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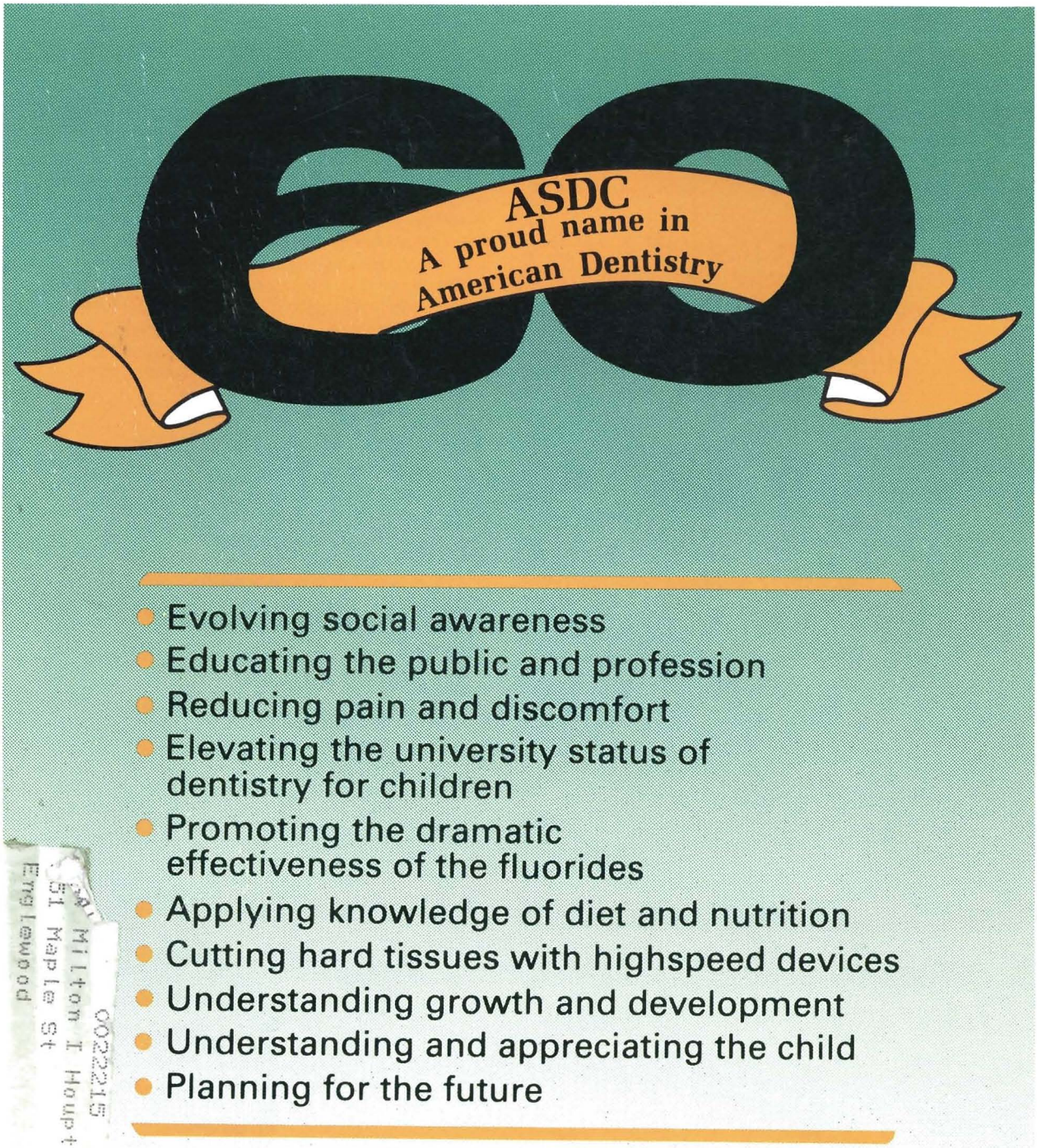

A HIGH PREVALENCE OF DENTAL CARIES TO A SIGNIFICANTLY LARGE CARIES-FREE POPULATION

ASDC AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

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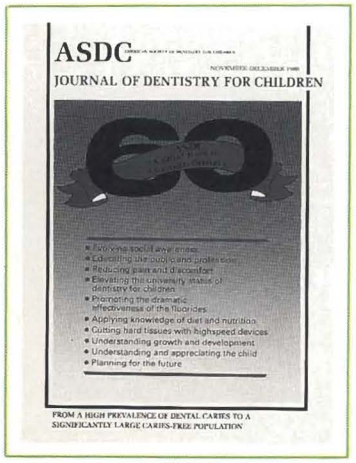
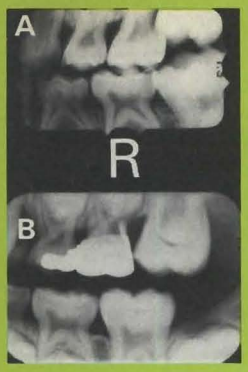


The cover introduces the 60th year since the founding of ASDC in 1927. The era sparkles with dramatic improvements in children's dental care, and is marked by the emergence of a large population of caries-free children.



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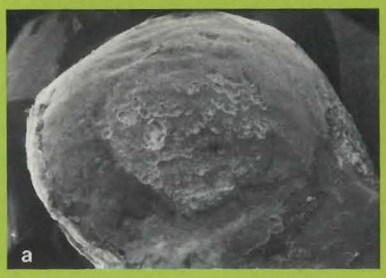
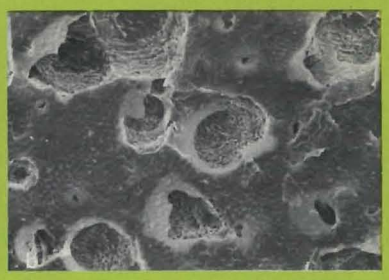




NUTRITION

CLINIC

For the busy reader

Bonding strengths of etched porcelain disks and three different bonding agents—page 409

The most popular porcelain laminate technique uses a porcelain laminate veneer baked against a tooth die made with porcelain-inlay investment. It is now known that the bond strengths of resin bonded to etched metal exceed the bond strengths of resin bonded to enamel; etched metal disks are therefore desirable surfaces for testing the bonded porcelain material. The bond strengths of all groups tend to be stronger than those reported for resin bonded to etched enamel.

Requests for reprints should be directed to Dr. David R. Avery, Indiana University School of Dentistry, 1121 W. Michigan Street, Indianapolis, IN 46202.

Solubility changes of human enamel treated with precipitate-free, aged SnF_2/amine fluoride 297 solutions—page 415

The optimal concentrations of SnF_2 to AmF 297 required to reduce the dissolution rate of enamel need not be the same as that necessary to achieve optimal antiplaque effects *in vivo* and *in vitro*. Applied clinically, the mouth could be rinsed with the test solution after conventional brushing with a dentifrice.

Requests for reprints should be directed to Dr. Fred Barbakow, Dental Institute, University of Zurich, Plattenstrasse 11, CH-8028 Zurich, Switzerland.

Incidence of temperature elevations after a full mouth dental rehabilitation under general anesthesia—page 420

Postoperative temperature elevations may occur as a consequence of general anesthetic procedures. The elevated temperatures observed in this study were compared for any association or correlation between the Oral Hygiene Index, Gingival Index Score, extraction of teeth, and degree of soft tissue trauma. The results indicated no cause-and-effect relationship.

Requests for reprints should be directed to Dr. N. Sue Seale, Baylor College of Dentistry, Department of Pedodontics, 3302 Gaston, Dallas, TX 75246.

The therapeutic effectiveness of nitrous oxide and chloral hydrate administered orally, rectally, and combined with hydroxyzine for pediatric dentistry—page 425

This study compared the effectiveness of three pediatric sedation modalities, based on a subjective evaluation of the overall quality of the sedation technique and data obtained from a pulse/oximeter (used to monitor the sedated patients). None of the three methods tested produced any significant respiratory depression.

Requests for reprints should be directed to Dr. Arthur P. Mourino, Department of Pediatric Dentistry, VCU-MCV School of Dentistry, Richmond, VA 23298.

Treatment of ectopically erupting maxillary permanent first molars with a distal extended stainless steel crown—page 430

This paper reports a technique to redirect into proper occlusion ectopically erupting maxillary first permanent molars. A stainless steel crown with a distal guide plane is placed on the primary second molar to correct the aberrant eruption angle of the permanent tooth.

Requests for reprints should be directed to Dr. Michael W. Roberts, National Institute of Dental Research, National Institutes of Health, Bldg. 10, Room 7C406, Bethesda, MD 20892.

Comparative analysis of ectopic eruption of maxillary permanent first molars in children with clefts—page 433

The purpose of this study was to analyze the prevalence of ectopic eruption among the following specifically defined cleft types: cleft lips, cleft lip and palate (including bilateral), cleft of the hard and soft palate, cleft of soft palate only, submucous cleft, and velopharyngeal insufficiency.

Requests for reprints should be directed to Dr. Dennis N. Ranalli, School of Dental Medicine, University of Pittsburgh, School of Dental Medicine, 3501 Terrace Street, Pittsburgh, PA 15261.

Prenatal factors and tooth eruption in children with oral clefts—page 436

The timing of eruption of primary central incisors was studied in seventy-six children with oral clefts. No previous data were available for this timing. The eruption timing of first primary teeth was found to be very close to that of healthy children.

Requests for reprints should be directed to Dr. Matti Pöyry, Cleft Center, Helsinki University Central Hospital, Helsinki, Finland.

Comparison of oral microflora between well-nourished and malnourished Nigerian children—page 439

Anaerobic microbes were rarely isolated, by the sampling techniques used, in well-nourished Nigerian children, in whom a wide spectrum of facultative microbial flora was displayed. In contrast, their malnourished counterparts almost always harbored colonies of *Bacteroides*, *Actinomyces*, and spirochetes. General oral health improved with a high-protein, balanced diet.

Requests for reprints should be directed to Dr. Danny R. Sawyer, Department of Pathology, Loyola University of Chicago, School of Dentistry, 2160 S. First Avenue, Maywood, IL 60153.

Physiological role of dietary fiber: a ten-year review—page 444

The past ten years have shown an upswing in interest in dietary fiber in both nutritional and medical communities. Dietary fiber is a very complex group of substances influencing the digestion and absorption of food in the upper alimentary canal and the formation of feces in the lower portion, the colon and rectum.

Reprints not available.

Undetected errors in cavity preparations from a preclinical course and their possible clinical implications—page 448

An evaluation of accepted class II cavity preparations from a preclinical pediatric dentistry course, taken after the course, showed significant levels of undetected errors in fifteen cavity criteria. Under clinical conditions the undetected errors would have caused failure of the restorations for a number of reasons.

Requests for reprints should be directed to Enrique Bimstein, C.D., Pedodontic Department, Hebrew University, Jerusalem, Israel.

Anterior space maintainer and regainer—page 452

A direct-bonded space maintainer and regainer is proposed here; the main goal is to correspond to the particular needs of the preschool child. By applying simple orthodontic principles and devices, the appliance can function more directly and effectively than a static space maintainer. It maintains arch symmetry and avoids occlusal impairment.

