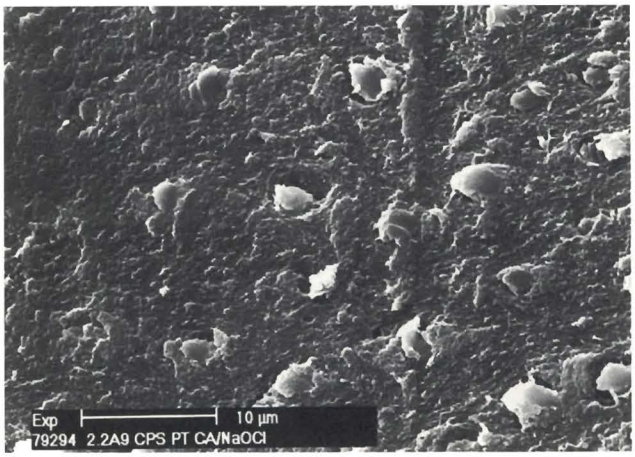

Figure 2. Resin cohesive failure close to the dentin surface with resin penetration into the dentin tubules (arrows) (Photo #79294).

The dentin specimen treated with the CPS conditioner showed SiO_2 precipitation on the surface, in spite of the thorough water spraying (Figure 8). The resin replica of the Gluma CPS treated dentin (Figure 9) shows numerous long resin tags. These strings are characterized by a conically-shaped irregular impression of the tubule entrances, which is considered the part with peritubular hybrid layer formation, and long smooth extensions into the deeper part of the tubules without peritubular sealing.

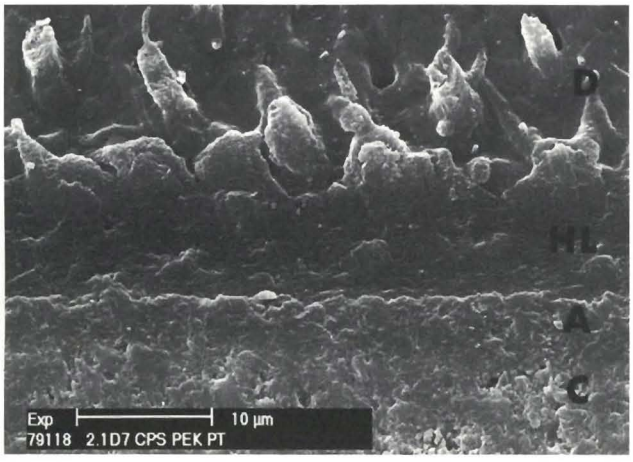

Figure 3. A hybrid layer of ± 10 µm thick was readily seen in all specimens. D=dentin; HL=hybrid layer; A=adhesive resin; C=composite resin (Photo #79118).

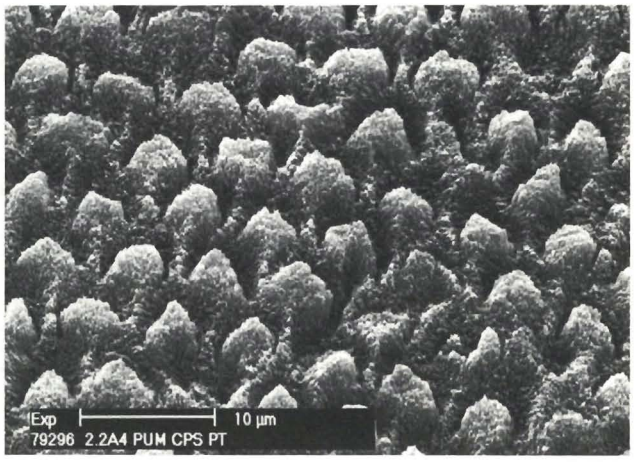

Figure 5. Etch patterns of a primary tooth enamel cleaned with pumice before treated with Gluma CPS Conditioner for thirty seconds. Note the shallow etching patterns (Photo #79296).

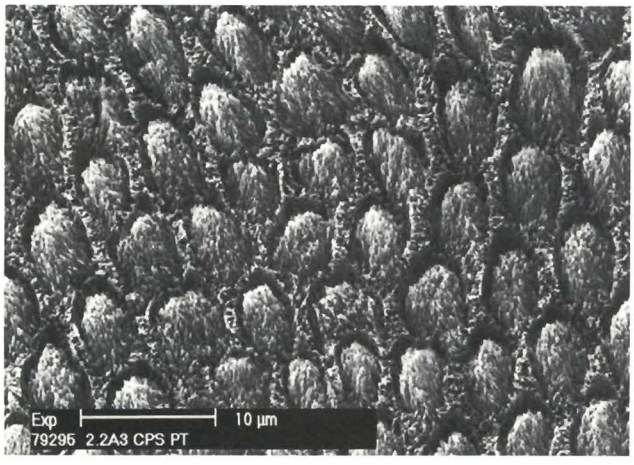

Figure 4. Etch patterns of a primary tooth enamel ground with SiC paper and treated with Gluma CPS Conditioner for thirty seconds. Note the uniform etching patterns (Photo #79295).

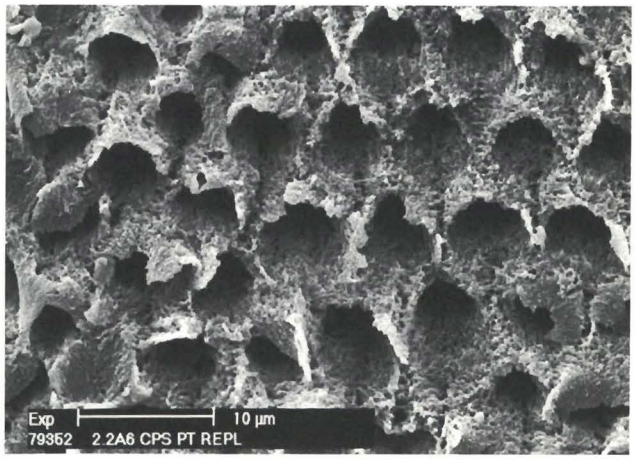

Figure 6. Resin replica of the enamel surface ground with SiC paper and treated with Gluma CPS Conditioner for thirty seconds. Note the uniform and deep etching patterns (Photo #79352).

DISCUSSION

The results of the present study are difficult to compare to previous ones conducted on primary dentin, not only because of different methodologies, but also because of the different bonding agents used. Elkins & McCourt, however, using All-Bond and Amalgambond, reported bond strengths of 11.60 and 12.62 MPa, respectively.[6] Our results on thermocycled samples showed values of 18.80 MPa in enamel and 13.70 MPa in dentin, which are similar to reports of previous studies. Hallett *et al*, using SMP in primary teeth enamel, reported 11.18 MPa.[12] The potential of these dentin bonding agents to equal or exceed the bond strength to enamel is evident in primary teeth. Another study, using a similar testing methodology as the present study, but employing Gluma 2000 adhesive system, reported a bond strength value of 8.2 MPa in primary dentin.[8]

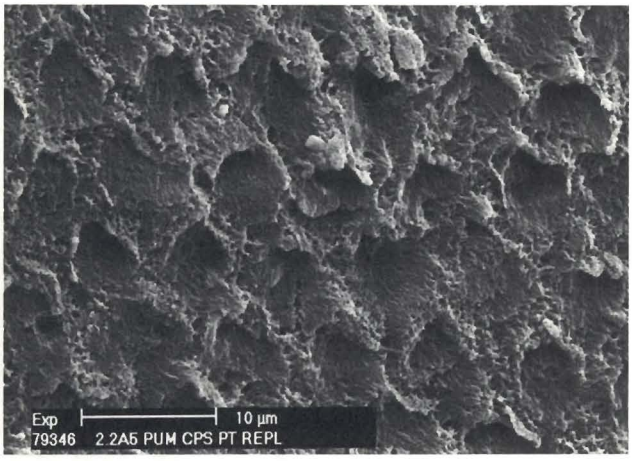

Figure 7. Resin replica of the enamel surface cleaned with pumice before treated with Gluma CPS Conditioner for thirty seconds. Note the shallow etching patterns (Photo #79346).

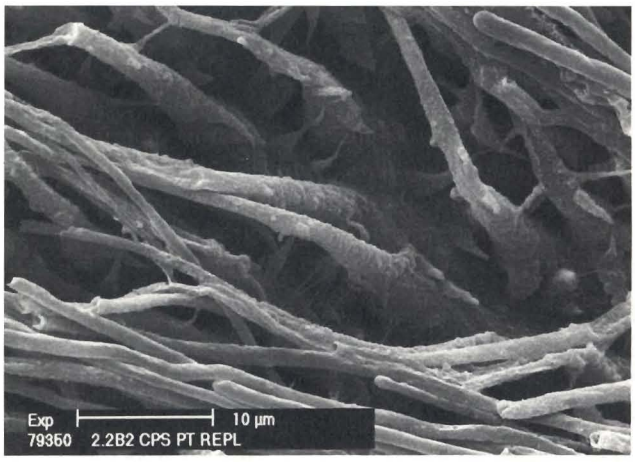

Figure 9. Resin replica of Gluma CPS treated dentin. The conical tag bases show peritubular hybrid layer formation. The smooth parts of the tags are impressions from deeper parts of the tubules where no resin impregnation of dentin is seen (Photo #79350).

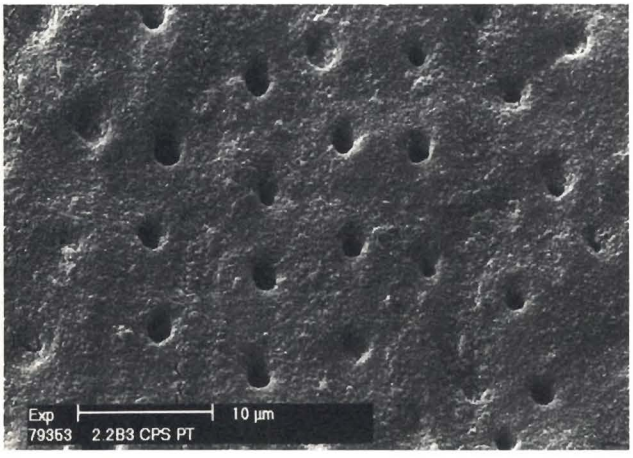

Figure 8. Dentin surface after treated with Gluma CPS Conditioner for thirty seconds. Notice SiO_2 precipitate on the surface (Photo #79353).

In this study, the bond strength value in primary dentin was not related to the failure mode recorded visually or with the SEM. For example, some specimens recorded values of 10.3 MPa and resin cohesive failures, while others had values of 16.9 MPa, also with resin cohesive failures. Dentin cohesive failures were not recorded in this study. Another study reported that in primary teeth, dentin cohesive values were only recorded with values above 14.10 MPa.[10]

In the present study, using Gluma CPS, all failures were recorded as resin cohesive failures; a higher bond strength would have been necessary, therefore, to produce a dentin cohesive failure. Malferrari *et al*, using Gluma 2000 in primary dentin, noted a similar trend: although the bond strength values were relatively low, evaluation with the SEM revealed that the true failure occurred in the resin, very near to the dentin surface.[8] Hallett *et al*, testing bond strength of SMP in primary enamel, reported that most failures occurred at the adhesive-resin interface or were resin cohesive. The few cases they recorded as failures at the enamel-adhesive interface displayed some resin coating the enamel surfaces.[12] García-Godoy and Finger, using Gluma 2000 in permanent teeth, also reported that in most cases, the bond failure was cohesive in resin, close to the dentin.[13] Due to this result they questioned the dye penetration technique for testing microleakage when using bonding agents capable of producing a hybrid layer.

Most recently, *in vitro* shear bond strengths to dentin have been tested, indicating that the current generation of dentin adhesive systems approached or exceeded the theoretical threshold value to resist contraction stresses during polymerization of resin materials, with SMP, All-Bond 2, and Optibond giving values of 23.1, 21.4, and 19.7 MPa, respectively.[14] Others have reported shear bond strengths to dentin of 22.2, and 20.2 MPa using Optibond.[15,16] The present study in primary tooth dentin would support the use of Gluma CPS in these teeth.

Because all samples in this study displayed resin cohesive failure, reporting the bond strength value *per se* is not as significant as describing the failure site. Future studies should strongly place emphasis on the failure site, while debonding the resin, and report this information with the bond strength value obtained.

When comparing the results of bond strengths obtained in primary and permanent tooth dentin, dentin thickness difference must be taken into consideration. Also, the bond strength of some dentin adhesives decreases as the occlusal dentin approaches the pulp, which was interpreted as the bond being dependent on the calcium level and on the total area of solid dentin available, both of which decrease toward the pulp.[4,17-19] Hirayama *et al* found no difference, however, in calcium or phosphorus content of the peritubular and intertubular dentin of primary and permanent teeth; but reported that the peritubular dentin, which is more mineralized but less crystalline than intertubular dentin was two to five times thicker compared to permanent dentin.[28] As Bordin-Aykroyd et al suggest, these differences could affect any chemical bonding of the adhesive or result in different effects of the pretreatment regimens on the dentin, which would also affect bonding.[4] On the other hand, because of the thinner primary dentin, more dentin cohesive failures could be recorded in primary teeth, even with lower bond strength values.

Another observation in this study was that in all specimens, a hybrid layer was readily seen. A similar finding has been reported, using other dentin adhesives in dentin of primary teeth.[9] A recent study has shown that when dentin is acid-etched, the subsequent moisture status of the collagen-rich outer zone is critical to achieving optimal shear bond strength.[21] As a result of drying, the organic rich collagen phase at the surface of conditioned dentin is morphologically altered.[22] These morphological changes impair the penetration of the primer, resulting in a reduction in bond strength.[23] In a previous study on primary dentin and the one by Barkmeier *et al*, on permanent dentin, the bond strength to dentin was not statistically significantly altered whether the dentin was wet or dry.[9,24] Perhaps the time needed to rehydrate the dentin should be increased before the primer is applied. This is possible because in one study, although there was no significant difference in bond strength, there was a trend to obtain higher values with the moist dentin.[9] Because the collagen-rich zone offers no direct quantitative contribution to bond strength, dentin rehydration (moist dentin) would then be mainly important to achieve a maximum porous dentin surface so the resin components can diffuse through the outermost demineralized collagen-rich zone into the partially demineralized dentin below, where it must polymerize.[22]

CONCLUSIONS

This study revealed that thermocycling did not affect the shear bond strength to enamel or dentin. There was a statistically significant difference ($p<0.05$) between the shear bond strength values obtained in enamel and dentin.

The maximal gap width was not significantly different between specimens that were stored in water for fifteen minutes or twenty-four hours. The average hybrid layer thickness was 11.7 ± 1.1 μm. All specimens revealed a resin cohesive failure very close to enamel or dentin surface.

The etch patterns produced on the abraded enamel surface were uniform and deeper as compared to the sample that was pumiced. This was also confirmed in the epoxy replicas.

