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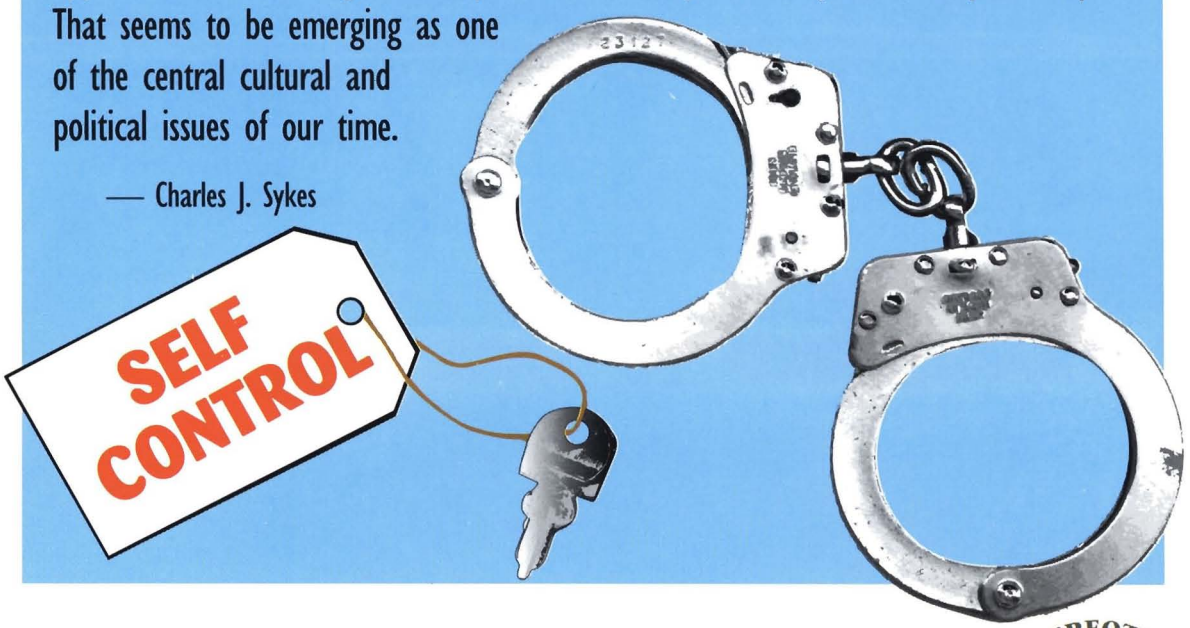
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN
JOURNAL OF DENTISTRY FOR CHILDREN
JANUARY– FEBRUARY 1994

In their monumental study of crime, Wilson and Herrnstein recognize the "complexity and mystery of human behavior" and argue that the important consequences of social policy may not be the direct effects they have but rather the example they set for society as a whole. By treating people "as if" they are personally responsible, society is "reaffirming the moral order of society and reminding people of what constitutes right conduct, in hopes that this reaffirmation and reminder will help people, especially in families, teach each other about virtue."

This principle extends to the way society allocates economic benefits, the way judges and juries dispose of legal claims, as well as to employment practices and educational policies. In each instance the question that must be asked is: "Do we appear to be rewarding the acceptance or the rejection of personal responsibility?" That seems to be emerging as one of the central cultural and political issues of our time.

— Charles J. Sykes



PERHAPS THE LESSON HERE IS ABOUT THE LIMITS OF GOVERNMENT'S POWER TO CHANGE BEHAVIOR, HOWEVER WORTHY THE REASON.

—*Joan Beck*

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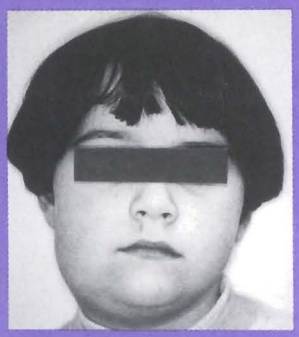
The advocacy of character is considered by some to be a simplistic way to approach complex problems. But the complexity of these problems may be one of the strongest arguments for a politics of personal responsibility.

CLINIC

BEHAVIOR

62



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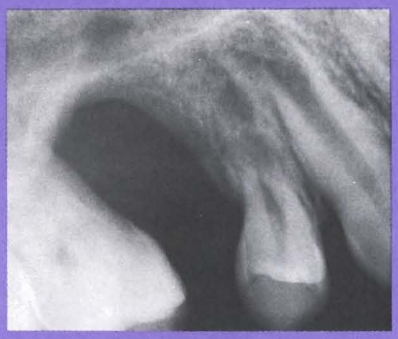

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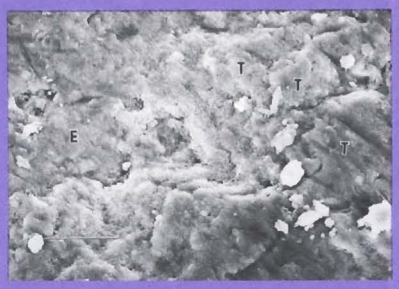

For the busy reader

Properties related to strength and resistance to abrasion of VariGlass VLC, Fuji II L.C., Ketac-Silver, and Z-100 composite resin—page 17

Light-cured Type II glass ionomers that offer improved physical properties for use as a final restorative material are new available. These new materials appear to be particularly beneficial in treating children. The authors evaluate some of the physical properties of two commercially available light-cured glass ionomer restorative materials, Variglass VLC and Fuji II L.C., and compare the results with Ketac-Silver, and Z-100 composite resin.

Requests for reprints should be directed to Dr. David R. Avery, Department of Pediatric Dentistry, 1121 West Michigan Street, Indianapolis, IN 46202.

Role of the acid-etch technique in remineralization of caries-like lesions of enamel: A polarized light and scanning electron microscopic study—page 21

It is possible that acid-etching of enamel lesions before exposure to calcifying fluids may facilitate remineralization. The purpose of this study was to determine the effect of acid-etching on remineralization of caries-like lesions of enamel with synthetic calcifying fluids containing relatively low and high levels of calcium using polarized light and scanning electron microscopic techniques.

Requests for reprints should be directed to Dr. Catherine M. Flaitz, Division of Oral Pathology, Department of Oral Diagnostic Sciences, 6516 John Freeman Avenue, P.O. Box 20068, Dental Branch, University of Texas Health Science Center, Houston, TX 77225-0068.

Evaluation of occlusal marginal adaptation of class II resin composite inlays— page 29

The authors observed the occlusal marginal adaptation of indirect Class II resin composite inlays, using a photographic technique. In this study the frequency of restorations with an *excellent* marginal adaptation along the occlusal outline is low compared to the value found in the study on marginal adaptation of direct Class II resin composite restorations.

Requests for reprints should be directed to Dr. C.M. Kreulen, Department of Pediatric Dentistry, Academic Centre for Dentistry Amsterdam (ACTA), Louwesweg 1, 1066 EA Amsterdam, The Netherlands.

Children who have hypoplastic enamel defects of primary incisors are not at increased risk of learning problem syndromes—page 35

Enamel defects of primary teeth have been seen in children with a wide variety of neurologic disturbances, raising the possibility that what disturbs tooth development also disturbs brain development. The authors comment on the plausibility of this observation. In the study the authors found no statistically significant association between hypoplastic enamel and seven learning-problem syndromes.

Requests for reprints should be directed to Dr. Alan Leviton, Children's Hospital, 300 Longwood Avenue, Boston, MA 02115.

Dental management of the child with developmental dyslexia—page 39

Dyslexia affects an estimated 3 percent to 10 percent of school-age children in the United States. Children with cleft palate are three times more likely to suffer dyslexia than their peers. Oral abnormalities in these patients include a short upper lip and wide mouth, steep palatal vault, fissured tongue, geographic tongue, aberrant frenula, and crowded or malformed teeth.

Request for reprints should be directed to Dr. Arthur H. Friedlander, Director: Quality Assurance, Hospital Dental Service, UCLA Medical Center, Los Angeles, CA 90024.

Effect of a preventive approach for the treatment of nursing bottle caries— page 46

The authors wished to determine whether a preventive methodology consisting of instructions on the use of

the bottle and daily fluoride treatment could arrest the progression of nursing-bottle caries. After three months the study was terminated for lack of compliance by the children's caretakers and because of an increase in the number of caries lesions. An interesting dissertation on caretaker cooperation.

Requests for reprints should be directed to Dr. Norman Tinanoff, Professor, Department of Pediatric Dentistry and Orthodontics, School of Dental Medicine, University of Connecticut Health Center, Farmington, CT 06030-1610.

Updating the changing number and distribution of pediatric dentists: 1982-1991—page 50

The author discusses the changes that occurred in the number of pediatric dentists and in the ratio of number of pediatric dentists to number of children, in the period 1982-1991. The regions are discussed by region and state. The author calls for an ongoing forecasts for various dental specialties, if the profession is to reach traditionally underserved populations.

Requests for reprints should be directed to Dr. H. Barry Waldman, Professor and Chairman, Department of Dental Health, School of Dental Medicine, State University of New York at Stony Brook, Stony Brook, NY 11794-8715.

Social security and providing for our children—page 57

At the end of 1991, more than 3.2 million children were receiving benefits under the OASDI program. In 1991 $10.6 billion in benefits were provided to children. In 1990 SSI dispensed $1.6 billion in benefits to blind and /or disabled children, an average of $404 per month. Pediatric dentists should be conversant with these programs.

Requests for reprints should be directed to Dr. H. Barry Waldman, Professor and Chairman, Department of Dental Health, School of Dental Medicine, State University of New York at Stony Brook, Stony Brook, NY 11794-8715.

Cervical adenitis: Report of two cases—page 62

This disorder can mimic an odontogenic infection. The etiology and clinical manifestations of cervical adenitis vary considerably. The symptoms are often preceded by a recent history of an upper respiratory infection. The dentist is often the first person to see the patient. A thorough examination will confirm or deny a dental etiology.

Requests for reprints should be directed to Dr. Brian J. Sanders, Assistant Professor of Pediatric Dentistry, Indiana University Hospitals, Department of Pediatric Dentistry, 702 Barnhill Drive, Room 1110, Indiana University Medical Center, Indianapolis, IN 46202-5200.

Pyogenic granuloma as a cause of bone loss in a twelve-year-old child: Report of case—page 65

This lesion can give an erroneous clinical impression of a malignant tumor. It is, however, well-circumscribed and benign, arising from connective tissue of skin or mucous membrane. The exact cause is unknown. These lesions grow rapidly and have the potential to destroy underlying bone. Treatment of choice is excisional biopsy.

Requests for reprints should be directed to Dr. Elizabeth D. Goodman-Topper, Department of Pedodontics, Hadassah Faculty of Dental Medicine, Jerusalem, POB 1172, Israel.

Lowe's syndrome: Review of literature and report of case—page 68

Because of their medical condition, these patients are very difficult to treat successfully. Oral conditions include crowding, delayed eruption, over-retained primary teeth, constricted palate, and taurodontism. The authors describe their treatment of a patient ten years old.

Requests for reprints should be directed to Dr. George P. Thomas, Howard University, College of Dentistry, 600 W Street, N.W., Washington, D.C. 20059.

Properties related to strength and resistance to abrasion of VariGlass VLC, Fuji II L.C., Ketac-Silver, and Z-100 composite resin

Sompit Dhummarungrong, DDS
B. Keith Moore, PhD
David R. Avery, DDS, MSD

In restorative dentistry, selecting the proper material is an important factor for clinical success. The properties of each specific material provide advantages and disadvantages. One advantage of glass ionomers is the release of fluoride, and as a result, less caries-susceptibility on the margins of the restorations.[1-8] Another advantage is its adherence to tooth structure.[9,10] The major disadvantages of conventional glass ionomers are short working time and relatively long setting time in the mouth. Composite resins offer easier clinical manipulation and superior esthetics when compared to glass ionomers.[11]

Research and development efforts for glass ionomers are currently directed toward eliminating the disadvantages of working time and setting time, by incorporating light-curing technology, while preserving the important benefits of fluoride release and dentine bonding.[10,12,13] The first development of a resin-modified glass ionomer material was a light-cured glass ionomer liner/base. Recently, light-cured Type ll glass ionomers that offer improved physical properties for use as a final restorative material are available. This new development of glass ionomers is beneficial in restorative treatment, especially in children with primary teeth when simple-to-use, fast setting, strong and fluoride releasing materials are most helpful.

The purpose of this study was to evaluate some of the physical properties of two commercially available light-cured glass ionomer restorative materials, VariGlass VLC and Fuji ll L.C., and to compare the results with Ketac-Silver, and Z-100 composite resin.

MATERIALS AND METHODS

The materials studied are shown in Table 1 (specifications received from the manufacturers).

VariGlass VLC. Fuji ll L.C.

VariGlass VLC and Fuji ll L.C. are visible light-cured glass ionomers with two components, powder and liquid. The powder and liquid are mixed in the proper ratio according to the manufacturers' instructions and cured with a visible curing light. Both are recommended for the following clinical uses:

- ☐ Lining and basing application.
- ☐ Restorative material.
- ☐ Core build-up.

The authors are indebted to the manufacturers for their cooperation in providing sufficient quantities of each product to allow us to conduct this *in vitro* evaluation.

Dr. Dhummarungrong is on the faculty of the Department of Pediatric Dentistry, Faculty of Dentistry, Khon Kaen University, Khon Kaen, Thailand. During this study, she was a research scholar at the School of Dentistry, Indiana University, Indianapolis, Indiana. Dr. Moore is a professor of Dental Materials, and Dr. Avery is a professor of Pediatric Dentistry, the School of Dentistry, Indiana University, Indianapolis, Indiana.

Table 1. ☐ The materials studied.

Product	Manufacturer	Batch No.
VariGlass VLC	L.D. Caulk	Powder-9205225-9206022 Liquid-920608
Fuji II L.C.	G.C.America	260223
Z-100 Composite	3 M	70-2010-0165-1/No. 5904A3.5
Ketac-Silver	ESPE-Premier	008A20 V315 004 V221

As restorative materials, they are indicated for use in non-occlusal stress-bearing restorations such as Class lll and V situations.

Ketac-Silver

Ketac-Silver is a capsulated poly-maleinate glass ionomer core and restorative cement, consisting of pure silver particles chemically bonded (sintered) into a glass ionomer matrix. It has been recommended for core build-ups, bases, restoring primary molars, conservative Class I and ll restorations, Class V restorations, and for retrograding fillings.

Z-100 composite resin

Z-100 composite is a visible light-activated, radiopaque restorative material. It has a maximum particle size of 4.5 microns. The filler is zirconia/ silica. Z-100 is packaged in traditional syringes and single dose capsules. It is designed for use in both anterior and posterior restorations.

Preparation of specimens

Specimens were prepared in stainless steel molds, with the exception of the specimens in the toothbrushing test, which were prepared in glass tubes to provide a smoother surface for testing. Twenty-two cylindrical specimens (6 mm in diameter, 12 mm in length) of each material were prepared. Ten were used for the compressive strength tests, while twelve were needed for the toothbrushing tests. An additional ten cylindrical specimens (6 mm in diameter, 3 mm in length) of each material were made for the diametral tensile strength tests. Finally, ten rectangular specimens (16x2x4 mm) were made for the transverse strength tests.

Ketac-Silver, in an encapsulated form, was mixed in an amalgamator (ESPE CAPMIX®) at high speed for ten seconds. The mixed cement was condensed in the mold by using the syringe that was supplied by the manufacturer. Three minutes after start of mixing, the mold was transferred to an oven, which was maintained at a temperature of 37 ± 1°C and relative humidity of at least 30 percent for 60 ± 5 min (according to ADA Specification No. 66). For VariGlass VLC and Fuji ll L.C., the powder/liquid was used in the recommended restorative ratio, mixed according to respective manufacturer's instruction, and placed in the mold. The materials were cured with visible curing light (COE-Lite, Model 4000) for 60 seconds. Z-100, which is packaged in a traditional syringe, was placed in the mold and cured with visible curing light (COE-Lite, Model 4000) for 60 seconds. All specimens were prepared at room temperature (22xC) and stored for fourteen days in distilled water at 37°C before testing.

Tests

- ☐ Compressive and diametral tensile strength tests. The specimens were tested with an Instron Universal Testing Machine* at a cross-head speed of 1.0 mm/min. The strength was calculated from the following formulas: Compressive strength = $4F/\pi\ d^2$; Diameter tensile strength = $2F/\pi\ dL$; F = maximum applied load (N); d = diameter of the specimen (mm); L = length of the specimen (mm).
- ☐ Transverse strength test. The samples were measured by using a three-point loading apparatus in an Instron Universal Testing Machine* at a cross-head speed of 0.5 mm/min. The strengths were calculated using the following formula: Transverse strength = $3WL/2bd^2$; W = maximum load before fracture (N); L = distance between the support (10 mm); b = width of the specimen (mm); d = thickness of the specimen (mm).
- ☐ Toothbrushing test.

The specimens were evaluated for their resistance to abrasion with a motor-driven toothbrushing machine. They were subjected to 20,000 brush strokes (two hours) with an abrasive slurry of 1:1 calcium carbonate (15 gm) and water (15 cc). The brushes had flat end nylon bristles in four rows with fifty tufts. The bristle diameter was 0.012 inch (0.3 mm). The samples were weighed before and after the testing period. Wear was calculated as weight loss and volume loss. The volume loss was calculated from the weight loss and the known density of the materials.

The data were analyzed by one-way analysis of var-

*Instron Universal Testing Machine, Instron Corporation, Cantor, MA 02021.

iance for detection of significant differences, followed by Newman-Keuls multiple comparison tests for pairwise comparisons.

RESULTS

Physical properties of each material are summarized in Table 2. The compressive, diametral tensile and transverse strengths of Z-100 were the highest. Among glass ionomers, Ketac-Silver achieved a significantly higher compressive strength than the others, and Fuji ll L.C. was significantly higher than VariGlass VLC ($p<0.01$). But for diametral and transverse strengths, Ketac-Silver was the lowest, Fuji ll L.C. had a significantly higher diametral tensile and transverse strength than VariGlass VLC ($p<0.01$).

Results of the toothbrushing tests are shown in Table 3. The greatest abrasion resistance was exhibited by VariGlass VLC, which was significantly higher than the other materials ($p<0.01$), followed by Z-100, Fuji ll L.C. and Ketac-Silver, respectively. Fuji ll L.C. and Ketac-Silver were not significantly different, when comparing the volume losses by the toothbrushing tests ($p<0.01$).

Table 2. ☐ Strength properties of each material.

	Compressive (MPa)		Diametral Tensile (MPa)		Tranverse (MPa)	
Materials	Mean	SD	Mean	SD	Mean	SD
VariGlass VLC	111.9	11.5	16.4	1.9	41.5	5.8
Fuji II L.C.	130.8	7.0	23.8	1.9	57.5	8.2
Z-100 Composite	248.7	33.2	44.6	12.7	139.4	20.5
Ketac-Silver	158.3	12.0	9.3	2.2	31.8	2.8

All are significantly differently at $p<0.01$.

Table 3. ☐ Abrasion resistance of each material.

	Weight loss (mg)		Volume loss (mm^3)	
Material	Mean	SD	Mean	SD
VariGlass VLC	13.80	3.18	6.62	1.53
Z-100 Composite	24.40	5.76	11.71	2.76
Fuji II L.C.*	32.59	5.67	15.65*	2.72
Ketac-Silver*	52.33	6.38	16.74*	2.04

* Fuji II L. C. and Ketac-Silver are not significantly different in volume loss ($p<0.01$). The others are significantly different.

DISCUSSION

This study supports the use of VariGlass VLC and Fuji ll L.C. in Class lll and V situations. They have higher diametral tensile and transverse strengths and better resistance to toothbrush abrasion than Ketac-Silver, which has been studied, used, and widely accepted.[14-17] Their compressive strengths are lower., however, than Ketac-Silver and Z-100. These results the reason that both VariGlass VLC and Fuji II L.C. are probably not indicated for stress-bearing restorations that need more compressive strength.

Interestingly, VariGlass VLC exhibits the greatest resistance to abrasion in the toothbrushing test. One possible explanation may be that its filler particles are smaller and harder than the other materials.[18,19] Although the manufacturer refers to VariGlass VLC as a light-cured glass ionomer, the mixed powder and liquid will not harden unless exposed to a curing light. This would indicate that the conventional acid-base curing reaction of a glass ionomer plays a limited role in the development of properties for VariGlass VLC and that it is primarily a resin-based system with some glass ionomer as a filler. It appears that VariGlass VLC is indicated for use as a filling material in eroded areas.

Fuji II L.C. has less abrasion resistance than VariGlass VLC, but compared to Ketac-Silver, no difference in

In selecting a material, the clinician must first determine the requirements of a particular restoration.

resistance to abrasion. Fuji II L.C. should be an acceptable alternative filling material, therefore, in eroded areas. Clinically, both VariGlass VLC and Fuji II L.C. provide more working time and set faster than Ketac-Silver and conventional glass ionomers.

No current restorative material is ideal. For clinical success, dentists should know the properties of materials, choose accordingly, and manipulate them properly. The newly developed Type II visible light-cured glass ionomers, VariGlass VLC and Fuji II L.C. have several desirable properties, including light-curing capability, lower water-sensitivity during the early setting period, fluoride release, and bonding to tooth structure.[20] Further research is indicated, however, for the development of additional improvements in esthetic restorative materials.

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SCREENING FOR CARIES DETECTION IN LOW-RISK CHILDREN

For many years, conventional radiography has been considered superior to clinical examination in the detection of approximal caries. With respect to occlusal caries detection, the reverse opinion has been predominant, and there has been a tradition of using a clinical examination alone for fissure caries diagnosis [King and Shaw, 1979; Weerheijm et al., 1989]. The current changes in behaviour of occlusal lesions, possibly due to the increased use of topical fluorides, seem, however, to have reduced the visual detection of such lesions [Sawle and Andlaw, 1988; Creanor et al., 1990]. In the present study, in which no true diagnosis was available, the accuracy of the diagnostic methods for detection of occlusal and approximal caries could not be evaluated. However, recent investigations using histological validation have demonstrated that conventional radiography performed more accurately than did visual inspection for the detection of dentinal occlusal lesions in non-cavitated molars and that digital radiographic techniques, particularly with the ability for contrast enhancement, performed as accurately or even better than film radiography [Wenzel et al., 1991a; Wenzel and Fejerskov, 1992].

Hintze, H.: Screening with conventional and digital bite-wing radiography compared to clinical examination alone for caries detection in low-risk children. Caries Res, 27:499-504, November-December 1993.

Role of the acid-etch technique in remineralization of caries-like lesions of enamel: A polarized light and scanning electron microscopic study

Catherine M. Flaitz, DDS, MS
M. John Hicks, DDS, MS, PhD, MD

Caries formation in enamel is a dynamic process with periods of demineralization by acidic byproducts from dental plaque interspersed with periods of remineralization by oral fluids containing various mineral ions, fluoride and salivary proteins.[1-5] Demineralization of enamel results in dissolution of mineral components from the subsurface enamel into acidic solutions and release into the oral environment. A portion of the dissolved mineral precipitates into the outer surface layer of enamel and results in a lesion composed of an intact, relatively unaffected surface zone with a subsurface region of considerable demineralization, the body of the lesion. With the white spot lesion of enamel, the surface zone is characterized by a pore volume varying from 1 to 5 percent and a surface topography that is quite similar to that for sound enamel.[1-3] Despite the fact that the surface zone has a pore volume that is ten to fifty times greater than that for sound enamel (pore volume 0.1 percent), adjacent areas of sound enamel and surface zone are indistinguishable by qualitative polarized light and scanning electron microscopic techniques.[5-8] These characteristics of the surface zone are quite remarkable when one considers that the underlying body of the lesion has a pore volume that ranges from a minimum of 5 percent at the periphery of the lesion to in excess of 25 percent at the lesion's center.[1-3,5]

Perhaps the intact, relatively unaffected surface zone may play a role in the difficulty involved with rapid remineralization of enamel caries. Remineralization with synthetic calcifying fluids, synthetic saliva and oral fluids both *in vitro* and *in vivo* may be accomplished; this procedure requires, however, relatively lengthy and numerous treatments.[4,9-13] While the intact surface zone may impede progression of the lesion and surface cavitation, remineralization of the lesion may also be hindered. Previous laboratory studies have shown that acid-etching of enamel caries results in minimal surface loss, typical etching patterns and increased surface porosity, while maintaining an intact surface zone.[6,7,14,15] It is possible that acid-etching of enamel lesions before exposure to calcifying fluids may facilitate remineralization.