Requests for reprints should be directed to Dr. Ruben E. Bayardo, Departments of Pedodontics and Orthodontics, Facultad de Odontologia, Universidad de Guadalajara, Mexico.

Tourette's syndrome: management of oral complications—page 456

This well-recognized clinical disorder appears regularly in clinical psychiatry; the onset usually occurs in childhood, between the ages of two and fifteen years. An oral characteristic of the syndrome is compulsive biting of the tongue. Treatment is strictly symptomatic.

Requests for reprints should be directed to Dr. Oariona Lowe, Director of Dental Services, City of Hope National Medical Center, 1500 E. Duarte Road, Duarte, CA 91010.

Bonding strengths of etched porcelain discs and three different bonding agents

Jong Gap Lee, DDS, MSD, PhD
B. Keith Moore, MS, PhD
David R. Avery, DDS, MSD
Suteera T. Hovijitra, DDS, MSD

Considerable interest has developed for the use of porcelain laminate veneer restorations bonded to enamel surfaces. Recent publications by Horn, Calamia, McLaughlin, Boksman *et al*, and Czarkowski describe various clinical techniques utilizing bonded porcelain laminates.[1-7] The clinicians utilizing these restorations believe that the porcelain laminates offer superior clinical results, when compared to the resin laminate veneer restorations.

The most popular porcelain laminate technique utilizes a porcelain laminate veneer baked against a tooth die made with porcelain-inlay investment. The inside or tooth contacting surface of the laminate is treated with a etchant containing hydrofluoric acid. The etched surface of the laminate is coated with silane to enhance the bond between the porcelain and the luting resin. Next the laminate is bonded to the enamel surface by conventional tooth-bonding techniques, utilizing a resin material to affix the etched porcelain surface to the etched enamel surface.

Dr. Lee is Professor and Chairman of Pedodontics, College of Dentistry, Yonsei University, Seoul, Korea. Dr. Moore is Professor of Dental materials; Dr. Avery is Professor and Chairman of Pediatric Dentistry; and Dr. Hovijitra is Associate Professor of Fixed and Removable Partial Prosthodontics at Indiana University School of Dentistry. Dr. Lee was Visiting Professor of Pedodontics at Indiana University School of Dentistry during the research phase of this project.

Although the clinical use of these restorations is increasing, few data are currently available to document the efficacy of the procedure. The available data, however, demonstrate good results. In 1983, Simonsen and Calamia reported tensile bond strengths of etched porcelain sufficient to cause fracture of both the porcelain and resin materials, when the specimens were stressed to failure in an Instron testing machine.[8] Further studies by the same co-workers showed that the etched porcelain surface was most important for satisfactory bond strengths, but a silane coupling agent added to the etched porcelain surface resulted in somewhat stronger bonds.[9] Additional work by Hsu, Stangel, and Nathanson demonstrated superior bond strengths, when the porcelain surfaces were first treated with silane before bonding.[10] More recently, a report by Hold, Bertolotti, and Lacy showed that resin bonded to silane-treated, etched porcelain resulted in bonds stronger than the tensile strength of the resin itself.[11]

This project was initiated to measure bond strengths of a particular porcelain system specifically designed for porcelain laminate veneer restorations.* The system has been used to restore to normal appearance discolored and malformed teeth; but bond-strength data for this system are lacking in the literature.

METHODS AND MATERIALS

The basic bond strength test used in this study was patterned after the tests reported by Holland, *et al*, in 1984.[12] The study just cited measured the retentive strengths of different luting agents, by cementing two cast and etched base-metal** discs together and then recording the tensile stress required to break the bond between them. The study reported here utilized the same testing system, but a flat etched porcelain disc (simulating a porcelain laminate veneer) was cemented between the two etched base metal surfaces. Adequate retentive strengths of resin luting agents bonded to etched enamel surfaces and to etched metal surfaces for clinical use have already been demonstrated. The purpose of this study was to measure the bond strengths of certain resin luting agents cured against the etched porcelain surfaces. Since it is now known that the bond strengths of resin bonded to etched metal exceed the bond strengths of resin bonded to etched enamel, the etched metal discs seemed to offer desirable surfaces for testing the bonded porcelain material.

Forty cylindrical porcelain discs 0.8 mm thick and 7.0 mm in diameter were used in the bond-strength tests with three different luting agents. The thickness of the discs was selected to represent approximately the maximum thickness of an actual laminate veneer restoration. This dimension may have an effect on the curing properties of light activated resin luting agents, since the curing light beam must pass through the porcelain material. The disc diameters were selected to match the base metal castings already available.

The supplier's materials and directions (Chameleon Dental Products) were followed throughout the process of fabricating and etching the porcelain discs. Many vinyl polysiloxane† impressions of a tooled stainless steel die with four holes 7.0 mm in diameter and 0.8 mm thick were made. These impressions were boxed in wax and poured with the porcelain investment, thus producing a positive replica of the original metal die with the four holes 8 mm apart. The investment die was then separated into four blocks so that each hole was centered in an individual block of investment. The porcelain discs were made by incremental additions of porcelain, mixed to a thick consistency, vibrated into the dies and firing them in a porcelain furnace. Thus the discs were fabricated in layers and required four or five firings each. Since both surfaces of each disc were to be flat, parallel and baked against investment (to simulate the tooth contacting surface of a porcelain laminate), the last increment of the porcelain slurry was vibrated into place and slightly overfilled; then another block of investment was vibrated against the slurry until it fit flat on the top of the investment die. The two blocks of investment were ligated tightly together and baked in the porcelain furnace for the last firing. The discs were then carefully devested, cleaned with a shell blaster and etched for ninety seconds according to the supplier's recommendations. After thorough rinsing and drying, the discs were stored in a clean and dry environment until used.

Each cast metal specimen used in the tests consisted of a cylindrical, 3 mm thick disc with a 7.0 mm diameter. A shaft extended vertically from the center of one surface of the disc and a hole was placed near the free end of the shaft. The shank portion of a high speed bur could pass through the hole allowing the bur to attach the metal specimen to a swiveling apparatus designed for tensile tests in an Instron testing machine. The remaining flat surface of each specimen was prepared for etching. Hand grinding each surface on 400 grit silicon-carbide paper was followed by grit-blasting with fifty micrometer alumina. Care was taken not to touch the

*Chameleon Porcelain Systems, Chameleon Dental Products, Kansas City, KS 66101.
**Rexillium III, Rx Jeneric Gold Co., Wallingford, CT 06492.
†Mirror 3, Sybron/Kerr, Romulus, MI 48174.

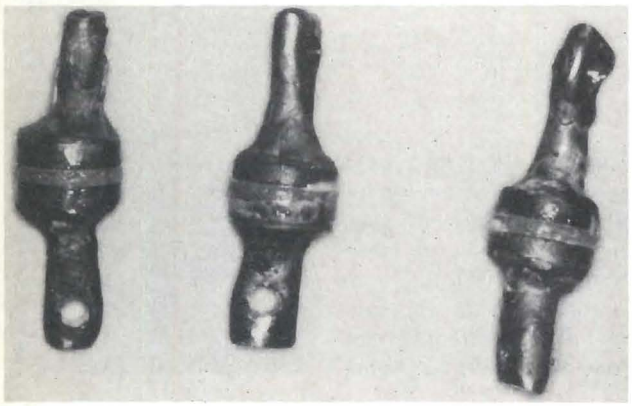


Figure 1. Three bonded test specimens ready for bond strength evaluation. The etched porcelain discs are centered between the two metal surfaces. No attempt was made to remove any of the thin resin flash extending onto the round, unetched, outside metal surfaces to insure that no inadvertent damage occurred to the porcelain discs.

test surfaces after blasting and the specimens were immediately stored in acetone until needed.

On the day that the porcelain discs and metal specimens were to be bonded together, the test metal surfaces were electrolytically etched, according to the procedure developed by Thompson and Livaditus.[13,14]

The procedure to produce each test specimen simply involved making a "sandwich" consisting of an etched porcelain disc bonded with resin to and between two etched metal discs (Figure 1). The two etched porcelain surfaces of each disc were coated with a silane coupling agent just before the resin bonding procedure. Three different resin luting systems were tested. All systems incorporated the initial use of an unfilled resin bonding material to coat the etched surfaces, followed by the application of a dilute composite resin for cementation. One resin system was designed originally for the cementation of resin bonded bridges.† † Another system was designed for bonding orthodontic brackets to enamel.‡ The third resin system was designed specifically for bonded porcelain laminate veneer restorations to enamel.‡ ‡ The first two resin systems are chemically cured, while the third is light-cured. The manufacturers' directions for their respective resin systems were followed during the bonding process.

A Teflon holding device, designed to stabilize and align the porcelain and metal discs together, during the bonding procedure, was used to standardize the cementation. Each specimen bonded with chemically cured materials was placed under a static load of 735 grams for five minutes. Only one surface of a porcelain disc could be bonded with the light cure system, since the light beam had to pass through one side of the porcelain and activate the polymerization of material on the other side; after the first surface was bonded to metal, therefore, the light could not be used for bonding the second surface. The other porcelain surface was then bonded to a metal surface with the orthodontic resin cement. Each porcelain surface bonded with the light cure material was exposed to the light for sixty seconds, while under pressure in the Teflon device.♣

Ten porcelain specimens (twenty surfaces) were bonded with each of the two chemically cured resin systems. Since only one surface of each porcelain disc could be bonded with the light-cure system, twenty porcelain specimens were bonded on one side with the light-cured material. Ten of these specimens were bonded with light-cured material on the porcelain surface baked against the investment die (LC Group A) and the other ten were bonded on the surface baked against the flat investment block placed during the last firing of each disc (LC Group B). Thus four groups of ten specimens each were tested for tensile strength and the test results were analyzed by analysis of variance. The Newman Keul's Test was employed for multiple comparison of test groups.

Before conducting the tensile tests, each prepared specimen was stored in distilled water at 37°C for one week. After three days, the specimens were removed from storage long enough to subject them to 2500 continuous thermal cycles between two water baths that varied by 40°C in temperature (15°C and 55°C).

At the end of the storage period, the specimens were loaded in tension until failure occurred, using a speed of .762 mm/minute with the Instron testing machine (Figures 2 and 3). The force required to cause separation of each test specimen was recorded as the bond strength. After failure, all specimens were examined with a dissection microscope to determine the nature of the break.

Before preparing the forty porcelain specimens used in these tests, many trials were required to develop a technique for producing the discs with satisfactory flat surfaces on both sides. A preliminary project involved assessing the quality of the etched porcelain surfaces

† †Conclude, Dental Products/3M, St. Paul, MN 55144.
‡Mar-Bond, Reliance Orthodontic Products, Inc., Itasca, IL 60143.
‡ ‡Chameleon Light Cure, Chameleon Dental Products, Kansas City, KS 66101.
♣Prisma-Lite, The L.D. Caulk Company, Milford, DE 19963.