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AN OUNCE OF PREVENTION

How are rituals, routines, and daily schedules going to solve any problems in day-to-day living? There are many reasons for using them and many ways in which they help. Think about how your own day goes. Your major activity is not punishing your children; you do not stand around waiting for someone to misbehave so you can take away a bicycle or imprison your child in his bedroom. Your major energies are probably devoted to running the family. This includes not only getting through the routines of dressing, washing, and eating breakfast, but also getting to work on time, working, planning meals, shopping, cooking, cleaning the house, cutting the grass, doing the laundry—the list is endless. Families are involved in the multitudinous activities of daily living. Punishing your children is only a very small part of life—even though on bad days it may not feel that way.

But *discipline* can be a part of everything the family does. Remember that the word means "teaching," and that what you are teaching your children is that they are competent to regulate themselves and to master social skills, control, and prediction. What better medium do you have for teaching them than the naturally occurring events of everyday living?

Williamson, Peter: *Good kids, bad behavior*; New York: Simon and Schuster, 1990, page 121.

Evolving "faces" of the next generations of pediatric patients

H. Barry Waldman, BA, DDS, MPH, PhD

In an extensive 1996 report, the U.S. Bureau of the Census projects that between 1990 and 2000 there will be an increase of 6.6 million children in the United States (a 10.3 percent increase).* In subsequent decades, there will be continuing increases in the number of children, (reaching 96 million by the middle of the next century—an increase of 50 percent since 1990). As a result of greater increases in the general population, however, there will be a slight decrease in the proportionate representation of children in the general resident population of the country (Table 1).

Even more significant than the overall general increases in the number of children, there will be dramatic changes in the "faces" of the youngsters in our nation and pediatric dental practices. By the middle of the next century, the overall numbers of minority group children (African-American, Native American, Asian, and Pacific Islander, and those of Hispanic origin**) will more than double, (for some populations, the numbers will more than quadruple). during the same period, the numbers of non-Hispanic white youngsters actually will decrease (Table 2).

*All data for this presentation were drawn from this Bureau of the Census report. In this review, unless otherwise specified, the reference to children will include all youngsters less than eighteen years of age.

**Persons of Hispanic origin may be of any race. The information in the Bureau of the Census report refers to data collected in the 50 States and the District of Columbia. It does not include data for Puerto Rico.[1]

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A previous presentation in the *Journal of Dentistry for Children* (based on an earlier report by the Bureau of the Census) provided a review of the future overall increases in the number of youngsters and the manpower needed to maintain pediatric dentist-to-youngster population ratios.[2] The following presentation will use updated projections with differentiations by race and Hispanic origin to consider the population changes that pediatric dentists and other health providers will experience as they provide care for the next generations of youngsters.

SOURCE OF INFORMATION

The Bureau of the Census report includes population projections by age, sex, race, and Hispanic origin. These numbers are based on estimates of the July 1, 1994 resident population. The estimates are consistent with the 1990 census as enumerated and are projected using age cohorts with alternative assumptions for future fertility, life expectancy, and net immigration levels. The Bureau provides three sets of population projections ("high range", "middle range", [or preferred range] and "low range"). The high and low ranges assume extremes in birth and death rates and immigration (legal and illegal) rates. The middle range (used in this presentation) assumes more moderate developments).

GENERAL POPULATION PROJECTIONS

The general resident population of the United States is projected to reach 275 million by 2000 (a growth of 12

Table 1 ☐ Number of children (in millions) and percent of total population by age: 1990 to 2050.[1]

	Number					Percent of total population			
	Age			Total		Age			
Year	Under 5 years	5-13 years	14-17 years	No.	Percent change since 1990	Under 5 years	5-13 years	14-17 years	Total
1990*	18.8	32.0	13.3	64.2		7.6%	12.8%	5.3%	25.7%
Projections									
1995	19.6	34.4	14.8	68.7	7.0%	7.5	13.1	5.6	26.2
2000	18.9	36.0	15.8	70.8	10.3%	6.9	13.1	5.7	25.7
2010	20.0	35.6	16.9	72.5	12.9%	6.7	12.0	5.7	24.4
2020	21.9	38.7	16.9	77.6	20.9%	6.8	12.0	5.3	24.1
2030	23.1	41.6	18.8	83.4	29.9%	6.6	12.0	5.4	24.0
2040	24.9	44.0	19.8	88.8	38.3%	6.8	11.9	5.4	24.1
2050	27.1	47.8	21.2	96.1	49.7%	6.9	12.1	5.4	24.4

* Estimate

Table 2 ☐ Number of children (in millions) by age, race and Hispanic origin: 1990 to 2050.[1]

	Age			Total		Age			Total	
Year	Under 5 years	5-13 years	14-17 years	No.	Percent change since 1990	Under 5 years	5-13 years	14-17 years	No.	Percent change since 1990
	White					African-American				
1990*	15.0	25.7	10.6	51.3		2.9	4.9	2.1	9.9	
Projections										
1995	15.4	27.3	11.7	54.5		3.1	5.3	2.3	10.7	
2000	14.7	28.3	12.4	55.4	8.0%	3.1	5.7	2.4	11.3	14.1%
2010	15.1	27.1	12.9	55.2		3.5	5.9	2.7	12.2	
2020	16.4	28.8	12.6	57.9	12.9%	3.8	6.7	2.9	13.4	35.4%
2030	16.9	30.5	13.8	61.1		4.1	7.3	3.3	14.7	
2040	18.0	31.6	14.2	63.9	24.6%	4.6	7.9	3.5	16.1	62.6%
2050	19.3	33.9	14.9	68.2		5.0	8.7	3.8	17.6	
	Native American					Asian & Pacific Islander				
1990*	0.2	0.4	0.2	0.7		0.6	1.1	0.5	2.2	
Projections										
1995	0.2	0.4	0.2	0.8		0.8	1.3	0.6	2.8	
2000	0.2	0.4	0.2	0.8	14.3%	0.9	1.6	0.7	3.3	50.0%
2010	0.2	0.4	0.2	0.9		1.2	2.1	1.0	4.3	
2020	0.3	0.5	0.2	0.9	28.6%	1.5	2.7	1.3	5.4	145.5%
2030	0.3	0.5	0.3	1.1		1.8	3.3	1.5	6.5	
2040	0.3	0.6	0.3	1.2	71.4%	2.1	3.8	1.8	7.7	250.0%
2050	0.4	0.6	0.3	1.3		2.4	4.4	2.1	8.9	
	Hispanic origin**					White, nonHispanic origin				
1990*	2.5	3.8	1.6	7.9		12.8	22.2	9.2	44.2	
Projections										
1995	3.1	4.6	1.9	9.6		12.6	23.1	10.0	45.7	
2000	3.2	5.7	2.2	11.0	39.2%	11.8	23.1	10.4	45.4	2.7%
2010	4.1	6.7	3.0	13.7		11.4	21.1	10.2	42.7	
2020	5.2	8.5	3.5	17.2	117.7%	11.7	21.2	9.5	42.3	−4.3%
2030	6.2	10.4	4.4	20.9		11.2	21.2	9.8	42.3	
2040	7.5	12.3	5.2	25.0	216.5%	11.3	20.5	9.6	41.4	−6.3%
2050	8.8	14.8	6.2	29.7		11.4	20.7	9.4	41.5	

* Estimate
** May be of any race
Note: Totals may differ due to rounding

million or 4.5 percent since 1995). The population may top 300 million shortly after 2010 and by the middle of the next century, the population may increase to 394 million (a 50 percent increase from the 1995 population).

By age

In 1995, there were 19.6 million youngsters less than five years of age (about as numerous as there have ever been in the last thirty years). Their numbers are projected to decline to fewer than 19 million around 2000, followed by increases throughout the first half of the next century.

There were 49 million five to seventeen-year-olds. This group may increase by about 3 million youngsters by 2000, then grow by another 17 million by 2050. This age-group's share of the total population, however, may never be any larger than it is in the mid 1990s.

- ☐ The post-World War II Baby Boomers will begin to turn fifty in 1996. During the next ten years, this group will represent more than half of the nation's total population increase.
- ☐ The percent of the population age sixty-five plus will remain near its current level during the next ten years. This slow growth is due almost entirely to a lack of growth in the sixty-five to seventy-four age-group. The number of people age sixty-five and over is projected to increase from 39 million in 2010 to 69 million in 2030 (when the surviving Baby Boomers will become sixty-five plus years).
- ☐ The most rapidly growing broad age-group will be the eight-five plus population, doubling its current size by 2025.

By race and Hispanic origin

Almost three-quarters of the population was non-Hispanic whites in 1995. This group will contribute only one quarter of the total population growth during the next ten years. During the following decades, this population will be declining in size.

- ☐ The non-Hispanic white share of the population will decline steadily from 74 percent in 1995, to 64 percent in 2020 and 53 percent in 2050.
- ☐ By the middle of the next century, the African-American population will nearly double its 1995 size. After 2016, more African-Americans than non-Hispanic whites will be added to the population each year.
- ☐ The race/ethnic groups with the highest rates of increase will be the Hispanic origin and the Asian and Pacific Islander populations.
- ☐ By 2010, the Hispanic origin population may become the second largest race/ethnic group.
- ☐ By the year 2030, the non-Hispanic white population will represent less than half of the population under eighteen years of age.
- ☐ In each of the years under review, it was projected that four of every ten people added to the population through net immigration would be Hispanic, three of every ten would be Asian and Pacific Islander, two in ten would be non-Hispanic white and one in ten would be African-American.

CHILD POPULATION PROJECTIONS

The number of births in the country is projected to decrease slightly as the century ends, then increase progressively throughout the first half of the 21st century. The number of births could reach over four million annually by 2005, as a result of the increasing number of women in the younger childbearing years. Many of these births will be the grandchildren of Baby Boomers. By 2012, the annual number of birth could exceed the highest annual number ever achieved during the 20th century.

Births by race and Hispanic origin

Birth trends are projected to differ substantially by race and Hispanic origin.

- ☐ White births will decrease during the rest of the 1990s and slowly start to increase in the early decades of the next century.
- ☐ African-American births are projected to increase steadily during the projected period, increasing by 60 percent by the middle of the next century.
- ☐ The Asian-American and Hispanic origin populations will experience the most dramatic increases in the number of births. For each group, the number of births will more than triple by the middle of the next century.
- ☐ In 1995, nearly four in six (about 66 percent) of all births were non-Hispanic whites, about one in six were African-Americans and one in six were of Hispanic origin. By the middle of the next century, two of every five births (about 40 percent) will be non-Hispanic white, one in three will be Hispanic, one in five will be African-American, and one in ten will be Asian and Pacific Islander.

Number of children by race and Hispanic origin

During the next decades, the numbers of minority children in our population will increase, while the numbers of non-Hispanic white children actually will decrease.

- ☐ Between 1990 and 2010, the number of minority children will increase by more than 10 million youngsters (including 5.9 million children of Hispanic origin). During the same period, the number of non-Hispanic white children will decrease by 1.5 million youngsters.
- ☐ By mid 21st century, the number of Hispanic origin children will increase by over 20 million youngsters. There will be an increase of 1) almost 8 million African-American children, 2) over 6 million Asian and Pacific Islander children, and 3) 0.6 million Native American children.

☐ By mid 21st century, the number of non-Hispanic white children will decrease by almost 3 million children (Table 2).

IMPACT ON PEDIATRIC DENTAL PRACTICE

The issues of language, culture and social environment, economics, the need for and use of health services, the availability of general health and dental insurance and any number of other variations among immigrant, minority and nonminority children have been reviewed in a series of previous presentations in the *Journal of Dentistry for Children*.[3-6]

Under current federal legislative arrangements, there will be an annual flow of hundreds of thousands of legal immigrants to this country during the next decades (in addition to an influx of hundreds of thousands of illegal immigrants). Nevertheless, most of the youngsters (including minority group children) who will receive dental services in your practice increasingly will be descendants of families that have been residents of this country for generations.

While many children will be raised in families that retain aspects of their particular traditions, the generations of youngsters will be affected by the "great homogenizers" of our society—the television, the movies, the playground and maybe even the school. As a result, the "faces" of pediatric patients will continue to evolve and practitioners and their staffs will need to reevaluate preconceived notions and conceptions regarding minority group children and the families in which they are raised. It is almost unnecessary to comment that the variability of minority group children will mirror the seeming infinite variability of nonminority children!

P.S. Surely legislators increasingly will be drawn to the needs of minority populations as these groups grow in number and exert their power in the voting booth.

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LOCALIZATION OF INFECTION

It is fascinating to observe, so many years since the suggestion of a link between chronic focal infection and chronic arthritis (Miller, 1890, 1891; Hunter, 1900, 1921; Billings, 1912, 1913), the advances in supporting evidence over the years (Davidson *et al.*, 1949; Ebringer *et al.*, 1989; Dixon, 1990). And yet earlier workers had considered that different organisms selectively colonized specific loci—the theory of elective localization (Rosenow, 1919, 1921, 1923). By this hypothesis, bacteria would localize from the source focus to the distant, systemic focus, and Rosenow demonstrated a targeting process in a series of experiments, reviewed by Hughes (1994). This should not surprise us. Micro-organisms cause infections of tissues, organs, or systems, clearly depending on an ability to survive and grow better in those loci—a clear extension of the principles of microbial ecology in general.

The mechanisms of spread are direct, through the local tissues and along fascial planes, along mucous surfaces, including ducts such as the salivary, and by inhalation and ingestion along respiratory and gastrointestinal mucosal surfaces, respectively, *via* the bloodstream, the lymphatics, and even, possibly, along nerves (Newman, 1968). Debelian *et al.* (1994) identify three pathways: metastatic infection from the oral cavity due to transient bacteremia, metastatic injury due to oral microbial toxins, and metastatic inflammation due to immunologic injury caused by oral micro-organisms.

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Inherited primary failure of eruption in the primary dentition: Report of five cases

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Angeliki Kotsaki, DDS

Most reports of primary failure of eruption concern the permanent dentition. The main causal factors are local, (lack of space, ectopic positions of teeth, supernumerary teeth, cysts etc). Systemic and genetic disorders, however, may have primary failure of eruption and retarded eruption as additional symptoms (cleidocranial dysplasia, osteopetrosis etc). Also inherited retarded eruption, where the delayed eruption is the only detectable manifestation of the inheritance, has been reported.[1]

Impaction of primary teeth, caused by primary failure of eruption, is claimed to be rare.[2-9] So far no epidemiological studies have been performed to evaluate its prevalence. Bianchi and Roccuzzo, however, found three cases by screening of 30,000 panoramic radiographs, indicating a prevalence of 1:10,000.[2] The few papers published concerning primary failure of eruption describe a single or only a few cases, and most of the impacted teeth are second molars.[2-9] Thus, Amir in a review includes only six cases reported after 1976 and adds one case, Bianchi and Roccuzzo adds three more cases, and Jarvinen in 1994 adds two cases, totalling twelve cases published during the last twenty years.[2,7,9] It is to be believed, however, that the condition is under-reported for several reasons: The impacted teeth are deeply seated, often below the reach of intraoral radiographs, and they are most often without symptoms.