The purpose of this *in vitro* study was to determine the effect of acid-etching on remineralization of caries-like lesions of enamel with synthetic calcifying fluids containing relatively low (1mM calcium) and high (3mM calcium) levels of calcium using polarized light and scanning electron microscopic techniques.

MATERIALS AND METHODS

A total of twenty caries-free human molar teeth were selected for this *in vitro* study. Following a fluoride-

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free prophylaxis, the specimens were varnished with an acid-resistant varnish, except for two 1x4mm windows of sound enamel on both the buccal and lingual surfaces. Caries-like lesions were created using a dialyzed–reconstituted, acidified gelatin gel containing 1.0mM calcium, 0.6mM phosphate, and 0.05mM fluoride at pH 4.75 ± 0.02.[16] Following a 12-week-exposure period, a single central longitudinal section (100 to 125μm in thickness) was taken from each specimen to serve as control lesions before treatment with calcifying fluids. The specimens were sectioned into quarters, longitudinally through the midpoint of the buccal/lingual surfaces and transversely through the midpoint of the mesial/distal surfaces. Each quarter from a single tooth was assigned to one of four treatment groups:

- ☐ 1mM calcium remineralization group (mesiobuccal quarters).
- ☐ Etched, 1mM calcium remineralization group (distobuccal quarters.
- ☐ 3mM calcium remineralization group (mesiolingual quarters).
- ☐ Etched, 3mM calcium remineralization group (distolingual quarters).

Following sectioning into tooth quarters, acid-resistant varnish was applied to the cut surfaces and adjacent to the exposed windows. The lesions in each group were rinsed in deionized, distilled water and a toothbrush prophylaxis with fluoride-free pumice was performed to remove any adherent gel. The lesions in the acid-etch group were then exposed to 30 percent phosphoric acid for a 30-second period. The etched surfaces were then rinsed with an air-water spray for 15 seconds. Synthetic calcifying fluids with calcium concentrations of 1mM and 3mM were prepared freshly from hydroxyapatite (calcium/phosphate ratio of 1.63).[17] Sodium chloride (200mM) was used to adjust the background ionic strength of the solution. The pH was adjusted to 7.0 using a potassium hydroxide solution. Fluoride at a concentration of 0.05mM (1ppm) was added to the calcifying fluids. The lesions in the remineralization groups were exposed to a total of ten separate, 60-second treatment periods with the appropriate calcifying fluid. Each treatment period was followed by a 30-minute, agitated rinse with deionized, distilled water to remove loosely adherent mineral reaction products. At each treatment period, a 2ml fresh aliquot of calcifying fluid was utilized with each specimen. Following the last treatment, the specimens were rinsed in deionized, distilled water for 30-minutes. Longitudinal sections (100 to 125μm in thickness) were prepared from the tooth quarters for polarized light study. The sections were imbibed with water and photomicrographs were taken. The photomicrographs were projected onto a computer-interfaced, digitized tablet and five measurements were made along the inner front of the surface zone and body of the lesion in order to determine mean depths. Depth measurements were made by drawing a traverse perpendicular to the enamel surface and to the point along the inner aspect of the surface zone or body of the lesion where pseudoisotropy occurred (transition from positive to negative birefringence). A total of forty paired lesions were available for statistical analyses in each of the four treatment groups (two lesion windows/tooth quarter) and two control groups (two lesion windows on each of the buccal and lingual surfaces for each control section). The mean depths were analyzed among groups using an ANOVA and a multiple comparisons test (Duncan Multiple Range) for a paired design. The surface topography of the lesions in the four treatment groups (twenty lesions/treatment group) were evaluated using standard scanning electron microscopic techniques.

RESULTS

Remineralization with 1mM and 3mM calcifying fluids with or without previous acid-etching of the lesions resulted in significant differences in both surface zone and body of the lesion depths, when compared with paired control lesions (Table, Figures 1 and 2). With the low calcium calcifying fluid group (1mM calcium), the body of the lesion depth was reduced by 12 percent following remineralization when compared with paired control lesions ($p<0.05$). When the lesion had been acid-etched before exposure to the calcifying fluid, the body of the lesion depth was approximately 25 percent less than that for the remineralized only lesions ($p<0.05$)

Table ☐ Effect of acid-etching on remineralization of caries-like lesions of enamel.

	Surface zone depth (mean ± sd)	Body of lesion depth (mean ± sd)
Low calcium calcifying fluid (1mM Calcium)		
Control lesions (n = 40)	18 ± 5μm^{A,B}	179 ± 18μm^{D,E}
Remineralized lesions (n = 40)	24 ± 6μm^{B,C}	157 ± 14μm^{D,F,L}
Etched, remineralized lesions (n = 40)	33 ± 5μm^{A,C}	118 ± 16μm^{E,F,M}
High calcium calcifying fluid (3mM Calcium)		
Control lesions (n = 40)	21 ± 6μm^{G,H}	188 ± 21μm^{J}
Remineralized lesions (n = 40)	29 ± 4μm^{G,I}	171 ± 17μm^{K,L}
Etched, remineralized lesions (n = 40)	39 ± 7μm^{H,I}	143 ± 14μm^{J,K,M}

Means with same letters are significantly different at $p<0.05$.

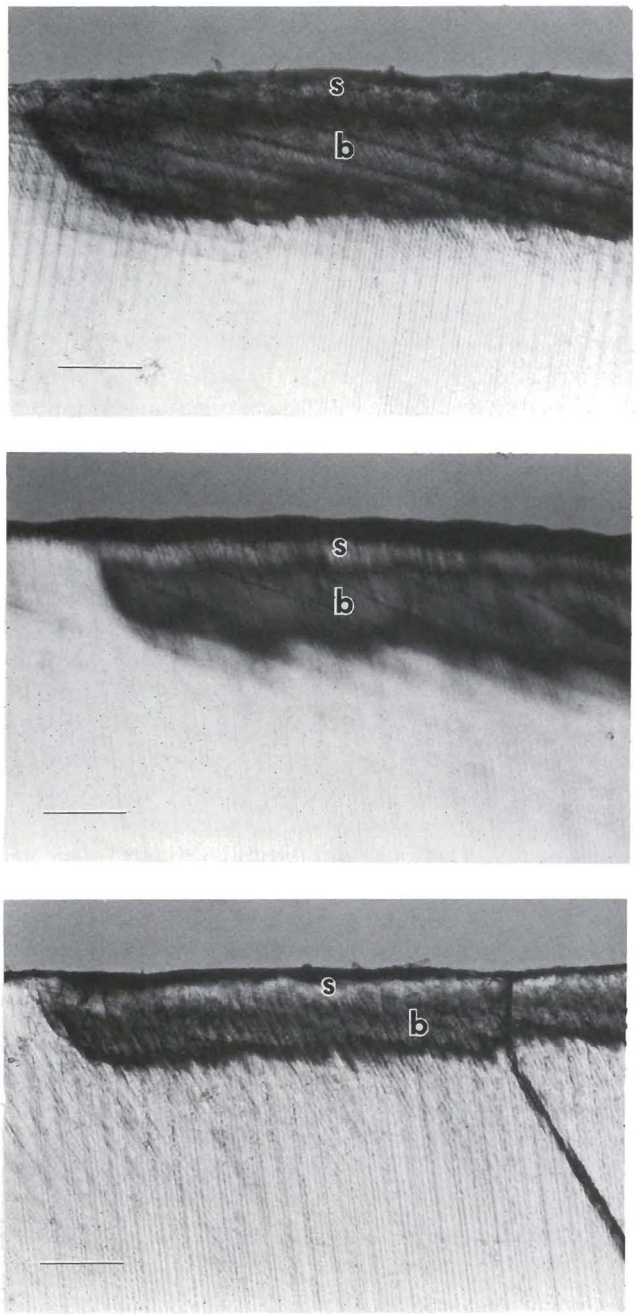

Figure 1. Low Calcium Calcifying Fluid Group (1mM Calcium). A. (Top) Representative Control Lesion with a mean body of the lesion depth of 172 μm and a mean surface zone depth of 26 μm. B. Representative Remineralized Lesion with a mean body of the lesion depth of 148 μm and a mean surface zone depth of 26 μm. C. Representative Etched, Reminalized Lesion with a mean body of the lesion depth of 121 μm and a mean surface zone depth of 30 μm. (Water imbibition, Polarized Light; S = Surface Zone; B = Body of Lesion; Space Bar = 100 μm)

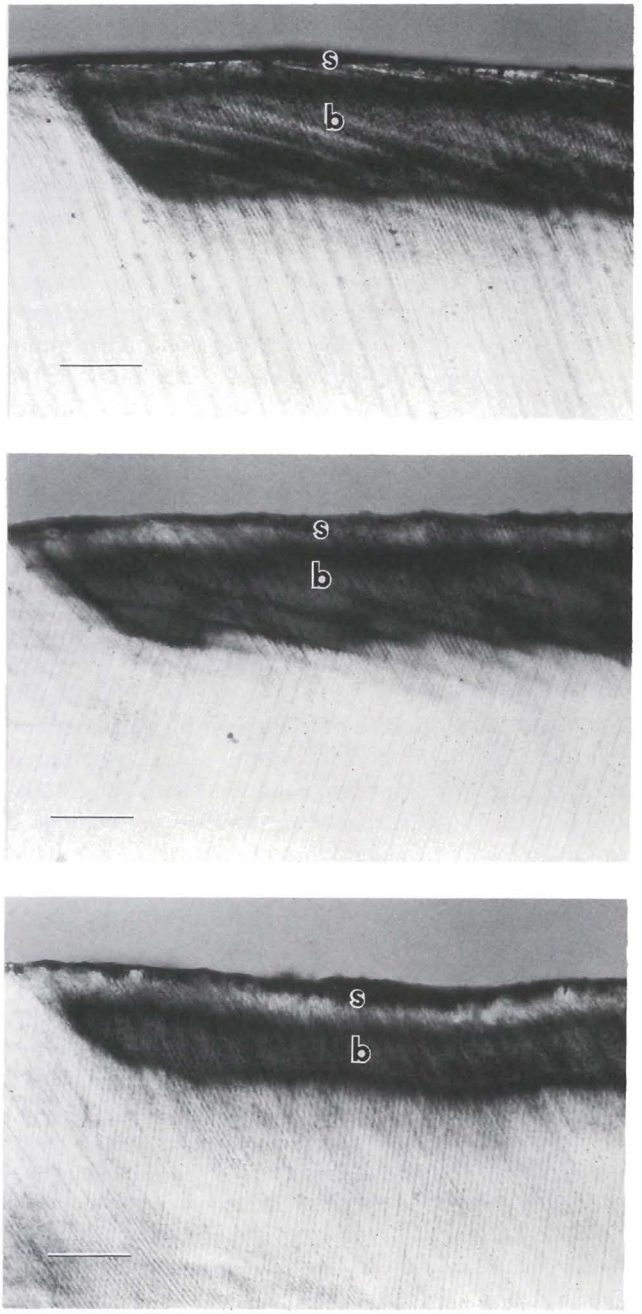

Figure 2. High Calcium Calcifying Fluid Group (3mM Calcium). A. (Top) Representative Control Lesion with a mean body of the lesion depth of 183 μm and a mean surface zone depth of 22 μm. B. Representative Remineralized Lesion with a mean body of the lesion depth of 165 μm and a mean surface zone depth of 32 μm. C. Representative Etched, Remineralized Lesion with a mean body of the lesion depth of 137 μm and a mean surface zone depth of 38 μm. (Water imbibition, Polarized Light; S = Surface Zone; B = Body of Lesion; Space Bar = 100 μm)

and 34 percent less than that for paired control lesions ($p<0.05$). The surface zone depths were also affected by both remineralization alone and in conjunction with previous acid-etching. Surface zone depth increased by one-third in the remineralized lesion group ($p<0.05$) and by over 80 percent in the etched, remineralized group ($p<0.05$) when compared with paired control lesions.

Following remineralization with the high calcium (3mM) calcifying fluid, similar results were obtained between the control, remineralized and etched/remineralized groups (Table, Figure 2). The body of the lesion depth was reduced by 9 percent following remineralization and by 24 percent following acid-etching and remineralization ($p<0.05$), when compared with paired control lesions. Acid-etching of the lesions prior to remineralization resulted in a further 16 percent reduction ($p<0.05$) in the body of the lesion depth, when compared with remineralization alone. The surface zone depth was increased substantially in both the remineralized and etched, remineralized groups when compared with paired control lesions. Surface zone depths increased by 38 percent for the remineralized group ($p<0.05$) and by 86 percent in the etched, remineralized groups ($p<0.05$) when compared with paired control lesions. When surface zone depths were compared between remineralized and etched, remineralized groups, a significant increase ($p<0.05$) occurred when acid-etching preceded remineralization with the 3mM calcifying fluid.

Comparison of the body of the lesion depth reductions between the 1mM and 3mM calcifying groups showed significant differences. With both remineralized and etched, remineralized groups, the body of the lesion depth reduction was considerably increased in the low calcium (1mM) calcifying group when compared with the high calcium (3mM) calcifying group ($p<0.05$). When the lesions were exposed to calcifying fluid alone, lesions in the low calcium calcifying group had a mean body of the lesion depth reduction of 12 percent compared with 9 percent for the high calcium calcifying group ($p<0.05$). When acid-etching of the lesion preceded remineralization, a 34 percent reduction in the body of the lesion depth occurred in the low calcium calcifying group compared with 24 percent for the high calcium calcifying group ($p<0.05$). In contrast, surface zone depths had increased by 33 percent and 38 percent following remineralization in the 1mM and 3mM groups, respectively. When remineralization was combined with acid-etching, the surface zone depth increases were 83 percent for the 1mM group and 86 percent for the 3mM group.

The surface morphology, as determined by scanning electron microscopic techniques, was quite different among the groups (Figure 3). With control lesions (Figure 3A), the typical lesion surface was characterized as one which had numerous depressions of 3 to 5μm in diameter, representing surface termination of enamel prisms. The lesion surfaces were intact and showed only slight etching of the prism cores. With exposure to the 1mM calcifying fluid (Figure 3B), many of the surface depressions were filled in with an adherent surface coating. Occasional surface depressions were identified. Lesion surfaces exposed to the 3mM calcifying fluid (Figure 3C) possessed a dense surface coating with occasional breaks in the coating, revealing the underlying lesion surface. The coating appeared to be laid down in layers and frequent areas showed a tiered-appearance with loss of more superficial layers. The prism depressions previously noted with control lesions and lesions exposed to the 1mM calcifying fluid were not identified and were apparently obliterated by the deposition of a dense surface coating. Remineralization of the acid-etched lesions resulted in an alteration in surface morphology for both the 1mM and 3mM calcifying groups. With the etched, remineralized lesions in the 1mM calcifying group (Figure 3D), the typical surface had an irregular contour with a finely globular, surface coating. The contour of the adherent coating gave the appearance of attachment to etched enamel prisms with numerous surface depressions and elevations, resembling "moguls". In contrast, the etched, remineralized lesions in the 3mM calcifying group (Figure 3E) possessed a finely granular, confluent surface coating with numerous microporosites. The effects of acid-etching were masked and the original surface contour of the etched lesion could not be appreciated.

DISCUSSION

Acid-etching of enamel was initially introduced as a means of increasing retention of dental restorative materials and to improve fluoride uptake.[18-20] The effect of etching on sound enamel includes:

- ☐ Loss of inert, fully-reacted surface enamel.
- ☐ Exposure of a more reactive enamel surface.
- ☐ Creation of surface porosities and etching patterns that result in an increased surface area for interaction with preventive and remineralizing agents, as well as for bonding of adhesive resins.

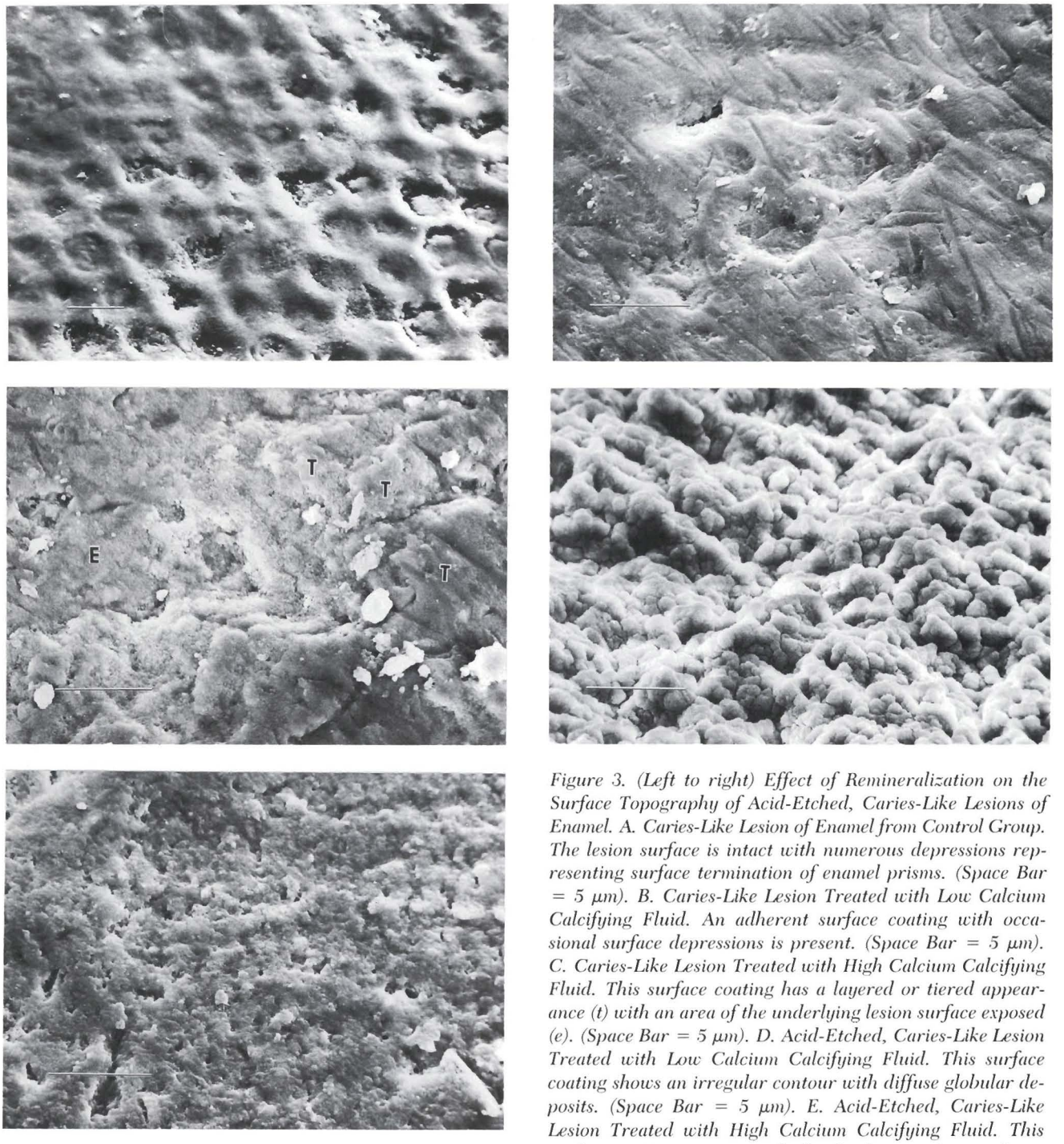

Figure 3. (Left to right) Effect of Remineralization on the Surface Topography of Acid-Etched, Caries-Like Lesions of Enamel. A. Caries-Like Lesion of Enamel from Control Group. The lesion surface is intact with numerous depressions representing surface termination of enamel prisms. (Space Bar = 5 μm). B. Caries-Like Lesion Treated with Low Calcium Calcifying Fluid. An adherent surface coating with occasional surface depressions is present. (Space Bar = 5 μm). C. Caries-Like Lesion Treated with High Calcium Calcifying Fluid. This surface coating has a layered or tiered appearance (t) with an area of the underlying lesion surface exposed (e). (Space Bar = 5 μm). D. Acid-Etched, Caries-Like Lesion Treated with Low Calcium Calcifying Fluid. This surface coating shows an irregular contour with diffuse globular deposits. (Space Bar = 5 μm). E. Acid-Etched, Caries-Like Lesion Treated with High Calcium Calcifying Fluid. This surface coating is composed of a finely granular confluent deposit with numerous microporosities. (Space Bar = 5 μm).

- ☐ Removal of organic debris and pellicle embedded within the superficial aspect of the enamel surface.[5-7]

The characteristic histologic pattern associated with etched, sound enamel has been described. Typically, removal of 5 to 10μm of fully-reacted sound enamel from the surface occurs following a 60-second exposure to phosphoric acid (etched zone). The underlying affected enamel shows qualitative increases in microporosity as determined by polarized light. These microporosites represent the surface porosities associated with etching patterns characterized by scanning electron microscopy. This qualitative porous zone extends into the underlying enamel to a depth of 20μm. An additional 20μm of subsurface enamel shows quantitative microporosites changes (quantitative porous zone). The overall depth of enamel affected by the etching procedure is usually 50μm, although resin tag lengths have been shown to extend into etched enamel up to a depth of 100μm.[21] Similar effects of acid-etching have been reported with caries-like lesions of enamel. Two of the three histologic zones, etched and qualitative porous zones, associated with etching of sound enamel and characteristic etching patterns are also present with acid-etched lesions.

It is apparent from the present study that acid-etching of lesion surfaces before treatment with a calcifying fluid affected the degree of remineralization significantly, when compared with paired control ($p<0.05$) and remineralized only ($p<0.05$) groups. The greatest effect on body of the lesion depth was found with etched lesions exposed to the low calcium calcifying fluid ($p<0.05$). In contrast, the greatest effect on surface zone depth was identified with etched lesions treated with the high calcium calcifying fluid ($p<0.05$). Similar patterns, although reduced, were also present within the remineralized only groups when compared between low and high calcium calcifying groups ($p<0.05$) and with control groups ($p<0.05$). The findings in the present study are in agreement with previous investigations.[4,9-11,16] Earlier remineralization studies using similar calcifying fluids have shown that 1mM calcium fluids affect the lesion to its entire depth and results in a significant reduction in lesional area (69 percent) and depth when compared with 3mM calcium fluids.[4,17,22] The effect of the 3mM calcium fluid was shown to affect primarily the superficial aspect of the lesion, resulting in an increase in surface zone depth and a moderate reduction in lesional area (20 percent). Both the 1mM and 3mM calcium fluids provided remarkable reductions in lesion depth when compared with the present study. One must consider that ten exposure periods of sixty minutes and twenty-four hours were utilized, however, in these studies. In contrast, ten exposure periods of sixty seconds in length were used in the present study. This would appear to be a more realistic time-frame if a remineralization protocol is to be introduced into clinical practice.