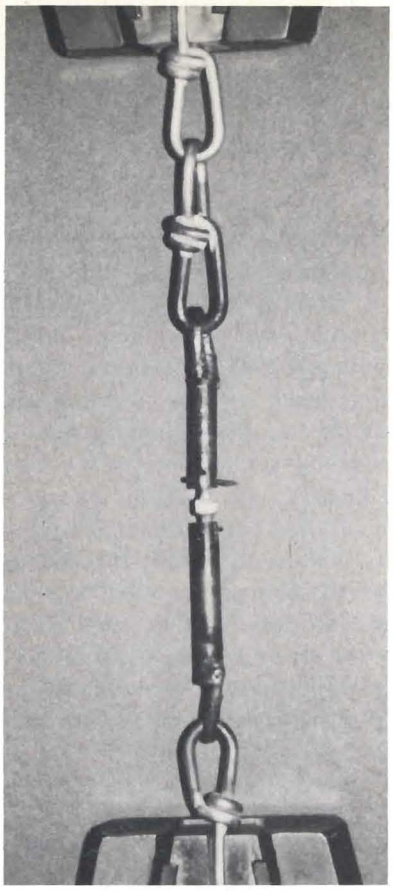


Figure 2. A specimen loaded for the tensile test. The chain-link apparatus permitted adjustment during loading to minimize the potential for shear forces.

with the scanning electron microscope (SEM) at different etching times. Porcelain surfaces fired against the investment, but not etched, and porcelain surfaces ground smooth were also observed under the SEM for comparison to etched surfaces. The SEM appearance of porcelain surfaces etched for 1.5 min, 2 min, 3 min, 5 min, and 10 min were compared. It was concluded that the supplier's recommended 1.5 min etch-time produced the desired surface as well as, or better than, longer etch-times (Figures 4 and 5).

A pilot project which involved making and breaking twelve bonded specimens to validate the testing procedure was conducted before the main project. This pilot project confirmed that placing a silane coating on the etched porcelain surface before the bonding procedure enhanced the retention strength of the specimens.

RESULTS

The mean tensile bond-strengths and their standard deviations of the test specimens are reported in the Table. Although the mean tensile bond-strength of the specimens in the orthodontic resin-group exceeded the mean strength of those in one of the light-cured groups

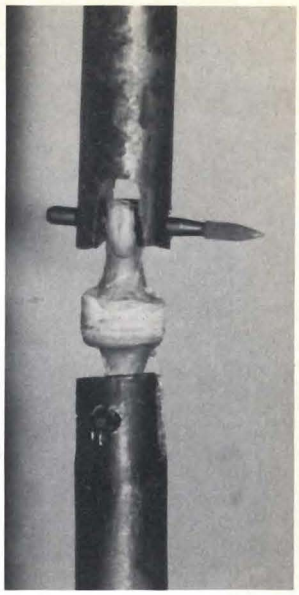


Figure 3. Close-up view of specimen loaded for the tensile test shows the high speed bur shank serving to attach the specimen to the testing apparatus. Both the apparatus and the specimen could rotate freely around the bur shank.

by over 700 psi, the analysis of variance indicated that there were no significant differences in the strength values at the $p \leq .05$ level. The nature of the bond failure of the two chemically cured resin-groups, however, compared to the two light-cured groups was noticeably different by direct observation. All bond failures in the chemically cured test groups showed evidence of porcelain fractures. In fact, seven specimens out of ten in each of the two chemically cured resin-groups (fourteen total specimens) showed complete or nearly complete failure through the porcelain material (Figures 6 and 7). Whereas the bond failures in the light-cured test groups typically showed failure through the resin interface (Figure 8). If the two light-cured groups are combined, four specimens out of twenty showed some evidence of porcelain fracture and only one was a complete failure through the porcelain material. Observation of the light-cured specimens under the dissection microscope suggested that the failure tended to occur between the unfilled, resin bonding-agent and the composite resin cement.

DISCUSSION

The fact that failure of the light-cured specimens occurred through the resin materials is not particularly disturbing, since the strength values certainly seem adequate. The nature of the failures, however, causes

Table ☐ Mean tensile bond strengths of test specimens in each group

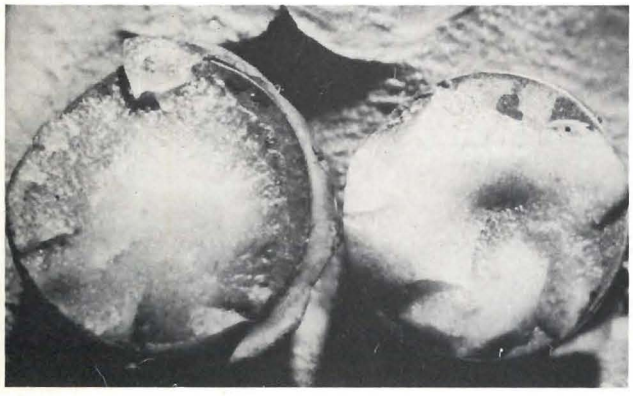
Test group	Mean strength, psi	Standard deviation, psi
Bridge bonding resin	3017	869
Orthodontic bonding resin	3737	693
LC resin group A	3352	506
LC resin group B	3009	578

Figure 4. SEM photograph of an unetched porcelain surface fired against the porcelain inlay investment. Magnification is 500x.

Figure 5. SEM photograph of an etched porcelain surface fired against the porcelain inlay investment. Surface was etched for 1.5 minutes. Magnification is 500x.

one to speculate about the adequacy of the polymerization between the resin bonding agent and the composite cement. The porcelain discs were thicker than most actual porcelain laminates and perhaps the 60 sec exposure of the light beam directed through the disc to the resin was only minimally adequate. Additional studies in which the thickness of the discs and the light exposure times varied would help to clarify this issue.

As indicated earlier in this report, considerable effort was required to develop a satisfactory technique for consistently producing porcelain discs with flat and parallel surfaces. A good surface on the side of the disc fired in the investment die could consistently be produced and this surface would be analogous to the tooth contacting surface of an actual laminate veneer restoration. Consistently producing a comparable surface on the "top" of the disc, however, was more difficult. Although the technique used in this project produced surfaces on both sides of the discs that appeared grossly comparable, subtle differences could be seen under the SEM. The fact that nearly all specimen failures in both chemically cured groups occurred in the porcelain near the "top" surface seems to bear out the authors' suspicions that this surface was weaker than the surface most similar to an actual laminate veneer. This pattern of relative weakness in bond strength on the "top" porcelain surface was even reflected in the data of the two light- cured groups, despite the fact that separation occurred in the resin



Figures 6 and 7. Photographs illustrating the type of fractures frequently observed in the two chemically cured test groups. The failure of the specimens tended to occur in the porcelain material rather than at the resin interface.

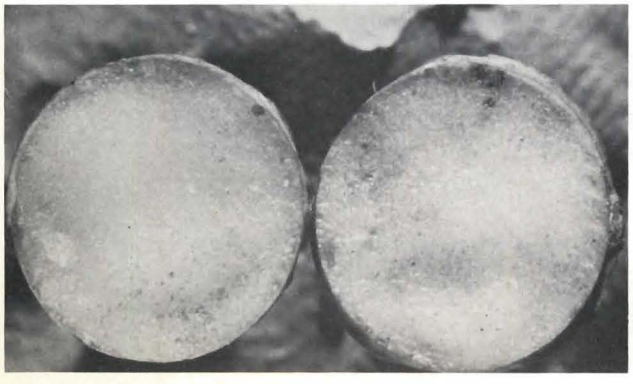
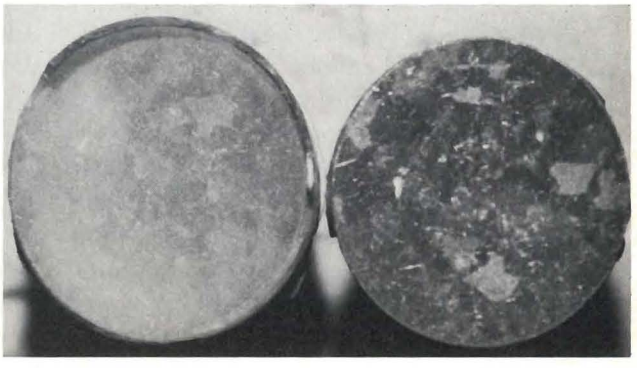


Figure 8. This photograph illustrates the typical type of failure observed in the light-cured test groups. The porcelain remained virtually, if not completely, intact and failure occurred by separation of the resin luting materials.



materials. These data do not reflect, therefore, the true resin-to-porcelain bond strength of the surfaces most similar to an actual laminate veneer restoration. Nevertheless, all mean bond strengths reported here exceed the best values reported by Bowen and Cobb for resin-to-etched-enamel (2750 psi) and would, therefore, be expected to be adequate in clinical situations.[15]

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SPEAKING OF ENGLISH

The rise of English is a remarkable success story. When Julius Caesar landed in Britian nearly two thousand years ago, English did not exist. Five hundred years later, English, incomprehensible to modern ears, was probably spoken by about as few people as currently speak Cherokee—and with about as little influence. Nearly a thousand years later, at the end of the sixteenth century, when William Shakespeare was in his prime, English was the native speech of between five and seven million Englishmen and it was, in the words of a contemporary, "of small reatch, it stretcheth no further than this iland of ours, naie not there over all".

Four hundred years later, the contrast is extraordinary. Between 1600 and the present, in armies, navies, companies and expeditions, the speakers of English—including Scots, Irish, Welsh, American and many more—travelled into every corner of the globe, carrying their language and culture with them. Today, English is used by at least 750 million people, and barely half of those speak it as a mother tongue. Some estimates have put that figure closer to one billion. Whatever the total, English at the end of the twentieth century is more widely scattered, more widely spoken and written, than any other language has ever been. It has become the language of the planet, the first truly global language.

McCrum, R.; Cran, W.; MacNeil, R.;
The Story of English. New York: Viking,
1986, pp 19,20

Solubility changes of human enamel treated with precipitate-free, aged SnF_2/amine fluoride 297 solutions

Fred Barbakow, BDS, HDD, MSc (Med)
Felix Lutz, MD, DMD, PhD
Walter Scherle, Snr Technician
Marinus Ransberger

Dental caries incidence is decreased by different fluoride salts at different concentrations, using various methods of application.[1] SnF_2 is frequently used topically and as a mouthwash for both children and adults, and is also used by patients requiring or having had head-and-neck radiation therapy. Its instability, however, in aqueous solution, is a limiting factor. Shannon stabilized SnF_2 by heating in anhydrous glycerine at 150° C and since then Mühlemann *et al* (1981) and Mühlemann and Schmid (1985) reported that aged aqueous solutions of SnF_2 and amine fluoride 297 (N'-Octadecyl-trimethyldiamine-N'N'N'-tris (2-ethanol)-dihydrofluoride) remained precipitate-free and had excellent anti-cariogenic properties.[2-4]

Previous studies showed that SnF_2 alone reduced the enamel dissolution rate *in vitro* and the products formed on enamel after topical SnF_2 application were characterized.[5-7] An *in vitro* pilot study indicated that the enamel dissolution rate, measured by the loss of phosphorus, was reduced after immersing enamel in aged precipitate-free SnF_2/AmF 297 solutions.[8] In a more recent study, enamel specimens were more frequently

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immersed in the SnF_2/AMF 297 solutions than in the pilot study cited above. These specimens required almost an 18 min exposure to 2N HCl to allow the detection of phosphorus in the etching solutions.