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Primary failure of eruption must be distinguished from the secondary impaction often observed as a consequence of ankylosis. In such cases the teeth have been in occlusion, but do not follow the adaptive secondary eruption needed to keep pace with the vertical growth of the alveolar process. This phenomenon is given many names: secondary impaction, secondary infraposition, submergence, reinclusion etc, and has a high prevalence, reaching a maximum of 14 percent at eight to nine years.[10] It is important to distinguish primary retention from secondary. Thus, Bianchi and Roccuzzo list the following criteria for diagnosing primary impacted teeth: Deeply positioned in the jaw; absence of caries or restorations; no resorption of the roots; frequent passing of the corresponding permanent tooth; possible retention and malposition of the adjacent permanent tooth.

In the present paper five cases of primary failure of eruption in the primary dentition are reported. The total number of impacted teeth is fourteen, varying from one to four second primary molars in each case. The etiological background appears to be inheritance.

CASE REPORTS

The report comprises five cases from a collection of fourteen (nine males, five females) which in addition to inherited retarded eruption in the permanent dentition also showed primary failure of eruption of one or more primary second molars. From their first visits at ages between 5.5 to 11 years, the patients were observed at

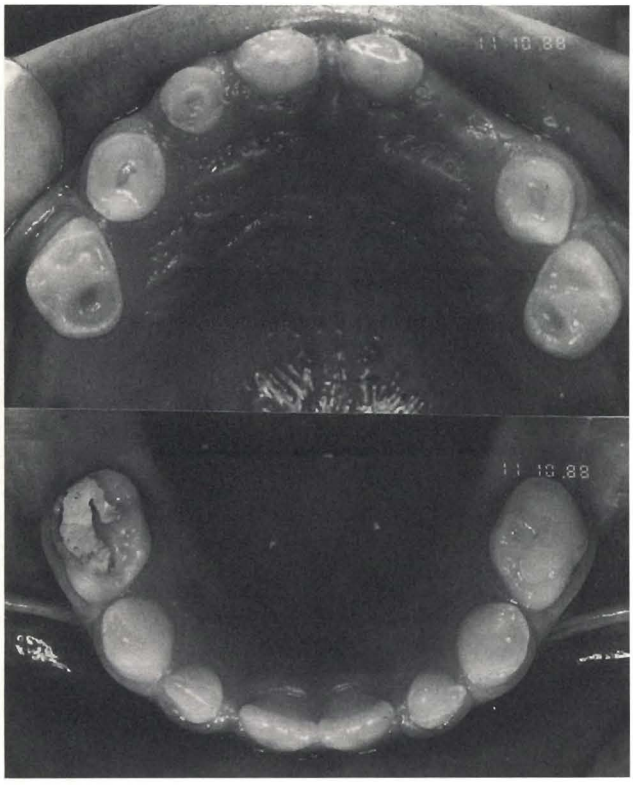

Figure 1. Case 1. Girl at age 8.5 years. Sixteen primary teeth erupted at normal age. All second primary molars are still unerupted.

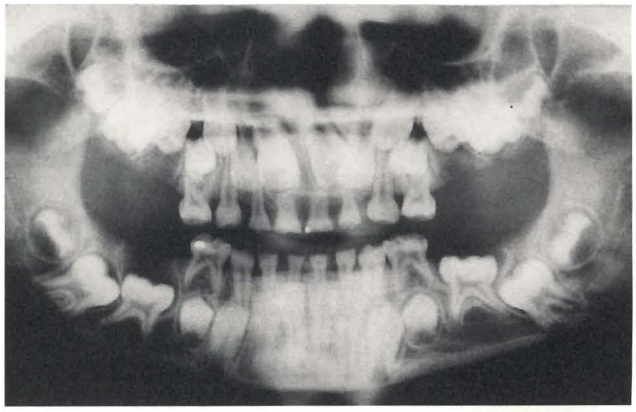

Figure 2. Orthopantomograph of same girl show all second primary molars deeply impacted in the jaw. The lower permanent first molars are locked below the distal curvature of the primary molars. Agenesis of all second premolars.

least every half year through several years. Orthopantomographs(OPG) were taken each year or every second year to disclose whether pathological conditions developed.

Case 1 (C.D.)

Female seen first at an age of 5.5 yrs. The patient had three siblings, two of them, a sister and a brother, were affected with retarded eruption in the permanent dentition, and one brother was unaffected. Father and grandfather were also affected. Clinical photographs (Figure 1) at 8.5 yrs show a dentition with only sixteen teeth, most of them with severe attrition. Orthopantomograph at the same age shows all second primary molars still deeply impacted in the jawbone and with completed root development (Figure 2). The teeth never erupted and had to be removed surgically at the age of 11.5 years because they were expected to prevent the eruption of the first permanent molars, which in cases with inherited retarded eruption have a mean eruption time of about twelve years of age. None of her second premolars developed.

Case 2 (T.A.)

Male seen first time at an age of 7.2 years. The patient had three siblings, one affected brother, one unaffected brother and one brother undiagnosed because of early age. His mother and grandfather were also affected. Orthopantomograph at 7.2 years shows all second molars deeply impacted (Figure 3). The teeth were surgically removed at 11.5 years. Lower second premolars did not develop.

Case 3 (I.G.)

Female seen first time at the age of 10.8 years. The patient had two unaffected siblings, but her mother was affected. Orthopantomograph at 10.8 years showed two maxillary and one mandibular second primary molar to be impacted (Figure 4). The teeth were removed at 11 years of age. In this case all second premolars developed, but it appears that the right mandibular second premolar was deformed because of crowding within the mandible during its development (Figure 5).

Case 4 (T.F.)

Male seen first time at an age of 11 years. None of his relatives was reported to be affected. Orthopantomo-

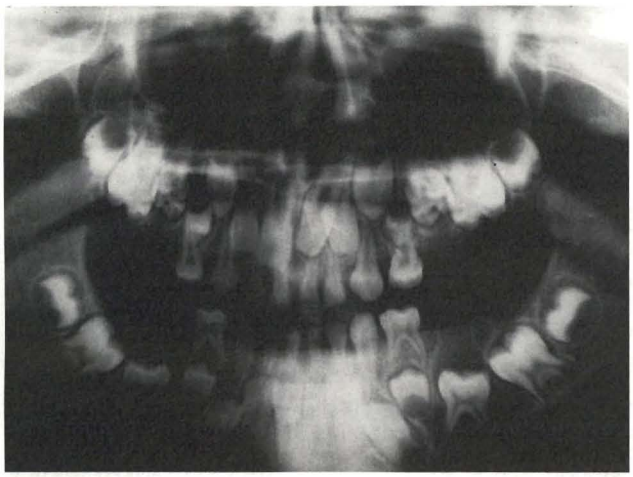

Figure 3. Case 2. Boy at age 7.2 years. All second primary molars unerupted. Agenesis of all second premolars. The distances between the occlusal surfaces of 75,85 and the alveolar margins are great. A bone-free open channel exists from tooth to surface. The apices of the lower second primary molars and first permanent molars are close to the mandibular margin.

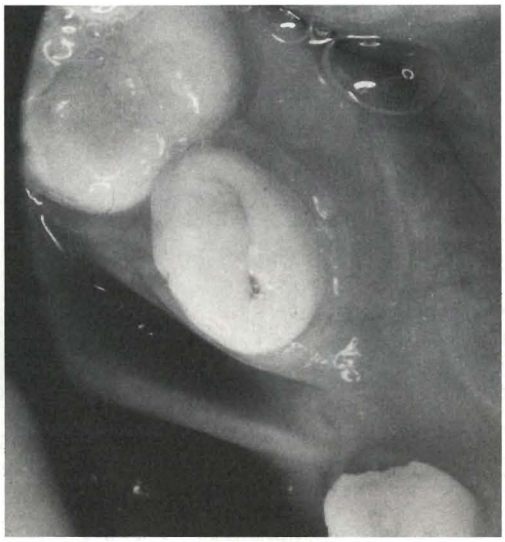

Figure 5. Same patient. Second premolar "flattened" by unerupted primary second molar during development.

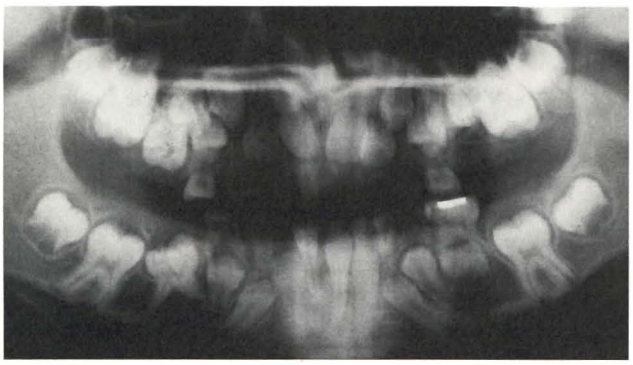

Figure 4. Case 3. Girl at age 10.8 years. Two maxillary and one mandibular second molar unerupted. All second premolars are developing.

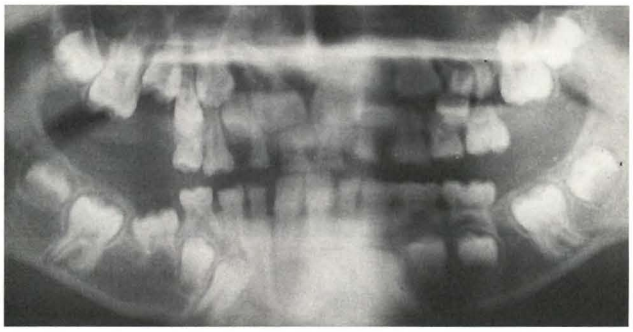

Figure 6. Case 4. Male at age 11 years. One maxillary and one mandibular second primary molar unerupted. Agenesia of the corresponding second premolar.

graph at 11 years shows one maxillary and one mandibular second primary molar impacted (Figure 6). The teeth where surgically removed at once. The corresponding premolars did not develop.

Case 5 (J.A.)

Brother of Case 2. First seen at the age of eight years. Orthopantomograph at 9.3 years shows one maxillary second primary molar impacted (Figure 7). The tooth was removed surgically at 10 years of age. All premolars developed.

In the present sample comprised of five cases with primary failure of eruption (total = 14 teeth), only the second molars were affected. Two of the patients had all four molars unerupted; the other patients had three, two and one unerupted tooth, respectively. The sex ratio was 3 males:2 females. Of the fourteen unerupted teeth seven belonged to males and seven to females. Eight of the unerupted teeth were maxillary, six were mandibular, eight were on the right side, and six on the left. Differences between sexes, jaws, and sides are not sig-

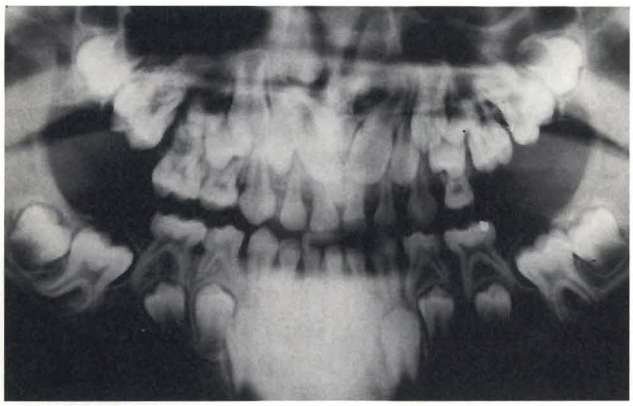

Figure 7. Case 5. Male at age 9.3 years. One maxillary second primary molar unerupted. All premolars are developing.

nificant). The teeth were always deeply seated, most often beyond the positions where they normally develop. In most cases their axial angulation was correct. There was no indication that the impacted teeth ever had been erupted and subsequently became impacted because of ankylosis. In nearly all cases the impacted teeth remained deeply buried, and increased their distance from the occlusal plane with increasing age. Only in one case evidence of a small occlusal movement was found, but the tooth never erupted. Often there remained an open "chimney" from the occlusal surfaces of the teeth to the margins of the alveolar processes, most visible in the mandible.

The eruption of the other primary teeth was reported by parents to have been uneventful, and some parents did not recognize that their child's primary dentition was incomplete. All of the patients with primary failure of primary teeth also suffered from severely retarded eruption of all teeth in the permanent dentition, constituting their greatest dental problem. Four of the presented cases (1,2,3,5) had siblings or parents/grandparents with retarded eruption in the permanent dentition, and two of the cases (2,5) were siblings.

None of the unerupted primary teeth ever erupted, contrary to the patients' permanent teeth, which finally erupted, however severely delayed (up to twenty years).

A conspicuous feature was the high prevalence of hypodontia, which afflicted three of the five cases with a total of eight missing teeth. All the missing teeth were second premolars, and always teeth succedaneous to unerupted primary molars. The hypodontia was most prevalent in the mandible, which occurred in five of the six sites with impacted primary molars. The figures for the maxilla were three missing premolars in eight possible sites.

DISCUSSION

Unerupted primary teeth are said to be a rarity and must be distinguished from the more prevalent secondary infraclusion caused by ankylosis.[9,10] The present report adds five new cases, for a total of fourteen unerupted second primary molars, to the sparse number of patients observed with primary failure of eruption in the primary dentition.[2-9] In none of our cases were the unerupted teeth ever observed to have been erupted, and they were never seen to erupt during the long period they were under observation (until an age of ten to eleven years). Thus, the condition may in all aspects be said to fulfil Bianchi's and Roccuzzo's criteria for being a primary failure of eruption.[2]

These five new cases are, however, in several aspects different from the earlier reported cases:

- ☐ While the earlier reported cases showed only one unerupted primary molar each, the present cases had from one to four unerupted teeth (mean 2.8 teeth).
- ☐ In the present cases no significant differences in sex distribution or in distribution between jaws and sides of jaw were found, contrary to the findings in earlier reported cases where the prevalence was highest in females (8:4), highest in maxilla (10:2), and highest on left side (8:4).
- ☐ In most of the present cases the positions of the unerupted primary molars were very deep, an observation also made in most of the earlier reported cases. The deep position may be explained by the vertical growth of the alveolar processes, which leave the impacted primary teeth progressively behind. Very often a "chimney" of soft tissues was left open between the occlusal surface and the crest of the alveolar process, most conspicuously in the mandible. This channel of soft tissues often remained open, while in many of the earlier case reports a filling in of bone tissue had occurred. The appearance of a radiograph translucency may well be mistaken for a follicular cyst.
- ☐ In the present cases two of the children were siblings, and in all cases a severely retarded eruption of the permanent dentition was found. This eruptional disturbance was also found in one or more of their relatives. In the earlier reported cases, no such familial relationships were found, and the authors offered no other etiological factor to explain

the failure in eruption. Also in the present cases no etiological factors other than inheritance could be documented.