Acid-etching affects degree of remineralization significantly.

The differences between the remineralizing abilities of the low and high calcium calcifying fluids may be due to differences in the saturation levels of various mineral phases and the mechanism of mineral deposition.[17-22] With the 3mM calcium fluid, it has been shown previously that the mineral phases hydroxyapatite, fluorapatite, octacalcium phosphate and tricalcium phosphate are supersaturated; while dicalcium phosphate dihydrate is saturated. In contrast, only the mineral phases hydroxyapatite and fluorapatite are supersaturated with the 1mM calcium fluid. Due to the degree of supersaturation of mineral phases in the 3mM calcium fluid, rapid deposition of mineral phases by a nucleation process would be favored. This mechanism of mineral deposition predicts that relatively small diameter mineral crystals would be formed and may result in rapid precipitation of these newly formed crystals within the pore structure of the lesion, effectively blocking access to the complete pore structure system of the lesion. In contrast, the mechanism of mineral deposition for the 1mM calcium fluid would favor crystal growth on existing demineralized enamel crystals lining the pore structures, and result in relatively large diameter crystals. With crystal growth, access to the pore structure system of the lesion would be available for prolonged remineralization. High resolution SEM studies evaluating crystal diameters sup-

port this hypothesis for the difference in degree of remineralization between relatively low and high calcium calcifying fluids.[4,17,22] Following remineralization, crystal sizes within the body of the lesion ranged from 50 to 150nm for the 1mM calcium fluid compared with 50 to 75nm for the 3mM calcium fluid. As a point of reference, sound enamel has a crystal diameter ranging from 35 to 40nm; whereas crystal diameters in the body of the lesion range from 10 to 30nm.

The enhancement of remineralization with calcifying fluids following acid-etching may be due to a number of factors. The surface of enamel caries lesions has been shown to be intact and difficult to distinguish from adjacent sound enamel.[6-8] The surface topography may demonstrate a slight increase in the prevalence of prism-end markings with minimal surface roughening. Similarly, the histopathologic appearance shows minimal qualitative changes from sound enamel although the pore volume may be increased anywhere from ten to fifty times over that for sound enamel.[6] With acid-etching of caries-like lesions, an increase in porosity has been demonstrated. In fact, the internal morphology of the etched surface zone has been described.[7] With etched lesions, funnel-shaped structures with maximum diameters of 3μm extended from the etched surface into the surface zone to a depth of 5 to 8μm. Etch pits of 0.5 to 0.75μm in size were found extending from the base of the funnel-shaped structures. Approximately 15 to 20μm from the lesion surface, these etch pits terminated and were replaced by microporosites of 100 to 300nm in size. It would appear as though the acid-etch technique provides a pathway to the body of the lesion for remineralizing fluids. With the intact surface overlying enamel caries, access to the underlying body of the lesion may be restricted and result in a lessened degree of remineralization when compared with etched lesions.[6,7,23] In addition, the deposition of a surface coating rich in mineral elements and fluoride may allow for continued prolonged remineralization.

The present study may have certain clinical implications. In past studies, extended exposure periods (hours to days) to calcifying fluids were utilized; the treatment protocol employed in the present study provided a significant degree of remineralization, however, while using an exposure period which would be feasible in the clinical setting. Acid-etching of clinically detectable white spot lesions followed by a series of 60-second exposures to calcifying fluid may provide protection against lesion progression and perhaps result in partial or substantial reversal of the lesion.

The conclusions drawn from this *in vitro* study are:

- ☐ Remineralization of caries-like lesions may be significantly enhanced by acid-etching of the lesion before exposure to calcifying fluids.
- ☐ Low calcium (1mM calcium) calcifying fluid provided the greatest degree of remineralization when compared with the high calcium (3mM calcium) fluid with both remineralization alone or in combination with acid-etching.
- ☐ Adherent surface coatings were present following either remineralization alone or acid-etching followed by remineralization.

These surface coatings may act as mineral reservoirs for prolonged remineralization of the lesions.

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IN SITU REMINERALIZATION OF ENAMEL LESIONS

There is widespread agreement that fluoridated drinking water has been effective in the prevention of dental decay, and similar caries reductions have been reported in the United States and other industrialized nations in regions with F-deficient drinking water (First International Conference on Declining Prevalance of Dental Decay, 1982) where topical F agents appeared to be major determinants in the decline of dental caries [Fejerskov et al., 1982; Anderson et al., 1982; Kalsbeek, 1982; von der Fehr, 1982; Koch, 1982; Glass, 1982; Brunelle and Carlos, 1982]. However, dental caries persists as a common disease, and it has been reported that in the United States 80% of the dental caries occurs in 20% of the population. This suggests a highly 'caries-susceptible' segment of the population, for whom the anticaries benefits which generally would be provided by conventionally available F agents/methods of application have not been realized. The reasons for these persistently high caries rates are not fully understood, but factors such as compromised salivary flow, frequent intake of fermentable carbohydrates, and bacterial virulence likely play a role [Featherstone, 1990]. Additionally, certain special populations who are physically disabled, chronically ill, or mentally impaired and for whom compliance may be difficult or who require assistance with oral hygiene procedures (i.e., toothbrushing or mouthrinsing) could derive significant anticaries benefits from alternative F agents such as F chewing gums and FRDs. Although F gum and FRDs achieved similar levels of remineralization in this study, practical differences in their use exist between them. F gums are cheap and relatively easy to use, but require patient compliance in order to be optimally effective, whereas with FRDs compliance following intraoral placement is minimal, but the use of FRDs is expensive and remains experimental.

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Evaluation of occlusal marginal adaptation of class II resin composite inlays

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The inlay technique has been assigned for use with resin composite materials in Class I and II cavities of posterior teeth and an overview of clinical applications has been given by Burke *et al* Extraoral postcuring of resin composite restorations would eliminate to a great extent a major disadvantage of resin based materials in direct applications, namely the polymerization shrinkage during direct placement. Improvements to mechanical properties of the material by additional curing have been reported, although other studies failed to demonstrate an increase as such.[2] Lutz *et al* concluded that polymerization contraction in directly bonded restorations interferes with the adhesion of the restorative material to the cavity wall and inlays should provide alternative ways to achieve optimal marginal adaptation.[3] The residual shrinkage of the composite cement was thought to be compensated for by deformation of the cavity walls. Feilzer reported on the wall-to-wall contraction of resin composites used in thin layers, however, and argued that the polymerization stress of luting cements in noncomplying structures is detrimental to the likelihood of obtaining a good adhesion.[4]

The aim of this clinical study was to observe the occlusal marginal adaptation of indirect Class II resin composite inlays, using a photographic technique. The results will be studied for influences related to the material, the dentists, and the cavity. Class II amalgam restorations were used for purposes of comparison and differences between indirect and direct Class II resin composite restorations are discussed.

MATERIAL AND METHODS

The overall design of the investigation and the placement techniques used for the indirect inlays have been described elsewhere.[5] Essentially, 'standard' sized, conservative, nonbevelled indirect inlays made of three resin composite materials→ were used in the treatment of 180 Class II cavities in fifty-eight patients (twenty-three years of age (s.d. 5)). Each material was applied to each patient and a fourth Class II cavity was filled with amalgam.● The four materials were randomly allocated within the patient, while the cavity distribution was dependent on the teeth that needed

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→Herculite XR (Kerr)
Clearfil CR Inlay (Cavex Holland / Kuraray)
Visiomolar (ESPE)

●Tytin (Kerr)

restorative treatment. Two of the fifty-eight patients received a total of eight restorations instead of the series of four. Three dentists performed the clinical procedures, all making equal numbers of restorations of each restorative. The dental laboratory work was done by one technician and the composite materials were postcured as recommended by the manufacturers. The inlays were placed using a dual-cure luting cement that matched the resin composite material used. Clinical finishing and polishing of the inlay were performed using x-fine diamond stones, rubber points and cups, and polishing paste. The amalgam restorations were finished using carbide finishing burs, rubber cups and points, and polishing paste.

Assessments of the occlusal marginal adaptation were performed according to a method described earlier.[6] After a mean period of seven months (s.d. 3.5) following polishing of the restorations, impressions were taken of the restorations using an individual impression tray. A stereomicroscope was used to take two black-and-white photographs of the occlusal surfaces in the impression; one with floodlighting from the right, and one from the left. Shadowing reveals deficiencies in the marginal adaptation of the restorations. The photographs (9x overall enlargement) were independently assessed by two trained and calibrated observers using a 'section-method' to locate the deficiencies observed (Figure 1). A standard series of a four-point scale was used (Table 1). Joint decision-making was applied if the assessed by two trained and calibrated observers using a 'section-method' to locate the deficiencies observed (Figure 1). A standard series of a four-point scale was used (Table 1). Joint decision-making was applied if the ratings differed by more than 1 scalepoint. Interexaminer agreement was 97 percent (kappa value 0.87), which was presented in a previous paper.[6]

Based on the average score awarded by the two observers per section, the mean marginal adaptation was determined; that is, the mean value for all sections included in the occlusal outline of the restoration. Excellent marginal adaptation is represented by a mean marginal adaptation of 1. Higher values of the mean indicate that at least one section of the restoration was given a rating of 2 (or more). The mean marginal adaptation provides an overall view of the marginal adaptation of the restorations and frequency distributions for both resin composite inlays and amalgam restorations are presented.

Table 1. ☐ Criteria applied during indirect, photographic evaluation of the marginal adaptation.

Rating	Description of the criterion
1	Margin not or slightly visible (shadowing/reflection* less than 0.5 mm)
2	Margin visible (shadowing/reflection between 0.5 and 1 mm)
3	Margin clearly visible (shadowing/reflection between 1 and 2 mm)
4	Margin and crevice highly visible (shadowing/reflection more than 2 mm)

* 'Shadowing/reflection' – width of the shadowing or reflection line

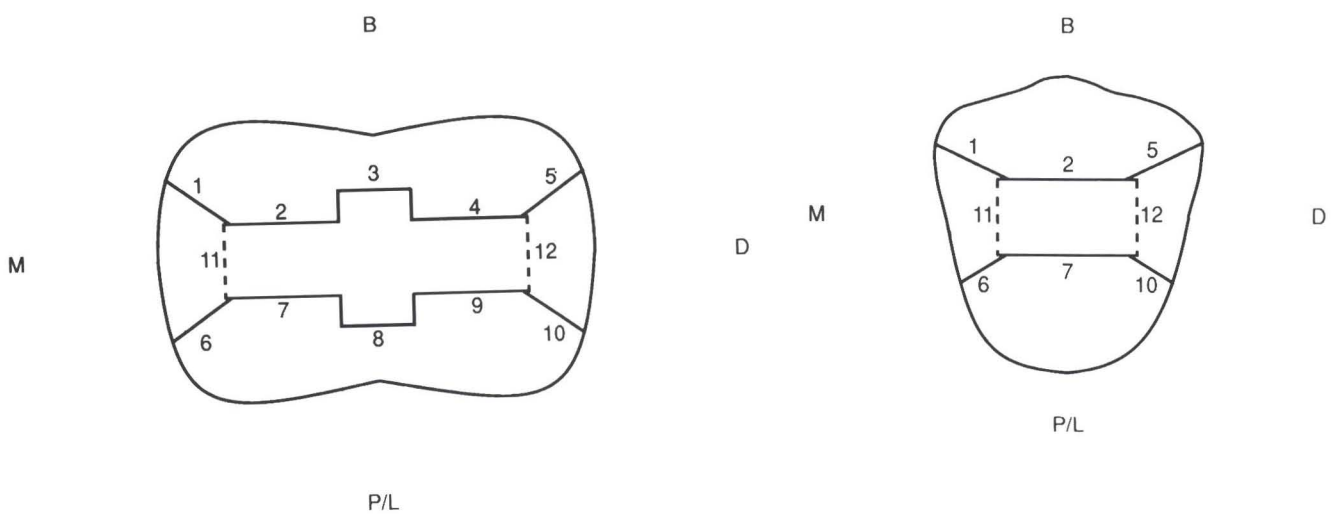

Figure 1. Division of the occlusal outline of a restoration into sections; molar (left) and premolar. B = Buccal, P/L = palatal/lingual, M = mesial, D = distal.

Factors that are considered to be of influence in this study are:

- ☐ The dentist who carried out the treatment.
- ☐ The type of resin composite.
- ☐ The type of restoration.
- ☐ The type of tooth.

Their influences are determined using Chi-square tests (SPSS/PC) and taking into account slightly skewed distributions of the variables, type of tooth and type of restoration; logistic regression analyses were also applied. The section method enables the marginal adaptation of various parts of the occlusal outline to be studied and frequency distributions of sectional assessments, therefore, are presented. In contrast to the mean marginal adaptation, the rating of each section is based on the least favorable score given by one of the observers.

RESULTS

Mean marginal adaptation

As shown in Figure 2 (left graph), the frequency distribution of the mean marginal adaptation of indirect Class II resin composite inlays is positively skewed. Nearly half of the restorations show *excellent* marginal adaptation (46.1 percent). The distribution of the mean marginal adaptation of amalgam restorations is less skewed (Figure 2). Only four restorations were rated as *excellent* (6.7 percent), while fifty-six included a section showing *nonexcellent* marginal adaptation at some point in the outline (93.3 percent). If the restorations are divided up into those showing *excellent* and *nonexcellent* occlusal marginal adaptation, a significant difference between indirect resin composite and amalgam Class II restorations results (Chi-square, $p<0.001$).

Table 2 shows the percentages of *excellent* and *nonexcellent* mean marginal adaptation of the composite inlays for each of the influencing factors. The differences among the dentists are significant, with the restorations made by Dentist 3 showing the least number of *excellent* margins (logistic regression, Dentist 1 versus 3, $p<0.001$; Dentist 2 versus 3, $p<0.05$). A difference was observed between premolars and molars (more *excellent* margins were found for premolars than for molars), while one composite material (Herculite) appears to yield the fewest *nonexcellent* margins of the three resin composites. Except for the difference between Herculite and Visiomolar (logistic regression, $p<0.05$), these differences are not significant (logistic regression, $p<0.05$). As only a few amalgam restorations were rated as *excellent*, no statistics were performed on the factors as mentioned above and their influence on the marginal adaptation.

Marginal adaptation per section

Of the 97 resin composite inlays rated *nonexcellent*, five restorations had sections rated as 3 (2.8 percent of the total number of resin composite inlays). Of those

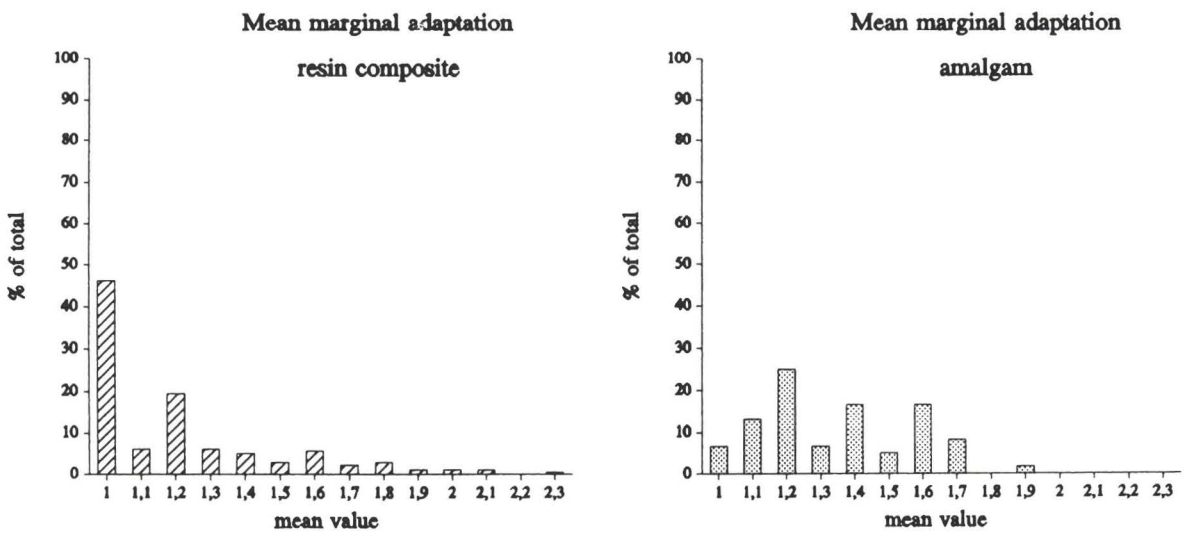

Figure 2. Frequencies of the mean marginal adaptation of Class II restorations: resin composite inlays (left), N = 180, and amalgam, N = 60.

five (four molars, one premolar) the maximum number of sections rated as 3 was three. For amalgam, one molar restoration had a section rated as 3 (1.7 percent of the total number of amalgam restorations).

A two-way division of the marginal adaptation of the sections (*excellent / nonexcellent*) yields the frequency distributions shown in Figure 3 (for premolars (left) and molars). Section 1 seems to have the most *excellent* ratings, except for resin composite inlays in molars. A 100 percent *nonexcellent* rating was found for section 11 with amalgam restorations (the mesial section of distoclusal restorations).

DISCUSSION

Hannig showed the Alpha ratings of the occlusal marginal adaptation of indirect Class II resin composite inlays declining from 100 percent to 77 percent after three months and 37.5 percent at a six-monthly check-up, assessed using modified USPHS criteria.[7] In a study on indirect Class II inlays in primary molars, Mutokawa observed 98 percent restorations with *excellent* margins become 67 percent after six months.[8] At that stage approximately 26 percent were rated 'Bravo', 3 percent 'Charlie', and 4 percent 'Delta'. Although the

Table 2. □ Influence of the factors examined on the mean marginal adaptation of indirect Class II resin composite inlays.

		Dentist			Type of restoration		Type of resin composite			Type of tooth	
		1	2	3	Two-surface	Three-surface	Herculite XR	Clearfil CR	Visiomolar	Premolar	Molar
	N	60	60	60	107	73	60	60	60	135	45
Resin composite	mean = 1	63.3%	48.3%	26.7%	47.7%	43.8%	58.3%	43.3%	36.7%	50.4%	33.3%
	mean > 1	36.7%	51.7%	73.3%	52.3%	56.2%	41.7%	56.7%	63.3%	49.6%	66.7%
	p-value	< 0.001 (Dentist 1 vs. 3)			n.s.*		<0.05 (Herculite vs. Visiomolar)			n.s.	

* n.s. = not significant
mean = 1 : mean marginal adaptation equals 1, refers to an excellent marginal adaptation
mean > 1 : mean marginal adaptation more than 1; at least one section is not rated excellent.

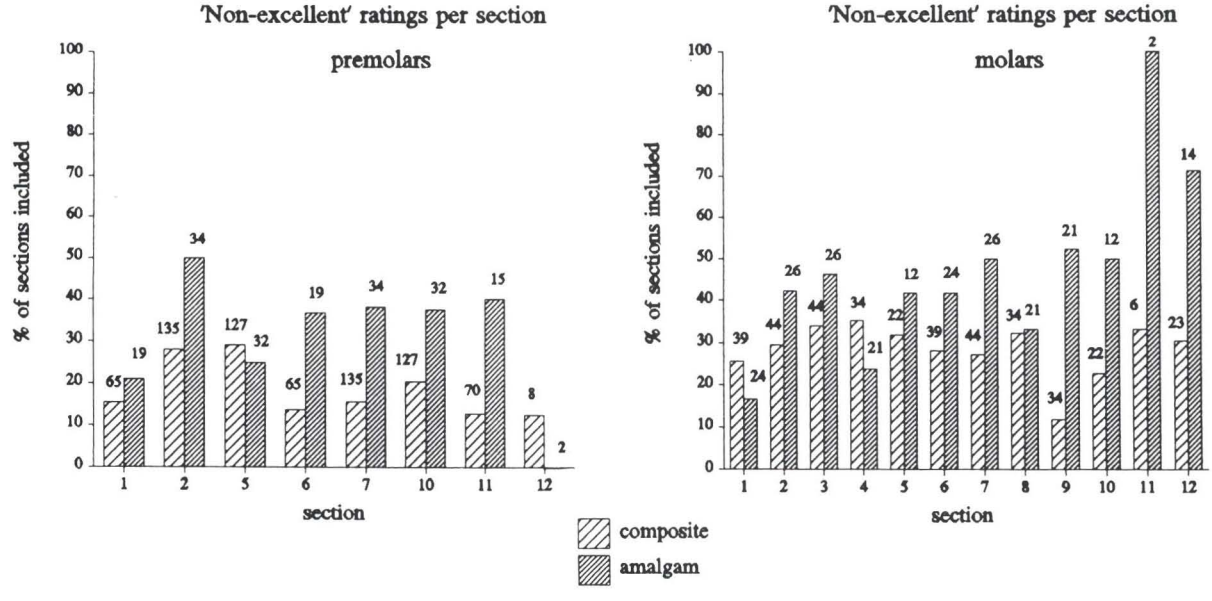

Figure 3. Frequencies of nonexcellent *ratings of premolar (left) and molar sections (right) for indirect Class II resin composite inlays and amalgam restorations. On top of each bar the total of each section is shown.*

behavior of restorations in primary molars may not be comparable, these figures show some correspondence. Conversely, Füllemann still found 100 percent Alpha ratings after one year.[9] This stability in marginal degradation is in agreement with Roulet *et al, who reported a slight decrease in the percentage of excellent* margins: from 72-87 percent to 61-82 percent after nine months.[10] The latter figures, however, show a moderate initial marginal adaptation of indirect resin composite inlays.

In the present study the frequency of restorations with an *excellent* marginal adaptation along the occlusal outline is low (46.1 percent) compared to the value found in our study on marginal adaptation of direct Class II resin composite restorations (64.5 percent *excellent* marginal adaptations).[11] The variation in the period elapsing between placement of the restorations and performance of the assessments is likely a distorting factor affecting the interpretation of the results; the 'oldest' restoration was approximately twelve months old, the 'youngest' two weeks (mean seven months). The distributions of the restorative materials, the dentist, the type of tooth, and the type of restoration are more or less homogeneously distributed, however, across the various periods of time. If the restorations are classified in quarterly age-categories, there appears to be no difference between *excellent* and *nonexcellent* restorations (Table 3). This is in agreement with the findings of Roulet and Fülleman. Nevertheless, we would not regard the percentages of marginal deficiencies observed as absolute figures that describe the situation at baseline; but by taking into account the homogeneous distribution over time, the factors influencing marginal adaptation can be considered.

The influences of the factors observed are in agreement with the results of the previous study on marginal adaptation of direct resin composite restorations. The dentist determined marginal adaptation to a certain extent. Thus, even if the body of the restoration is not produced by the dentist himself, his influence on the finishing and polishing of the margins appears to be clear. Füllemann *et al* have discussed the differences that can be expected between marginal adaptation of premolars and molars, ascribed to an operating problem: the occlusal surface of molars is more fissured than that of premolars and it is to be expected that the dentist will not be able to remove excess cement completely.[9] This excess cement can easily fracture under loading, leaving a detectable margin. The present results appear to show an influence accorded the type of tooth, but the difference between premolars and molars was not significant. The type of restoration also did not have an influence on the results.

Table 3. ☐ Classification of the numbers of restorations according to quarters of a year and their mean marginal adaptation (excellent / non-excellent), $p>0.05$ (Chi-square).