The purpose of this study was to examine the surface morphology of enamel specimens in the SEM that had been repeatedly immersed in aged, precipitate-free aqueous SnF_2/AmF 297 solutions and then acid-etched for 17¾ min in 2N HCl. The amounts of phosphorus in the etching solutions were determined, and correlated with the SEM observations.

MATERIALS AND METHODS

Partially or unerupted human third molars that had been stored in 0.1 percent thymol were used in this study. The teeth were cleaned with pumice and kept in a moist environment (0.1 percent thymol) during the entire experiment. As part of a larger concurrent study, ten enamel specimens were immersed twice daily in one of the test solutions listed in the Table and water-washed in fast-flowing water for 3 min after each immersion. The immersions and water-washings were repeated five days per week for 10 weeks. Only the aqueous SnF_2 solution was freshly mixed prior to each immersion; the other solutions were aged. Uniform areas of enamel (3 mm in diameter) were successively acid-etched in 2N HCl*, for a total of 17 ¾ min and the cumulative phosphorus concentration determined calorimetrically.[9] The demarcated enamel was successively acid-etched eight times (5, 10, 15, 15 sec and 4, 4, 4 and 5 min; total etching time, 17 ¾ min) each time in separate containers, each with 5 ml 2N HCl. Two enamel areas were etched in this way on each specimen.

The etched enamel specimens were subsequently gold-coated (200 - 500 A°) at 15 mA in a Balzer BA 109 Shadow Caster** and examined in a Cambridge Stereoscan 180*** operating at 16 KV.

RESULTS

Figures 1 to 4, respectively, are photomicrographs of acid-etched enamel specimens that had been immersed in the SnF alone; or in the SnF_2/AmF 297 solutions at molar fluoride ratios of 3:1; 1:1; and 1:3. Figures 5 a and b are photomicrographs of an enamel specimen treated with water. Figure 6a details the etched enamel surface

Table ☐ List of test solutions used, their pH values, fluoride concentrations and mean cumulative phosphorus (P μg) dissolved after the 17¾ min exposure to acid.

Composition (molar ratio)	pH	ppm $\bar{F}$	N	Mean P (± SD)
SnF_2 (fresh)	3.4	250	20	0.017 (0.042)
SnF_2/AmF 297 (3 : 1) (aged)	3.6	250	20	0.387 (1.091)
SnF_2/AmF 297 (1 : 1) (aged)	4.0	250	20	0.335 (0.582)
SnF_2/AmF 297 (1 : 3) (aged)	4.4	250	20	1.437 (0.892)
Water (dist)	6.8	—	20	12.543 (3.348)

after immersion in the SnF_2 solution alone, indicating that a preexisting enamel defect was selectively acid-etched even after immersion in SnF_2 solution. Figure 6b

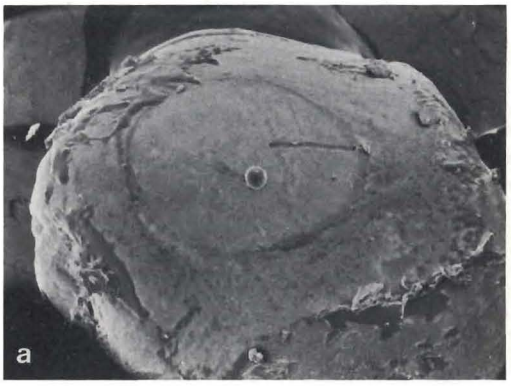

Figure 1a. Enamel surface treated with freshly mixed SnF_2 alone and acid-etched for 17 ¾ min in 2N HCl. (Mag. x 20 x 0.54).

Figure 1b. Detail of Figure 1a showing the pitted enamel surface and perikymata, without evidence of etching. (Mag. x 20 x 0.54).

*Merck, Darmstadt, West Germany
**Balzers, Liechtenstein
***Cambridge Instruments, Cambridge, UK

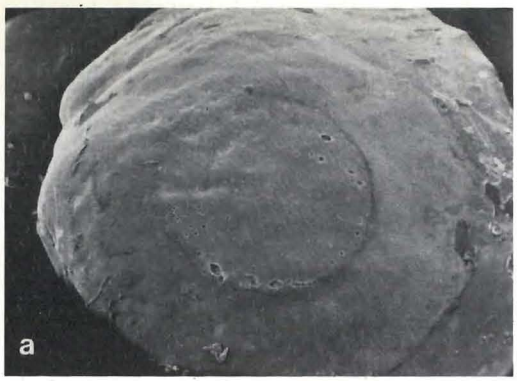

Figure 2a. Enamel surface treated with aged aqueous SnF_2 /AmF 297 solution (molar fluoride ratio 3:1) and acid-etched for 17 ¾ min in 2N HCl showing various sized etched areas. (Mag. x 20 x 0.54).

Figure 2b. Detail of Figure 2a showing the etched area with undermining of the reaction products on the enamel. (Mag. x 300 x 0.54).

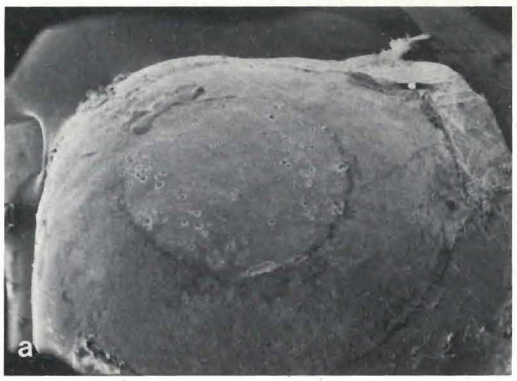

Figure 3a. Enamel surface treated with aged aqueous SnF_2/ AmF 297 solution (molar fluoride ratio 1:1) and acid-etched for 17 ¾ min in 2N HCl showing various sized etched areas. (Mag. x 20 x 0.54).

Figure 3b. Detail of Figure 3a showing the etched area with undermining of the reaction products on the enamel. (Mag. x 300 x 0.54).

shows details of the enamel etched after immersion in a SnF_2/AmF 297 solution at a 1:3 molar fluoride ratio. Undermining of the reaction products on the enamel can be seen.

The cumulative amounts of phosphorus (μg) dissolved from the treated enamel surfaces, acid-etched for 17¾ min, are listed in the Table.

DISCUSSION

Both SnF_2 and AmF 297 have excellent cariogenic properties, particularly in reducing the enamel dissolution rate and in the remineralization of white-spot lesions.[10-16] Because the SnF_2/AmF 297 mixture yielded precipitate-free aqueous solutions, any possible synergistic effect of this combination on the *in vitro* enamel dissolution rate (measured by the amount of dissolved phosphorus) was investigated.

Previously, SnF_2 was used clinically in combination with other products and recently Crall *et al* (1983) and Crall and Bjerga (1984) reported changes on enamel following topical application of SnF_2 and acidulated phosphofluoride (APF) in a two-step procedure.[17,18] Among others, these studies indicated that the APF/ SnF_2 combination effectively protected human enamel from forming artificial caries-like lesions in an acidified gel. There has been, to date, no report detailing the surface morphology of enamel immersed in aged precipitate-free aqueous SnF_2/AmF 297 solutions and then acid-etched. Nor have its effects on artificial white-spot lesions been studied.

Under the conditions of the present study, freshly-mixed SnF_2 provided maximum protection to the enamel. Although the SnF_2/AmF 297 mixture produced no synergistically decreased enamel dissolution rate *in vitro*, the protection provided against acid dissolution was similar to that of SnF_2alone. The enamel dissolution rate, measured by the amount of phosphorus dissolved from the enamel appeared to be inversely proportional to the quantity of SnF_2 in the SnF_2/AmF 297 solution. Further, the enamel dissolution rate appeared to increase with increasing pH value of the test solutions (3.4 to 4.4). One must, therefore, question the possible changes of the chemical composition of the reaction

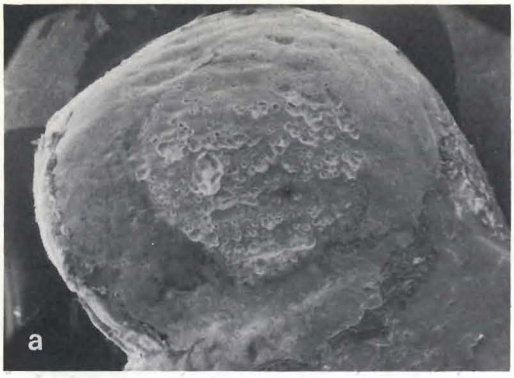

Figure 4a. Enamel surface treated with aged aqueous SnF_2/AmF 297 solution (molar fluoride ratio 1:3) and acid-etched for 17 ¾min in 2N HCl showing various sized etched areas. (Mag. x 20 x 0.54).

Figure 4b. Detail of Figure 4a showing the coalesced etched areas with undermining of the reaction products on the enamel. (Mag. x 300 x 0.54).

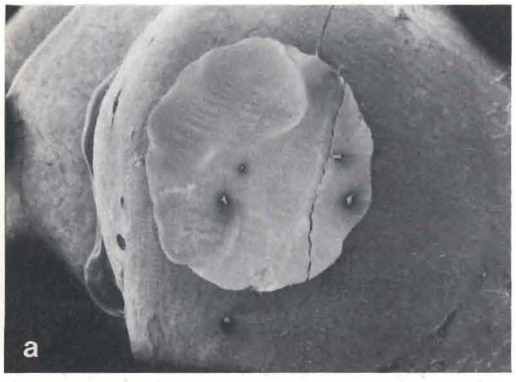

Figure 5a. Control enamel surface treated with distilled water showing a crater-like etched area after being acid-etched for 17 ¾ min in 2N HCl. (Mag. x 20 x 0.54).

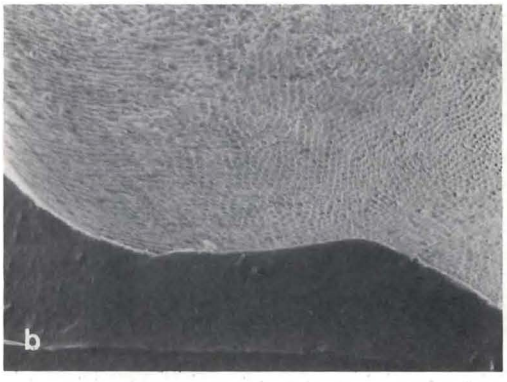

Figure 5b. Detail of Figure 5a showing the edge between the etched and nonetched enamel surface.

products on the surface enamel at different pH values. Finally, the surface products that formed on the enamel appeared to be more resistant to the etching than the enamel itself, because the former nearly always produced overhanging edges over the etched enamel (Figures 2b - 4b, 6b).