Pedigree analyses of families with inherited primary failure of eruption in the primary dentition combined with severely retarded eruption in the permanent dentition point to a single gene effect with autosomal dominant transmission.[1] Thus it may be speculated that this is the gene that produces signal substances to initiate and control the eruption mechanism, which according to recent theories is believed to be linked to processes in the dental follicle or in the periodontal ligament.[11] As the parents reported that the first teeth, including the canines, erupted at normal time, it is reasonable to assume that this genetic/biological mechanism may have been working satisfactorily the first two years of life and then switched off before eruption of the primary dentition was completed, thus affecting only the last erupting primary teeth, in addition to causing a severe retardation in the eruption of the permanent dentition.

The increased prevalence of hypodontia affecting corresponding second premolars observed when primary molars were impacted (three of five cases), are much higher than may be expected by chance. Thus, it is reasonable to believe that the hypodontia is secondary to the impaction of the primary molars, and caused by crowding in the jaws. The fact that the space is scarcer inside the mandible, corresponds well with the finding that only one of six mandibular premolars managed to survive in the present cases. In this case the second premolar was, however, found compressed in the buccolingual dimension. Hypodontia was also observed by Jarvinen, but not in any of the other earlier reported cases.[9]

The inconveniences observed in inherited primary failure of eruption in the primary dentition are limited. In the present cases none of the patients complained of pain, and often the parents had not recognized that their children's dentitions were incomplete. The following aberrancies were observed:

- ☐ Suppression of the development of the corresponding premolars as found in cases 1,2,4. The present authors considered this phenomenon as a positive consequence, which prevented compression and eruptional problems for the premolars.
- ☐ Preventing the premolars to erupt. This seems to have been a problem in several of the earlier reported cases, irrespective of the position of the unerupted primary molar, below, parallel to, or above the premolar.
- ☐ Preventing the eruption of the permanent first molar. This may happen if the permanent first molar is locked beneath the distal curvature of the primary molar as seen in Case 1, left lower side.
- ☐ Preventing early mesial migration of the first permanent molar, which would be favorable when the second premolar is not developing, as seen in cases 1,2,4.
- ☐ Pathological developments as cyst formation or infection, not seen, however, in the present cases.

Treatment will always be extraction, but will not be needed immediately unless the problems listed above appear. Often the unerupted molars are very deeply seated and need surgical intervention most often using general anesthesia. Thus, the surgery may be postponed some years, it should be beneficial to the child. In the present cases the authors found the proper time for intervention to be at an age of ten to eleven years, because the patients also had retarded eruption of the permanent dentition, and none of the neighboring teeth were expected to erupt until the child was past twelve years. If the time schedule in eruption of permanent teeth is normal, intervention must occur earlier to allow normal eruption of the first molars and to prepare the way for the corresponding premolars. The adverse effects on the developing premolars, however, may be impossible to prevent, because this condition occurs very early and a long time before it has been diagnosed.

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BEHAVIOR

Intravenous sedation for outpatient treatment of child dental patients: An exploratory study

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R.J.M. Gruythuysen, DDS, PhD

Dental treatment of very young children, under four years of age, is known to be difficult. Most of the children showing disruptive behavior during dental treatment are between three and six years old.[1,2] The difficult behavior tends to decline with increasing age.[2] This is easily explained from a developmental view: the small child's brain is in the early stages of cognitive development; a complex matter like a dental treatment cannot be fully comprehended. Furthermore the attachment to (mostly) the mother is too strong to allow other people to assume control. The active and passive abilities to communicate orally are still immature.[3,4,5]

THE NECESSITY OF SEDATION

From this point of view, parenteral sedation or general anesthesia offers an adequate solution for extensive dental treatment of those who are certainly unable to adapt their coping skills to the necessary treatment, such as young or mentally handicapped children. Some conditions favor treatment strategies that do not require awareness and understanding by the patient, such as by a young child. Retrospective research attributes most of adolescent and adult dental fear to treatment received during early childhood and perhaps conditioned by unpleasant treatment experiences in childhood.[6,7] The possibility of a latent development of dental anxiety is suggested and supports, therefore, the idea of performing dentistry in young children in the least provocative way.[8] In older people it is questionable whether they are able to overcome their dental fears after treatment using intravenous drugs, because they lack the conscious exposure to training that enables them to increase their coping skills.[9]

ACCEPTANCE AND LEGISLATION

When dental treatment is needed for children showing disruptive behavior, parents are inclined to choose the least invasive treatment strategy possible. They dislike obstructive behavioral techniques, although when thoroughly informed they are more inclined to approve.[10-12] Dentists themselves are inclined not to use drastic behavioral management techniques, but rather to wait until the child is able to control its tantrum behavior.[1] The choice of sedation as a treatment strategy might be related to the experience and training of the dentist. In an Australian survey it was apparent that especially the younger dentists more frequently use behavioral strategies.[13] The use of general anesthesia seems to depend there on the availability of nearby hospital facilities, especially when compared to parenteral sedation.[14] The line of treatment chosen also depends on the legislation in a specific country and on the attitudes of its population. In many European countries, like the Netherlands, the combination of physical restraint (mouth prop and papoose board) with the use of oral, nasal or intravenous sedation is not permitted in general practice, only in combination with the services of an anesthetist. Often dental schools do not have a specific sedation training program. When training facilities are available, the other line of treatment comes up automatically: a survey identified that approximately 75 percent of American pediatric dentists relied on a variety of sedatives in selected

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patients, although their use is somewhat reduced, possibly due to recent guidelines or increasing malpractice insurance costs.[15]

DRUG REGIMEN

Sedating the young child is a complex matter. Often the line of treatment chosen is oral or intranasal sedation and must be combined with some form of physical restraint and the use of nitrous oxide.[15,16] Nitrous oxide is used to augment the effect of the sedation of young children, to weaken the child's behavioral responses; but positive effects do not apply uniformly for all young children.[17-19] Moreover, in measuring the atmospheric concentrations of nitrous oxide during the treatment of young children, concentrations in the room air that exceed the safety limits applied in most countries are reported.[20,21] Oral and intranasal sedation techniques have their specific benefits, but also drawbacks.[14,22,23] Various sedation regimens are studied, with reported success rates up to 90 percent.[15-24] No relationship was found between a patient's behavior and a specific drug regimen.[25] Several cases involving the patient's morbidity were reported, when increasing drug dosages or adding coagents.[14]

If sedation is not possible for medical reasons or the child's behavior demands a higher dose of the sedative drug, resulting in difficulties in maintaining an open airway or protective reflexes, the preference should be given to full anesthesia.[26,27] On the other hand, several fatal accidents were also reported after using general anesthesia (post-extubation aspiration; cardiac arrhythmia). All cases involved young dental patients between nineteen and sixty months of age.[14] Based on comparable results, a national report in Great Britain stated that general anesthesia should be avoided wherever possible and alternatives used.[27]

Propofol, a single drug

Combinations of drugs are usually administered to ensure the safe use of general anesthesia. For sedation, a single drug procedure may have benefits; because the effect of the sedative agent is not obscured by other drugs, there is no interference with its action and the risk of a delayed recovery is avoided. With the modern drug propofol (2,6 di-isopropophenol, Diprivan®) these benefits become apparent. Propofol is developed as an anesthetic for induction and maintenance of general anesthesia and sedation. It is a fast-working sedative, with few known side effects and a short half-life of 0.5-1 hour. There are no known reports of nausea during recovery and patients have no recollection of treatment.[28] It is vital that an anesthetist, controlling the treatment of the sedated patient, is fully aware of the signs and symptoms of both sedation and anesthesia.[29] Propofol has proven its superiority in recent studies using it for conscious sedation and ambulatory surgery.[30] It is in fact useful in minor procedures with spontaneous ventilation and it is sometimes combined with nitrous oxide, although no randomized clinical trials have been done concerning this supplement.[31,32] In children between four and ten years of age, a high infusion rate and, as a consequence, a slower recovery is reported.[21] The introduction of propofol into clinical practice as a shorter-acting intravenous drug, with fewer side-effects and decreasing recovery time has demonstrated its value as a sedative agent for outpatient treatment.[28]

SEDATION OR ANESTHESIA?

Sedation in pediatric dentistry is a fairly well-documented area, but the development of drugs with capacities for both sedation and anesthesia, like propofol, have changed the dividing line between sedation and anesthesia. Recently, guidelines have been developed for anesthesia and sedation, but neglecting the problem of differentiating between them in young children.[26,33,34] Literature on propofol sedation in children is scarce. Most of the reports on intravenous sedation are about adult patients. Whether sedation in toddlers and preschoolers is possible using propofol still has to be studied. Because the literature and the psychological concepts seem to be uncertain about the sedation concept for young children and to favor general anesthesia, further study is needed to take a closer look at the power of a single-drug-supported dental treatment procedure, in a completely controlled situation.

The aim of this pilot study was to investigate the possibilities and effects of propofol as an intravenous agent for treatment of the youngest group of dental patients. We took a close look at the treatment itself, its safety and its convenience. Recommendations for further use and research are made.

MATERIALS AND METHODS

Patient selection

For this pilot study nineteen healthy (ASA I) children from twenty-four to forty-five months of age were selected. All children were referred for treatment because

the referring dentist could not manage the patient. They were selected during a separate screening visit and their anxiety was assessed, using a special (Likert-type) anxiety scale.[35] The children were considered to be too young to enter a normal restorative treatment program. They had severe dental decay, most of it caused by misuse of the nursing bottle. Stopping the habit of giving their child a sweetened pacifier was conditional to entering the restorative program. The treatment was based on normal standard routines: the affected teeth were treated conservatively, if possible, or otherwise extracted. Every child required restorative or surgical dental treatment in four to six sextants.

The treatments were performed by a dentist and an anesthetist, after informed consent from the parents and a thorough medical check-up. Medical check-up included possible contraindications like recent airway illness, known allergies like nut-allergy, or sickle cell anemia.[36,37]

Treatment procedure and level of sedation

The protocol of the propofol infusion was performed according to the guidelines of the council of the European Federation for the Advancement of Anaesthesia in Dentistry (EFAAD), the British Society of Paediatric Dentistry and the guidelines for deep sedation in pediatric dental patients, as defined by the American Academy of Pediatric Dentistry.[33,38,39] The parents and the child were asked to be in the clinic for a final check-up and to remove the topical anesthetic cream (EMLA®) thirty minutes before starting the treatment. The cream was administered at home by the parents to the dorsum of the right hand and foot and protected by dressings, according to written instructions. Treatment is started by the anesthetist placing an intravenous access. The intravenous line was kept in place for the propofol infusion during treatment. The sedation was induced by administering a propofol bolus via a Graseby anesthesia syringe pump, sufficient to sedate the child to a level that enabled the dentist to perform dental treatment without physical restraint. If the child still proved to be too agitated to perform routine dental treatment, additional propofol was given via the infusion pump. The anesthetist's aim was to keep the propofol concentration as low as possible, but provide anesthesia sufficiently deep to ensure an undisturbed dental procedure. Lack of crying or movement was evidence of good sedation. All patients were allowed to breath spontaneously. The treatment was performed in a standard dental chair, with the patient's head somewhat overstretched, to guarantee an open airway (Figure).

Blood pressure, pulse rate, and arterial oxygen saturation (S_aO_2) were monitored. The quality of the sedation was assessed by the anesthetist. The following specifications were recorded: age, body weight, pulse rate, S_aO_2, child behavior during treatment, amount of propofol used, induction time, treatment time, and recovery time. Also the data concerning oxygen desaturation, possible intraoperative and postoperative complications were registered. The behavior was registered using the Venham modified behavior rating scale, a six-point numeric scale on which the two extremes were labelled 0 = not afraid, or highly treatable; and 5

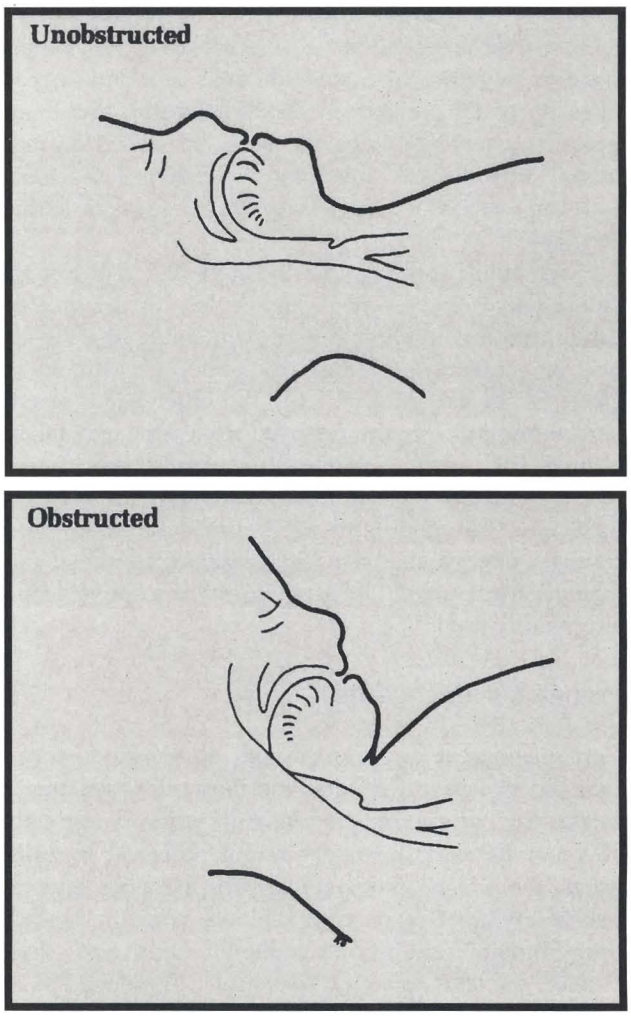


Figure. Graphic representation of a child's positioning to guarantee an unobstructed airway.

Table 1 ☐ Distribution of dental procedures.

	Ex	Rest	Pulp	Ssc	Seal
Maxilla	15	71	8	24	16
Mandible	5	23	4	13	20

= panic, or untreatable, as originally used by Venham.[40] Following known criteria we defined desaturation at every oxygen saturation level ≤ 90 percent for a period 2 ≥ 20 seconds. When desaturation occurred, the position of the head and neck was checked and the chin lifted, if necessary. All treatment procedures were performed, using local anesthesia and the rubber dam.

End of the treatment

After ending the dental treatment the anesthetist stopped the infusion. The child was monitored until it awakened and was able to walk around freely and reacted adequately to mother's questions. The anesthetist contacted the parents six hours later, to check on the child's condition. The children returned for check-up visits approximately four weeks later.