Quarter of a year	mean = 1 *	mean > 1	Total
1st	14	18	32
2nd	17	23	40
3rd	26	30	56
4th	26	26	52
Total	83	97	180

* mean = mean marginal adaptation, two-way division into excellent and non-excellent

Some studies valued the thickness of the cement layer at 50 to 150 μm, although Reich *et al* stated that layers of 200 to 400 μm are conceivable in the average dental practice.[1,9,12] Abrasion of this interfacial low filled resin resulted in an underextended margin of 20-40 μm in the study by Füllemann *et al*. This is an indication that resin composite inlays may even exhibit a marginal ditch after polishing due to differences in abrasion of the relatively soft cement, surrounded by the enamel and the inlay material. On the other hand, a worn cement layer that is detected clinically by catching of the probe or photographically by shadowing does not necessarily indicate a leaking restoration.[12]

CONCLUSIONS

- ☐ The quality of the occlusal marginal adaptation of indirect Class II resin composite inlays is determined to a great extent by the dentist who provides the treatment.
- ☐ In this study, indirect Class II resin composite inlays show more *excellent* ratings in terms of marginal adaptation compared with Class II amalgam restorations, after a mean period of seven months.

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EFFECT OF MILK ON CARIOGENESIS

These results confirm and extend previous observations. Our data showed that neither milk, nor lactose-reduced milk is cariogenic. Equally, it is apparent that they do not possess cariostatic properties when the dietary challenge and milk are administered separately. However, if milk is taken simultaneously with the caries challenge (sucrose), it is clear that milk exerts some protection against the cariogenic challenge of sucrose. Nevertheless, milk containing sucrose is more cariogenic than milk alone.

Superficially, it may appear difficult to compare the effect of one fluid with another because significantly different amounts were consumed in the experimental groups. However, where more or an equal amount of test fluid (e.g. milk) is consumed with the positive control (sucrose), valid comparisons can be made. If, on the other hand, more sucrose solutions than milk had been consumed, it would have been essential to resort to programmed feeding to achieve meaningful data, as we have done previously [Bowen et al., 1990b]. In our experiments, the animals consumed significantly more milk than any other solution. This observation is consistent with previous observations in desalivated animals [Bowen et al., 1991]. It is clear, therefore, that milk does not affect the cariogenic potential of diet 2000. Diet 2000 contains a significant amount of dried milk powder; nevertheless, the diet on its own is highly cariogenic. Ingestion of liquid milk does not confer protection against caries using this challenge. The animals consumed equal volumes of sucrose and fructose solutions. Therefore, valid comparisons of data from those groups can be made. Fructose in solution can help to enhance the development of smooth-surface caries in animals fed a cariogenic diet. This observation is consistent with previous reports [Bowen et al., 1990a] which showed that fructose is highly cariogenic and can induce smooth-surface caries.

Bowen, W.H. and Pearson, S.K.: Effect of milk on cariogenesis.
Caries Res, 27:461-466, November-December 1993.

Children with hypoplastic enamel defects of primary incisors are not at increased risk of learning-problem syndromes

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David Bellinger, PhD
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Children who had supernumerary or congenitally-missing teeth or who had delays in permanent dentition did less well than their peers on assessments of function in school (Silvestro, 1977; Oliver *et al.*, 1979). Enamel defects of primary teeth have been seen more commonly than expected in children with a wide variety of neurologic abnormalities, including cerebral palsy, mental retardation, developmental delay, and psychometric abnormalities (Bhat and Nelson, 1989). These observations raised the possibility that what disturbs tooth development also disturbs brain development. This hypothesis is plausible for two reasons:

- ☐ The time interval from the beginning to the end of enamel formation of the crown of primary incisors (i.e., the end of the first trimester to the end of the third postnatal month) (Levine *et al.*, 1979) is a time when the brain is viewed as vulnerable to perturbation (Gilles *et al.*, 1983; Bhat *et al.*, 1992).
- ☐ Both brain and teeth are derived from the same embryonic tissue, neuroectoderm.

To our knowledge no study has investigated the relationship between enamel defects of the primary dentition and learning problems. In this report we compare the prevalence of learning-problem syndromes in children whose primary incisors had hypoplastic enamel defects (HED) to the prevalence in their peers whose incisors did not have HED.

This investigation was supported in part by grants RO1-HD20381 and P30-HD18655 from the National Institute of Child Health and Human Development.

METHODS

Sample

The sample for this study was drawn from 3,814 newborns delivered at one hospital between April 1, 1979 and March 31, 1980, whose umbilical cord lead content was measured, for a study of the effects of prenatal lead exposure (Leviton *et al.*, 1993a). Mothers were interviewed after birth about demographic, reproductive, pregnancy, and lifestyle characteristics. This is basically a middle class sample with less than 4 percent of families receiving public assistance near the time of delivery. Approximately 5 percent of these children were born before the 37th week of gestation.

Teeth

When the subjects were approximately six years old, their parents were sent a two dollar bill ("from the tooth fairy") and a request to send the investigators one of the child's exfoliated primary teeth. Only the

first tooth was included in this study. Teeth were submitted for 1,982 children. The last 670 teeth submitted were examined for defects before they were split in preparation for lead content analysis (Needleman *et al.*, 1991). We excluded sixty-two teeth that were not incisors and 153 incisors that were damaged or had caries or dental restorations.

The teeth were prepared for visual examination by cleaning the outer surfaces with dental scalers and a standard dental prophylaxis cup on a slow speed handpiece using lead-free flour of pumice. After drying, the teeth were examined by one of the authors (HLN), a pediatric dentist, using a 4x magnifying loop under direct illumination.

Defects were recorded using the classification established by the Commission on Oral Health, Research and Epidemiology of the Federation Dentaire Internationale (1982). A full description of the macroscopic enamel defects of teeth in this sample has been reported (Needleman *et al.*, 1991). Two of the seven defects in this classification, hypoplastic pits and hypoplastic grooves, were combined into one category of defect, which we call hypoplastic enamel defect (HED). Hypoplasia, defined as a quantitative defect of enamel associated with a reduced thickness of enamel, is considered to be the enamel defect most likely to result from a systemic insult (Small and Murray, 1978; Cutress and Suckling, 1982; Pindborg, 1982). Thus, it is the focus of this study.

Intra-observer variability was assessed with 123 teeth reexamined among the primary examinations of the remainder of the teeth. The kappa for identification of hypoplastic defects was 0.66, which is viewed as acceptable (Siegel *et al.*, 1992).

Boston Teacher Questionnaire

The eighteen-item Boston Teacher Questionnaire (Leviton *et al.*, 1993b) was sent to the child's school during the second half of the school year in which the child celebrated the eighth anniversary of her/his birth. Clustering of fifteen items of this questionnaire resulted in seven syndromes of problems in school (Leviton *et al.*, 1993c). These syndromes are identified in Table 1. Of the 455 children who provided an undamaged incisor, information about function in school was available for 345.

ANALYSIS

The basic form of the null hypothesis tested was that the prevalence of each learning-problem syndrome among children with HED did not differ from the prevalence among children who did not have HED of their single exfoliated incisor. Only simple descriptive statistics are presented.

RESULTS

Of the 345 primary incisors evaluated, 18.6 percent had HED. These defects were more common among maxillary incisors (25 percent) than among mandibular incisors (10.1 percent).

Of the seven learning-problem syndromes evaluated

Table 1. ☐ The prevalence (per 100 children) of learning-problem syndromes presented separately for girls and boys whose primary incisor had HED and for those whose incisor did not have HED.

		GIRLS Hypoplastic enamel defect			BOYS Hypoplastic enamel defect		
Syndrome	Components	Yes (N=31)	No (N=118)	p value	Yes (N=36)	No (N=160)	p value
Behavior	Easily frustrated Has peer difficulties	6.5	7.6	1.00	8.3	0.0	.42
Hyperactive	Hyperactive Overexcitable and impulsive	0.0	2.5	1.00	0.0	5.6	.22
Reading	Reading is slow, hard work Not a good speller	6.5	7.6	1.00	11.1	10.6	1.00
Arithmetic	Doesn't know tables Can't apply these easily	16.1	13.6	.77	16.7	8.1	.13
Directions	Can't follow simple directions Can't follow sequence of directions	0.0	8.5	.12	8.3	7.5	.74
Daydream	Daydreamer Easily distracted	9.7	17.8	.41	22.2	20.0	.82
Tasks	Impersistent Dependent Inflexible and inappropriate approach to tasks	6.5	11.0	.74	5.6	7.5	1.00

separately among girls and boys, none was associated with HED at a p value of 0.05 or less (Table 1). Six of the seven syndromes occurred more frequently in girls whose incisor did not have HED, and three of the seven syndromes occurred more frequently in boys whose incisor did not have HED. Of the fourteen comparisons made, only two had a probability value of less than 0.2. Among girls, those who had HED had an appreciably lower prevalence of the directions syndrome than did those whose teeth did not have HED ($p = 0.12$). Among boys, the math syndrome was twice as common among those whose incisors had HED than among those whose incisors did not have these defects ($p = 0.13$).

Both girls and boys with HED were more likely than their peers to have repeated a grade, but these differences did not achieve nominal statistical significance (Table 2). Receipt of special services was not more common among children with HED.

Table 2. ☐ The prevalence (per 100 children) of other ways teachers acknowledge a child might not be functioning as well as peers.

	Girls			Boys		
	Yes	No	p value	Yes	No	p value
Teacher report: "Not functioning as well as other children"	12.9	15.2	1.00	27.8	16.3	.15
Repeated a grade	9.7	5.9	.43	11.1	7.5	.50
Assessed for remedial services	9.7	15.2	.57	27.8	22.5	.52
Received special services this year	16.1	19.5	.80	27.8	28.1	1.00
Received special services last year	12.9	11.9	1.00	16.7	24.4	.39

DISCUSSION

Our study failed to find any statistically significant association between HED and seven learning-problem syndromes, repeating a grade, and receipt of special services. Although others have assessed the relationship between dental characteristics and psychological function, ours is the first to evaluate the relationship between enamel defects in exfoliated primary incisors and selected learning-problem syndromes.

This study has a number of strengths. First, the recruitment of children eight years before assessing dysfunction in school minimized selection bias. This was a predominantly middle class sample, presumed to be at low risk of postnatal adversities that might confound the assessment of a relationship between enamel defects and learning handicaps. Second, all teeth were examined by one dentist, whose intra-observer variability was comparable to that of experts in other diagnostic fields (Koran 1975, Feinstein 1985). Third, we assessed dysfunction in the classroom rather than performance on a standardized test administered in a setting deprived of distractions. We acknowledge the limitations of teacher reports (Shaywitz *et al.*, 1990), but feel that problems related to attention are best appreciated in a child's normal classroom setting.

Our study also has a number of limitations. Only one tooth per child was examined. Thus, the possibility exists that some children may have been misclassified as not having any HED, when in fact, examination of another tooth might have identified a HED. Bhat and his colleagues (1989) have suggested that the optimum time for studying developmental defects of enamel is

This is the first study to evaluate the relationship between enamel defects and learning-problem syndromes.

as soon as possible after teeth erupt because defects may become difficult to identify due to excessive wear, caries or restorations. In light of this, our examination of the teeth more than five years after eruption may have been another limitation of this study.

Our failure to find any statistically significant association between HED and learning disorders leads to the inference that the biologic processes contributing appreciably to the occurrence of educational problems tend not to influence tooth enamel development. This is reasonable, but should not be accepted as fact until our findings are confirmed by others.

An alternative inference is that HED and learning disorders are associated, but methodologic considerations limit our perceiving the association. For example, misclassification of children by HED status and by learning capability status would reduce both accuracy and precision, thereby obscuring any association between HED and learning problems. An additional consideration is that the large contribution of inherited phenomena to the occurrence of learning problems (Pennington, 1991) might diminish the opportunity to identify exposures that make a smaller contribution (Tsai and Won, 1984).

Confounding might account for the perceived lack of association between HED and learning disorders, if the epiphenomenon (HED) was negatively associated with a correlate of the outcomes (learning-problem syndromes). This seems unlikely. The major correlates of HED (e.g. preterm birth, trauma, nutritional disturbances, etc.) (Bhat and Nelson, 1989; Needleman *et al.*, 1992) seem more likely to be positively associated with learning problems. Thus, we consider confounding unlikely to account for the absence of an association between HED and learning disorders.

Although our study had lower power than we would have liked, the absence of any appreciable relationship between a learning-problem syndrome (other than the arithmetic syndrome in boys and the directions syndrome in girls) suggests that a moderately larger sample would not have provided nominal statistical significance. Even if these associations did achieve statistical significance, we would be reluctant to make much of them in light of our failure to see any such relationship in the other sex, and because they would have been the only two statistically significant findings out of fourteen individual comparisons. Thus the possibility exists that these observations might reflect random phenomena. Our conclusion that HED and learning problems are not associated is also supported by our failure to find any consistent relationship between HED and repeating a grade or receiving special services.

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Dental management of the child with developmental dyslexia

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Dyslexia most often presents as a biologically determined reading disorder in children of otherwise normal intelligence. Dyslexia may adversely affect academic attainment and self esteem, and the associated frustration and failure may lead to behavioral difficulties. Children with dyslexia are also at significantly higher risk of developing attention-deficit-hyperactivity disorder, asthma, thyroiditis, and inflammatory bowel disease. The presence of these disorders and the medications used to treat them often require modification of standard pediatric dental protocols.

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DYSLEXIA

Children with dyslexia have trouble learning to read in spite of sufficient intelligence, adequate visual and auditory acuity, and appropriate schooling.[1,2] While reading aloud they tend to commit numerous coding errors (omissions, distortions, and word substitutions), and while reading silently their manner is slow and halting.[3,4] On standardized reading tests they score two or three grade levels below expectation. These children may also have difficulty learning the alphabet and multiplication tables and frequently mix the order of letters (most are extremely poor spellers) and numbers while writing. Children with dyslexia commonly have language problems. Often late talkers, they have problems recognizing (phonological coding) individual speech sounds (phonemes) and using them appropriately. They cannot remove the "c" sound from "cow" and say "ow," nor can they count the number of syllables in a word. Verbal memory processes may also be impaired, with the child unable to repeat back an unfamiliar phrase.[5,6] An immature handwriting, a lack of manual dexterity, and poor coordination may accompany these deficits.[7,8] Dyslexia adversely affects academic attainment, occupational choice, social status, and self-esteem. Frustration, failure, withdrawal, and behavioral difficulties can ensue.[9,10]

Children with dyslexia are at significantly higher risk of developing attention-deficit hyperactivity disorder

(ADHD) and certain autoimmune diseases [asthma, hyperthyroidism, hypothyroidism, and inflammatory bowel disease (ulcerative colitis and Crohn's disease)] than their peers.[11,12] The presence of these disorders often requires modification of standard pediatric dental protocols.

Associated Medical Disorders

Attention-deficit hyperactivity disorder afflicts approximately 40 percent of children with dyslexia.[13] In addition to having difficulty reading at grade level, these children also have chronic problems concentrating, restraining movements, and controlling impulses. They do poorly in the classroom setting, where they are extremely distracted by extraneous stimuli, are unable to follow instructions, and lack the necessary persistence to complete assigned tasks.[14,15]

Asthma develops when susceptible children are exposed to inhalant allergens (i.e., dust mites, pollen, mold spores) and their B-cell lymphocytes respond by producing IgE antibodies. These antibodies bind to receptors on the surface of mast cells located in the lungs (adjacent to blood vessels, submucosal glands, muscle bundles, and in the bronchial lumen) and sensitize them. On subsequent exposure to the allergen, sensitized mast cells degranulate and release mediators that reduce small-airway diameter by causing muscle spasm, mucosal edema, and increased mucus secretion. Increased airway resistance traps previously inspired air within the lungs. Severe asthma often interferes with a child's attendance at school and frustrates remedial attempts at reading education.

Grave's disease is a form of hyperthyroidism that occurs when an immune defect precipitates the production of antibodies to thyroid-stimulating hormone (TSH) receptors. These antibodies bind to and stimulate the TSH receptors causing an increase in size and function of the thyroid gland.[17,18] Children with this disorder manifest tachycardia, nervousness, exophthalmos, increased appetite, and weight loss. More than two-thirds of childhood cases occur between the ages of ten and fifteen years. A family history of the disorder is present in up to 60 percent of patients.

Hashimoto's thyroiditis is a form of hypothyroidism that results from an immune defect in which there is the production of antibodies directed against thyroglobulin and the thyroid peroxidase enzyme. This reaction is accompanied by lymphocytic infiltration and varying degrees of thyroid cell atrophy and fibrosis. These changes almost always result in bradycardia and moderate weight gain.

Inflammatory bowel disease is thought to arise when an immune disturbance permits the production of antibodies to the bacteria normally found in the gastrointestinal (GI) tract. The antibodies attack GI epithelial cells and mucin-producing glands, causing inflammation and tissue injury.[19] Ulcerative colitis is characterized by superficial ulcerations of the lining in the rectum and adjacent large intestine. Clinical manifestations include an urge to have repeated bowel movements, diarrhea, and rectal bleeding. Long-standing disease may predispose the development of carcinoma. Crohn's disease is characterized by transmural ulcerations of the terminal ileum and ascending large bowel. Diarrhea, weakness, fatigue, anorexia, and fever are common manifestations of active disease. Approximately 25 percent of children with Crohn's disease suffer an alteration in their growth pattern.

Epidemiology

Dyslexia affects an estimated 3 percent to 10 percent of school-age children in the United States. In most instances early reading difficulties improve over time, however; severe cases may persist into adolescence and adulthood. Historically, it was believed that boys were three times more likely than girls to manifest the disorder, but recent studies show an almost even distribution between the sexes.[20] Familial and genetic studies demonstrate an approximate 40 percent inheritability rate, but the exact mode of transmission remains elusive. Identical twins are concordant for the disorder nearly all the time and fraternal twins 33 percent of time.[21]

Etiology

Anatomical studies (necropsy, computed tomography, magnetic resonance imaging) and neurological assessments of individuals without dyslexia demonstrate that in most instances, the left cerebral hemisphere is larger than the right, is dominant, and is responsible for right-handedness and language function. Among those with dyslexia, however, there is reduced or reversed asymmetry such that the hemispheres are of relatively equal size, and there is an unusually high prevalence of left-handedness. Positron emission tomographic scans demonstrate that individuals with dyslexia (unlike normal readers) are unable to increase blood flow appro-

priately (and increase oxygen use and glucose metabolism) in the left temporal lobe, when challenged to identify paired words that rhyme.[22]

These changes have been hypothesized to arise when unusually high levels of testosterone bathe the fetus *in utero*. Testosterone is suspected of slowing development of the left hemisphere of the brain and allowing compensatory overdevelopment and dominance of the right cerebral hemisphere. This excess of right brain growth may permit anomalous neuronal connections ("miswiring" of the brain) and impair reading skills. Increased testosterone activity likewise may adversely affect development of the thymus gland and lead to a faulty immune system.[23-28]

MEDICAL MANAGEMENT

The comprehensive evaluation of a child with dyslexia often requires the services of a pediatrician and consultants from the fields of pediatric neurology, child psychiatry and psychology, ophthalmology, and audiology. These clinicians search the medical history for evidence of familial patterns and risk factors. Clinical evaluation determines the presence of any local or systemic diseases and includes screening for both vision and hearing deficits. There are no laboratory investigations specific or required for the diagnosis.[29]

In 1975, Congress passed and the President signed the *Education for All Handicapped Children Act* (PL 94-142), which mandated a free and appropriate education in the least restrictive setting for those afflicted with specific learning disabilities (e.g., dyslexia). These educational therapies include small classes, intensive tutoring, and interventions that balance "sight reading" (visual recognition of the whole word and its meaning) and "sounding out" (the phonetic letter-by-letter approach, which emphasizes an awareness of the constituent sounds of words).[30,31] Family therapy, while not mandated by law, is often indicated to assist parents troubled by their child's academic and behavioral problems.

Piracetam, an experimental medication, has been in some studies found to increase alertness, left hemisphere responsiveness, and verbal memory; double-blind, randomized trials are still needed, however, to determine it's clinical usefulness.[32,33] Some clinicians have proposed therapies consisting of sensory stimulation, optometric visual training, vestibular treatments, megavitamins, trace elements (hair analysis), and hypoglycemic diets, but there is little or no scientific evidence to recommend their use.[34]

Medical Management of Associated Disorders

Attention-deficit hyperactivity disorder is usually treated with the administration of stimulant medications such as methylphenidate.[35] These drugs improve the child's ability to focus attention and concentration, decrease motor activity, relieves anxiety, and elevate the patient's morale.[36]

The treatment of asthma varies with the severity of the condition. Children with infrequent episodes of bronchospasm are often given β2-adrenergic agonists by inhalation during acute attacks. Agents such as albuterol, bitolterol, and terbutaline effectively relax bronchial smooth muscle, while producing fewer cardiovascular side effects than do older, nonselective β agonists. Chronic asthma is increasingly being managed by drugs with anti-inflammatory activity. Cromolyn sodium inhibits mast cell degranulation and is a preferred drug in children for maintenance therapy. Although ineffective against acute attacks, inhalation of

Approximately 12 percent of individuals with ulcerative colitis develop painful aphthous ulcerations.

cromolyn has the fewest side effects of any treatment for asthma available. Aerosolized inhaled corticosteroids, such as beclomethasone dipropionate and budesonide, are generally effective in the long-term control of symptoms in patients with chronic asthma. These anti-inflammatory agents inhibit IgE and histamine synthesis and release, and reduce vascular permeability and mucus production. In children, growth retardation is a concern with continuous daily use of inhaled corticosteroids. Oral corticosteroids and theophylline are potentially more toxic and are generally reserved for refractory cases.

Hyperthyroidism is managed in the short term by the administration of methimazole or propylthiouracil to inhibit thyroid hormone synthesis. Acute manifestations of the disease are also controlled by propranolol (which blunts cardiac stimulation and relieves anxiety) and occasionally iodine (which inhibits thyroid hormone release). If spontaneous remission does not follow the use of antithyroid drugs, definitive therapy in the form of radioactive iodine (^{131} I) ablation or subtotal surgical thyroidectomy is indicated. Hypothyroidism is managed by replacement therapy with synthetic or natural preparations of thyroxine and/or levothyronine.

Therapy for inflammatory bowel disease consists of suppression of the inflammatory reaction with sulfasalazine and corticosteroids. Immunosuppressants such as mercaptopurine, may be required for refractory patients.

OROFACIAL FINDINGS

Children with a cleft palate are approximately three times more likely to suffer dyslexia than their peers. For reasons that remain elusive, children with both a cleft palate and cleft lip are not, however, at heightened risk for dyslexia.[37,38]

Some children with dyslexia and concurrent ADHD may manifest numerous oral abnormalities.[39] These anomalies can include a short upper lip and wide mouth, steep palatal vault, fissured tongue, geographic tongue, aberrant frenula, and crowded or malformed teeth.[40,41] Orofacial injury is common because children with ADHD are incessantly running, climbing, and occasionally falling.[42] Injury is unfortunately not always accidental. A hyperactive child is five to seven times more likely to suffer physical abuse from a parent than a child in the general population.[43]

Children with dyslexia and concurrent asthma are often treated with a beta$_2$-adrenergic agonist for management of the respiratory disorder. These medications are associated with a decrease in stimulated whole saliva (by 20 percent) and parotid saliva (by 35 percent) and an increase in the number of lactobacilli. These adverse changes in the oral environment result in an increased caries susceptibility.[44] Children with allergic asthma are also prone to develop maxillary sinusitis.

Children with dyslexia and concurrent hyperthyroidism may manifest premature loss of primary teeth and early eruption of permanent teeth. The thyroid gland is usually enlarged, and exophthalmus may be present. Children with hypothyroidism manifest delayed shedding of the primary teeth, and retarded development (completion of root formation) and eruption of the permanent dentition.