The 3 min postimmersion, water-washing period selected for this study is short and the effects of longer washing periods of up to 50 h should be investigated.[19] The chemical nature of the products formed on the enamel after immersion in the precipitate-free SnF_2/AmF 297 must be analyzed to optimize the molar concentration of SnF_2 to AmF 297. The optimal concentration of $SnF_2$to AmF 297 required to reduce the dissolution rate of enamel need not be the same as that necessary to achieve optimal antiplaque effects *in vivo* and *in vitro*. The possible clinical application of such a test solution would be to rinse the mouth after conventional brushing with a dentifrice. Because the enamel dissolution rate is reduced, the rate at which white-spot lesions are formed clinically could be reduced. In addition, both SnF_2 and AmF 297 have proven antiplaque properties *in vitro* and *in vivo*.[13,15]

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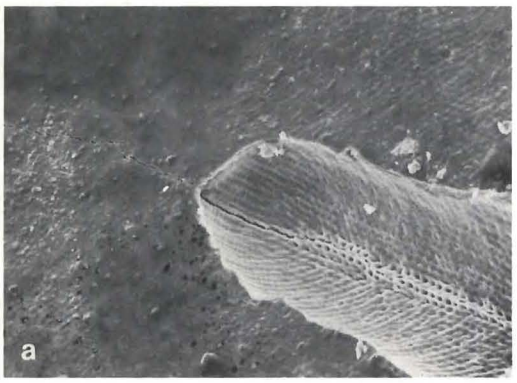

Figure 6a. Detail of enamel treated with SnF_2 alone and subsequently acid-etched for 17¾ min showing etched enamel along a preexisting defect. (Mag. x 300 x 0.54).

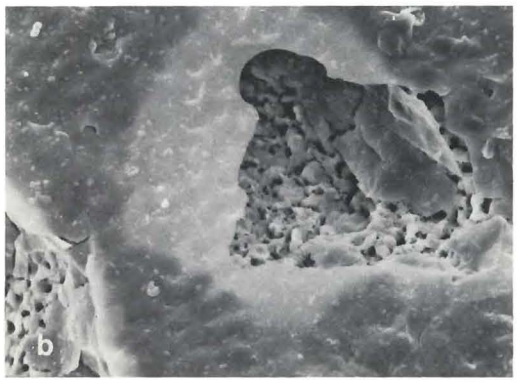

Figure 6b. Detail of Figure 4b showing the undermining of the reaction products. The etched enamel is not typical and is partially covered by remnants of the reaction products. (Mag. x 1000 x 0.54).

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THE IMMENSITY OF THE DRUG PROBLEM

The inhabitants of the earth spend more money on illegal drugs than they spend on food. More than they spend on housing, clothes, education, medical care, or any other product or service. The international narcotics industry is the largest growth industry in the world. Its annual revenues exceed half a trillion dollars—three times the value of all United States currency in circulation, more than the gross national products of all but a half dozen of the major industrialized nations. To imagine the immensity of such wealth consider this: A million dollars in gold would weigh as much as a large man. A half-trillion dollars would weigh more than the entire population of Washington, D.C.

Narcotics industry profits, secretly stockpiled in countries competing for the business, draw interest exceeding $3 million per hour. To what use will this money eventually be put? What will be its ultimate effect?

Mills, J.: The Underground Empire.
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Behavior

Incidence of temperature elevations after a full mouth dental rehabilitation under general anesthesia

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William D. Love, DMD, MPH

In the past decades, there have been advances in availability and delivery of dental services to the general public. Most dental patients can receive care in a conventional office environment. There are exceptions, however, that make an alternative method of treatment delivery necessary. The use of general anesthesia in a hospital setting has become a routine procedure for the young patient who would not be treatable in a conventional dental office environment.[1,2] Any patient subjected to the use of general anesthesia is at risk to accompanying complications. There are a number of reports which reference temperature elevations during or after surgery as a common complication.[3-5] These studies indicate that postoperative temperature elevations in pedodontic patients are common when dental procedures are performed under general anesthesia.

Many factors may be responsible for the temperature increases. General anesthetics may cause temperature fluctuations during and after surgery by inactivating the hypothalamic temperature-regulating mechanism.[6] Other causes include septicemias, bacteremias, trauma to tissues or destruction of soft tissues, environmental extremes during and after surgery, certain drugs such as atropine or scopolamine, and dehydration.[7-10]

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It is a known fact that various dental treatment modalities such as extractions, fillings, scaling and prophylaxis, and pulpotomies may cause a bacteremia.[11-14] Bacteremia may occur in procedures performed by the patient, such as brushing or even chewing.[15] It is also possible that dental manipulation of the teeth or oral tissues may result in detectable levels of bacteria in the blood, especially when bleeding is induced.

The objectives of this clinical study were to:

- ☐ Determine the incidence of temperature elevations after a dental rehabilitation under general anesthesia.
- ☐ Relate temperature increases to the dental procedures performed or the condition of the oral cavity.

MATERIALS AND METHODS

The sample in this study consisted of fifty-seven patients from Texas Scottish Rite Hospital for Crippled Children (Dallas, TX) who were scheduled for dental rehabilitation under general anesthesia. All subjects received a preoperative physical by a pediatrician, and those patients with a current history of otitis media, respiratory problems, colds, illness or antibiotic therapy were excluded from the study, since these predisposing conditions could induce postoperative temperature fluctuations.

Rectal temperature was monitored every four hours before surgery and then every two hours after surgery for a minimum of twenty hours. If a subject's temperature reached 38°C, the pediatrician required that the patient be treated with antipyretic agents until the temperature went below 38°C. A significant elevation had occurred, therefore, when the temperature reached 38°C.

At the time of the dental rehabilitation, the following information was recorded for each patient:

- ☐ Simplified Oral Hygiene Index Score (OHI).[16]
- ☐ Löe and Silness Gingival Index Score.[17]
- ☐ Number of extractions performed.
- ☐ Degree of soft tissue trauma inflicted by dental procedures.
- ☐ American Society of Anesthesiologist classification.[18]

Soft tissue trauma was arbitrarily classified as:

- ☐ Mild—rubber dam used and restorations (crowns and amalgams) placed.
- ☐ Moderate—all procedures just named plus extractions of one to two primary teeth with no more than two-thirds root present.
- ☐ Severe—all procedures named above plus extraction of fully rooted primary teeth and permanent teeth.

Complete sets of data were collected for thirty-eight of the fifty-seven subjects in the study. Patients with complete data ranged in age from 1.5 years to 17 years, with a mean age of 4.7 years. There were eighteen females and twenty males.

The subjects were assigned to one of two groups consisting of those attaining a significant temperature elevation of 38°C or greater and those with a temperature that did not reach 38°C (designated as the control group).

The preoperative OHI and Gingival Index scores were compared between the two groups. The tissue trauma resulting from the extraction of teeth and other dental procedures during the oral rehabilitation were also compared between the two groups. By statistical analysis, significant differences between the two groups were determined. Specifically, the Student's t test was used to identify a difference in the preoperative OHI scores. Due to the unlimited or nonspecific nature of the data, a Chi- square analysis was used to test for differences in the preoperative Gingival Index scores, extraction of teeth and degree of soft tissue trauma during the dental rehabilitation. The significance level was set at $p<0.05$.

RESULTS

Temperatures were measured and recorded at thirteen intervals including the admission temperature, the preoperative temperature, the postoperative temperature and an additional ten measurements every two hours postoperatively. All patients had temperature elevations. In seventeen subjects, the temperature elevation reached 38°C or above, and in the remaining twenty-one, the temperature elevations did not reach 38°C. Thus, 45 percent of the subjects had a significant temperature elevation.

The general temperature pattern showed a decrease at the preoperative and immediate postoperative periods, followed by a rise through the four-to-eight-hour period. Then a gradual temperature decrease occurred back to the admission temperature. By the twenty-hour measurement period, the temperature had returned to the admission level for all subjects.

Sixteen of the seventeen subjects with temperature elevations were between the ages of 1.5 and five years. The other subject was nine years of age. Nine subjects were male and eight were female.

Eleven of the seventeen patients (65 percent) with significant temperature elevations to 38°C had the elevation occur in the two-to-eight-hour measurement period after surgery. The other six occurred at various

Table 1 ☐ OHI mean values.

Group	Mean OHI
Normal temperature N = 21	2.365 ± 0.71*
Elevated temperature N = 17	2.036 ± 0.58*

*Standard Deviation
Calculated t value = 1.53, 36d.f. (N.S. $p<.05$)

Table 2 ☐ Chi-square analysis of gingival index.

Group	Inflammation	
	Mild	Moderate
Control group N = 21	5	16
Elevated temp group N = 17	8	9

Calculated Chi-square value = 2.25, 1d.f. (N.S. $p<.05$)

Table 3 ☐ Chi-square analysis of extractions.

Group	#Without extractions	#With extractions
Control group N = 21	6	15
Elevated temp group N = 17	8	9

Calculated Chi-square value = 1.39, 1d.f. (N.S. $p<.05$)

Table 4 ☐ Chi-square analysis of soft tissue trauma.

Group	Mild	Moderate	Severe
Control group N = 21	4	12	5
Elevated temp group N = 17	5	7	5

Calculated Chi-square Value-1.02, 3d.f. (N.S. $p<.05$)

times, mostly in the ten-to-eighteen-hour period after surgery.

The duration of the significant temperature elevation for the seventeen patients varied from two to eight hours. Eight subjects showed a temperature elevation for two hours, three for four hours, five for six hours, and one for eight hours. The elevations did not always occur at successive measurement periods for all subjects.

The two groups were compared for preoperative OHI scores, using the Student's t test. Table 1 shows the means and standard deviations for the elevated- temperature group and the control group. There were no significant differences between the groups.

The two groups were also compared for preoperative Gingival Index scores, using a Chi-square analysis. Table 2 shows the frequencies for mild and moderate inflammation in both groups, and there were no significant differences between the groups.

The two groups were compared for extraction of teeth, using a Chi-square analysis. Table 3 compares the frequencies for patients with and without extractions in both groups. There were no significant differences between the groups.

The amount of soft tissue trauma from the dental procedures was also a factor for comparison, between the two groups, using a Chi-square Analysis. Table 4 shows the frequencies of mild, moderate, and severe tissue trauma in both groups. There were no significant differences between the groups.

These data indicated that the temperature elevations were sequelae to the general anesthesia and not necessarily to the dental procedures.

DISCUSSION

This study monitored temperature closely and consistently for up to twenty hours after dental surgery. The results showed 45 percent of the subjects had temperature elevations to 38°C or above, at which point antipyretic therapy was begun. Temperatures decreased within a few hours after antipyretic therapy; thus, this study was unable to determine whether the temperature elevation would decrease spontaneously or whether antipyretic therapy was actually needed. This study showed that postoperative temperature elevations can be more prevalent than previously reported. These findings are in agreement with the findings of other investigators who reported temperature elevations in from 12 percent to 25 percent of their subjects.[3-5] None of these previous studies stated, however, how closely or often the temperatures were monitored after surgery. Thus, there may have been more temperature elevations than reported.