Statistical analysis

All treatment results were analyzed, using a correlation matrix, SPSS/PC+V4.0.[41]

RESULTS

Most of the children were treated because of nursing bottle caries, with the major part of the treatment performed on the maxillary teeth. Most of the affected teeth were restored, using stainless steel crowns, amalgam or composite resin restorations. The injection of the local anesthetic and the placing of the rubber dam, pain and an increase of the pulse rate were frequent occurrences. All children reacted by slight movements of the hands or feet in response to the pain, but not of their heads. By ending the stimuli, movements and pulse rate quickly returned to normal.

Table 2 gives the graphic representation of the amount of propofol given to sedate the child (initial dose) and the dose to continue the sedation (maintenance dose). The initial propofol bolus varied extensively, between 2.0 and 5.3 mg propofol/kg body weight (average 4.0 mg/kg body weight, speed: 200ml./hr). The variation is not related to the body weight (p=n.s.) or the age of the child. The graph gives a fairly constant

Table 2 ☐ Initial and maintenance concentration propofol, needed for sedation, per kilogram bodyweight, in relation to the age of the child. The maintenance concentration is given per minute.

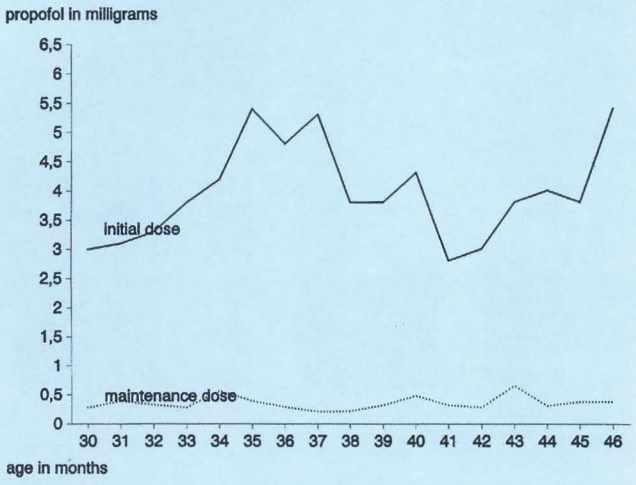
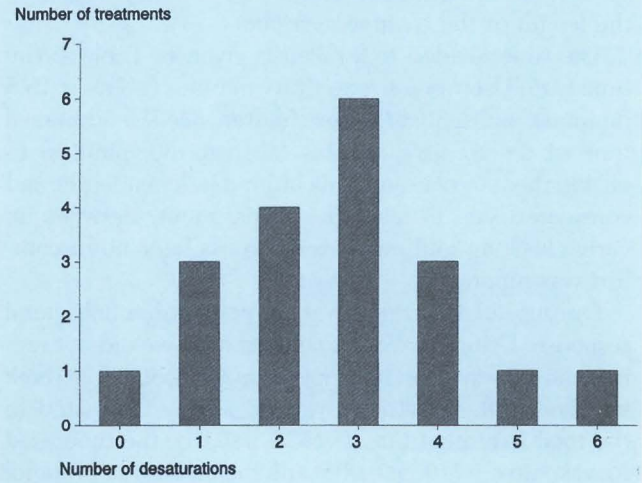


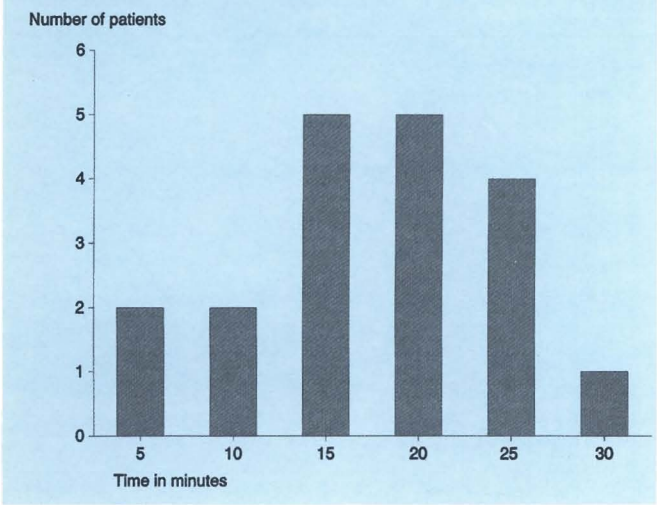
Table 3 ☐ Number of desaturations during treatment.



amount of propofol used for the maintenance of the sedation, after the administration of the induction bolus. The concentration for the maintenance of the sedation varied between 0.3 and 0.9 mg/min/kg bodyweight (average: 0.49 mg/min/kg b.w., speed 24ml/hr). We found no relation between age or body weight. The maintenance ratio was eight times slower than the induction bolus. No other relation was found between the maintenance ratio and the age of the child or the induction bolus.

Table 3 represents the number of desaturation periods during each treatment. No significant relationship was found between the oxygen saturation and the pro-

Table 4 ☐ Time taken to wake up.



pofol concentration, the child's age or bodyweight, or the length of the treatment period.

The time needed to awaken is given in Table 4: the time varied between five to thirty minutes (average: 18.5 minutes). Subsequently the children needed additional time of ten to sixty minutes (average: 30 minutes) to enable them to play and walk around independently, and considered safe to leave the Department. Between an early check-up and one several weeks later no discomfort was reported.

During sedation there was no perceptible behavioral response. Using the Venham rating scale we did not register any anxious or uncooperative behavior. The rank number of the treatment was negatively correlated to the total treatment time ($-.583$, $p \leq 0.01$), the time used to wake up ($-.710$, $p \leq 0.001$) and the amount of propofol used ($-.623$, $p \leq 0.01$). A weak correlation was also found between the sedation time and the time necessary for awakening (.469, $p \leq 0.05$). Except for an obvious correlation between the total treatment time and the total amount of propofol used (.778, $p \leq 0.001$) no correlations of any importance were found.

DISCUSSION

In this group of children most of the affected teeth were destroyed by nursing bottle caries. The pattern can be recognized by difference in numbers of restored teeth in the maxilla and mandible and by similarity to other signs of the disease.[42]

In young children, toddlers for instance, sedation is difficult to define. In general, a sedated patient should be able to respond and to maintain his protective reflexes.[26,27,33,34,38] Young children or mentally handicapped people of comparable developmental level are not able, however, to recognize or understand the relaxed, dreamy feeling of sedation. The child is not yet able to lie down in an unfamiliar surrounding and focus on a dental treatment for several minutes. As a consequence, the feeling that accompanies sedation is interpreted by the child as offering one of two possibilities: being awake or being asleep and the child wants to act accordingly.

The child's behavior was recorded in the preoperative appointment only as a means of entering the sedation program. Since the reason for the referral was a negative dental experience, it was considered to be in conflict with the aim of the introductory visit to do more than this. No further attempts were made to treat the child in a normal routine way. Considering the child's limited mental capacities, it did not seem purposeful to repeat this dental procedure. The use of nitrous oxide was not allowed, because of the possible high concentrations of the gas mixture in the operating room, due to the child's disruptive behavior. The presence of an anesthetist is in conformity with the European guidelines on sedation and anesthesia.[20,33,34]

It was not considered effective to choose an instrument for behavioral analysis. In this exploratory study, where the treatment was performed without mouthprop and papoose-board, the behavior could be influenced only by the propofol titration. Normally evidence of an adequate sedation is a lack of crying or movement. If oral sedatives are given, behavioral scales like Venham's are useful.[40] In this study the lack of crying and movement was the only level of sedation possible to treat the young child. The anesthetist had to adapt his dose to the response of the child, resulting in a deep sedation: the child was able to breath spontaneously and reflexes like holding hands up during local anesthesia were present but random and erratic. It is suggested that the child does not understand the concept of being sedated because of its inability to comprehend. This might mean that the concept of intravenous sedation has to be reformulated for young children. Recent publications express rather strongly that the level of sedation as created in this study in fact should be named anesthesia.[26,33,34] It would seem inappropriate, even unethical, however, in situations like this to fix the propofol dose and wait for the behavioral response. The aim to maintain the propofol dosage as light as possible pays back in the shorter recovery time than the recovery times reported elsewhere.[31] Additional studies based on clear protocols are certainly mandatory.

On choosing the treatment modality, most of the parents preferred sedation, favoring the treatment in the dental fear clinic and the shorter recovery time for the child. The fear many adults have of general anesthesia, might be the result of a negative choice.[43] Also it must be stated that the choice of a treatment procedure is primarily the decision of the medical team: the patients safety must be the primary concern. Intravenous sedation will be a possibility, only when the patient's physical condition and the nature of the dental treatment make it feasible.

The amount of propofol per kilogram bodyweight used in this study was higher than in sedation procedures of adult patients.[28] This is known from the few studies on pediatric patients. As a consequence, the recovery time for children is longer compared to adult patients.[31] On the one hand the higher propofol dose might result in a longer recovery time, on the other hand the child's different perception of the actual sedated state might cause a resistance to awakening. In fact the slow recovery might be caused by the same mechanism that causes the child to be disinclined to allow herself to be sedated. In this case, the child wants to continue his sleep, because he feels good when asleep. Partially, this might be caused by the propofol itself, since it causes an euphoric state.[32]

The negative correlation between the amount of propofol used, the time the child needed to awaken and recover and the total amount of propofol used seem to be part of the learning process of the operating team. The same applies to the weak but significant correlation between the length of the treatment and the time necessary for awakening. During the first treatment procedures the anesthetist had to get used to the idea of keeping the child as lightly sedated as possible. Further research on the subject is necessary.

The lack of correlation between other factors stress the need for further research. First, from these results it seems advisable to find an instrument for registering the child's emotional response. Since in this study no significant correlation was found between the propofol dose and the child's body weight, it seems that other factors are of vital importance in assessing a dose-response curve. Second, the lack of a relationship between the initial propofol dose and the dose required to maintain the sedation, underlines the complex nature of dental anxiety or arousal during dental treatment. An initial dose does not mean the child needs more or less during the rest of the treatment. If a high initial propofol bolus is needed to sedate the child, a high dose might also be necessary later, just the same as a lower dose might be needed because the child becomes more relaxed during the course of treatment.

The short recovery time might be influenced by the course of the treatment planned for that visit. Possibly certain additional treatment elements (pulpotomies, extractions) were introduced after treatment was begun, and because the propofol dose was reduced toward the end of the treatment, the recovery time was usually short. Results correspond with a study where it was found that the number of extractions was related to the ineffectiveness of the sedation.[16] During treatment several moments of desaturation occurred. One of the most important aspects of deep sedation/anesthesia is the guarantee of an open airway. The rating of an open airway is clearly a dichotomous evaluation: clear or obstructed.[30] When an anesthetist is responsible for the administration of the anesthetic, different rules must be maintained: the recommended strategy is the registration of oxygen desaturation. The definition of desaturation is based on earlier studies.[29,30] As explained, no baseline saturation values could be recorded, since we were not able to perform undisturbed relaxed registrations, because of the children's ages. Desaturation is an important factor in determining the success, however, of the anesthesia when supported by an anesthetist. A successful anesthesia should maintain a maximum saturation during the full course of treatment. Especially young children, however, are reported to show an unstable S_aO_2 during treatment.[44] Since no correlations with age, bodyweight, the length of the treatment or the concentration of the propofol were found, further studies should try to create circumstances in which this goal can be achieved.

In this explorative study we looked at the possibilities of propofol as a single sedative drug for toddlers and preschoolers, since the simultaneous use of several sedative drugs might create an extra risk factor in an ambulant treatment situation. In an ambulant situation every security precaution must be observed. The additional attention to pretreatment diagnosis and the use of a single drug should be approached from this point of view. Nevertheless, propofol seems to be particularly useful in an ambulatory setting. Its quick onset, patient's short recovery time, its effectiveness in all patients treated and the broad limitations of potential risk patients, are all promising. Young children especially seem to be good candidates for ambulatory procedures, since they are generally healthy, emerge from anesthesia more rapidly than adults, and benefit greatly from the psychological support of a familiar environment.[45]

CONCLUSION

The use of propofol may be a useful support in the treatment of patients with a limited cognitive level. If depth of sedation is guided by the dosage of the drug as a response to the behavior displayed and no physical restraint or mouthprop is used, the level of sedation seems more difficult to define and tends to lean toward anesthesia. More studies on protocols are mandatory.

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EARLY CHILDHOOD CARIES

Soft drink logos on baby bottles: Do they influence what is fed to children?

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The marketing of baby bottles decorated with name-brand soda pop logos (Pepsi, diet and regular, 7UP, Dr. Pepper, Orange Slice, and Mountain Dew), noncarbonated soft drink (Kool-Aid) and juice logos (Mott's, Welch's, V8, Veryfine, and Perrier) is raising concern among many health care professionals who fear that the recognizable logos will encourage parents to give children soda and other inappropriate beverages in baby bottles. Health professionals report that increased consumption of sweetened beverages could increase the child's susceptibility to potentially severe and costly nutritional and dental problems.[1,2]

Sodas have negligible nutritional value, frequently contain caffeine, and are high in sugars, often sucrose, the most cariogenic.[3] In addition, sodas contain carbonic acid, which dissolves tooth enamel. Soft drinks and juices, which have a minimal nutrient content, have a carbohydrate content equal to that of sodas. All contain negligible amounts of the recommended daily value for vitamins, minerals, and protein.

Nutritionists warn that sweetened beverages can curb a child's appetite and displace protein and calcium rich foods needed for proper growth. Excessive intake of fruit juice has been associated with chronic diarrhea and failure to thrive.[4]

Dental professionals are concerned about a condition known as Baby Bottle Tooth Decay (BBTD), more recently called Early Childhood Caries (ECC), a serious pattern of dental caries that begins with a child's maxillary incisors, where the nipple of baby bottles is placed. The decay can be caused by a combination of improper bottle feeding habits and any sugar-containing liquids put in the bottle.[5,6] BBTD can result in pain, infection, and premature tooth loss. Treatment is costly, possibly traumatic, and may require crowns, extractions, hospitalization and general anesthesia.[7]

The Dental Health Foundation and the California Department of Health Services designed this study to determine whether young children drank soda and Kool-Aid*

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Authors want to acknowledge those who contributed to the study through their expertise, funding, and time:
☐ The Dental Health Foundation
☐ California Dental Association
☐ University of California, Berkeley, School of Public Health
☐ Sacramento, San Bernardino and Yuba County Interviewers
☐ Sacramento Kiwanis Club
☐ Richard Sun, MD, MPH
☐ Zipora Weinbaum, Ph.D.

*Soda pop and juice were used generically because it was cumbersome to ask which of the six in the soda pop series and of the five in the juice series children drank. Kool-Aid was specifically asked in the survey because it was the only noncarbonated soft drink with its logo on a baby bottle.

from baby bottles**, and if so, whether there was a positive association between those children who drank soda or Kool-Aid and those who owned the bottles with soda or Kool-Aid logos respectively.***

MATERIALS AND METHODS

From September 1994 through February 1995, a convenience sample of 314 women was interviewed in northern and southern California at major grocery and discount stores, and at a day-care center. All females who appeared to be within child bearing age were approached and asked whether they had a child presently using a baby bottle. Those that responded positively or said their child had used a baby bottle within the previous three months were asked to complete a face to face survey. Grandmothers were interviewed, if they were principal caregivers. Tippy cups and inexpensive children's toys were given as incentives to participate. Interviews were conducted weekdays and weekends using nine trained interviewers.