Children with dyslexia and concurrent inflammatory bowel disease in an acute phase may manifest oral ulcerations of varying configurations. Approximately 12 percent of individuals with ulcerative colitis develop painful aphthous ulcerations (10 mm or less in diameter) on the nonkeratinized mucosa of the lips, cheek, oral vestibule, and tongue. Approximately 15 percent of children and adolescents with Crohn's disease also develop aphthous ulcers. Some individuals also develop "cobblestone" surface lesions on the attached gingiva or inflammatory hyperplastic tissue in the mucobuccal fold and retromolar pad. These oral lesions, irrespective of appearance, tend to regress as the bowel disorder is controlled.[45]

DENTAL MANAGEMENT

The pediatric dental patient with known dyslexia must be afforded a comprehensive medical history and dental examination capable of identifying occult autoimmune disease. Specific interventions for those free of compounding medical disorders can be limited to an appreciation of the child's psychosocial well-being and the presence of motor/coordination problems that may interfere with efficient toothbrushing.

Youngsters suffering from dyslexia and ADHD should have their dental appointments scheduled in the morning, when they are least fatigued, most attentive, and best able to remain seated in the dental chair.[46] Morning appointments are also appropriate because most medication regimens target maximal drug effect (enhanced cognition and behavior) to occur during the early part of the school day. Parents or guardians should be reminded to administer the child's medication prior to the office visit, especially if the child is seen during a school holiday or summer vacation. Compliance with an age-appropriate home-care regimen is difficult for

these children. Understanding and adherence are enhanced when colorful or highly stimulating educational materials are employed and instructions are simplified and repeated numerous times during the dental treatment visit. Emphasis should be placed upon the brushing of teeth rather than the specifics of a brushing technique. Undo emphasis on technique is likely to result in cessation of all toothbrushing activities. Methylphenidate therapy is associated with a number of adverse side effects of concern to dentists. Elevation in both systolic and diastolic blood pressures and an increased heart rate are common. Prudent care necessitates obtaining profound local anesthesia, thus limiting pain and the endogenous production of epinephrine, which may exacerbate these findings. An aspirating syringe must be used in order to avoid an intravascular injection and the possibility of an adverse interaction between the pressor agents (epinephrine, levonordefrin) used with local anesthesia and methylphenidate.

Children with dyslexia and concurrent asthma may be at risk of severe adverse reactions to medications commonly used in dental practice. Aspirin, other nonsteroidal anti-inflammatory agents (e.g., ibuprofen) and sulfite antioxidants used to preserve the vascostrictors found in dental anesthetic solutions can trigger an allergic asthma attack and significantly reduce pulmonary function. Acetaminophen and local anesthetic agents without a vasoconstrictor are recommended for these individuals.[43,49] Nitrous oxide sedation is a satisfactory adjunct for those patients whose asthma is worsened by emotional upset; children with obstructed nasal passages or enlarged nasal membranes, however, may find breathing through a nasal mask difficult. Administration of an antihistamine before beginning dental treatment is an alternative approach. These medications open airways and sedate the anxious child. Deep sedation should be avoided because asthmatics have reduced control of their airway. Opioid agents should also be avoided because of their respiratory depressant effects and, with some drugs (e.g., meperidine, morphin) their propensity to release histamine.[50]

Children with dyslexia and concurrent *uncontrolled* hyperthyroidism should not receive routine dental care. Local anesthetic agents containing epinephrine or other pressor amines can theoretically precipitate severe tachycardia, and possibly a fatal arrhythmia. Emergency care should only be provided in a hospital, and routine care should await control of the disorder. Children receiving propylthiouracil are at risk of developing drug-induced agranulocytosis and hypoprothrombinemia. When planning oral surgery for these patients, a complete blood count with a "differential" and a prothrombin time must be included in the presurgical evaluation.

Children with dyslexia and concurrent hypothyroidism are hyperresponsive to even small doses of analgesics, anesthetics, antianxiety drugs, and sedative-hypnotics because of central nervous system depression and a lowered metabolic rate. Consultation with the patient's pediatrician regarding proper dosing and venue of dental procedures is advisable. Thyroid supplements used in excess may sensitize the heart to exogenous epinephrine. Two cartridges containing 1/100,000 epinephrine (0.036 mg) should be the maximum dose administered at each dental appointment.[51,52]

Children with dyslexia and concurrent inflammatory bowel disease may require shorter appointments and nitrous oxide sedation to reduce stress. Evaluation of the patient's hemoglobin and hematocrit levels is indicated prior to oral surgery, because anemia may have arisen secondary to chronic GI blood loss. A white blood cell count and "differential" are indicated for those children receiving immunosuppressive therapy, because of

Children with dyslexia and ADHD should be scheduled for morning appointments.

the potential for bone-marrow suppression. Patients concurrently receiving corticosteroids or those with a history of therapy within the past year may require supplementation to avoid an adrenal crisis brought about by the stress of dental care. The child's pediatrician should be consulted as to the need for administering additional medication.

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DYSLEXIA

Does your adolescent have reading problems that cause him to reverse letters or numbers or see them upside down? Does he read at a slow rate, really struggle to decode words, or continually misspell fairly simple words?

These kinds of problems fall into a category commonly called dyslexia, which is a specific reading disorder. Even though dyslexia is more likely to be identified properly than many other learning deficiencies, it still sometimes goes undetected by both teachers and parents unless the child undergoes specific diagnostic tests.

What causes dyslexia? According to some specialists it may be due to a combination of delayed developmental factors—physical, emotional, motivational—that cumulatively influence the central nervous system and impede the brain's capacity to interpret the written word properly. In many cases it may run in families.

Whatever the cause, the symptoms are quite familiar to most teachers. A dyslexic adolescent often:

- confuses the order of letters in words
- doesn't look carefully at all the letters in a word, guessing what the word is from the first letter
- loses his place on a page while reading, sometimes in the middle of a line
- doesn't remember common words he's learned from one day to the next
- has no systematic way to figure out a word he doesn't know; instead, he guesses or says, "I don't know"
- reads word by word, struggling with almost every one of them
- reads without expression and ignores punctuation
- reads very slowly and tires significantly from reading
- adds, deletes, or substitutes words in a sentence

If your adolescent's teachers suspect dyslexia, make sure that any formal evaluation rules out other possible problems. For instance, does he have difficulties with his eyesight? Are there family problems that might be placing strain on his learning ability? Has he developed slowly on an emotional level?

Once dyslexia is diagnosed, several treatment options are available, and adolescents often make good progress.

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Effect of a preventive approach for the treatment of nursing bottle caries

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Nursing-bottle caries, also known as baby-bottle tooth decay, is a dental caries pattern associated most often with prolonged feeding with milk or sweetened liquids in a nursing-bottle. Despite considerable advances in the prevention of dental caries, nursing-bottle caries continues to have high prevalence, especially in certain cultures.[1,2] Preventive approaches, such as counseling parents to wean children by age one, progressively diluting the liquid in the nursing bottle with water, and modifying the parents' behavior of giving a nighttime bottle to their children, have been suggested.[3-5]

The purpose of this study was to determine whether a preventive approach consisting of instructions on the use of the bottle and daily fluoride treatment could arrest the progression of nursing-bottle caries.

MATERIALS AND METHODS

After obtaining informed consent forms from the caretakers, seventeen children between the ages of twenty-one and thirty-six months with incipient, untreated nursing-bottle caries lesions (white/brown spots on enamel and/or small cavitations in dentin on one or more teeth) were recruited at the Pediatric Dental Clinic at the University of Connecticut Health Center in Farmington and at the Pediatrics Clinic at Burgdorf Health Clinic in Hartford. Inclusion criteria were based on evidence of nursing-bottle caries pattern and on a history of the child sleeping with a bottle. Children with hypoplastic enamel defects, but no carious component to the lesion, were not included in the study.

At baseline, a clinical examination was performed to evaluate the lesions (teeth involved, surfaces, size, color and consistency). The affected teeth of each subject also were photographed using an intraoral macro lens and color prints were made from the negatives. Each child was sampled for salivary mutans streptococci levels using a modification of the Kohler and Bratthall method.[6] This microbial screening test involved sampling saliva from each child by having the child moisten a sterile wooden tongue depressor in his/her mouth. The moistened tongue depressor was then impressed onto plates with growth media selective for mutans streptococci.[7] After two days of incubation (35°C) in a CO_2 enriched environment, the numbers of mutans streptococci colony forming units were recorded semi-quantitatively as zero, low (1-9), moderate (10-99), or high ($\geq$100).

After the clinical parameters were gathered, the caretakers were verbally administered questionnaires regarding their child's oral hygiene and nursing habits;

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and the caretaker's oral health status, demographic characteristics, and knowledge of dental prevention.

This initial visit was concluded by giving the caretakers specific instructions to brush their children's teeth with a "pea size" amount of 0.4 percent SnF_2 gel (Gel Kam, Dallas, TX) applied to the affected teeth twice a day after regular tooth brushing. Frequent use of stannous fluoride is known to reduce the numbers of mutans streptococci in the oral cavity.[8] The caretakers also were instructed to wean their children from the nighttime use of the nursing-bottle or to substitute water for the liquid in the bottle. Written instructions for gel use and weaning were provided to each participant. All subjects were contacted by phone between data collection visits for the express purpose of reinforcing the importance of complying with the regimen.

Children were placed on a three- and six-month recall schedule. At three months the children's teeth were rephotographed, and microbial screenings again performed. A self-evaluation questionnaire assessing the use of the 0.4 percent SnF_2 gel was administered and caretakers were instructed to return the unused portion of the SnF_2 gel. The procedures at six months were to be identical to those performed at three months.

RESULTS

The study was terminated after three months because it was apparent that carious lesions increased in number and severity in most of the children during this interval. In addition, analysis of the preliminary compliance data revealed that the caretakers generally were not complying with the study protocol. Thus it was felt that the risks of continuing the study were high and the benefits to the children would be low.

Of the seventeen children, eleven children were male and six were female, with eleven being the first born. Twelve children came from single caretaker households with the majority of these family units consisting of a mother with one child (Table 1).

All children reportedly were bottle fed and thirteen were using the bottle most often at night. When asked before the study about awareness of potential cariogenicity of sleeping with a nursing-bottle, twelve respondents answered positively. The caretakers initially reported that brushing of their child's teeth started at an average age of ten months, with ten caretakers indicating that the child's teeth were brushed once a day. In all cases, caretakers reported that they were the ones who did the brushing (Table 2).

After three months only seven caretakers reported that their respective children completely stopped the bottle habit, and only two caretakers reportedly followed the requested regimen of twice daily brushing with SnF_2 gel (Table 3). The photographs reveal obvious caries progression in most of the children. Two children who reportedly stopped the bottle habit and were using the fluoride gel as prescribed had no caries progression and their mutans streptococci counts were

Table 1. ☐ Demographic data of the seventeen children with nursing caries and their families.

Sex	11
Male	
Female	6
Age	
Mean	31.5 months
Range	21-36 months
Sibling rank	
1	11
2	3
3	3
Who cares for the child?	
Mother	12
Grandmother	1
Other relative	4

Table 2. ☐ Reported brushing and nursing habits of the seventeen children.

Age brushing started	
Mean	10.5 months
Range	6-18 months
Who brushes child's teeth?	
Mother	17
Child	0
How many times a day?	
Less than 1	0
1	10
2	5
3	1
More than 3	1
Nursing practices:	
Bottle during the day	4
Bottle during the night	13
Breast-fed	0

Table 3. ☐ Self reported compliance of the caretakers with discontinuing nursing habit and use of fluoride gel.

Substitution of water in bottle	
Attempted	5
Rejected	7
Not attempted	5
Completely stop use of the bottle	
Yes	7
No	10
Fluoride Gel	
Used twice a day as instructed	2
Used once a day	7
Used less than once a day	4
Did not use	2
Used other toothpaste	2

reduced from high to moderate (Figure 1). None of the caretakers returned the gel at the three-month visit as instructed.

DISCUSSION

Children with a prolonged nursing habit most often have been found to come from single caretaker homes of low socioeconomic status.[9-11] The present study conforms with such a profile in that most of the children were from single caretaker households, and the caretakers were regarded to be in low socioeconomic levels.[12] That the subjects involved in this study were predominantly firstborn children suggests that prolonged nursing habit may be higher in firstborns, when caretaker knowledge of, and experience in, child rearing is limited.

It is generally recommended that by one year of age, a child should be weaned from either the breast or the bottle[13] The mean age of children in this study was 31.5 months and all of them were still using the bottle, most frequently at night. The finding that twelve out of the seventeen caretakers admitted being aware, before the study, of the potential cariogenicity of sweet liquids in the bottle suggests that simply informing the caretakers of the hazard of a behavior is not enough to prevent that behavior.[2,14] Overindulgence or lack of parental control has been associated with nursing caries.[10] The present study confirms that at least in the sample we studied, there may be a lack of persistence and control of the children by the caretakers.

Compliance is an important consideration in the design and implementation of any preventive program. The three compliance indicators in this study, mutans streptococci scores, self reports on utilization of the fluoride gel, and returning the bottle of fluoride gel, were consistent in indicating that in general the caretakers in this study could not or would not comply with the preventive regimen.

The lack of compliance with the preventive instruction hindered this investigation from examining the effectiveness of a preventive program for nursing-bottle caries. Perhaps the traditional approaches that dental professionals use to affect behavioral changes, i.e., education and instruction, may not be effective in altering inappropriate habits or improving preventive behaviors in some populations, especially in those that are not health-oriented. Additional methods to induce behavioral change, such as self-efficacy training or behavioral modification techniques, may be necessary. Alternatively, preventive interventions that do not rely on patient compliance, such as frequent professional

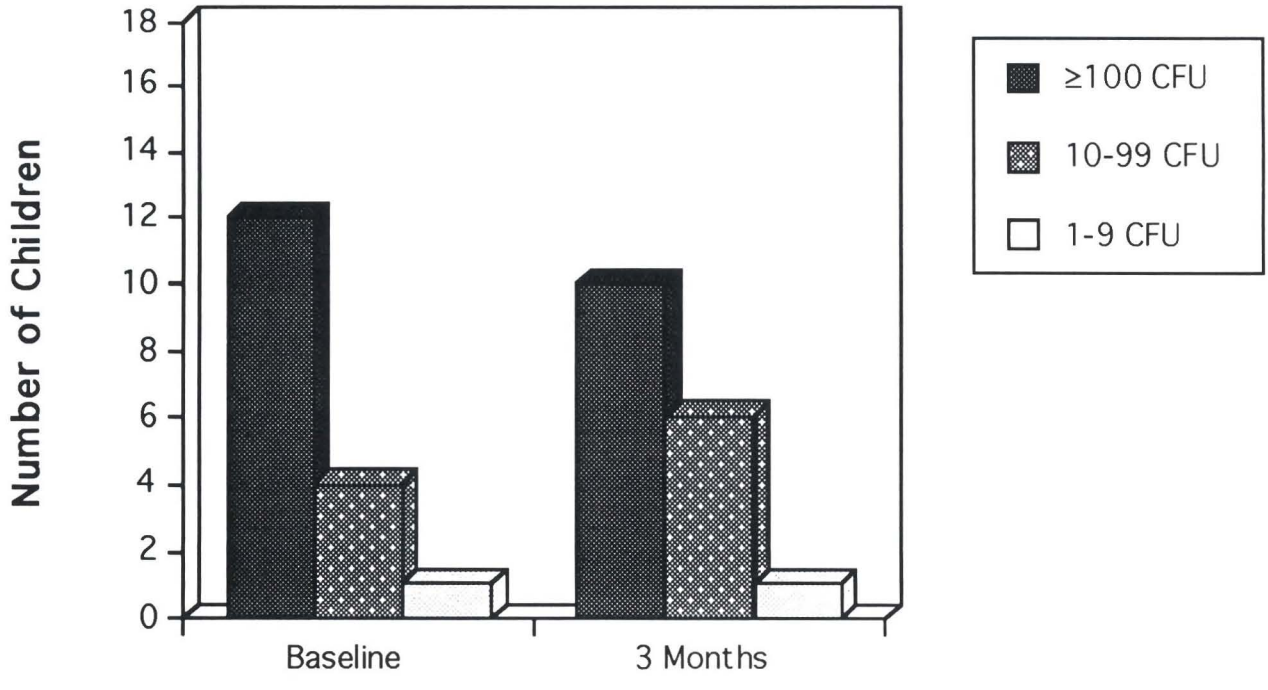

Figure. Change in salivary mutans streptococci levels in children over a three-month period.

fluoride treatment or daily supervised use of fluoride gels, may need to be considered to reduce caries incidence in such high risk groups.

CONCLUSIONS

- ☐ This study, which attempted a preventive intervention for the treatment of nursing-bottle caries, was discontinued after three months because caries noticeably progressed in most of the children, and only two caretakers complied fully with the prevention program.
- ☐ Two children, who reportedly stopped the bottle habit and were using the fluoride gel as prescribed, had no caries progression and their mutans streptococci counts were reduced from high to moderate.
- ☐ The traditional approaches of education and instruction may not be effective in correcting inappropriate habits or improving preventive behaviors in certain high caries-risk populations. Interventions that do not rely as much on patient compliance may need to be considered as preventive approaches in these situations.

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EFFECT OF XYLITOL CHEWING GUM: FIVE YEARS LATER

About 65 percent of the original 258 children who participated in 1982-1984 in a caries prevention program involving the use of xylitol chewing gum were retrieved in 1989 for a follow-up study. Ninety-five subjects from the original xylitol (X) group and 70 subjects from the original control (no-gum, C) group were available. In 1984, when the children completed the program at the age of 13-14 years, the caries scores were significantly lower in children who had used xylitol gums daily, compared with the C group. In 1989, five years after the discontinuation of the gum program, the difference between the X and C groups had continued to increase in favor of the X group. These effects were explained by assuming that the X gum program had facilitated the establishment of a low-virulent bacterial flora on the surfaces of the teeth, and especially on those teeth that erupted during the trial proper. This type of results are possibly helpful when evaluating cost-benefit ratios of caries prevention.

Isogangas, P. *et al*: Long-term effect of xylitol chewing gum in the prevention of dental caries: A follow-up 5 years after termination of a prevention program. Caries Res, 27:495-498, November-December 1993.

Updating the changing number and distribution of pediatric dentists: 1982-1991

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The dental profession in general, and pediatric dentistry in particular, finally may have begun to get the message out that the '...job is far from finished and the future of pediatric dentistry is most favorable.'"[1]

The recent publication of the American Dental Association (ADA) report, "Distribution of Dentists in the United States by Region and State: 1991" is the latest in a series of periodic surveys to track the evolving general picture of dental personnel on a regional and state-by-state basis.[2] An earlier presentation in the *Journal of Dentistry for Children* that compared the then available data, reviewed pediatric practitioner personnel during the first half of the 1980s.[3] Available data now permit a review through 1991 on the number and distribution of 1) professionally active pediatric dentists,* and 2) "younger" (less than 40 years of age) and "older" (40 years or more) pediatric dentists in private practice.

NUMBER OF PEDIATRIC DENTISTS

Between 1982 and 1987 there was an increase of only 140 professionally active pediatric dentists (4.7 percent increase). Between 1987 and 1991, there was, however, an increase of 374 pediatric dentists (12.1 percent increase). Overall between 1982 and 1991 the number of pediatric dentists increased from 2,949 to 3,463. The ratio of pediatric dentists per 100,000 children (less than 18 years of age) increased from 4.7 to 5.3 pediatric dentists per 100,000 children (Table 1).

By region

In 1982, the Mountain region (AZ, CO, ID, MT, NV, NM, UT, WY) had the smallest number of pediatric dentists (144), the Pacific region (AK, CA, HI, OR, WA) had the greatest number (593). By 1991, in each of the nine regions in the nation there were increased numbers of pediatric dentists. Once again, the Mountain region had the smallest number of pediatric dentists (162) and the Pacific region had the greatest number (621) (Table 1).

By state

In 1991, the number of pediatric dentists ranged from four in North Dakota and Wyoming to 253 in New York and 461 in California. Between 1982 and 1991, in forty-two states and the District of Columbia, there were increases in the number of pediatric dentists. During the same period, the number of pediatric dentists decreased in six states (IA, KS, MI, MT, OK, VT) and remained the same in two states (NC, WY) (Table 2).

* Includes clinical practitioners, dental school faculty or staff, armed forces dentists, government employed dentists at the federal, state or local levels, interns and residents, and other health or dental organization staff members.

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Table 1. ☐ Number of professionally active pediatric dentists, number per 100,000 children (<18 years of age) by region and state: 1982, 1987, 1991.[2,4]

Region & State	Number pediatric dentists			Change in number	Number of pediatric dentists per 100,000 children < 18 yrs.	
	1982	1987	1991	1982 to 1991	1982	1991
New England	204	222	263	59	6.5	8.5
Connecticut	57	71	84	27	7.2	11.1
Maine	9	8	10	1	2.9	3.2
Massachusetts	113	112	138	25	7.8	10.0
New Hampshire	8	12	11	3	3.1	3.9
Rhode Island	10	13	14	4	4.3	6.1
Vermont	7	6	6	−1	5.0	4.1
Middle Atlantic	400	467	545	145	4.3	6.0
New Jersey	87	112	125	38	4.6	6.8
New York	181	208	253	72	4.0	5.8
Pennsylvania	132	147	168	36	4.0	5.9
South Atlantic	445	497	567	122	4.5	5.2
Delaware	5	9	9	4	3.1	5.3
Dist. of Columbia	11	17	22	11	7.9	18.1
Florida	111	137	143	32	4.2	4.8
Georgia	69	84	94	25	4.2	5.3
Maryland	77	77	107	30	6.9	8.9
North Carolina	63	61	63	–	3.9	3.8
South Carolina	34	37	41	7	3.7	4.4
Virginia	65	67	76	11	4.5	4.9
West Virginia	10	9	12	2	1.8	2.7
East South Central	190	213	219	29	4.5	5.4
Alabama	55	60	58	3	4.9	5.4
Kentucky	52	57	61	9	4.9	5.9
Mississippi	18	23	24	6	2.3	3.2
Tennessee	65	74	76	11	5.2	6.1
East North Central	458	467	510	52	3.9	4.6
Illinois	115	112	143	28	3.7	4.8
Indiana	81	94	85	4	5.2	5.8
Michigan	83	81	79	−4	3.2	3.2
Ohio	123	127	137	14	4.1	4.9
Wisconsin	56	54	66	10	4.3	5.0
West North Central	201	181	215	14	4.2	4.5
Iowa	48	43	47	−1	6.0	6.5
Kansas	29	25	20	−9	4.5	3.0
Minnesota	41	40	50	9	3.6	4.2
Missouri	45	44	55	10	3.4	4.1
Nebraska	33	22	34	1	7.5	7.8
North Dakota	2	3	4	2	1.0	2.3
South Dakota	3	4	5	2	1.5	2.5
West South Central	314	344	361	47	4.2	4.7
Arkansas	19	24	27	8	2.9	4.3
Louisiana	55	62	61	6	4.1	4.9
Oklahoma	39	36	35	−4	4.4	4.1
Texas	201	222	238	37	4.4	4.8
Mountain	144	148	162	18	4.1	4.1
Arizona	32	34	39	7	3.9	3.9
Colorado	49	55	58	9	6.0	6.6
Idaho	8	8	9	1	2.6	2.8
Montana	15	9	7	−8	6.5	3.1
Nevada	12	9	13	1	5.2	4.0
New Mexico	12	15	15	3	2.8	3.3
Utah	12	14	17	5	2.1	2.6
Wyoming	4	4	4	–	2.6	2.9
Pacific	593	552	621	28	6.8	5.8
Alaska	4	8	12	8	2.9	6.7
California	457	411	461	4	7.0	5.6
Hawaii	22	27	32	10	7.9	11.1
Oregon	44	43	48	4	6.2	6.4
Washington	66	63	68	2	5.8	5.2
United States	2,949	3,089	3,463*	514	4.7	5.3

*Total differs by one pediatric dentist from total in report

Table 2. ☐ High and low range and change in professionally active pediatric dentist counts by state: 1982, 1991.[2]

	1982		1991	
	Number of pediatric dentists			
High	California	457	California	461
	Texas	201	New York	253
	New York	181	Texas	238
Low	Alaska	4	Vermont	6
	Wyoming	4	South Dakota	5
	South Dakota	3	North Dakota	4
	North Dakota	2	Wyoming	4
	Pediatric dentists per 100,000 children			
High	Hawaii	7.9	Dist. of Col.	18.1
	Nebraska	7.5	Connecticut	11.1
	California	7.0	Hawaii	11.1
Low	West Virginia	1.8	Utah	2.6
	South Dakota	1.5	South Dakota	2.5
	North Dakota	1.0	North Dakota	2.3

	Change between 1982 and 1991	
	Number of pediatric dentists	Pediatric dentists per 100,000 children
Increase	42 states Dist. Columbia	39 states Dist. Columbia
Decrease	6 states	9 states
No change	2 states	2 states

RATIO OF PEDIATRIC DENTISTS TO THE NUMBER OF CHILDREN

By region

In 1982, the East North Central region (IL, IN, MI, OH, WI) had the lowest ratio of pediatric dentists to the number of children (3.9 per 100,000 children). The Pacific region had the highest ratio (6.8 pediatric dentists per 100,000 children). By 1991, the Mountain region had the lowest ratio (4.1 pediatric dentists per 100,000 children) and the New England region (CT, ME, MA, NH, RI, VT) had the highest ratio (8.5 pediatric dentists per 100,000 children).