The sex of the subjects had no effect, since the latter were almost evenly divided between male and female. The ages of the patients showed a definite pattern. With one exception, all subjects with a significant temperature elevation were five years of age or under. The exception was nine years of age. These findings are in agreement with the findings of other studies that reported that most of their subjects with temperature elevations were under age ten.[4,5] These reports indicate that temperature elevations are more prevalent in younger age-groups. This study confirms the tendency for temperature elevations to occur in the younger age-group.

Most of the subjects (65 percent) with significant temperature elevations had the elevation occur in the four-hour-to-eight-hour measurement period. The other 35 percent had the elevations occur mainly in the ten- hour to eighteen-hour measurement period. The duration of the temperature elevations varied from two to eight hours.

If the temperature elevation occurred in the immediate postoperative period, it would likely be caused by the lingering effects of the general anesthesia on the hypothalamus.[6] In the four-hour-to-eight-hour measurement period, the general anesthetic could still have a lingering effect on the hypothalamus, but the effects of a bacteremia or septicemia could become a factor. It appears that the time for a temperature elevation to occur after a bacteremia or septicemia is indefinite. Major factors could be the subject's natural immune response and ability to resist the bacteremia or septicemia. The type and severity of the septicemia or bacteremia would play roles. This may be why the temperature elevations did not occur at specific postsurgical times. It did appear that by twenty hours postsurgically, the temperature was back at a normal level and the effects of the surgery were over.

Dehydration should not have been a postsurgical problem, in the study. The anesthesiologist usually ordered patients to have no food or liquids by mouth after midnight. This was adjusted according to the age of the patient. The fluid deficit before surgery was calculated and replaced during surgery, so that by the end of surgery the patient would be well hydrated. After the patients were returned to the room, oral fluids were encouraged. Some patients had episodes of postoperative nausea and vomiting, but showed no increased incidence of temperature elevation, due to lack or loss of fluids.

The comparison of the two groups brought some surprises. According to previous studies, the more periodontal disease or gingival inflammation present, the more likely a bacteremia is to occur.[7,9,16,18] This study showed there were no differences between the groups in the Gingival Index scores or OHI scores. One would have expected the elevated temperature group to have higher OHI and Gingival Index scores, because these conditions are more likely to lead to a bacteremia and subsequent temperature elevation.

The extraction of teeth can cause a bacteremia as well as tissue damage. Since tissue damage can also influence a potential temperature elevation, it would be expected these two factors would be more prevalent in the elevated temperature group. This study showed there was no difference in the groups for either factor.

This study showed no association between the dental procedures performed or the condition of the oral cavity with a temperature elevation. Since so many factors play a role in the body temperature, it would be logical that all the factors mentioned had the potential to have some effect. To what degree each factor influenced temperature elevation was unclear.

SUMMARY AND CONCLUSIONS

Fifty-seven patients underwent a full-mouth dental rehabilitation under general anesthesia at Texas Scottish Rite Hospital for Crippled Children. Thirty-eight subjects had complete data collected to monitor for temperature elevation. Nineteen subjects were discarded from the study due to incomplete data. The results showed 45 percent (seventeen subjects) had a significant temperature elevation to 38°C or above. Sex of the subject made no difference. The significant temperature elevations occurred in the younger age-groups, especially in the one- to-five-year age-group. In the subjects with a significant temperature elevation after surgery, no definite pattern was evident as to when it would occur or how long it would last.

The study population was divided into two groups, one with a significant temperature elevation to 38°C or above, and the control group with temperatures below 38°C. These groups were then compared to see whether their preoperative Oral Hygiene Index, Gingival Index, and soft tissue trauma or extraction of teeth showed a difference. No significant difference was found. This study is the first designed specifically to monitor temperature. It reported the highest percentage of subjects (45 percent) to have a postoperative temperature elevation, in comparison to other studies. Although no single factor could be found as the cause of the postoperative temperature elevation, combinations of the factors mentioned or the effects of anesthetic may have caused the elevations.

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THE BRAIN AND ITS MESSENGERS

Of all the momentous revolutions of twentieth-century science, two hold particular promise for bringing the mystery of human consciousness into the realm of human understanding. One of these revolutions is the development of new groups of drugs that produce extraordinary effects upon the mind; the other is the explosion in our understanding—at the cellular and molecular levels—of just how the human brain works.

These two fields of inquiry are intimately related. Drugs that affect the brain in novel and unexpected ways reveal hitherto unsuspected facets of brain organization and function. An example is the introduction in 1960 of benzodiazepine drugs, such as the widely used Valium, for the treatment of anxiety. Early in the twentieth century, barbiturates were used to quiet down agitated mental functions; in higher doses, they put the user to sleep—an inconvenient, often dangerous, but presumedly inevitable aspect of any tranquilizing drug activity. The development of benzodiazepine drugs was a startling breakthrough: they were able to calm anxiety without inducing the marked sleepiness brought on by barbiturates. This unique therapeutic utility of the benzodiazepines raises important questions about brain function. The fact that they relieve anxiety without causing much sedation suggests that the alertness system of the brain is different from the anxiety system. Barbiturates and benzodiazepines have provided chemical probes that scientists can use to distinguish between these two functions so as to study them separately.

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American Library, 1986. p 1.

The therapeutic effectiveness of nitrous oxide and chloral hydrate administered orally, rectally, and combined with hydroxyzine for pediatric dentistry

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Some form of premedication is often used in providing dental treatment for children who are difficult to manage. Premedication has a variety of applications, among them for the child who, due to age or some type of handicap, lacks the ability to cooperate and for the older child who is so psychologically stressed by the dental environment that he simply refuses to permit dental treatment.[1-6] The drugs used to premedicate the child patient are usually administered orally, intramuscularly, intravenously, or subcutaneously. Each of these routes has its own particular advantages and limitations.

Due to its relative simplicity, oral administration is most often used. One particular drug, chloral hydrate, has been popular among pediatric dentists for use in children, since its introduction, following its development by Liebig in 1832.[6,7] Chloral hydrate is considered to be a relatively fast acting drug, produces few side effects, and has a wide safety margin.[4,7-9] Dosages of the drug may also be adjusted to produce various depths of sedation. The hypnotic dose, that amount which produces a light stage of arousable sleep, is considered to be 50 mg/kg, while the sedative dose ranges from 25 to 35 mg/kg. The drug is widely adaptable, therefore, to a range of sedation needs.

Oral administration of chloral hydrate has inherent disadvantages, not the least of which is the unpredictable absorption rate of the drug.[5] This is especially pertinent when the patient has had food or drink before the appointment and reinforces the importance of requiring patients scheduled for premedication to have nothing by mouth (NPO) for a period of four to six hours before administration of the drug. Chloral hydrate, a congener of ethanol, is also a source of gastric irritation, occasionally leading to episodes of nausea and vomiting.[3,5,6,8]

For the dentist who prefers to administer the drug in the office, a problem often arises with the child of preschool age who refuses to take the medication, even with parental attempts at persuasion or efforts to "disguise" the taste by combining it with orange juice or soft drinks. If this occurs, the dentist is faced with a decision to postpone the procedure, persist in his attempts to premedicate the child orally, attempt premedication by an alternate route using a different drug, or perform the dental procedure under general anesthesia. In many cases, because of insufficient training or experience, the dentist may not feel comfortable using an alternate route or drug and the child may not be a good candidate for general anesthesia. If the decision is made to use oral premedication, a clinical estimate of the drug dosage, reliability of drug absorption, and drug effectiveness must be made. If the dose is considered to be insuffi-

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cient after a set period of time (thirty to forty-five minutes) then administration of an additional amount of medication must be considered. At this point, the practitioner is faced with both an agitated child and the need for making a decision regarding how much additional time must pass before achieving an optimal drug level and an adequately sedated patient. It may now appear that the entire medication sequence has progressed from one of known dosages and rigid guidelines to one of estimates, total guesswork, and unreliability, creating the possibility of a potentially dangerous overdose. Considering these consequences, it appears that the clinician should observe Hayden's principle that oral medication should never be forcefully administered to a child who is struggling and uncooperative.[11] This is further supported by a report of laryngospasm and cardiac arrest, when a 250 mg dose of chloral hydrate was rapidly administered orally, using a syringe.[12]

Although commonly used for premedicating children in the hospital, the rectal route is rarely used for premedicating the pediatric dental patient in the private office. This route of administration may provide, however, a rational alternative to forced oral administration. The principle disadvantages of the rectal route are the inability to control precisely the quantity of medication, because of the preset dosages of the suppositories, and the variable rate of absorption, which may occur because of the presence or absence of bowel contents.[3,4,13] It offers to the dentist who chooses not to use the intramuscular, intravenous, or subcutaneous routes, however, a means by which to deliver a specific dose to a child who will not take oral medication. By adhering to the same food and drink restrictions imposed upon patients who receive oral premedication, the amount of stomach contents can be controlled, reducing the incidence of nausea or gastrointestinal upset often seen when chloral hydrate is used. Prohibiting the oral intake also decreases the esophageal and rectal peristaltic activity to a minimum, which may decrease the tendency toward defecation from rectal drug administration. The chief advantage of rectal administration is the utilization of a route over which the patient has a lesser rejection ability, assuring the delivery of the desired dose of the drug. By using chloral hydrate, the dentist, furthermore, has the advantage and confidence provided by a drug that has been shown to be both safe and effective for pediatric dental sedation.

This study was designed primarily to compare the effectiveness of nitrous oxide in combination with the oral administration of chloral hydrate, rectal administration of chloral hydrate, and the oral administration of a mixture of chloral hydrate and hydroxyzine, in the preschool age dental patient. In addition, vital signs were measured and recorded to assess the safety of these drug combinations in the sedated patients.

METHODS AND MATERIALS

Thirty patients, aged twenty-seven to seventy-four months, were selected from the Virginia Commonwealth University - Medical College of Virginia Department of Pediatric Dentistry clinic population. Their medical histories were negative for abnormal emotional or mental development and none had medical conditions, such as hepatic or renal disease, which would contraindicate the use of either chloral hydrate or hydroxyzine. Children were selected for the study because of their lack of cooperation at a previous restorative treatment visit and because of their need for amalgam or stainless steel crown restorations. Parental consent for using sedation and data collection was obtained before the child's restorative treatment visit.

All patients presented to the clinic for their restorative treatment appointment, having had nothing by mouth (NPO) for a minimum of four hours. At this appointment, they were randomly placed into one of three groups. Ten patients, average age 39.6 months, were given a dose of 50 mg/kg of chloral hydrate orally.* Ten patients, average age forty-two months, received the same dose (50 mg/kg) of chloral hydrate in the form of suppositories.** The remaining ten patients, average age 38.4 months, were given an oral dose of 30 mg/kg/ of chloral hydrate in combination with a standard dose of 25 mg of hydroxyzine.† All patients waited thirty minutes before any restorative treatment was begun. Nitrous oxide was used in concentrations of 50 percent for the completion of the injection phase of treatment and then was reduced to approximately 30 percent to 40 percent for the remainder of the treatment.