Thirteen questions, requiring approximately three to five minutes to answer, were asked about bottle feeding practices, opinions of the beverage logos on baby bottles, and personal demographics. Questions pertaining to feeding practices included what the child drank from the bottle and the frequency with which the child drank each liquid. If more than one child was bottle-fed, these two questions were asked about the oldest child only. Then, being shown a display of bottles with soda pop, juice and Kool-Aid logos, respondents were asked whether they had similar bottles, and if so, which types specifically.

Respondents' opinions were sought on whether the logos can influence what parents put in the bottles; whether the logos were a form of advertisement; and whether the logos were intended to get parents to buy the beverages for the whole family, including the youngest. Demographic questions included respondent's age, highest school grade completed, ethnicity, and age of child using the bottle.

Data were entered and analyzed, using Epi-Info software to calculate chi-square tests for independence.

**When the report refers to the consumption of soda, Kool-Aid, and juice, it is always in the context of consuming the beverage from a baby bottle. The report does not take into account consumption of beverages from cups or other containers.

***Respective beverage refers to the child drinking Kool-Aid from baby bottles if the family owned a "Kool-Aid logo" bottle and drinking soda if they owned a "soda logo" bottle. It does not imply, however, that the Kool-Aid or soda was always consumed from the respective "logo bottles" and not from other bottles.

Table 1 ☐ Demographic Variables (n = 314)

	(n)	(%)
County		
Sacramento	(101)	32
Sutter	(43)	14
San Bernardino	(170)	54
Respondent's ethnicity		
White	(170)	54
Black	(42)	14
Hispanic	(80)	25
Other	(19)	6
Unknown	(3)	1
Respondent's education		
<HS*	(58)	18
grad HS	(95)	30
>HS	(158)	51
Unknown	(3)	1
Respondent's age (yrs)		
15-20	(50)	16
21-30	(141)	45
31-40	(85)	27
41-65	(36)	11
Unknown	(2)	1
Children's age (mos)		
0-6	(61)	19
7-12	(99)	32
13-24	(132)	42
25-48	(22)	7

*HS = High School

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RESULTS

Specific response rates were not determined for this convenience sample. Interviewers estimated, however, that there was a 90 percent response rate among those eligible.

Demographics

Three hundred fourteen respondents in three California counties were interviewed (Table 1). The ethnic mix resembled that of California's population.[8]

Respondents' ages ranged from fifteen to sixty-five years with a mean of twenty-nine. Respondents' education by highest grade completed was as follows: 18.6 percent did not graduate from high school; 30.5 percent graduated from high school; and 50.8 percent attended college. This resembled the educational distribution found in California's 1994 unpublished Behavioral Risk Factor Survey.

Questions regarding liquids children drank from the bottle were asked in the following order: milk (breast milk, formula, bovine), water, juice, Kool-Aid, soda pop, and others. As expected, virtually all parents gave milk from bottles, and almost three-fourths provided water and juice. The study found that as many as a third of the respondents gave children Kool-Aid or soda. When respondents were asked about the frequency with which specific liquids were consumed from a baby bottle, seven respondents who said the child did not drink soda and

Table 2 ☐ Percent of children by age-group drinking juice, soda, Kool-Aid from baby bottles.

Child's age (mos)	(n)	*Percent drinking juice, soda, Kool-Aid			
		Juice	Soda	Soda Kool-Aid	or Kool-Aid
0-6	(61)	44.3	1.6	1.6	3.3
7-12	(99)	76.8	17.2	22.2	26.3
13-24	(132)	77.3	35.6	38.6	45.5
25-48	(22)	72.7	40.9	36.4	45.5
Total	(314)	70.4	23.6	25.8	31.2

*Percents signify row percentages

Table 3 ☐ Frequencies with which children drink soda and Kool-Aid from baby bottles.

	(n)	Number days per week			
		6-7/wk	3-5/wk	<3/wk	Total
Soda	(74)	28%*	13%	59%	100%
Kool-Aid	(81)	42%	32%	26%	100%

*Percents signify row percentages

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four who said the child did not drink Kool-Aid in the previous question, replied that the child did drink soda or Kool-Aid from a baby bottle occasionally. These second responses were considered more accurate and were included, therefore, in the analysis.

Although some statistics about juice are included in this report, to allow comparisons, juice consumption was not analyzed for association with owning "juice logo" bottles. The authors believe that because of the high frequency with which families give children juice and the generally (not medically) held belief that juice is beneficial to children, the "juice logos" would not be a key influencing factor in determining whether families give children juice from baby bottles.[9]

Juice was given as early as one month of age. With the exception of two children, however, neither soda nor Kool-Aid was given before eight months of age. After eight months, the percentage drinking soda and Kool-Aid increases with age, whereas the percentage drinking juice levels off (Table 2).

The frequency with which children drank specific beverages in baby bottles was measured by the number of days per week these liquids were consumed. The study found that of those who drank soda from baby bottles, 28 percent did so daily, and of those who drank Kool-Aid, 42 percent drank it daily (Table 3).

Respondents who owned "Logo Bottles"

The study found that 53 percent of the respondents owned baby bottles with soda, Kool-Aid, and/or juice logos. Specifically, 46 percent owned soda, 16.6 percent Kool-Aid, and 13.7 percent juice bottles. The analysis revealed little demographic variation between families who owned "Kool-Aid bottles", or who owned "soda bottles", with the exception of the thirty-one to forty age-group in which a lower percentage owned "soda bottles" (Table 4).

Table 4 ☐ Own "logo bottles" and give children soda and/or Kool-Aid from baby bottles.

Respondents (n=314)		Soda		Kook-Aid	
		Own soda bottle %	Give soda from bottle %	Own Kool-Aid bottle %	Give Kool-Aid from bottle %
Ethnicity					
White	(n=170)	45.6	17.8	16.0	16.5
Black	(n=42)	52.4	33.3	23.8	42.9
Hispanic	(n=80)	47.5	30.0	15.0	37.5
Other	(n=19)	21.1	21.1	10.5	15.8
p value ⊞		—	—	—	***
Age					
15-20	(n=50)	60.0	38.0	20.0	44.0
21-30	(n=141)	47.5	16.3	16.3	19.9
31-40	(n=85)	31.8	24.7	10.6	22.4
41-65	(n=36)	54.3	25.7	25.7	27.8
p value ⊞		**	*	—	**
Education					
<HS**	(n=58)	50.0	46.6	19.0	44.8
HS grad	(n=95)	49.5	24.2	18.9	31.3
>HS	(n=158)	41.8	14.6	13.9	15.2
p value ⊞		—	***	—	**
Totals		46.0	23.6	16.6	25.8

* Percents signify row percentages
□ HS = High School
⊞p values
* = <.05
** = <.01
*** = <.001
— = not significant

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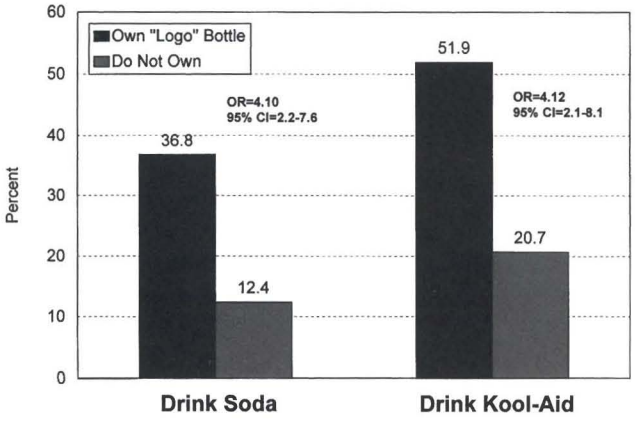

Figure. Percent of children who drink soda or Kool-Aid from baby bottles, by ownership of "logo" bottle.

Respondents who gave children soda or Kool-Aid

Respondents in all ethnic, age, and educational groups gave soda and Kool-Aid to children in baby bottles. There was a significant increase in the number of respondents who gave children these beverages in the black and Hispanic groups, the youngest age-group (15-20 years), and if respondents did not have a high school diploma (Table 4). A multiple logistic regression found that the two most significant predictors of whether respondents gave children soda or Kool-Aid were

☐ If respondents owned the respective "logo bottle",
☐ Respondents did not have a high school diploma.

Children owned "Logo Bottles" and drank respective beverage

The prevalence of owning "soda bottles" and giving children soda was highest among blacks and Hispanics. The prevalence of owning Kool-Aid bottles and giving Kool-Aid was highest among blacks. The percent of Hispanic children who drank Kool-Aid from bottles was almost that of blacks, but fewer Hispanic families owned the "Kool-Aid" bottle. By respondent's age, the prevalence of owning "soda or Kool-Aid bottles" and giving the respective beverage was highest among the teen parents, followed by those forty-one years and older. By education, prevalence was highest among respondents who did not have a high school diploma. The higher the education, the lower the prevalence found.

A statistically significant association was found between those who own a baby bottle with a soda logo and those whose children drink soda. A statistically significant association was also found between those who own a bottle with a Kool-Aid logo and those whose children drink Kool-Aid (Figure 1). In every ethnic, age, and educational group, a higher percent, and in most cases a statistically significantly higher percentage, of children were given soda and Kool-Aid when they owned the respective logo bottles (Table 5).

Table 5 ☐ Percent of children drinking soda or Kool-Aid from baby bottles.

Respondents (n=314)		Drink soda			Drink Kook-Aid		
		Own "soda bottle" %*	Do not own "soda bottle" %	p value ▣	Own "Kool-Aid Bottle" %	Do not own "Kool-Aid bottle" %	p value ▣
Ethnicity							
White	(n=170)	23.4	13.0	—	40.7	12.0	***
Black	(n=42)	54.5	10.0	**	90.0	28.1	***
Hispanic	(n=80)	47.4	14.3	**	41.7	36.8	—
Age							
15-20	(n=50)	50.0	20.0	*	60.0	40.0	—
21-30	(n=141)	29.9	4.1	***	39.1	16.1	**
31-40	(n=85)	40.7	17.2	*	55.6	18.4	**
41-65	(n=36)	31.6	18.8	—	66.7	15.4	**
Education							
<HS**	(n=58)	65.5	27.6	**	72.7	38.3	*
HS grad	(n=95)	36.2	12.5	**	44.4	28.6	—
>HS	(n=158)	24.2	7.6	**	45.5	10.3	***
Total		36.8	12.4	***	51.9	20.7	***

* Percents signify row percentages
□ HS = High School
▣p values
* = <.05
** = <.01
*** = <.001
— = not significant

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Logos

When asked whether logos can influence what parents put in bottles, 70.2 percent of the respondents said they would; 25.6 percent said they would not; and 4.2 percent did not know. When asked whether logos are a form of advertisement, 92.3 percent said yes, 5.8 percent said no, and 1.9 percent said they do not know. When asked whether logos are intended to get the parents to buy the beverages for the whole family, including the youngest, 68.1 percent replied yes; 27.1 percent said no; 4.8 percent did not know.

DISCUSSION

This study used a convenience sample as opposed to a random sample, and results, therefore, are not generalizable. The study was conducted in three counties (Table 1), however, and the respondents' ethnicity and educational levels are similar to those of California's.

"Soda bottles", specifically, proved to be a commonly owned baby item by all demographic groups with almost half the population surveyed owning them. This was almost three times more than the number who owned Kool-Aid "logo bottles". Parents were not asked why they owned the "logo bottles", but interviewers mentioned that respondents commonly reported that they were "cute" and occasionally said they got them as gifts.

Regardless of whether families owned a "logo bottle", the study found that almost a third of families studied gave children either soda or Kool-Aid from bottles and, surprisingly, almost as many children drank soda as drank Kool-Aid from bottles. Kool-Aid is marketed as a child's beverage, but soda with caffeine and carbonation has not traditionally been targeted to the very young.

The authors believe that the percent of children who drank soda was a conservative estimate for several reasons. First, respondents might not have been honest in a face to face interview where caregivers might have perceived they were being judged. Second, Sutter county (Yuba City) was the only area where no respondents reported giving soda. Further exploration revealed that many residents in the Sutter county area attended Women Infants and Children (WIC) and perinatal classes where they received nutritional instruction. This may have influenced both what parents consequently fed children as well as how honestly they reported.

Third, because people generally consume less liquids in winter and due to California's unusually cold and rainy winter, the results (both of numbers of children who drank soda or Kool-Aid and frequency with which they drank) may not reflect what would be found in California's warmer months.

Overall, families who owned "soda" or "Kool-Aid bottles" in every demographic group were four times more likely to give children the respective beverage from baby bottles, if they owned bottles with the respective logos. Because of the lack of baseline data on soda and Kool-Aid consumption from baby bottles before these "logo bottles" were introduced to the market, the results could not be used to determine conclusively whether the logos influenced families to give children soda or Kool-Aid. Some families, as documented, gave soda and Kool-Aid and did not own the "logo bottles".

Because of the strong association, however, the authors hypothesize that logos may tempt some families to give sweetened beverages in baby bottles instead of in cups, and/or at younger ages or more often than they otherwise would. Also, the "cute" logos may encourage families to use the bottles as pacifiers between meals, all increasing the risk of BBTD and of children drinking sweetened beverages instead of milk with protein, calcium, and other essential nutrients.

The study alerts dental and medical professionals that a significant number of families are giving children inappropriate beverages in baby bottles and that "logo bottles" could be influencing this. These results emphasize the need in all groups, and especially in designated high-risk groups, blacks, Hispanics, teen parents, and those without a high school diploma, for increased education about bottle-feeding.

The authors surmise that the look-alike bottles and packaging (Dr. Pepper packaging reads "Just what the doctor ordered", the Pepsi bottle says "Gotta have it") may create a confusing message for parents who attend prenatal, WIC, and nutrition classes where they receive a set of dietary guidelines. At home, parents then repeatedly see the bottles with logos that may subliminally encourage them to buy the beverage, and may give the message that it is okay to feed the child what is on the logo.[10] Whatever the intent of the logos, the majority of respondents believed the logos were to get the parents to buy the beverage for the whole family, including the youngest.

Campaigns that educate families about nutrition, BBTD, and proper dental care should incorporate a statement that addresses the popularity of the bottles and the possible misleading message they may give. Messages given by practitioners should include feeding children a nutritionally well-balanced diet; if giving sweetened liquids, to do so moderately and when the child is old enough to drink them from a cup.[11] The baby

bottle should be used for milk and water only. The study results show that parents begin feeding a sweetened beverage at approximately eight months. This is a significant time, because it correlates with the eruption of the primary maxillary central incisors, the teeth most susceptible to the pattern of baby bottle tooth decay. This is also the time children can use tippy cups.