It is significant to note that the Pacific region had a decrease in the ratio of pediatric dentists to the number of children between 1982 and 1991 (from 6.8 to 5.8 pediatric dentists per 100,000 children). In 1991, the Middle Atlantic region (NJ, NY, PA) with 6.0 pediatric dentists per 100,000 children had a higher ratio than the Pacific region (Table 1).

By state

In 1991, at the state level, there continued to be major differences in the ratio of pediatric dentists per number of children. The range was from 2.3, 2.5 and 2.6 per 100,000 children, respectively, in North Dakota, South Dakota and Utah, to 11.1 per 100,000 children in Connecticut and Hawaii and 18.1 per 100,000 in the District of Columbia.

There were marked changes at the state level between 1982 and 1991 in the ratio of pediatric dentists per number of children. For example:

Increases

Alaska, from 2.9 to 6.7

Connecticut, from 7.2 to 11.1

Hawaii, from 7.9 to 11.1

District of Columbia, from 7.9 to 18.1 pediatric dentists per 100,000 children.

Decreases

Kansas, from 4.5 to 3.0

Montana, from 6.5 to 3.1 pediatric dentists per 100,000 children.

Because of the great number of children in the State of California, however, (in 1991, there were 8.2 million or 12.5 percent of all U.S. children less than eighteen years of age) one of the more important developments in the availability of pediatric dentists was the decrease in the ratio of pediatric dentists per number of children (between 1982 and 1991, from 7.0 to 5.6 pediatric dentists per 100,000 children).

Numerically, between 1982 and 1987 there was a decrease of forty-six and then an increase of fifty pediatric dentists between 1987 and 1991. Thus at the beginning and end of the overall period, at a time when the number of children in the State of California increased from 6.5 to 8.2 million, the total number of pediatric dentists remained the same (457 vs. 461) (Table 1). (See the following section on age of private practice pediatric dentists for a further discussion of developments in the State of California.)

PEDIATRIC DENTISTS BY AGE

The ADA report on the 1991 distribution of dentists by region and state (for the first time) dichotomized specialist private practitioners in "younger" (less than age forty) and "older" (forty years and older) categories. Of the 2,948 private practice pediatric dentists, 825 (or 27.9 percent) were less than forty years of age.[2]

By region

In seven of the nine regions there were minor differences between the percent of younger pediatric dentists in the particular region and the national average (27.9 percent). In the Middle Atlantic region, one third (33.8 percent) of pediatric dentists were less than age forty, 20.1 percent in the Pacific region (Table 3).

Table 3. ☐ Number and percent private practice pediatric dentists by age, region and state: 1991.[2,4]

	Number & percent of pediatric dentists in region & state				Percent of ALL pediatric dentists in country	
	Under age 40		Age 40 and over	Total	Under age 40	Age 40 and over
Region & State	Number	Percent	Number	Number	Percent	Percent
New England	62	26.7	170	232	7.5	8.0
Connecticut	21	29.5	50	71	2.5	2.3
Maine	3	33.3	6	9	0.4	0.3
Massachusetts	32	26.4	89	121	3.9	4.1
New Hampshire	3	27.2	8	11	0.4	0.3
Rhode Island	3	21.4	11	14	0.4	0.5
Vermont	0	0.0	6	6	0.0	0.2
Middle Atlantic	152	33.8	297	449	18.4	13.9
New Jersey	32	29.0	78	110	3.9	3.7
New York	75	38.2	121	196	9.0	5.7
Pennsylvania	45	31.4	98	143	5.4	4.6
South Atlantic	128	27.8	332	460	15.5	15.6
Delaware	3	37.5	5	18	0.4	0.1
Dist. of Columbia	7	46.6	8	15	0.8	0.4
Florida	37	28.2	94	131	4.5	4.4
Georgia	20	27.3	53	73	2.4	2.5
Maryland	22	27.1	59	81	2.6	2.8
North Carolina	13	26.5	36	49	1.6	1.7
South Carolina	7	20.5	27	34	0.8	1.3
Virginia	19	30.6	43	62	2.3	2.0
West Virginia	1	14.2	6	7	0.1	0.3
East South Central	54	28.7	134	188	6.5	6.3
Albama	8	16.0	42	50	1.0	1.9
Kentucky	14	31.8	30	44	1.7	1.4
Mississippi	7	29.1	17	24	0.8	0.8
Tennessee	25	36.2	44	69	3.0	2.1
East North Central	134	30.1	311	445	16.2	14.6
Illinois	4	33.6	83	125	5.1	3.9
Indiana	21	25.6	61	82	2.5	2.9
Michigan	17	26.9	46	63	2.1	2.2
Ohio	38	32.4	79	117	4.6	3.7
Wisconsin	16	27.1	43	59	1.9	2.0
West North Central	54	30.3	124	178	6.5	5.8
Iowa	11	28.9	27	38	1.3	1.3
Kansas	2	11.7	15	17	0.2	0.7
Minnesota	12	29.2	29	41	1.4	1.4
Missouri	15	31.9	32	47	1.8	1.5
Nebraska	9	36.0	16	25	1.1	0.8
North Dakota	2	50.0	2	4	0.2	0.1
South Dakota	3	60.0	2		0.4	0.1
West South Central	94	30.0	219	313	11.4	10.3
Arkansas	9	34.6	17	26	1.1	1.1
Louisiana	14	25.4	41	55	1.7	1.9
Oklahoma	4	14.8	23	27	0.5	1.1
Texas	67	32.6	138	205	8.1	6.5
Moutain	40	27.9	103	143	4.8	4.8
Arizona	6	20.6	23	29	0.7	1.1
Colorado	16	30.7	36	52	1.9	1.7
Idaho	2	22.2	7	9	0.2	0.3
Montana	2	28.5	5	7	0.2	0.2
Nevada	5	38.4	8	13	0.6	0.4
New Mexico	3	23.0	10	13	0.4	0.5
Utah	6	37.5	10	16	0.7	0.5
Wyoming	0	0.0	4	4	0.0	0.2
Pacific	109	20.1	432	541	13.2	20.3
Alaska	1	20.0	4	5	0.1	0.2
California	87	20.8	331	418	10.5	15.6
Hawaii	8	29.6	19	27	1.0	0.9
Oregon	8	20.5	31	39	1.0	1.5
Washington	5	9.6	47	52	0.6	2.2
United States	825	27.9%	2,123	2,948	100.0%	100.0%

Except for the Middle Atlantic and Pacific regions, there were minor differences in the other regions between the number of younger and older pediatric dentists as a percent of all pediatric dentists in the respective age-groups (Table 3).

Middle Atlantic region: 18.4 percent of all younger pediatric (NJ, NY, PA) dentists 13.9 percent of all older pediatric dentists;

Pacific region: 113.2 percent of all younger pediatric (AK, CA, HI, OR, WA) dentists; 20.3 percent of all older pediatric dentists.

In terms of practitioner age differences reviewed from within and between regions, in 1991:

The Middle Atlantic had a relatively greater concentration of younger pediatric dentists than any other region;

The Pacific region had a relatively greater concentration of older pediatric dentists than any other region (Table 3).

By state

In 1991, there was a wide range by state in the percent of pediatric dentists that were under forty years of age, from 46.6 percent, 38.4 percent and 38.2 percent, respectively, for the District of Columbia, Nevada and New York, to 14.2 for West Virginia, 11.7 percent for Kansas and 9.6 percent for Washington. There were no private practice pediatric dentists under the age of forty in Vermont and Wyoming (Table 3).

The particular changes in the State of California should be considered separately because of the numbers of pediatric dentists within its jurisdiction. In 1991, there were 418 private practice pediatric dentists (14.1 percent of the national total and more than double the number in Texas, the state with the next highest total [205 private practice pediatric dentists]). Between 1982, 1987 and 1991, (during a period when there was an increase of 1.7 million children) the number of private practice pediatric dentists decreased from 419 to 379 and then rebounded to virtually the same count in 1991 (418)[2]**. In addition, in 1991 younger pediatric dentists represented 20.8 percent of all pediatric dentists (compared to the national average of 27.9 percent) (Table 3). Undoubtedly selecting a practice location is more complex than the ratio of the number of dentists and children. "The decision to locate a dental practice and live in one region, state, community street or even building, is based upon a highly complex series of personal, family, cultural, economic and a seemingly infinite series of other interrelated variables.[7]

PROSPECTS FOR THE FUTURE NUMBER OF PEDIATRIC DENTISTS

"A general slowing down in the increasing numbers of pediatric dentists and an essentially constant pediatric dentist-to-population ratio should continue in the future."[3]

**Discussions with representatives of the ADA, and the American Academy of Pediatric Dentistry and its California Chapter were unable to provide a satisfactory explanation (other than possible survey difficulties) for the decrease in the number of pediatric dentists in 1987. Nevertheless, the fact does remain that the reported number of pediatric dentists in 1982 and 1991 remained essentially unchanged during a period of marked increases in the number of children within the state.

Downturns are expected to continue in the ratio of overall number of dentists to the general population and a numeric downturn in the number of dentists during the final years of this decade.

That was one of the earlier conclusions by this writer, based upon the results of

- ☐ The 1987 ADA report on the distribution of dentists,
- ☐ The enrollment patterns in pediatric training programs during the first two-thirds of the 1980s. Specifically, between 1982 and 1987 the ratio of pediatric dentists to the number of children had increased slightly from 4.7 to 4.9 pediatric dentists per 100,000 children (Table 4). During the same period, there were minimal changes in the number of first-year enrollments in pediatric training programs (Table 5).

The more recent data indicate

- ☐ A somewhat faster increase in the ratio of professionally active pediatric dentists to the number of children (reaching 5.3 pediatric dentists per 100,000 (Table 4),
- ☐ Continuing limited changes (through the early 1990s) in the number of first year enrollees in pediatric training programs (Table 5).***

The 1991 Survey provided an initial count of "younger" and "older" pediatric dentists. Further comparative age specific data from subsequent surveys will provide necessary information to determine the "aging" of the population of pediatric dentists and the potential availability of practitioners.

Table 4. ☐ Pediatric dentists per 100,000 children.[2,4]

	1982	1987	1991
Number of professionally active pediatric dentists	2,949	3,089	3,463
Number of children (< 18 yrs) (in millions)	62.6	63.0	65.1
Number of professionally active pediatric dentists per 100,000 children	4.7	4.9	5.3

Table 5. ☐ Number of students enrolled in first year of pediatric training programs: selected years 1972–1992.[5,6]

Year*	First year enrollment
1972	163
1974	177
1976	165
1978	173
1980	190
1982	158
1984	164
1986	152
1988	162
1990	161
1991	177
1992	161

* Represents the start of the academic

OUTLOOKS

"Tracking the numbers of pediatric dentists at the national and state levels provides a necessary but only a partial appreciation of developments in the specialty. Far more in depth reviews at a community level are essential to develop a greater understanding of local exigencies."[3]

- ☐ Increasing numbers of children in the population that traditionally have been underserved.
- ☐ Increasing use of dental services by young children.[9]

The question of determining the adequacy of the number of pediatric dentists (and the profession in general) to provide necessary services is beyond the scope of the ADA surveys. Nevertheless, the reports provide directional insight for necessary manpower forecasts. For example: current projections indicate a continuing downturn (since 1987) in the ratio of the overall number of dentists to the general population and a numeric downturn in the number of dentists during the final years of the current decade.[10]

The need is for comparable ongoing forecasts for the various dental specialties. Until that time, ADA surveys and their retrospective analysis will continue to be the basis for much of the decision making process for pediatric dentistry manpower determination.

But if the profession is to reach traditionally underserved populations, (and these are very same populations that the Bureau of the Census is projecting to experience marked growth during the next decades) does it not seem reasonable for us to develop a system of forecasting the availability of pediatric dental personnel to provide the necessary care?[11]

*** See a previous presentation in the *Journal of Dentistry for Children* for a detailed review of the changing interest in pediatric dentistry.[1]

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CARIES-PREVENTIVE EFFECT OF TWO FLUORIDE CONCENTRATIONS

Fluoride uptake data have shown that amine fluoride (and APF) deposits more fluoride in enamel than NaF or SnF_2 [Gron, 1977; Kirkegaard, 1977; Mellberg, 1990]. ten Cate [1990] concluded that fluoride from depots in porous parts of the dentition and plaque may gradually redissolve. The results of this present study suggest that monthly applications of amine fluoride, even with a lower fluoride concentration, ensure that enough fluoride is present for the direct or indirect interaction with the de- or remineralization components of the caries process and thereby reduce its progression in the deciduous dentition.

The use of preparations with lower fluoride concentrations could further increase the safety of professional topical fluoride applications in kindergarten children.

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Social security and providing for our children

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Social Security ... is a relatively unknown but crucial component in the public sector effort to aid children."[1]

"(In the final month of 1991) the Social Security Administration ... provided $970 million in benefits to nearly 3 million children under the age of 18. That's nearly as much money as the Aid to Families with Dependent Children (AFDC) program provides to children in a typical month."[1]

The national Old-Age, Survivors and Disability Insurance (OASDI) program, generally referred to as Social Security, is the largest income-maintenance program in the United States. The OASDI program provides monthly benefits to retired and disabled workers, to their dependents and to survivors of insured workers. Retirement benefits were established by the original Social Security Act of 1935.

While most people consider Social Security to be a program for the aged (particularly with the addition of Medicare in 1965 for the sixty-five and over population), the Social Security Act and its subsequent amendments include a wide range of services. For example, $17.5 billion in federal and state funds were expended in 1989 to 3.8 million families (with an average monthly payment of $383) under the Aid to Families with Dependent Children (AFDC) program.[2]* The OASDI program under the Social Security Administration was broadened to include benefits for dependents and survivors (in 1939), the disabled (in 1956) and dependents of disabled workers (in 1958).

Since 1972 the federally administered Supplemental Security Income (SSI) program (which replaced the Old Age Assistance, Aid to the Blind and Aid to the Permanently and Totally Disabled programs) provides monthly benefits to individuals who meet specific criteria.**

While the media emphasizes the "Old Age" (OA) retirement component of the OASDI Social Security program, almost 20 percent of the 39 million people (including almost two million children) who receive

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*AFDC is a program for aid to dependent children. "A dependent child is defined as a child under age 16 who is deprived of parental support or care by reason of the death, continued absence from home, or physical or mental incapacity of a parent, and who is living with a father, mother, grandparent, brother, sister, stepparent, stepsister or stepbrother, uncle, or aunt in a residence maintained by such relative as his or her home."[2]

**In addition to financial regulations, (see below) an individual may qualify for payments on the basis of age, blindness or disability. *Aged*: Any person aged 65 or older. *Blindness*: Any person with 20/200 or less vision in the better eye with the use of correcting lenses, or with tunnel vision of 20 degrees or less. *Disabled*: Any person unable to engage in any substantial gainful activity by reason of any medically determinable physical or mental impairment expected to result in death or that has lasted or can be expected to last for a continuous period of at least 12 months. For a child under age 18, eligibility is based on disability or severity comparable with that of an adult.[2]

OASDI benefits receive "Survivor" (S) benefits. The survivor benefit program is so extensive that 98 percent of all children could get benefits, if a working parent should die.[1]

And further, in 1990, more than a third of a million children received in excess of $1.6 billion in benefits under the SSI program for the blind and disabled.[3]

In an effort to provide pediatric dental practitioners with some general appreciation of the magnitude of these entitlement programs, the following presentation will review various national and state OASDI and SSI data on the benefits provided annually to more than three million children — many of whom are patients in our practices. (Note: it is unfortunate that some commentary is necessary regarding the differences in the public's general perceptions of the beneficiaries of OASDI and SSI entitlement programs and various support programs that often are classified under the "welfare" rubric [e.g. AFDC, Medicaid]. Nevertheless, the reality is that all too frequently there is a series of "negative assumptions" associated with the recipients of welfare programs, which for the most part are not reflected on the recipients of OASDI and SSI benefits.)

NUMBER OF CHILDREN RECEIVING BENEFITS

OASDI—At the end of 1991, more than 3.2 million children were receiving benefits under the OASDI program, (1.8 million were the children of deceased workers and 1 million were the children of disabled workers) (Table 1).

In line with the major differences in the general population of the various states, the number of children receiving OASDI benefits at the end of 1990 ranged from 275 thousand in the State of California, 216 thousand in the State of Texas and 202 thousand in the State of New York, to six thousand in the District of Columbia, and somewhat more than five thousand in the States of Alaska and Wyoming (Table 2). *SSI*—At the end of 1991, 438 thousand children were receiving benefits under the SSI program, (430 thousand were disabled and eight thousand were blind). To be eligible for SSI payments as a child, an individual must be under age eighteen (or under age twenty-two, if he or she is a full-time student), unmarried, and must meet the applicable SSI disability, income and resource criteria.[6] (see below)

Boys were more likely than girls to be SSI recipients, by about three to two. Seventy-five percent of child SSI recipients lived with their parent(s). Three percent were patients in a medical facility where more than half of the cost of their care was covered by the Medicaid program. An additional 18 percent lived in other types of hospitals, nursing homes, residential schools, foster care facilities or independently. More than one

Table 1. ☐ Number of children receiving OASDI benefits: 1960–1991.[4]

At the end of December	Children of: Retired workers	Deceased workers	Disabled workers	All workers
		(in thousands)		
1960	268.2	1,576.8	155.5	2,000.5
1970	545.7	2,687.9	888.6	4,122.3
1980	642.4	2,608.7	1,358.7	4,609.8
1990	422.2	1,776.0	988.8	3,187.0
1991	425.5	1,790.8	1,051.9	3,268.3

Notes: To some extent, the variations in the number of children reflect changes in eligibility definitions.

Data include students age 18–19 who received benefits (95 thousand in 1991) and disabled children 18 years and over (616 thousand in 1991). In order for disabled children to receive benefits under the OASDI program, they must be eligible under one of the three parental categories (i.e. retired, deceased or disabled worker). Based upon an income review, disabled children who are eligible and not eligible under OASDI may be eligible under the SSI program.

More than half (56 percent) of a ten percent sample of SSI children were disabled based on a mental disorder.

Table 2. ☐ Number of children receiving OASDI and federally administered SSI payments by state, December 1990.[3,5]

	OASDI entitlement			SSI entitlement	
	Retirement benefits	Survivor benefits	Disability benefits	Blind	Disabled
United States*	422,184	1,776,035	988,765	8,240	331,990
	(in thousands)				
Alabama	10.2	41.4	26.1	98	9,248
Alaska	.6	3.8	1.1	14	346
Arizona	6.0	24.3	13.8	90	4,224
Arkansas	5.8	22.5	17.9	118	5,808
California	40.1	157.9	76.6	1,510	30,498
Colorado	3.2	18.5	10.8	66	3,668
Connecticut	4.2	17.5	7.7	72	2,238
Delaware	.9	4.3	2.2	14	776
Dist. of Col.	.7	4.9	.9	12	1,020
Florida	21.6	83.3	46.4	326	16,162
Georgia	9.4	55.3	31.9	172	10,436
Hawaii	3.8	6.2	2.3	22	518
Idaho	1.6	7.2	3.9	34	1,386
Illinois	16.2	77.8	37.4	284	15,102
Indiana	8.5	40.2	24.9	182	6,664
Iowa	4.2	17.4	9.5	144	3,074
Kansas	3.3	15.8	7.3	64	2,460
Kentucky	7.6	32.3	27.8	146	7,226
Louisiana	9.4	44.8	31.4	232	12,838
Maine	1.9	7.9	5.3	26	1,198
Maryland	5.1	29.3	9.8	74	4,024
Massachusetts	7.5	31.6	18.0	496	6,224
Michigan	15.5	67.9	40.5	246	11,064
Minnesota	5.9	23.1	11.1	126	3,140
Mississippi	7.6	30.0	21.9	80	8,850
Missouri	8.4	38.9	23.3	140	7,026
Montana	1.3	6.2	4.1	18	1,008
Nebraska	2.0	9.6	4.9	30	1,754
Nevada	1.6	6.9	3.4	58	936
New Hampshire	1.2	5.9	3.3	12	532
New Jersey	9.5	47.1	20.4	128	8,230
New Mexico	3.2	13.6	7.6	52	2,594
New York	28.3	116.9	56.5	402	29,054
North Carolina	9.9	52.7	29.1	224	9,454
North Dakota	1.2	4.4	1.9	12	540
Ohio	17.4	137.4	45.8	398	14,836
Oklahoma	5.0	36.9	12.3	120	4,370
Oregon	4.1	31.5	8.8	78	2,616
Pennsylvania	17.4	137.6	36.6	306	16,588
Rhode Island	1.3	13.4	3.3	28	1,094
South Carolina	5.8	55.9	18.1	176	6,262
South Dakota	1.3	5.1	2.7	30	1,204
Tennessee	8.9	41.4	26.4	182	8,630
Texas	30.6	130.1	55.8	652	23,972
Utah	2.5	11.6	5.5	82	1,754
Vermont	.8	3.6	2.0	10	512
Virginia	7.9	38.5	21.9	156	6,090
Washington	5.9	26.7	15.0	100	4,522
West Virginia	4.9	17.4	15.4	76	3,136
Wisconsin	7.6	29.6	19.1	118	6,700
Wyoming	.5	3.2	1.6	6	334

* National totals include American Samoa, Guam, North Mariana Islands, Puerto Rico, Virgin Islands, those living abroad and unknown residence.

quarter (27 percent) were between five and nine years of age; 30 percent were between ten and fourteen years of age (Table 3).

A study of a 10 percent sample of recipients indicated that more than half (56 percent) of SSI children were disabled based on a mental disorder; 44 percent were mentally retarded; and 19 percent had diseases of the nervous system and sense organs (including all the blind children.)[6]

In 1990, the number of disabled children receiving SSI benefits ranged from more than thirty thousand disabled children in the State of California, twenty-nine thousand in the State of New York and twenty-three thousand in the State of Texas to slightly more than five hundred in the States Hawaii, New Hampshire, North Dakota and Vermont and somewhat more than three hundred in the States of Alaska and Wyoming.