All patients were monitored by the operator, using a precordial stethoscope and a pulse/oxygen saturation meter.‡ This device uses a fingertip sensor to measure transmission of light through the nail bed of the finger. Arterial oxygen saturation is measured in relation to the amount of systolic expansion of the capillary bed.[14] The pulse and oxygen saturation readings were recorded by the dental assistant at five-minute intervals throughout the appointment.

* Noctec - Squibb, Princeton, NJ
** Aquachlor - Alcon; Humumacao, Puerto Rico
† Vistaril - Pfizer; New York, NY
‡ Nellcor - Nellcor, Co.; Hayward, CA

A subjective appraisal of the overall quality of the sedation technique was made by the operator and used to determine the relative effectiveness of the premedicants. The appraisals considered the status of the patient before, during, and after the treatment and consisted of a modification of a sedation scoring system first suggested by Barker and later modified by Barr, Wynn, and Spedding.[5,15]

RESULTS

Each sedation was scored as shown in Table 1 and classified as *Excellent* if the score totaled 6 or 7, *Good* if it was 4 or 5, *Fair* if it was 3 or 2, and *Poor* if it was 1 or 0. These results are presented in Table 2. The group members receiving chloral hydrate orally were classified as *Good* or *Excellent* in four of the ten cases (40 percent) and six were classified as *Fair* or *Poor*. The members of the groups receiving the chloral hydrate rectally and the chloral hydrate and hydroxyzine orally were classified as *Good* or *Excellent* in seven of the ten cases (70 percent) and only three were classified as *Fair* or *Poor*.

Pulse and oxygen saturation readings for each patient were averaged individually and as a combined group. These are presented in Tables 3 and 4. Those patients receiving the chloral hydrate orally had an average baseline pulse of 117.1 beats/min with a range of 98 to 182 beats/min. Their baseline oxygen saturation averaged 99.3 percent and during the appointment was 98.7 percent with a range of 94 percent to 100 percent. Patients receiving chloral hydrate rectally had an average baseline pulse of 116.4 beats/min and an average pulse rate during the appointment of 122 beats/min with a range for the group of 99 to 150 beats/min. Their baseline oxygen saturation level averaged 99.8 percent and during the appointment ranged from 94 percent to 100 percent with an average of 99.0 percent. The patients who received oral chloral hydrate in combination with hydroxyzine had an average baseline pulse of 118.9 beats/min and an average pulse rate during the appointment of 121 beats/min with a range of 98 to 145 beats/min. Their baseline oxygen saturation level averaged 98.2 percent and during the appointment was 98.6 percent with an average range of 94 percent to 100 percent.

Statistical comparison of these groups, using a one-way analysis of variance and a p value of 0.05 showed no significant differences between the average baseline pulse rates, baseline oxygen saturation values, oxygen saturation values during the appointment, or average ages of the three groups. There were significant differences, however, between the average pulses recorded during the appointments for the three groups. Further analysis of the average pulse values obtained during the appointments using the Student-Newman-Keuls Multiple Comparison Test revealed that at $p<0.05$, the group

Table 1 ☐ Modified scoring system.

Factor	Score
Reception area	
Calm or apprehensive, but cooperative	1
Apprehensive and uncooperative	0
Reception Area to Operatory	
Drowsy or asleep, cooperative or accepting instructions	1
Crying, struggling, resistant to instructions or postures other than supine	0
Reaction to injection	
No resistance, gross motor or verbal response, is asleep, or exhibits only slight wince or vocalization	1
Crying, struggling, or physical resistance	0
Reaction to restorative treatment	
Asleep or cooperative, procedure accomplished easily	3
Cooperative but restless, procedure accomplished with minimal difficulty	2
Uncooperative with mild resistance, procedure accomplished with difficulty	1
Uncooperative with marked resistance, procedure unable to be completed	0
Completion of procedure	
Drowsy or asleep, cooperative or accepting instructions	1
Struggling, crying, resistant to instructions	0

Table 2 ☐ Scoring of subjective sedation evaluations.

Drugs and routes	Scores			
	Excellent (7-6)	Good (5-4)	Fair (3-2)	Poor (1-0)
Nitrous oxide (30-50%)				
Chloral hydrate orally	1	3	3	3
Chloral hydrate rectally	2	5	2	1
Chloral hydrate and hydroxyzine orally	2	5	1	2

Table 3 ☐ Average pulse rates.

Drugs and routes	Pulse rates (beats/min)		
	Baseline	Procedure	Range
Nitrous oxide (30-50%)			
Chloral hydrate orally	117.1	133	98-132
Chloral hydrate rectally	116.4	122	99-150
Chloral hydrate and hydroxyzine orally	118.9	121	98-145

Table 4 ☐ Average oxygen saturation.

Drugs and routes	Oxygen saturation (%)		
	Baseline	Procedure	Range
Nitrous oxide (30-50%)			
Chloral hydrate orally	99.3	98.7	94-100
Chloral hydrate rectally	99.8	99.0	94-100
Chloral hydrate and hydroxyzine orally	98.2	98.6	94-100

receiving the chloral hydrate orally differed significantly from both the group receiving the chloral hydrate rectally and the group receiving the combination of chloral hydrate and hydroxyzine orally. It also showed that the values for the two groups receiving the rectal administration and the oral administration of the combination of drugs were not significantly different.

DISCUSSION

The scores obtained from the subjective evaluations of patients administered chloral hydrate rectally and those given chloral hydrate orally in combination with hydroxyzine indicate that these two methods are similarly effective. In each instance, seven of the ten sedations (70 percent) were considered *Good* or *Excellent* and only three (30 percent) were classified *Fair* or *Poor*. The scores from the patients given chloral hydrate orally, however, showed only four of the ten (40 percent) classified as *Good* or *Excellent* and six of the ten (60 percent) considered *Fair* or *Poor*, implying that it was a less effective sedation technique. Although the use of nitrous oxide represented an additional variable, which could have masked some of the results, these differences observed between the groups are simply too large to be accounted for merely by a small difference in concentrations between the patients.

These observations are supported by the average pulse rates seen during the appointments for the groups. The patients in the group that received chloral hydrate orally had significantly higher average pulse rates (133 beats/min) throughout the procedure than did those patients in the groups receiving either chloral hydrate rectally or orally in combination with hydroxyzine, which were 122 and 121 beats/min, respectively. This group also had a much wider range of pulse values observed during the procedure. The higher average pulse rates recorded were not limited only to those sedations classified as *Fair* or *Poor*, but were seen uniformly throughout the group sedated using oral chloral hydrate. This uniform increase, along with the greater ranges observed, would imply that there was an increased amount of sympathetic nervous system activity manifested as patient restlessness and agitation, and indicating a lesser depth of sedation. Based upon the data and observations, it would appear that in this particular study, chloral hydrate orally administered was not as effective in calming or sedating the preschool age child as was either chloral hydrate rectally administered or a combination of chloral hydrate and hydroxyzine given orally. It also appears that chloral hydrate administered rectally is as effective as oral administration of a combination of chloral hydrate and hydroxyzine.

When examining the oxygen saturation data obtained for each of the three groups, it may be noted that no significant differences are observed among them in either baseline oxygen saturation, levels recorded during the appointments, or ranges of the values. Since there was no significant decrease in oxygen saturation observed in either technique involving chloral hydrate given alone in doses of 50 mg/kg, and in fact there was a slight increase in oxygen saturation observed during the procedure involving chloral hydrate given orally in combination with hydroxyzine, it may be inferred that no significant respiratory depression or airway obstruction was produced during any of the sedation modalities studied. A possible explanation for this observation may be that since all patients were treated using no more than 50 percent nitrous oxide, and thus were receiving at least 50 percent oxygen, they were receiving well over the 21 percent oxygen in ambient air, as long as their airways remained unobstructed. This explanation may also provide a possible reason for the similar ranges seen for all three groups and for the increases seen in the oxygen saturation levels during the procedures for those patients sedated orally with the combined chloral hydrate and hydroxyzine. No explanation can be offered as to why there was not a similar slight increase seen in the sedations involving the chloral hydrate alone.

CONCLUSIONS

On the basis of the subjective evaluations of the effectiveness of the techniques and the pulse and oxygen saturation data provided by the monitoring equipment, it may be concluded that:

- ☐ The rectal administration of chloral hydrate at doses of 50 mg/kg is as effective in sedating the preschool age child for dental restorations as is the oral administration of a dose of 30 mg/kg of chloral hydrate and 25 mg of hydroxyzine.
- ☐ The oral administration of 50 mg/kg of chloral hydrate is somewhat less effective than either the oral administration of 30 mg/kg of chloral hydrate and 25 mg of hydroxyzine or the rectal administration of chloral hydrate at a dose of 50 mg/kg in the preschool-age dental patient.
- ☐ None of the sedation techniques examined produced any significant respiratory depression or airway obstruction, manifested as hypoxia and bradycardia, in any of the patients studied.

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EASING ANXIETY

Mice, rats, cats, and monkeys all refuse to die. When Librium and Valium were introduced to the market, a number of suicidal patients consumed their bottle's entire contents—a hundred pills or more. They would sleep for two or three days but awaken with no apparent ill effects.

Since benzodiazepines are to some extent sedating, patients treated with Valium and Librium do not require any additional sleeping medication, even if they suffered previously from severe insomnia. All that is usually needed to transform a minimally sedating anxiety-relieving dose of the drug into a sleeping-inducing dose is to take two tablets instead of one. Many patients on an antianxiety regimen routinely do this at bedtime. Some benzodiazepines are marketed predominantly for use as sleeping pills, although their general pharmacological effects are not much different from those of Valium or Librium.

As already mentioned, benzodiazepines are not totally innocuous. Their addictive potential, though milder than that of barbiturates, is nevertheless disturbing. Additionally, perhaps because benzodiazepines are somewhat sedating, they potentiate the depressive effects of alcohol and barbiturates (more on this mechanism below). When a benzodiazepine is taken with a sedative substance, the result can be lethal. Many deaths have been attributed to such a combination. Judy Garland's demise in 1969 resulted from a synergistic alcohol-Valium interaction. What is particularly disconcerting is the finding that the amount of a benzodiazepine that will be lethal when combined with alcohol is quite unpredictable and varies tremendously from person to person. Sometimes ingestion of alcohol with as little as two or three times the recommended benzodiazepine dose can halt the patient's breathing.

Snyder Solomon H.; Drugs
And the Brain. New York:
Scientific American Library, 1986, p 161.

Growth aberrations

Treatment of ectopically erupting maxillary permanent first molars with a distal extended stainless steel crown

Michael W. Roberts, DDS, MScD

The exaggerated mesial eruption of the first permanent molar can result in

- ☐ The tooth becoming impacted under the distal contour of the primary second molar.
- ☐ The premature atypical resorption and exfoliation of the primary molar with resultant loss of space.