Although educating parents about feeding practices is crucial and should be a high priority for health care professionals, often times the education that takes place between practitioner and parents is not enough to counteract the effects of repeated advertising.[12] Responsible use of logos, in combination with education, would most likely have a greater impact in influencing families about what is most healthful for small children.

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TOTAL FLUORIDE INTAKE IN CHILDREN, AGES 3 TO 4 YEARS

The findings of this study support the view by others that the systemic fluoride dosage schedule, based on McClure's estimates, is too high for children older than two years, in view of the other current sources of fluoride in children. This study supports the reduction both in total supplement use and in the fluoride doses of the supplements in children to decrease fluorosis in the permanent anterior teeth and first molars. Fluoride supplement dosages for older children have been reduced recently in Canada and the United States, and the use of fluoride supplements and their doses are currently being reevaluated by the Public Health Commission in New Zealand. Studies such as this current one are important in monitoring levels of systemic fluoride intake, especially during the period of enamel development in the first 20 to 36 months of life, when the teeth are susceptible to dental fluorosis. Even if fluoride use were to become totally topical, foods and drinks would always contribute to systemic fluoride intake and would need to be monitored regularly.

Guha-Choudhury, N.: Total fluoride intake in children, aged 3 to 4 years—A longitudinal study. J Dent Res, 75:1451–1457, July 1996.

ECONOMICS

Favorable dental economics into the middle of the next decade, including pediatric dentistry

H. Barry Waldman, BA, DDS, MPH, PhD

"Average net income among U.S. independent dentists in 1994 topped $127,000—up 10.5 percent over the previous year ..."[1]

The 1995 ADA Survey of Dental Practice reported continuing increases in "real income" (after adjustment for inflation) since 1991. The improving economics of dental practice during the 1990s has been in contrast to the late 1980s when real income growth was flat due to inflation and rising practice expense.[1] While American Dental Association (ADA) reports provide a view of improving economics during the most recent past, a series of projections by various federal agencies and organizations of the profession, offer a continuing favorable picture for the next ten years. These projections include 1) increases in national and per capita expenditures for dental services, and 2) changes in the numbers of professionally active dentists; resulting in 1) increases in practitioner business activity and 2) improvements in net income.[2,3]

The year is now 2005. How are you and your colleagues doing?

SOURCE OF INFORMATION

The Health Care and Financing Administration (HCFA) periodically publishes health expenditure projections (based on current laws and practice activity) using trend analysis, including actuarial, statistical, economic and judgment factors. The most recent review provides projections through 2005.[2]

The Bureau of the Census offers a continuing series of population estimates over extended periods of time.[4] Per capita expenditure data were developed using HCFA and Bureau of the Census estimates. Corrections for rates of inflation were developed using HCFA projections.[2]

The ADA, the American Association of Dental Schools, and the Bureau of Health Professions of the U.S. Public Health Service (BHP) provide varying estimates of the current and future numbers of professionally active dentists. While all projections indicated an increase in the numbers of dentists through the late 1990s, followed by decreases during the next decade, BHP projections indicate greater future numbers of professionally active dentists. In an effort to develop a conservative estimate of expenditures per dentist and dentist income, these larger estimates were used in conjunction with national dental expenditure estimates.

Notes: 1. In addition to practicing dentists, "professionally active dentists" includes administrators, researchers and educators. The inclusion of these added numbers of dentists, who may practice to a limited extent, will decrease further the conservative estimates of expenditure per dentist and dentist income.

2. The dentist-to-population ratio reached its peak in 1987 and has started a significant decrease (reflecting a continued increase in the general population and a lev-

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Table 1 ☐ Per person expenditures for dental services: selected years: 1980-2005.[2,4,7]

				Projected		
	1980	1990	1994	1995	2000	2005
Dental expenditure (billions)	$13.6	$30.4	$40.0	$42.9	$59.1	$79.1
Population (millions)	226.5	248.7	260.7	263.4	276.2	288.3
Expenditure per person (current dollars)	$60.04	$122.23	$153.43	$162.87	$213.61	$274.37
Price deflator (1987=100)	71.7	113.3	126.2	129.7	150.1	174.0
Expenditure per person (constant dollars)	$83.70	$107.89	$121.58	$125.60	$142.30	$157.68

Table 2 ☐ Net income of active dentists: selected years 1980-2005.[2,3,6]

				Projected		
	1980	1990	1994	1995	2000	2005
Dental expenditure (billions)	$13.6	$30.4	$40.0	$42.9	$59.1	$79.1
Number of active dentists	126,200	150,760	155,200	156,300	154,500	151,650
Expenditures per dentist (000s)	$107.8	$201.6	$257.7	$274.5	$382.5	$521.6
Net income as a percent of gross receipts	40.1%*	33.2%	35.1%	35.0%	30.0%	26.0%
Net income per dentist (000s) (current dollars)	$43.2	$66.9	$90.4	$96.1	$114.8	$135.6
Price deflator (1987=100)	71.7	113.3	126.2	129.7	150.1	174.0
Net income per dentist (000s) (constant dollars)	$60.3	$59.1	$71.7	$74.1	$76.5	$77.9

* 1981 datum

eling down in number of graduates from schools of dentistry) which is expected to continue well into the next century, reaching levels not seen since the early years of the twentieth century.[5]

The *ADA Survey of Dental Practice* provides a detailed review of the business aspects of dental practice.[6] For purposes of this presentation, data for business receipt and overhead costs were used to develop net income to gross receipt ratios. Once again, in an effort to develop conservative estimates, progressive decreases in the net income ratios (far below past and current estimates) were used for future projections.

FINDINGS

Dental expenditures

Between 1994 and 2005, national expenditures will double, with more than half of the increase resulting from continuing changes in the rates of inflation. Nevertheless, constant dollar expenditures (removing the effects of inflation) are projected to increase by 43 percent during the period reflecting an increasing use of dental services by an enlarging population.

- ☐ Current dollar per capita dental expenditures are projected to increase from $153 in 1994 to $274 in 2005.
- ☐ Constant dollar per capita expenditures are projected to increase from $121 to $157 (Table 1).

Dentist income

During the same period, expenditures per professionally active dentist are projected to increase from $257,000 in 1994 to $521,600 in 2005. (ADA estimate for gross receipts per independent *practicing* dentist in 1994 was $362,780.[6])

- ☐ Current dollar net income for all professionally active dentists is projected to increase from $90,400 in 1994 (compared to ADA estimate of $127,000 per independent dentist) to $135,600 in 2005.[1]
- ☐ Constant dollar net income would increase from $71,700 to $77,900 (an 8.6 percent increase)—despite the use of a projected marked decrease in the net income to gross receipt ratio from 35 percent to 26 percent (Table 2).

SOME THOUGHTS

Projections based on current laws and circumstances are subject to numbers of unforeseen exigencies. But if we are to await any and all developments before establishing future estimates, we would have to await the time for which we are planning. By forecasting developments for

a relatively short period of time, together with a series of overly conservative estimates to compensate for unforeseen events, the projections should provide a reasonable sense of reliability.

Therefore, if this was 2000 or 2005, there would be:

- ☐ A continued decrease in the dentist-to-population ratio.
- ☐ A decrease in the number of dentists.
- ☐ An increase in per capita expenditures for dental services.
- ☐ An increase in the income of dentists.

It would appear that you and your colleagues (including other pediatric dentists) should be doing quite well!

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STRESS ANALYSIS OF THREE FILLING TECHNIQUES

An important disadvantage of current dental resin composites is polymerization shrinkage. This shrinkage has clinical repercussions such as sensitivity, marginal discoloration, and secondary caries. The objective of this study was to compare three filling techniques in terms of the transient stresses induced at the resin composite/tooth interface during polymerization. The techniques were: bulk filling (B), three horizontal increments (HI), and three wedge increments (WI). A simple Class V cavity preparation was modeled in finite element analysis. Polymerization shrinkage was simulated by a thermal stress analogy, thereby causing 1 percent shrinkage due to an arbitrary coefficient of thermal expansion. Interface normal and shear stresses were calculated at nine steps during polymerization, proceeding from 0 percent to 100 percent volume of cured resin. The importance of the interface transient stresses was revealed by the finding that, in most cases, their peak values exceeded the final or residual stress. Also, the WI and B techniques consistently exhibited the highest and lowest maximum transient stresses, respectively. These results from the simple model of a Class V restoration suggest that bulk filling of light-cured resin composites should be used in restorations which are sufficiently shallow to be cured to their full depth. Winkler, M.M. *et al*: Finite element stress analysis of three filling techniques for class V light-cured composite restorations. J Dent Res, 75:1477–1483, July 1996.

REPORT

Moebius syndrome: Report of case

Kaw Jue Lin, DDS
Wei Nan Wang, DDS

In 1892, Moebius described bilateral congenital facial weakness, loss of abduction of the eyes, and associated deformity of the hand, later called Moebius syndrome.[1,2] The syndrome is uncommon. It includes the following features:

- ☐ Bilateral or unilateral, complete or incomplete muscular weakness of the face.
- ☐ Lateral rectus muscle palsy.
- ☐ Primary or secondary congenital deformities of the extremities (club foot, syndactyly or other digital deformities.
- ☐ Sometimes other involvement of the brachial musculature.
- ☐ Frequent involvement of other cranial nerves.
- ☐ Occasional anomalies of the ears, including defective pinna and/or deafness.
- ☐ Mental retardation in 10 percent of the patients (usually not severe).[2,3,4]

Henderson presented detailed analysis of the clinical features in sixty-one cases of Moebius syndrome: associated features comprise other cranial nerve palsies, predominant bulb; structural abnormalities of the ear; defects of brachial musculature; and mild mental retardation.[5] The syndrome involves other cranial nerves, most often the hypoglossal, and the most commonly associated skeletal anomaly is club foot.[6] Various authors have altered the criteria for diagnosis, accepting cases with weakness of one side of facial muscle, unilateral or no weakness of the eye muscles, and adding various deformities of the limbs (syndactyly, polydactyly, brachydactyly, agenesis of digits, or talipes equinovarus).[7] Association of Moebius syndrome with the absence of the pectoralis major muscle (the Poland anomaly) has also been reported.[8]

The etiology of this neurological defect remains obscure. Some authors feel that it has a genetic basis. Harrison and Parker thought that the Mendelian characteristics of a dominant gene were involved and, in some cases, a sex-linked gene might be the offended agent.[9,10] Becker and Lund presented one of the eleven families with Moebius syndrome described in the literature and postulated a recessive mode of inheritance.[11] Other authors, including Reed and Grant, state that heredity plays no role in Moebius syndrome.[5,10,12] Elsahy reported a case in which the mother took thalidomide during pregnancy.[10] Henderson reviewed sixty-one cases and could demonstrate no hereditary etiology.[5] The sexes appear to be equally affected.

The cause of most cases of Moebius syndrome is probably a transient ischemic/hypothic insult to the fetus.[13] It may be due to any event that interferes with uterine/fetal circulation, whether trauma, rupture of membranes, pharmacological effect, hyperthermia, or other as yet undefined events. Webster *et al* have shown that uterine artery occlusion, handling the uterine vessels, uterine handling, and hyperthermia are associated with bilateral brain stem ischemic lesions and limb deficiency in a rat animal model.[14,15] The association of Moebius syndrome with unilateral cerebellar hypoplasia is unusual, but consistent with a vascular disruption occurring in the basilar artery in its development.[16]

Treatment of the malformation aims at improving esthetics and function. There have been a few reported attempts to relieve the facial paralysis of Moebius syndrome. The most successful principle of treatment is the transfer of functioning striated muscle to support and animate the paralyzed face. When the paralysis spares the platysma muscles, strong consideration should be given to using this valuable true "mimetic" muscle for reanimation of the face and correction of the lower lip paralysis after trauma or surgery.[6]

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CASE REPORT

A premature male newborn (gestational age: thirty-six weeks, six days) with Moebius syndrome received consultation in the Pediatric Dental Department at TSGH for evaluation of a maxillofacial and oral condition on the 21st day after birth. The mother had neither a history of prenatal illness nor use of any medication, and nothing of special note in her maternal and family history. The baby was her first child, and was delivered after a natural spontaneous delivery. The birth weight was 2450 gm. O_2 inhalation was given after birth due to poor crying with frequent apnea; and the infant was admitted to the pediatric intensive care unit for respiratory distress.

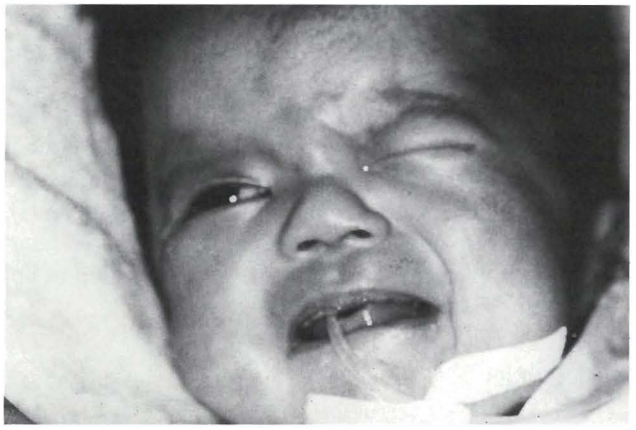

Figure 1. Asymmetric facial expression was found on the right face during crying. The baby received G-I feeding for poor sucking ability and dysphagia.

Figure 2. Polydactyly, right thumb.

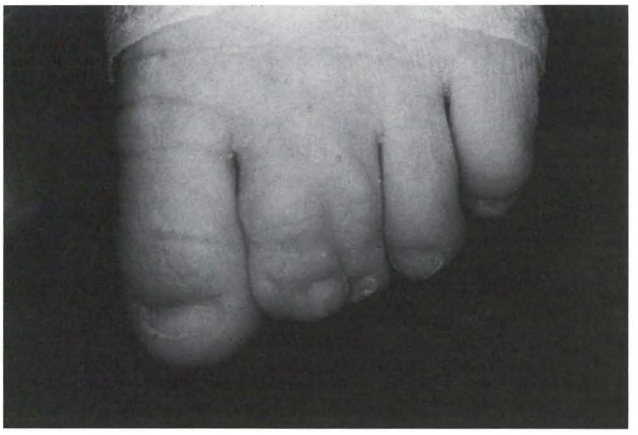

Figure 3. Syndactyly, left 2nd and 3rd toes.

The patient showed unusual facial features during crying: poor facial expression, no wrinkling of the forehead, inability to close the eyelids, and frequent drooling on the right side (Figure 1), an abnormal finger (polydactyly, right thumb) (Figure 2), and abnormal toes (syndactyly, left 2nd and 3rd toes) (Figure 3). Occasional cyanosis and difficulty in sucking were also found.