Table 3. ☐ Number and percent distribution of children receiving federally administered Supplemental Security Income payments by age and category: December 1991.[3]

	Blind	Disabled	Totals
Total number	8,660	430,193	438,853
Total percent	100%	100%	100%
Under 5 yrs	16.9	16.1	16.9
5-9 yrs	26.3	27.4	27.4
10-14 yrs	27.6	30.1	30.0
15-17 yrs	15.2	16.2	16.2
18-21 yrs*	14.8	9.4	9.5

* Individuals age 18-21 years may be classified as either children or adults depending on their student status.

Table 4. ☐ OASDI benefits: average monthly benefits for children 1960–1991.[4]

	Children of:		
Year	Retired workers	Deceased workers	Disabled workers
1960	$30.37	$50.87	$30.25
1970	45.45	79.91	37.00
1980	105.82	238.80	116.80
1990	248.31	403.49	153.78
1991	257.67	414.86	155.80
Total benefits in 1991 (in billions)	$1.4	$9.0	$.2

In addition, 1,510 blind children in the State of California received benefits under the SSI program (2.3 times the number in the state with the next highest number , 652 in the State of Texas) (Table 2).

BENEFITS RECEIVED

OASDI—In 1991, $10.6 billion in benefits were provided to children. Average monthly benefits ranged from $156 for children of disabled workers, to $258 for children of retired workers and $415 for children of deceased workers (Table 4).

SSI—In 1990, $1.6 billion in benefits were provided to blind and/or disabled children, an average of $404 per month (in December 1990).[3] The specific benefit level for a particular child under the Supplemental Security Income program is based upon the child's income.***

***Under the SSI program, the federal payment is based on the individual's countable income. Under the SSI program, each eligible person living in his or her own household and having no other income is provided, as of January 1992, a monthly cash payment of $422 ($633 for a couple if both members are eligible). If an individual or couple, is living in another person's household and is receiving food and shelter there, the federal benefits are reduced by one-third. If the individual is institutionalized, eligibility and benefits vary depending upon the type of facility. The benefits generally increase at the same rate as the cost-of-living increases in OASDI benefits. The first $20 monthly in OASDI benefits or other earned or unearned income is not counted. Also excluded is $65 monthly of earnings plus one half of any earnings above $65. For example:

☐ A person living in his or her own household, whose sole income is a $200 monthly OASDI benefit, would receive $242.00 in federal SSI payments:

$422 - ($200 - $20) = $242

☐ A person whose income consists of $500 in gross monthly earnings would receive $214.50 in federal SSI payments:

$422 - ([$500 - $85]) ÷ 2) = $214.50

Note: Individuals generally are not eligible for SSI if they have resources in excess of $2,000, $3000 for a couple (in 1992). Certain resources are excluded (e.g. a home). A state may supplement the federal SSI benefits.[7] In 1992, twenty-seven states supplemented federal benefits by an average of $32 per month.[9]

Overall— "What people don't know about Social Security is the full extent of what it does for children and families."[1] For example:

☐ The protection offered to families through Old Age Survivor protection is equivalent to a life insurance policy with a face value of $85,000 and could be as high as $390,000 for young families with two or more children.[1]

☐ Total survivor protection offered to all children in the United States is estimated to be $7.6 trillion.[1]

☐ Changed guidelines for childhood disability recognize the particular problems with childhood HIV infection and childhood AIDS. Flexible policies permit greater assurance of SSI support.

Nevertheless, there are great difficulties in locating potentially eligible children with information about the Social Security program. Estimates in 1990 indicate that families with children constitute 34 percent of the urban homeless. Many of these children or their families could benefit from SSI support but because they have no fixed addresses they are difficult to locate and assist.

GENERAL THOUGHTS

"Welfare is money, just like other money. But it is also money that is associated with and perhaps even encourages parents who do not work. Income from welfare may not have the same positive effect on children as income from other sources."[8]

An extended series of reports indicate that "it matters where income comes from."[8] Advocates for child support programs (which guarantee a minimum amount of financial support per child) emphasize the associated increased educational attainment, stabilization of the family structure and reduction in early childbearing. The OASDI and SSI entitlement programs provide operating examples of an essential child support system that functions beyond the "shadow" of welfare.

(Note: much has been written about the relationship of early childbearing and associated difficulties of poverty. In 1990, the poverty rate for female headed household was 45 percent.[8] The intensity of this issue may be emphasized further with the report that "in 1988, over 20,000 teenagers gave birth to not their first, not their second, but their third child.")[1]

Old Age, Survivors and Disability Insurance, Supplemental Security Insurance, and a seemingly endless alphabet soup of governmental support programs may seem well beyond the confines of the average dental practice. The reality is that in excess of $12 billion annually are provided to more than three million children, many of whom frequent dental offices. It would seem reasonable for pediatric dental practitioners to be more conversant with these programs.

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RECENT REFORMS IN BRITISH NATIONAL HEALTH SERVICE: LESSIONS FOR U.S.

President Clinton recently announced his reform plan for the health care system in the United States. This plan is similar to the British National Health Service as established in 1948 in that it aims to introduce a system of health care for all. It is intended to eliminate fraud, abuse, and inappropriate medical procedures. However, there will be nothing other than rhetoric to ensure that resources are spent on effective and appropriate services. The financing of the plan is naturally being questioned, since President Clinton is promising to give 37 million uninsured people a single package of health benefits while, in the long term, reducing the ever-increasing costs of health care.

The common problems facing our countries' health services make it instructive for the United States to consider the lessons of the British health reforms. The inexorable demands and expectations placed on health services with limited resources and their inability to expand to meet these demands force them to make choices as to which services can and should be provided. Tinkering with structures, improving efficiency, and increasing accountability do not resolve this fundamental dilemma. One solution—rationing health care—has been attempted in Oregon and the Netherlands. All societies will eventually have to find a way of choosing which services should be universally provided.[26,27] These services will then have to prove their effectiveness.

The United States and the United Kingdom need to confront two major issues. The first is that neither country can afford the health service that it needs and wants. Both countries have to decide which services should be provided to whom and how. Second, many actions outside of health care could have a greater impact on improving health status than an increase in health expenditures. In general, the United States needs to decide whether there should be universal access to equal care, while the United Kingdom has to consider increasing its spending on health care. Both countries need to decide what their priorities are with regard to health care and related expenditures.

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Cervical adenitis: Report of two cases

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Cervical adenitis is an inflammation of one or more of the lymph nodes of the neck. In children it is a common finding that can mimic an odontogenic infection. The etiology and the clinical manifestations of cervical adenitis vary considerably. Seventy to 80 percent of the cases are attributed to staphylococcus and streptococcus infections and often they are preceded by a recent history of an upper respiratory infection. In more than 80 percent of the cases the submaxillary and the deep cervical lymph nodes are affected because they are the major routes of lymphatic drainage of the head and neck.[1,2]

CASE ONE

A five-year-old female presented to the hospital dental clinic with a history of a rapidly enlarging mass on the right side, of one day duration. The patient's medical history was negative. Physical examination revealed a firm palpable swelling along the right border of the mandible (Figure 1). The swelling was slightly sensitive to palpation, did not cross the midline and was not compromising the patient's airway; and the patient was afebrile. A clinical and radiographic examination (panorex and bitewings) ruled out a dental etiology and the patient was referred to the hospital ENT service, who admitted the patient under their care. Admission blood values were RBC, 4.4cu^3; WBC, 16,600; Hct, 360; Hgb, 11.9; Polys, 63 percent; Lymphs, 31 percent; Monos, 2 percent; Eosinophils, 2 percent; Basophils, 2 percent; and Platelets, 386,000. The chest X-ray was clear and the patient had normal electrolytes. The infectious disease service was consulted and a tentative diagnosis of cervical adenitis was made. Bacterial and viral cultures were obtained and the patient was empirically placed on I.V. Nafcillin (150mg/kg/day) and Clindamycin (40mg/kg/day) prior to the results of the cultures. After three days there was a spontaneous resolution of the cervical swelling and the patient was discharged on a home course of Augmentum (250mg) three times a day for ten days. The results of the viral culture were negative, but the bacterial throat culture was positive for Hemophilus parahaemolyticus. Follow up was with the family pediatrician.

CASE TWO

A five-year-old-male had been followed by his family pediatrician for a slowly developing right submandibular mass (Figure 2). History was significant for a similar facial swelling on the left side two years previously. No definitive diagnosis was made at that time and treatment consisted of I.V. Zinocef (500mg) followed by a home course of Ceclor. The facial swelling resolved without complications. For this most recent episode the patient had been placed on Ceclor without resolution, and after eight days the patient was admitted to the hospital infectious disease service for further

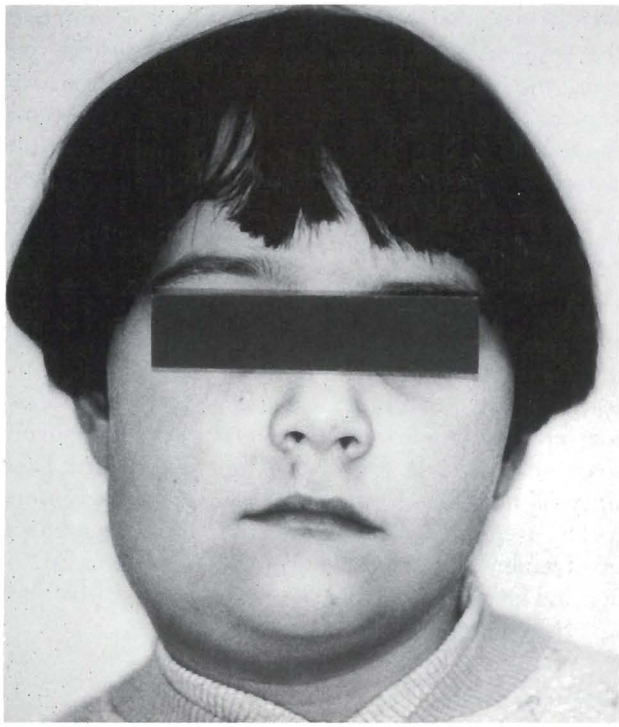

Figure 1. Child presenting to dental clinic with right facial swelling.

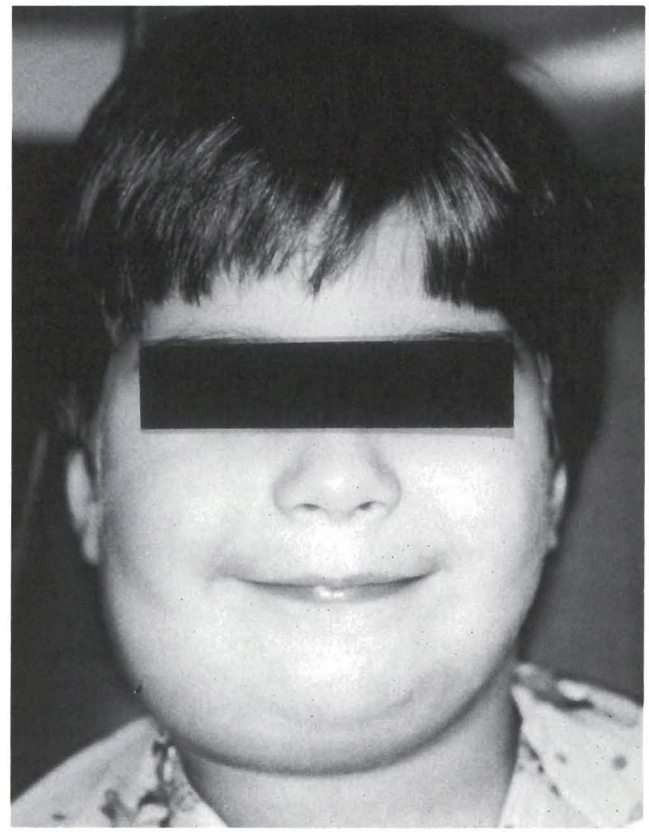

Figure 2. Right-sided facial swelling noted at time of consultation.

evaluation. At the time of admission the patient was in no apparent distress and had an oral temperature of 102°F.

Physical examination revealed a right submandibular mass that was tender to palpation. The patient had a history of pharyngitis one week before the onset of the swelling. Blood values were as follows: RBC, 5.4 cu^3; WBC, 8,900; Polys, 51percent; Lymphs, 89 percent; Monos, 8 percent; Eosinophils, 1 percent; and Platelets, 357,000. The chest film was clear and an ultrasound revealed no abscess. The patients immunoglobulin levels were normal and the patient had a negative tuberculin (PPD) test. No throat cultures were taken. The patient was placed on I.V. Nafcillin (750mg) every six hours. The day following admission a consultation was scheduled with the dental department .

Examination revealed no obvious carious teeth and the soft tissues were within normal limits. A panoramic radiograph was obtained and showed no obvious dental etiology. Following the dental evaluation, a pediatric surgery consultation was obtained; their recommendation was to leave the area untouched, since no obvious suppuration was present and the swelling was beginning to resolve with the antibiotic therapy.

The patient's hospital course remained unremarkable until the ninth day, when the patient began to develop a papular, pruritic rash. The pediatricians discontinued the I.V. antibiotics. The patient was afebrile at this time and the swelling was resolving; the decision was made, therefore, to discharge the patient on Cleocin (75mg), two teaspoons every six hours for ten days. The provisional diagnosis at discharge was cervical adenitis and the patient was scheduled for follow up with the hospital infectious disease department in one week.

DISCUSSION

Cervical adenitis is a common pediatric problem with multiple etiologies. Because of the swelling that resembles odontogenic infections and its rapid onset, with or without other clinical signs, the dentist may be the first person to see the patient. A thorough examination will confirm or deny a dental etiology.

A thorough history and physical examination are completed noting the duration of the cervical lymph

node enlargement, any preceding respiratory infection, pneumonia, otitis media, or association with pets. A family history of tuberculosis, conjunctival, oropharyngeal, skin or scalp lesions should be noted. A differential diagnosis should include such conditions as Hodgkin's disease, leukemia, thyroid tumors, juvenile rheumatoid arthritis and systemic lupus erythematous. Congenital masses such as a thyroglossal duct cyst or branchial cyst may cause a similar swelling. In addition, miscellaneous causes such as cat scratch fever, histiocytosis X, sarcoidosis and mucocutaneous Iymph node syndrome should be considered. Viral and bacterial infections may be responsible for cervical neck swelling. These may include rubella, measles, mumps, infectious mononucleosis, herpes simplex, cytomegalovirus, S.aureus. group A strept.., H. influenzae, Actinomyces, atypical mycobacteria, and gram negative enterics.[1]

A thorough dental clinical examination with dental radiographs is performed next, to rule out any dental etiology. If upon completion of the dental examination you are confident that the patients clinical finding are not consistent with a dental infection, immediate referral to the family physician or hospital is indicated.

As previously stated many infectious agents have been associated with cervical Iymphadenitis. Staphylococcus aureus and group A streptococcus have been isolated in a large number of cases; although no significant differences in patients with streptococcus or staphylococcus with respect to sex, race, symptoms, site, size or presence or absence of fever has been noted. Staphylococcus infections tend to have a longer duration and a greater percentage of fluctuant swelling of the Iymph nodes.[3] The decision to aspirate the cervical node is made by the medical service attending the patient. In both of the cases reported, no aspiration was deemed necessary, because they responded to the antibiotic therapy. Had there been little change in the swelling after starting antibiotics, aspiration would have been indicated.

In most of cases of cervical swelling, the patient is placed immediately on antibiotics when admitted to the hospital. An antibiotic is selected empirically because the results of culture and sensitivity tests are not known. Penicillin is usually not given initially, because the incidence of staphylococcus organisms resistant to penicillin is high. The average course of antibiotic therapy is ten days, unless abscess formation occurs; then incision and drainage are indicated. Surgical removal of the Iymph node is rarely necessary.

Because of the increased incidence of tuberculosis, the dentist must be aware of the clinical manifestations of the illness. Typical and atypical mycobacterium infections have clinical features similar to cervical adenitis; but typical mycobacterial infections are more likely to occur in patients greater than four years of age, have bilateral node involvement, and a history of exposure to TB. Atypical mycobacterium rarely has bilateral node involvement, occurs in children less than four years of age and gives a tuberculin skin test that is positive, but less than 10mm.[4]

CONCLUSION

Cervical adenitis has multiple etiologies and clinical presentations. Because of its similarity to odontogenic infections, a dental evaluation is warranted. If the clinician has ruled out a dental etiology, the patient should be referred to a medical service for further evaluation. Early intervention with antimicrobial therapy results in rapid resolution in the majority of the cases of cervical adenitis.

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Pyogenic granuloma as a cause of bone loss in a twelve-year-old child: Report of case

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The pyogenic granuloma is a common reactive lesion that generally develops rapidly, bleeds easily, and ulcerates causing the erroneous clinical impression of a malignant tumor.[1] It is, however, a well-circumscribed, benign, soft-tissue tumor of inflammatory rather than neoplastic nature, arising from the connective tissue of skin or mucous membranes.[2]

The exact cause is unknown, although it is thought to represent a localized, overexuberant reaction to a minor irritation.[3] Hormonal and other factors seem to modify the response under certain circumstances and promote the development of hyperplastic granulation tissue.[4]

The incidence of the pyogenic granuloma has been described as between 26.8 percent to 32 percent of all reactive gingival lesions.[5,6] Although it has been reported in all age-groups, it occurs mainly between the ages of eleven and forty years, with the peak incidence in the third decade.[7] Females are more frequently affected: a study by Skinner *et al* (1986) revealed a 3:2 predilection for females over males.[8]

The lesion most frequently arises in the gingivae of the maxillary anterior facial region, but has also originated in the lips, tongue, buccal mucosa, and edentulous alveolar mucosa.[3] Moriconi and Popowich report a case of a large exophytic lesion, causing significant alveolar bone resorption.[9]

Dr. Goodman-Topper is an instructor and Dr. Bimstein is an Associate Professor, Department of Pediatric Dentistry of the Hebrew University, Hadassah Faculty of Dental Medicine, Jerusalem.

It usually appears as an elevated, pedunculated or sessile soft-tissue mass. The surface may be smooth, granular or lobulated, and the color may range from pink to red or brownish-red. The surface may be ulcerated, depending on the site and its exposure to traumatic irritation. The surface may thus be necrotic and covered by a white slough that clinically resembles pus—hence the name. There is usually no pus in the lesion.

The clinical features of the pyogenic granuloma are indicative but not specific. Differential diagnosis would include: peripheral giant cell granuloma; capillary hemangioma; metastatic tumor, especially renal cell and bronchogenic carcinomas; Kaposi's sarcoma; angiosarcoma; and malignant melanoma. Definitive diagnosis can only be made by histopathologic examination of biopsied tissue.

Histopathologically, the lesion is composed of a large number of epithelial-lined vascular spaces with an infiltrate of lymphocytes, plasma cells, and frequently polymorphonuclear neutrophils. It is covered by a thin, often ulcerated, layer of stratified squamous epithelial cells. Superficially there may be infiltration of microorganisms from the oral cavity, via the breach; but the deeper layers show no microbial infiltration. Collagen is sparse in the lesion. The treatment of these lesions is by conservative local excision. A recurrence rate of 16 percent, however, has been reported.[5]

CASE REPORT

A twelve-year-old Arab male presented at the Pedo-

dontic Clinic of the Dental School of the Hadassah Faculty of Dental Medicine, complaining of two problems: a carious tooth in the left maxillary area; and a red lesion in the palate in the maxillary right area. He was a normal child with no relevant medical history. The dental history, however, revealed that some seven to eight months previously, the maxillary right second primary molar had been extracted.

Clinical findings

Extra-oral examination revealed symmetry of the face and neck. There were palpable but non-tender submandibular lymph nodes on the right. Intra-oral examination revealed: poor oral hygiene; an extremely carious and mobile maxillary left second primary molar, with an associated draining fistula; and a red lesion with yellow speckles, on the palate between the right first premolar and the right first permanent molar with some exposure of the roots of the latter (Figure 1).

Radiographic findings

Radiographic examination disclosed that the roots of the abscessed maxillary left second primary molar had undergone extensive resorption (Figure 2). On the right, there was absence of the maxillary second premolar, and an extensive bony defect in the basal bone between the maxillary first premolar and the first permanent molar (Figure 3).

Treatment

The abscessed maxillary left second primary molar was extracted using local anesthesia. The child was then

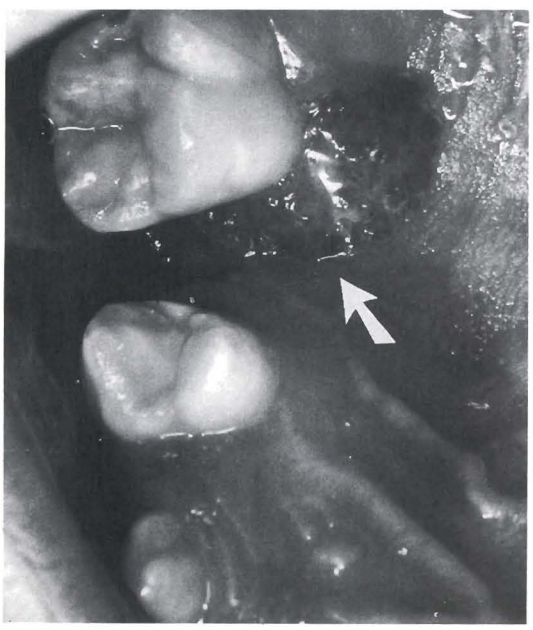

Figure 1. Clinical photograph of the maxillary right premolar/molar area. Note (arrow) the speckled lesion appearing on the mesiopalatal aspect.

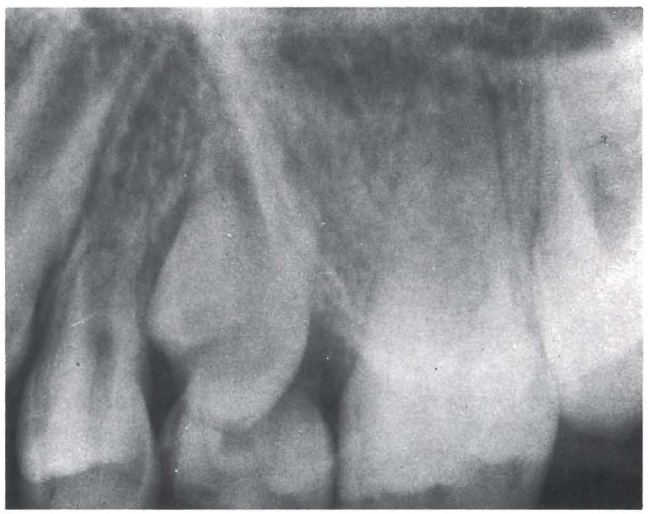

Figure 2. Periapical radiograph of the maxillary left quadrant showing resorption of the roots of the second primary molar.