Physical examination revealed right facial paralysis with free movement of the eyeball, bilateral cryptorchism, and no abnormal intraoral characteristics. Magnetic resonance imaging of the brain revealed a small pons. The cardioechogram revealed patent ductus, arteriostenosis and coarctation of the aorta. The auditory brainstem reflex test was negative. He had suffered with dysphagia, dyslaria, and frequent apnea. The baby was diagnosed with Moebius syndrome, which may involve the cranial nerves VIII, IX, X, and XI. The baby died on the 41st day, of airway obstruction.

DISCUSSION

The question of a hypoplasia of the cranial nervous system versus a primary peripheral muscle defect as the site of the primary lesion has not been settled. One theory holds that the primary defect is an ectodermal dysplasia within the brain, involving the sixth and seventh cranial nerve nuclei. According to his school of thought, the peripheral palsies may be secondary to this nuclear defect.[17-19] Another theory is based on a primary defect in the neural crest mesenchyme, resulting in aplasia of the mimetic muscles of the face. Hypoplasia of the cranial nerve nuclei is believed by the latter school to be a secondary degeneration.[20,21] But most authors agree that it represents a dysplasia rather than a degenerative pro-

cess, while a loss of cells from motor nuclei has been demonstrated in some cases. Another study has suggested that the primary defect is in the muscle, and the victim of the Moebius syndrome is devoid of facial muscles in the upper three fourths of the face and that the facial nerve is usually intact.

The facial muscle paralysis can be localized in the portion of the motor pathway that is affected through the knowledge that muscles of the forehead and eyelid receive innervation from facial regions of motor areas of both cerebral cortices. But the musculature of the lower part of the face receives innervation mainly from the contralateral motor cortex. A lesion that involves one motor cortex will cause the paralysis of the lower facial muscles of the contralateral side. The muscles of the forehead and eyelids, however, will be unaffected bilaterally, which is described as central facial weakness. The lesions that may involve the nucleus of the facial area in the motor cortex or its upper motor neuron include a tumor, encephalitides, multiple sclerosis, or vascular lesions. If there is a total hemifacial paralysis, as in this case, including an inability to wrinkle the forehead or to close the eye, this would indicate a disease affecting the ipsilateral peripheral distribution of the facial nerve. The lesion could affect any part of the nerve along its pathway from the motor nucleus in the pons to the terminal branches. This is called peripheral facial paralysis.[24] Agenesis of the motor nucleus in the pons, meningitis, subarachnoid hemorrhage, neuritis, skull fractures, and tumors of the neck may cause this type of paralysis. Moebius syndrome fits into this category.[23,24]

A rare unilateral presentation of Moebius syndrome (as in this case) may be incorrectly diagnosed as Bell's palsy. The lack of ability to abduct either eye beyond the midline would be used to differentiate diagnosis of these two conditions. This baby, however, also had free eyeball movement. Hence, the other criteria, acquired or congenital, involvement of other cranial nerve or other musculoskeletal anomalies, may be considered.

A newborn baby with Moebius syndrome has inability to suck and take in adequate nutrition. Obviously, it is difficult to suck when a good perioral seal cannot be maintained. When using a nursing bottle for more than a year after birth, the baby could conceivably develop a severe type of "nursing bottle caries" more common in these patients.[2] Our case was fed with a nasogastric tube, due to poor sucking ability and dysphagia (Figure 1). As the child develops, the indistinct speech and mask-like face become a source of parental concern. Drooling is occasionally a major problem. The sixth nerve palsy may not be noticed, because the child will follow objects by turning his head, thus camouflaging the lateral rectus palsy. There are also limitations to talking without the ability to move the lips.

Speech is affected when one cannot move the lips to form certain sounds, like "m", "b", "f", or "p". If it is hard to communicate with others, he must depend on lip reading because of the associated hearing impairment.[8]

Adult patients with Moebius syndrome may also have problems with complete dentures, due to absent or reduced facial muscle control, which makes it very difficult to wear a complete denture.[2]

SUMMARY

This is a review of diagnosis, etiology and abnormalities of Moebius syndrome. The Moebius syndrome is rare and the cause is still unclear. It presents a case of a premature newborn baby with Moebius syndrome, showing unilateral facial nerve palsy, asymmetry of facial expression, inability to tightly close the right eyelids, asymmetry of the angles of the mouth with frequent drooling, poor sucking ability, dysphagia, extremity abnormalities and other cranial nerve involvement (VIII, IX, X, XI). He also had hypoplasia of the pons, a heart defect, and bilateral cryptorchism. The baby died of apnea on the 41st day after birth.

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CELLULAR-TELEPHONE CALLS AND MOTOR VEHICLE COLLISIONS

Our study indicates an association but not necessarily a causal relation between the use of cellular telephones while driving and a subsequent motor vehicle collision. For example, emotional stress may lead to both increased use of a cellular telephone and decreased driving ability. If so, individual calls may do nothing to alter the chances of a collision. In addition, our study did not include serious injuries; hence, we do not know how—or whether—cellular-telephone use is associated with motor vehicle fatalities. Finally, the data do not indicate that the drivers were at fault in the collisions; it may be that cellular telephones merely decrease a driver's ability to avoid a collision caused by someone else.

We caution against interpreting our data as showing that cellular telephones are harmful and that their use should be restricted. Even if a causal relation with motor vehicle collisions were to be established, drivers are vulnerable to other distractions that could offset the potential reductions in risk due to restricting the use of cellular telephones. Regulations would also mean reducing benefits; in Canada, for example, half a million calls to 911 emergency services are made from cellular telephones each year. Yet proposals for regulation are not unreasonable, since poor driving imposes risks on others. Public debate is needed, given that cellular telephones contribute to improvements in productivity, the quality of life, and peace of mind for more than 30 million people in North America alone.

The role of regulation is controversial, but the role of individual responsibility is clear. Drivers who use a cellular telephone are at increased risk for a motor vehicle collision and should consider road-safety precautions. For them as for all other drivers, these include abstaining from alcohol, avoiding excessive speed, and minimizing other distractions. Additional strategies might include refraining from placing or receiving unnecessary calls, interrupting telephone conversations if necessary, and keeping calls brief—particularly in hazardous driving situations. Physicians should also learn to recognize patients who are at risk for a collision and who may benefit from advice regarding safety. Even limited success in reducing risk may prevent some of the death, disability, and property damage related to motor vehicle collisions.

Redelmeier, D.A. and Tibshirami, R.J.: Association between cellular-telephone calls and motor vehicle collisions. N Engl J Med, 336:453–458, February 17, 1997.

ABSTRACTS

Cagidiaco, Maria Crysanti; Ferrari, Marco; Davidson, Carel: Comparison of in vivo and in vitro demineralized dentin with phosphoric and maleic acid. J Dent Child, 64:17-21, January-February 1997.

This article evaluated possible differences between dentin conditioned *in vivo* and *in vitro* with 10 percent maleic acid or with 36 percent phosphoric acid. Semispherical Class V cavities were prepared *in vivo* and *in vitro* at the cementum-enamel junction and were divided into four groups. After etching procedures, the *in vivo* samples were extracted and fixated in 10 percent buffered formaldehyde. Both the *in vivo* and *in vitro* samples were then fractured in two parts along their long axis, critical-point dried, and subsequently examined with SEM. Both the acids removed completely the smear layer and demineralized the dentin, leaving a layer of collagenous network. No morphological differences were found between *in vivo* and *in vitro* dentin samples.

Dentin; Collagen layer; Demineralized dental structures; Dental bonding systems; Demineralization *in vivo*; Demineralization *in vitro*

Kohli, Kavita; Houpt, Milton; Shey, Zia: Fluoride uptake by proximal surfaces from professionally applied fluorides: An in vitro study. J Dent Child, 64:28-31, January-February 1997.

This study was performed to examine the uptake of fluoride by mesial enamel surfaces of extracted teeth from different types of topical fluoride. Forty-eight extracted human molars and twelve premolars were randomly divided into four groups and mounted in twelve wax blocks with five teeth in proximal contact in each block. Each group was treated for four minutes with either 1.23 percent acidulated phosphofluoride conventional gel, thixotropic gel, or foam in lined trays, or 2 percent neutral sodium fluoride solution applied with a cotton tip applicator. Acid-etch biopsies were performed three times to indicate the amount of fluoride uptake at three different depths. The results demonstrated that there was a statistically significant increase in fluoride uptake in the surface layer with both gels and foam (1150, 1058 and 1120 ppm F), whereas there was an insignificant increase with the fluoride solution (27 ppm F). In the deepest layer, the thixotropic fluoride produced the greatest fluoride uptake (919 ppm F) in comparison with the uptake from conventional gel (383 ppm F), foam (297 ppm F), or solution (118 ppm F).

Topical fluoride; Fluoride uptake

Fritz, Ulrike; García-Godoy, Franklin; Finger, Werner J.: Enamel and dentin bond strength and bonding mechanism to dentin of gluma CPS to primary teeth. J Dent Child, 64: 32-38, January-February 1997.

This study evaluated the shear bond strength to enamel and dentin and bonding mechanism to dentin of Gluma CPS bonding system to primary teeth enamel and dentin. Pekafill hybrid composite was used. Ten specimens were immersed in deionized water at 37°C for twenty-four hours and ten other specimens were subjected to a thermocycling procedure (2000 cycles between 5° and 55°C, dwell time fifteen seconds). After water storage or thermocycling, the bonded cylinders were sheared and the bond strength calculated. After debonding, the failure sites of all samples were evaluated microscopically. The data were analyzed with an ANOVA and Student-Newman-Keuls test. In ten other primary molars, the buccal surfaces were ground flat on wet SiC papers to expose peripheral dentin. Cylindrical buttjoint cavities were prepared with a cylindrical diamond bur. The cavities were treated with Gluma CPS and filled with Pekafill as described above. After fifteen-minute water storage, filling excess was removed and marginal integrity and bonding mechanism of five restorations was examined with a light microscope. Five other restorations were evaluated after storage in water at 37°C for twenty-four hours. Maximal gap widths and hybrid layer thickness were determined. The results showed that thermocycling did not affect the shear bond strength to enamel or dentin. There was a statistically significant difference ($p<0.05$) between the shear bond strength values obtained in enamel and dentin. The maximal gap width was not significantly different between specimens that were stored in water for fifteen minutes or twenty-four hours. The average hybrid layer thickness was 11.7 $\pm$ 1.1 µm. All specimens revealed a resin cohesive failure very close to enamel or dentin surface. The etch patterns produced on the abraded enamel surface were uniform and deeper as compared to the sample that was pumiced. This was also confirmed in the epoxy replicas.

Bond strength; Primary teeth; Gluma CPS; Pekafill hybrid composite

Waldman, Barry H.: Evolving "faces" of the next generations of pediatric patients. J Dent Child, 64: 39-42, January-February 1997.

Bureau of the Census projections for the next decades indicated dramatic increases in the numbers of minority children in our population and actual decreases in the numbers of non-Hispanic white children.

A summary of these projections is provided.

Minority children; Population growth

Rasmussen, Per and Kotsaki, Angeliki: Inherited primary failure of eruption in the primary dentition: Report of five cases. J Dent Child, 64:43-47, January-February 1997.

Unerupted primary teeth are a rare event and in most cases involve the sec-

ond primary molars. The impaction may be primary, meaning that the teeth never have been erupted (also called primary failure of eruption), or it may be secondary, meaning that the teeth after eruption are reimpacted. The present report describes five cases (three boys and two girls) with primary failure of eruption of totally fourteen second primary molars. None of these teeth ever erupted and were removed surgically at an age of 10-11 yrs. Additionally, in all five cases the eruption of the entire permanent dentition was severely retarded. Consequences of the nonerupting primary molars put a more functional strain, with severe attrition, on the remaining teeth. The unerupted primary molars also seemed to prevent the development of the corresponding premolars. In addition the impacted primary molars were in some cases a threat to the eruption of first permanent molars. The etiology of the present primary failure of eruption seems to be inheritance, and the mode of transmission is judged to be autosomal dominant.
Primary dentition; Primary failure of eruption; Impaction

Veerkamp, J.S.J.; Porcelijn, T.; Gruythuysen, R.J.M.: Intravenous sedation for outpatient treatment of child dental patients: An exploratory study. J Dent Child, 64:48-54, January-February 1997.
Dental treatment of very young children (toddlers) is known to be difficult. One of the new pharmacological strategies is intravenous sedation with the modern drug Propofol (2,6 diisopropophenol, Diprivan®). This article briefly describes the state of the art and a pilot study of dental treatment of 19 toddlers under intravenous sedation. The treatments were performed by a dentist and an anesthetist. Results suggest that sedation is difficult to achieve in young children. Recommendations for further research are made.
Anesthesia; Sedation; Behavior; Children

Siener, Karen; Rothman, David; Farrar, Jeff: Soft drink logos on baby bottles: Do they influence what is fed to children? J Dent Child, 64: 55-60, January-February 1997.
Baby bottles with popular soda pop and soft drink logos are on market shelves. A descriptive study was conducted to determine their prevalence among families and to determine whether the logos could be influencing what families put in baby bottles. A convenience sample of 314 mothers (and grandmothers if they were primary caregivers) with children using baby bottles was interviewed in three California counties. The results were analyzed for significance, using the chi-square test for independence.

The ethnicities and educational levels of the sample population matched the distribution of the State. Overall, 31 percent of the children drank either soda pop or Kool-Aid from baby bottles. Forty-six percent of the respondents owned a baby bottle with a soda pop logo and 17 percent owned a bottle with a Kool-Aid logo. Families who owned bottles with popular beverage logos were four times more likely to give children the respective beverage in bottles than families without "logo bottles." Populations most likely to drink these beverages were those in the black and Hispanic ethnic groups, in the youngest age-group (15-20 years of age), and those without a high school diploma.

Health professionals are concerned that the logos could cause an increase in children's consumption of sweetened beverages in baby bottles and consequently an increase in Baby Bottle Tooth Decay and nutritional problems.
Baby-bottle tooth decay; Logos on bottles

Lin, Kaw Jue and Wang, Wei Nan: Moebius syndrome: Report of case. J Dent Child, 64:64-67, January-February 1997.
The Moebius syndrome is a rare case which consists of congenital facial nerve and other cranial nerve palsies, most often of the sixth cranial nerve (abducent nerve), and or musculoskeletal abnormalities. The etiology of this syndrome remains obscure, and may be correlated with transient ischemic/hypothic insult to the fetus. A premature newborn with Moebius syndrome is presented, who had unilateral facial nerve palsy. Abnormal toes and fingers, brain hypoplasia, heart defect, and bilateral cryptorchisms were also found. Other cranial nerves (VIII, IX, X, XI) may be involved. The orofacial conditions were asymmetry of facial expression, inability to close the right eyelids tightly, frequently weakness and drooling of the right corner of the mouth, and poor sucking ability. He died on the 41st day after birth from apnea.
Moebius syndrome; Facial palsy; Cryptorchism; Polydactyly; Syndactyly