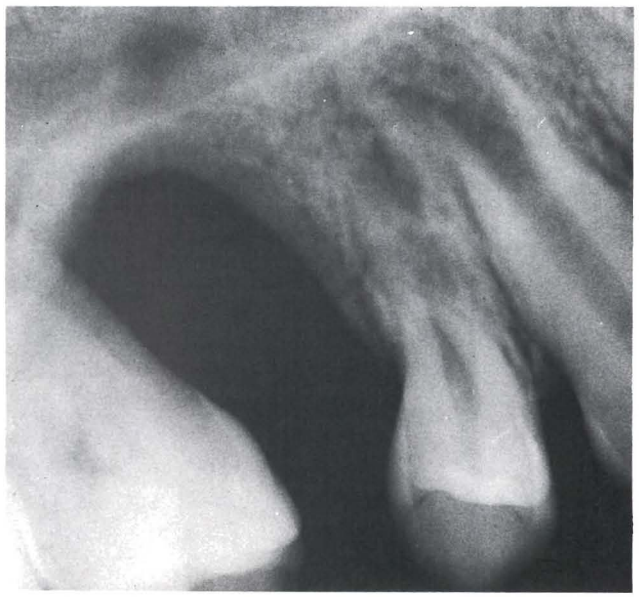

Figure 3. Periapical radiograph of the maxillary right quadrant showing the extensive bony defect between the first premolar and the first permanent molar.

referred to the Periodontology Department. An excisional biopsy was subsequently carried out using local anesthesia. The tissue mass was sent for histopathologic examination. The patient was instructed to maintain good oral hygiene and re-appointed one week later. At the subsequent visit, the palatal wound was healing well. A further appointment was scheduled two months later to assess the bony repair of the alveolar defect. Clinically the palatal lesion was healed. The radiograph revealed, however, no significant change in the bone. The patient was referred to the periodontal department for assessment and treatment. It was envisaged that, although the maxillary right first premolar might be lost, the right first permanent molar might be saved, possibly by resection of the involved root.

The patient failed, however, to attend that and subsequent appointments. Despite careful explanations of the underlying bony damage, it seemed that once the external lesion had healed, the parents were no longer concerned with further treatment. This illustrates the importance of early diagnosis of this lesion.

Histopathologic findings

A typical pyogenic granuloma was revealed with overlying stratified squamous epithelium, proliferation of capillary endothelium with an accompanying fibrous tissue element. The surface tissue was ulcerated and covered with fibrinous exudate (Figure 4).

DISCUSSION

Oral pyogenic granuloma is a relatively common lesion, although less so among the pediatric population. The rapid growth of these lesions, in addition to their potential to destroy underlying bone, may give a sinister clinical appearance of malignancy.

Recognition of the pyogenic granuloma is important in order to avoid patient (or parent) alarm. Its morphology and location are distinctive aids in arriving at a correct clinical diagnosis: an erythematous tumor of rubbery to firm consistency arising from the gingiva is characteristic. A history of trauma or local infection may contribute to the diagnosis, i.e. factors that could give rise to exuberant granulation tissue. Oral malignancies, both primary and secondary, however, can only be differentiated from the pyogenic granuloma by histopathologic examination. Thus the treatment of choice is excisional biopsy, which, if total, is curative.

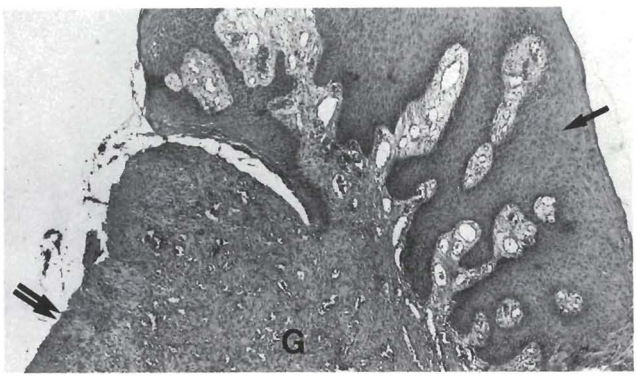

Figure 4. Photomicrograph of the pyogenic granuloma. Note overlying stratified squamous epithelium (arrow), granulation tissue (G) with vast numbers of vascular spaces and ulcer covered with fibrinous exudate (double arrow). Hemotoxylin and Eosin x 120.

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Lowe's syndrome: Review of literature and report of case

George P. Thomas, DDS
Stephen E. Grimm, III, DDS

The oculocerebrorenal syndrome of Lowe is an x-linked recessive disorder commonly found in males of Caucasian or Asian ancestry. This condition is associated with three phases of the biomedical problem.[1] The first or neonatal phase is associated with major ophthalmologic problems such as congenital cataracts. The second phase, which lasts from infancy to mid-childhood, is associated with renal tubular dysfunction. The third phase is usually associated with less severe metabolic problems, even though some patients have had progressive renal failure.

Clinical manifestations of these patients may include failure to thrive, congenital cataracts, mental retardation, and renal tubular dysfunction.[1,2,4] These children tend to be short in stature, which may be secondary to their renal disease. Deaths at all ages have been reported from renal disease, dehydration, and recurrent infections. This report describes the oral findings of a ten-year-old child with Lowe's syndrome.

REPORT OF CASE

In February of 1991, the patient was referred to the Pediatric Dental Clinic from Pediatric Endocrinology at Howard University. Their chief concern was "a loose tooth in his mouth," and "double row dentition." His chronologic age was ten years and he had been diagnosed with Lowe's syndrome (oculocerebrorenal) at birth. This necessitated a more involved medical consultation with specific recommendations than were present on the terse referral form.

Yet we commenced our cursory examination and radiographs to establish his preliminary dental needs. No overt carious lesions were noted. Teeth lettered N, P and Q were overretained by the fact that their successors were erupting ectopically lingually, and only root resorption was seen radiographically (Figures 1 and 2). Tooth lettered D was truly loose and due to exfoliate in short time. The stage of eruption was slightly delayed from the norm. The anterior alveolar gingiva was edematous and fibrotic, attributable to mouth breathing and an open mouth habitus. This may be contributory to the delayed eruption of the anterior teeth, as well as his condition of rickets (metabolic). The palatal vault was high with an anterior open bite and excessive overjet. A Class I molar relationship was noted. The examination was accomplished with the patient sitting on his mother's lap and using mild restraint. Behavior shaping was needed to alter a precooperative type pattern due to this developmental delay. He was reappointed for a conditioning prophylaxis with the recommended SBE regimen, because of his cardiac defect, aortic stenosis. At that visit we would decipher the myriad medical problems present in Lowe's syndrome and establish a coordinated schedule of proposed dental treatment. In April of 1991, the consultation revealed the following findings:

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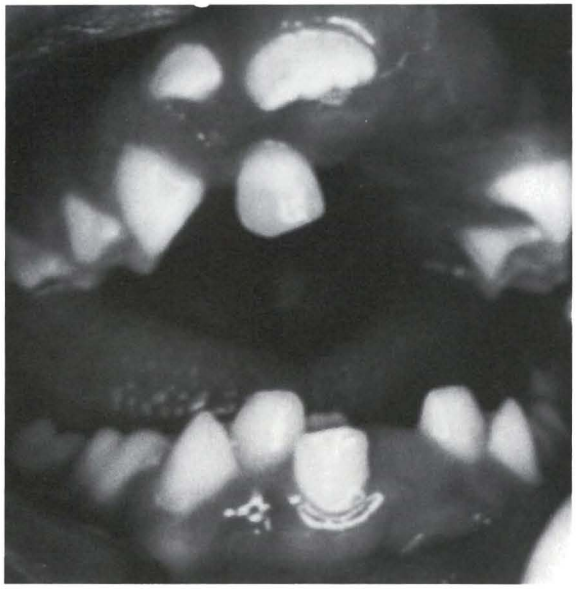

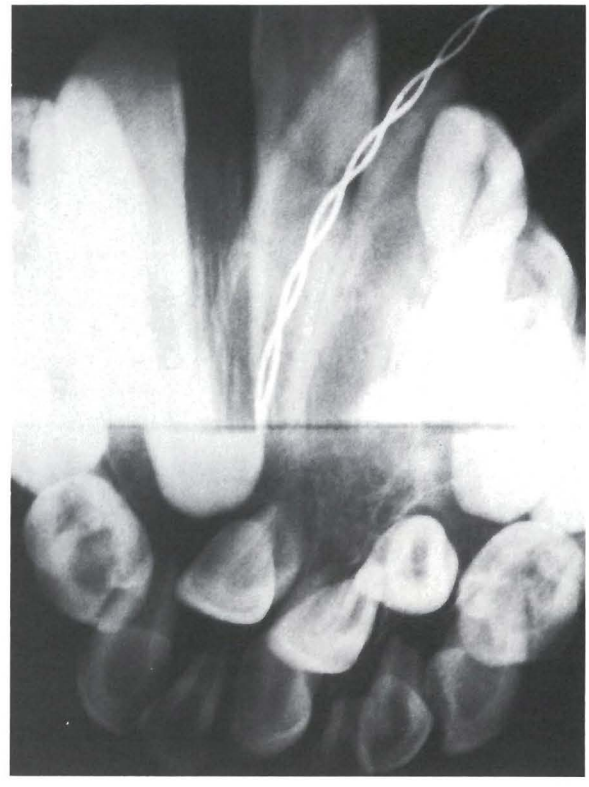

Figures 1 & 2. (Respectively) Intraoral photograph and occlusal radiographs of maxillary and mandibular anterior regions indicating the over-retention of primary teeth with the ectopic eruption of the succedaneous teeth. Duly noted is the fibrotic gingiva and the constricted, deep palatal vault.

LOWE'S SYNDROME (OCULOCEREBRORENAL)

Occular Microopthalmus (non-functional eye) with congenital glaucoma (functional eye). (Figure 3).

Cerebral Microcephaly with developmental delay.

Renal Organic aciduria/aminoaciduria/diminished production of ammonia with medications of bicitra, neutrophos, and diamox, to reverse metabolic acidosis/rickets.

CV (not part of syndrome): Aortic stenosis with mild LVH.

We attempted our oral prophylaxis with only slight success using restraint due to head movement. We chose not to use pharmacologic or complex restraint, to avoid raising ocular pressures or insulting his medical anomalies. Yet, with our slight progress, we chose to make another attempt in May 1991, which proved futile. After a discussion with his pediatrician, the venue of general anesthesia was selected. In June 1991, the patient was anesthetized for comprehensive dental treatment. SBE prophylaxis with IV ampicillin was administered after the patient was induced, and an IV was started. A nasotracheal tube was placed, patient draped, and throat packed with a 4x4 gauze pad. Periapical radiographs were taken of the four posterior quadrants to supplement the anterior occlusal radiographs. No interproximal decay was noted, but an interesting finding of taurodontism was duly noted (Figure 4). Sealants were placed on all posterior molars as a preventive service. A thorough scaling and prophylaxis with attendant fluoride treatment were completed, to eliminate all calcarious deposits as well as plaque. Tooth lettered D exfoliated before our operative procedure. Teeth lettered N, P and Q were extracted after elevation with forceps. Profuse bleeding was encountered. Pressure was not sufficient to encourage clot formation. Gelfoam was placed in the extraction alveolus and chromic sutures were placed to prevent dislodgement and afford hemostasis. This bleeding was more profuse than normally encountered, even though no local anesthetic with epinephrine was used in conjunction with other anesthetic agents. Slight oozing was noted one to two days postoperatively. It is our impression that due to the renal disease and altered electrolytes, the bleeding/clotting times were extended, even though no laboratory studies were considered before the procedure. The patient was extubated, taken to recovery in stable condition, and discharged to home later that day with an appropriate postoperative dose of amoxicillin.

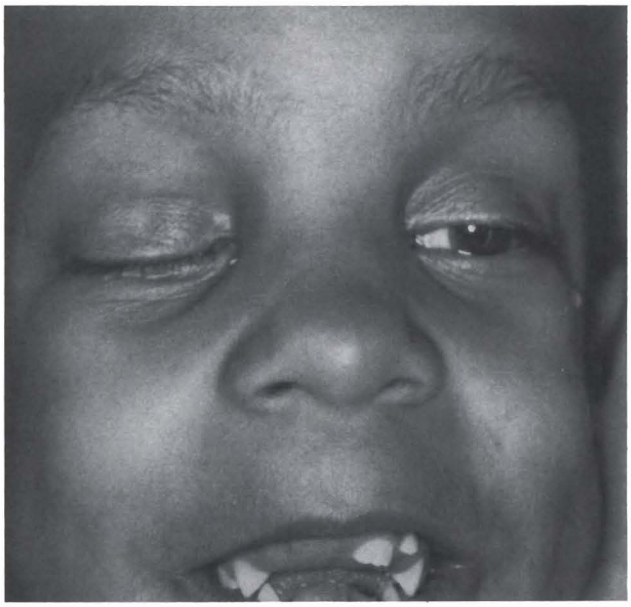

Figure 3. Frontal photograph showing closed eyelid of microophthalmic globe (postoperative).

The one-week follow-up appointment was satisfactory with no subjective complaints. The extraction sites clotted and were healing. The bleeding reported was when he "picks" at the sutures. The patient was placed on periodic follow-up to ascertain healing and labial movement of the lingually erupted mandibular incisors.

CONCLUSION

In 1952, Lowe, Terry and Maclachlan described this medical condition.[4] Since then, more than 150 cases have been reported in the medical literature. It appears that no oral and dental aspects of this syndrome, however, have been reported. The oral manifestations include crowding, delay of eruption, over-retained primary teeth, constricted palate and taurodontism of the molars. Delay of eruption may be related to the rickets

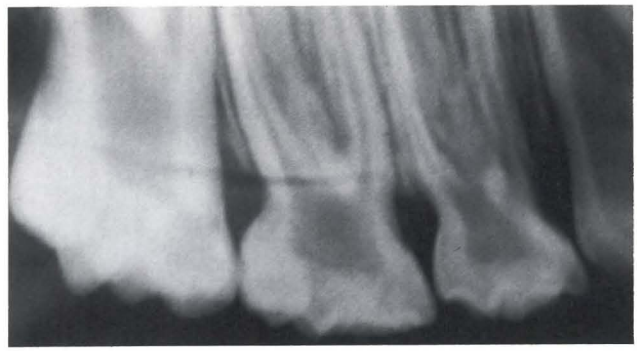

Figure 4. Intraoperative posterior periapical radiograph, illustrating taurodontism of the primary dentition.

associated with OCR syndrome. The patient in this report did not exhibit any unusual caries pattern for children of his age. The parent was informed of the importance of establishing a good oral hygiene program at home, because patients with Lowe's syndrome exhibit variable degrees of mental retardation. Although Lowe's syndrome is not a common heritable disorder, the occurrence of more than 150 cases in the literature suggests that, as dental practitioners, we should know the characteristics of this condition and establish effective management guidelines.

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EARTHQUAKE

And for a salesman there is no rock bottom to the life. He don't put a bolt to a nut, he don't tell you the law or give you medicine. He's a man 'way out there in the blue, riding on a smile and a shoeshine. And when they start not smiling back—that's an earthquake.

ARTHUR MILLER, *Death of a Salesman*

ABSTRACTS

Dhummarungrong, Sompit; Moore, B. Keith; Avery, David R.: Properties related to strength and resistance to abrasion of VariGlass VLC, Fuji II l.C., Ketac-Silver, and Z-100 composite resin. J Dent Child, 61:17-20, January-February 1994.

The purpose of this study was to evaluate the properties related to the strength and resistance to abrasion of new Type ll lightcured glass ionomers, VariGlass VLC, and Fuji ll L.C., and to compare the results with Ketac-Silver and Z-100 composite resin. Each material was tested for strength with an Instron Universal Testing Machine and for resistance to abrasion with a motor-driven machine. The strengths of Z-100 were the highest. Among glass ionomers, KetacSilver had higher compressive strength, but lower diametral tensile and tranverse strengths than the others; and Fuji ll L.C. had higher strengths than VariGlass VLC. VariGlass VLC showed the greatest resistance to abrasion, followed respectively by Z-100, Fuji ll L.C. and Ketac-Silver. ANOVA indicated significant differences in strengths and abrasion resistance, except, the volume losses of Fuji ll L.C. and Ketac-Silver in the toothbrushing test were not significantly different ($p<0.01$). The results support VariGlass VLC and Fuji ll L.C. as alternative filling materials in Class lll and V situations, but they are not recommended for stress-bearing restorations.

Abrasion; Strength; VariGlass VLC; Fuji II L.C.

Flaitz, Catherine M. and Hicks, M. John: Role of the acid-etch technique in remineralization of caries-like lesions of enamel: A polarized light and scanning electron microscopic study. J Dent Child, 61:21-28, January-February 1994.

The aim of this *in vitro* study was to determine the effect of acid-etching on remineralization of caries-like lesions with calcifying fluids (CF). Caries-like lesions were created in two windows of sound enamel on both buccal and lingual surfaces of human molars. A central longitudinal section was taken from each tooth to serve as control lesions (CL). Each tooth was then sectioned into quarters and each quarter assigned to one of four groups: 1) lmM Ca CF Remineralized (RC1); 2) 1mM Ca CF Remineralized Etch (RE1); 3) 3mM Ca CF Remineralized (RC3); 4) 3mM Ca CF Remineralized Etch (RE3). Lesions in the etch groups were exposed to 30 percent H_3PO_4 for 30s prior to CF. Etch and non-etch groups were treated with 1mM or 3mM Ca CFs (prepared from HAP [Ca/P ratio = 1.63], pH 7.0, 0.05mM F^-) for ten 60-second periods, interspersed with deionized water rinses. Mean lesion depths were determined using a digitized tablet. Following 1mM Ca CF, mean lesion depths were: 179μm for CL, 157μm for RC1 and 118μm for RE1. Following 3mM Ca CF, mean lesion depths were: 188μm for CL, 171μm for RC3 and 143μm for RE3. SEM topographic features of the remineralized etched lesions consisted of adherent surface coatings with finely globular patterns in the 1mM Ca CF group and densely adherent surface coatings which masked the effects of etching in the 3mM Ca CF group. Acid-etching of caries-like lesions before treatment with calcifying fluids appears to facilitate remineralization, resulting in adherent surface coatings, that may provide a source for prolonged remineralization and may increase resistance to lesion progression.

Enamel caries; Acid-Etch; Remineralization; Calcifying fluids

Kreulen, C.M.; van Amerongen, W.E.; Borgmeijer, P.J.; Gruythuysen, R.J.M.: Evaluation of occlusal marginal adaptation of class II resin composite inlays. J Dent Child, 61:29-34, January-February 1994.

In this paper, the results of a clinical study of the occlusal marginal adaptation of indirect Class II resin composite inlays are presented. The margins of 180 resin composite and 60 amalgam restorations, made by three dentists, were assessed, shortly following their placement. An indirect, photographic method has been applied to assess marginal adaptations. The restorations were classified into excellent and non-excellent marginal adaptation categories and on this basis influencing factors were determined.

Resin composite inlays appeared to have a greater percentage of 'excellent' margins than amalgam restorations (46.1 percent and 6.7 percent, respectively). The dentist was the variable that most influenced the marginal adaptation. Variability in the period elapsing between applying the restoration and conducting the assessments is discussed as a factor that may impair a fair comparison with initial results for direct composites. Nevertheless, based on the literature, differences in initial marginal adaptation between direct and indirect restorations are described.

Occlusal marginal adaptation; Resin composite inlays and amalgam; Clinical trial; Indirect evaluation

Leviton, Alan; Needleman, Howard; Bellinger, David *et al*: Children who have hypoplastic enamel defects of primary incisors are not at increased risk of learning problem syndromes. J Dent Child, 61:35-38, January-February 1994.

Developmental enamel defects of primary teeth have been associated in earlier reports with motor, hearing and psychological disturbances. The relationship between hypoplastic enamel defects and learning problems had not been evaluated previously. For a total of 149 girls and 196 boys we collected a primary tooth shortly after shedding and approximately 2 years later a teacher-completed questionnaire about function in school. Children with a hypoplastic enamel defect of one of their primary incisors were no more likely than their peers without an enamel de-

fect to have any of 7 different learning problem syndromes. In the absence of reason to suspect selection bias, confounding, or misclassification of hypoplastic defects or school problems, we conclude that hypoplastic enamel defects are not associated with teacher-identified learning problems.

Enamel defects, primary teeth; Motor, hearing, and psychological disturbances

Friedlander, Arthur H.; Friedlander, Ida Kreinik; Yagiela, John A. *et al*: Dental management of the child with developmental dyslexia. J Dent Child, 61:39-45, January-February 1994.

Dyslexia, a biologically determined reading disorder effects an estimated 3 percent to 10 percent of school-age children in the United States. Standard pediatric dental protocols frequently have to be modified because many of these children concurrently suffer attention-deficit-hyperactivity disorder (ADHD), asthma, thyroiditis and inflammatory bowel disease (ulcerative colitis and Crohn's disease). Youngsters suffering from dyslexia and ADHD should have their dental appointments scheduled in the morning when they are most attentive and best able to remain seated in the dental chair. An aspirating dental syringe must be used in order to avoid an intravascular injection and the possibility of an adverse interaction between the pressor agents used with local anesthesia and the medication used to treat ADHD. Aspirin, other nonsteroidal antiinflammatory agents (e.g., ibuprofen) and local anesthetic agents containing vasoconstrictor and preservative (antioxidants, i.e., sulfite) agents should be avoided in children with concurrent asthma because of their propensity to trigger an asthmatic attack. Children with uncontrolled hyperthyroidism should receive only emergency care and this care should be provided in a hospital. Children with hypothyroidism are hyperresponsive to even small dosages of analgesics and anesthetic agents; proper dosing and venue of dental procedures should be a joint decision between the child's dentist and pediatrician. Children with dyslexia and concurrent inflammatory bowel disease may require shorter appointments and nitrous oxide sedation to reduce stress. Those receiving corticosteroids or with a history of steroid therapy within the past year may need supplementation to avoid an adrenal crisis brought about by the stress of dental care.

Dyslexia; ADHD; Drug interacting

Benitez, Claudia; O'Sullivan, David; Tinanoff, Norman: Effect of a preventive approach for the treatment of nursing bottle caries. J Dent Child, 61:46-49, January-February 1994.

Seventeen children with nursing bottle caries and their caretakers were studied to assess whether a preventive program would arrest the progression of the caries. Prior to the study the caretakers were asked questions regarding their child's oral hygiene practices and nursing habits, as well as the caretakers' demographic characteristics. All caretakers were given instructions on how to control the use of their child's nursing bottle and how to brush their child's teeth twice daily with 0.4 percent stannous fluoride gel. After the instruction the children and caretakers were placed on 3-month recalls. Compliance was determined through a self-evaluation questionnaire assessing preventive behaviors. In general, the caretakers were unmarried 20- to 30-year-old women subsisting on welfare with one child. The majority of the caretakers were aware of the potential dangers of the child sleeping with milk or sweet liquids in the bottle. The study was discontinued after three months because caries noticeably progressed in all but two of the children, and only two caretakers complied fully with the prevention program.

Nursing-bottle caries; Prevention; Caretaker response

Waldman, H. Barry: Updating the changing number and distribution of pediatric dentists: 1982-1991. J Dent Child, 61:50-56, January-February 1994.

A review of the 1991 and past two ADA studies on the distribution of dentists provides specific information on the evolving numbers of pediatric dentists at the national, regional and state levels.

Pediatric dentists, numbers of; ADA studies

Waldman, H. Barry: Social security and providing for our children. J Dent Child, 61:57-61, January-February 1994.

The scope and magnitude of OASDI and SSI social programs for children are reviewed. More than $12 billion in benefits are provided annually to three million children. These "nonwelfare" programs could serve as a model for financial child support arrangements which could foster family responsibility and reduce family reliance of welfare.

Social security; OASDI program; SSI program; Family responsibility

Goodman-Topper, Elizabeth D. and Bimstein, Enrique: Pyogenic granuloma as a cause of bone loss in a twelve-year-old child: Report of case. J Dent Child, 61:65-67, January-February 1994.

The pyogenic granuloma frequently appears between the ages of eleven and forty years. It is a benign soft tissue lesion of inflammatory origin, which may be misdiagnosed as a neoplastic tumor due to its rapid development. Definitive diagnosis can only be made by histopathologic examination. Treatment is by conservative local excision. The present manuscript reports on the diagnosis and treatment of such a lesion, which caused significant maxillary bone loss in a twelve-year-old child.

Pyogenic granuloma; Bone loss; Histopathologic examination