REVIEW OF THE LITERATURE

This aberrant molar eruption pattern affects 3 percent of the population. No sex differences or preference of sides have been observed, and pain has rarely been reported.[1] The etiology of the condition has been described as a combination of several factors, including abnormally large teeth, small maxilla, posterior positioning of the maxilla in the cranial base, and severe eruption angle of the maxillary first permanent molar.

The presence of an impacted maxillary first permanent molar can usually be detected during visual examination of the oral cavity, but is most often confirmed by dental radiographs. Numerous techniques have been described to treat this problem including

- ☐ Extract the second primary molar, followed by moving the maxillary first permanent molar distally with cervical traction.[2]
- ☐ Move the impacted tooth distally with a brass separating wire.[3]

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- ☐ Move the permanent molar posteriorly using an orthodontic band with a variety of attached springs.[4-7]
- ☐ Insert an elastic separator at the contact area to permit the ectopically erupting molar to be unlocked.[8]
- ☐ Place a band with an edgewise bracket on the second primary molar and a bonded bracket on the exposed cusp of the permanent tooth. Distal movement is achieved by activating an open coil spring over a multistrand twisted orthodontic wire.[9]

All of these procedures have been proved effective in selected cases. They require the first permanent molar to be at least partially erupted, however, or to be exposed surgically. The purpose of this paper is to describe a technique that can be successfully incorporated, when the maxillary first permanent molar is unerupted and indisputably impacted against the distal aspect of the second primary molar.

The procedure incorporates the use of a distal extension attached to a stainless steel crown. The primary second molar is prepared in the usual manner and a crown adapted. A piece of orthodontic band material (.005-inch) extending 1/4 inch apically is spot welded to the distal of the crown. The mesial of the permanent molar is identified by the use of a periodontal probe. Upon cementation, the distal extension is carefully guided to engage the mesial surface of the impacted permanent molar.

The pressure of the erupting permanent tooth can initiate ectopic resorption of the distal portion of the primary molar. This can occasionally result in a pathological pulp exposure and necessitate a pulpotomy before using an orthodontic technique to reposition the errant permanent tooth.

CASE REPORTS

Case A

The patient was a female, six years and eleven months of age, in no acute distress. The dental occlusion was class I and the arch length was apparently adequate. Bitewing radiographs revealed that both maxillary second primary molars had abnormal resorption on the distal surfaces, associated with the presence of mesially erupting permanent first molars. The left permanent tooth was not impacted and erupted into normal occlusion. The right permanent molar, however, had become engaged under the distal contour of the second primary molar.

The pulp of the right primary second molar had been exposed by ectopic resorption and a pulpotomy was required. A stainless steel crown was adapted, with a distal extension guide plane spot-welded to its distal surface and cemented with the guide plane engaging the mesial surface of the first permanent molar. The patient was observed periodically for eleven months, until the permanent tooth had erupted into normal occlusion. Ectopic resorption of the remaining root structure of the primary molar appears to have continued. A conventional band-loop space maintainer, however, can now be placed, should the tooth exfoliate prematurely (Figure 1).

Case B

This healthy female patient, seven years and one month of age, was noted to be small in stature for her age. Her primary molars were in a class I relationship, but there was an obvious gross bimaxillary arch-length insufficiency. The ectopic eruption of the right maxillary first permanent molar was detected upon radiographic examination.

Although there had been extensive damage to the distal of the second molar, a pulpotomy was not re-

Figure 1a. Ectopic resorption can be seen on the distal surfaces of both primary second molars. The right permanent molar, however, was the only one that became impacted.

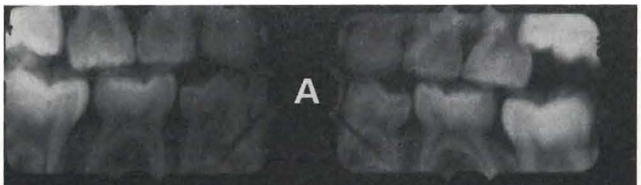
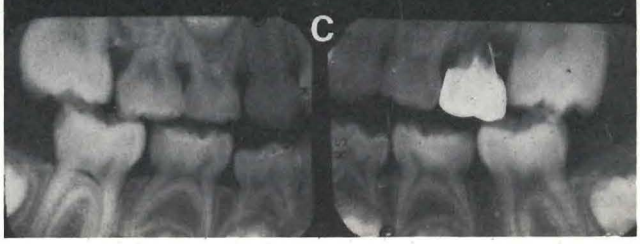


Figure 1b. A pulpotomy was performed and a stainless steel crown with a distal guide plane attached was placed on the primary second molar.

Figure 1c. Eleven months later. The first permanent molar has been unlocked and erupted into occlusion.



quired. The tooth was reduced and a stainless steel crown adapted. A distal extension was attached to the crown, as previously described, prior to cementation. The first permanent molar was guided into occlusion uneventfully, within eight months (Figure 2).

DISCUSSION

Techniques previously described to correct mesially erupting maxillary first permanent molars require that the offending tooth be at least partially erupted. This is necessary to allow engagement of various devices, springs, wires, and brackets. The technique of using a stainless steel crown with a welded distal extension guide plane allows treatment, when the permanent molar has become totally impacted. The crown provides excellent retention and rarely becomes dislodged or requires recementation.

It has been observed, however, that 66 percent of the ectopically erupting maxillary first permanent molars do not become impacted.[10] It is advisable, therefore, that treatment not begin without an adequate observation period. Preventive orthodontic intervention should be started only after the dentist is convinced that the permanent tooth is unable to erupt past the distal surface of the second primary molar and into normal occlusion.

Figure 2a. The maxillary first permanent molar is impacted at the distal aspect of the second primary molar.

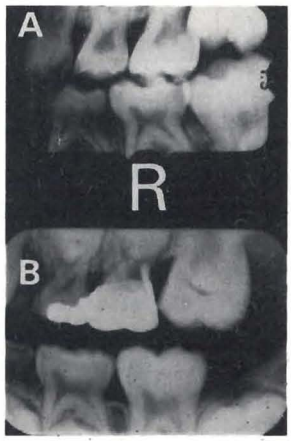


Figure 2b. The stainless steel crown with a distal extension has redirected the permanent molar into occlusion.

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QUALITY AND DECENCY OF LIFE

The quality and decency of life in the United States, I contended, were incomparably greater than in years past. And yet there was a segment of our population whose lives had somehow not been touched by the general success, save to the extent they had fallen even further behind. Increasingly, family structure defined this population. It could be that the seeming anomaly was nothing of the sort but rather a process. It was, I thought, entirely possible that many of the processes that were producing prosperity were also producing much of our poverty; it might be that both sets of problems were in fact part of a single phenomenon: the pathology of postindustrial society.

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Comparative analysis of ectopic eruption of maxillary permanent first molars in children with clefts

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Ectopic eruption is the process by which the maxillary permanent first molar moves mesially from the normal path of eruption, resulting in abnormal resorption of the distal aspect of the maxillary second primary molar. This aberrant path of eruption may result in the maxillary first permanent molar becoming locked into position (noncorrected) within the maxillary second primary molar and thus causing space loss, or it may correct spontaneously (self-corrected) with few or no sequelae.

While the etiology of ectopic eruption of the maxillary permanent first molar has not been determined definitely, several factors have been suggested as representative mechanisms. Chapman (1923) listed small arches, abnormal path of eruption, lack of forward movement of the primary teeth, and early eruption of the maxillary first permanent molar as contributing factors.[1] Additionally, Pulver (1968) concluded that the etiology was multifactorial and included: large mean sizes of all maxillary primary and permanent teeth, large first permanent molars and second primary molars, small maxilla, posterior position of the maxilla, abnormal angulation of eruption of the maxillary first permanent molar, delayed calcification of the first permanent molar.[2] Cheyne and Wessels (1947) suggested lack of bony growth in the tuberosity region at the correct time, while O'Meara (1962) stated that the major etiologic factor was insufficient intercanine and anteroposterior jaw growth.[3,4]

The prevalence of ectopic eruption in noncleft samples was reported to range from 2.0 percent to 4.3 percent, Cheyne and Wessels (1947), Young (1957), Kurol and Bjerklin (1982); and 22.9 percent to 29.1 percent, Carr and Mink (1965) in children with various cleft forms.[3,5-7]

The purpose of this study is to analyze the prevalence of ectopic eruption among the following specifically defined cleft types: cleft lip (left side, right side), cleft lip and palate (left side, right side), cleft lip and palate (bilateral), cleft of the hard and soft palate, cleft of the soft palate only, submucous cleft, velopharyngeal insufficiency.

METHODS

A retrospective review of the available dental records at the University of Pittsburgh Cleft Palate Center was conducted independently by two investigators. All charts with age-appropriate panoramic or bilateral bitewing radiographs were reviewed for the presence of ectopic eruption, either noncorrected or self-corrected. For each patient with radiographic evidence of ectopic

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eruption, the following additional data were collected: age, sex, race, cleft type, cleft side, unilateral or bilateral involvement. Patients with clefts associated with syndromes were excluded from the study.

The data were described by means of frequency distributions and analyzed by the Chi-Square Test.

RESULTS

A total of 933 charts of patients with clefts were available for initial review. Based on the previously stated criteria established for inclusion, the final sample consisted of 118 patients with a combined mean age of 9.3 years. The sex and racial distribution of the sample appears in Table 1.

Radiographs of thirty-one (26.3 percent) of the 118 patients with clefts showed evidence of ectopic eruption. The distribution for ectopic eruption by sex is presented in Table 2. Intrajudge and interjudge reliability were accepted above the 95 percent level.

When maxillary permanent first molars were considered as individual units, there were 236 teeth evaluated for the presence of ectopic eruption in this cleft sample. A total of forty-nine (20.8 percent) maxillary permanent first molars were diagnosed as ectopically erupted. Of these, eleven (22.4 percent) were noncorrected (Figure 1), while thirty-eight (77.6 percent) corrected spontaneously (Figure 2).

The distribution of ectopically erupted teeth by specific cleft type is presented in Table 3. The greatest number of ectopically erupted teeth was found in patients with left side cleft lip and palate (N = 11), while the smallest number of ectopically erupted teeth was diagnosed in left side cleft lip (N = 2), followed by patients with velopharyngeal insufficiency (N = 0).

Of all of the maxillary permanent first molars diagnosed as ectopically erupted, the following represents a frequency distribution by specifically defined cleft types: cleft lip and palate (left side), 22.5 percent; cleft lip and palate (bilateral), 18.4 percent; cleft of the hard and soft palate, 14.3 percent; clefts of the soft palate, 12.2 percent; submucous clefts, 12.2 percent; cleft lip and palate (right side), 10.2 percent; cleft lip (right side), 6.1 percent; cleft lip (left side), 4.1 percent; velopharyngeal insufficiency, 0 percent. When the distribution of ectopically erupted maxillary permanent first molars was analyzed, however, by means of Chi-Square Tests for cleft type, no statistically significant relationships were found.

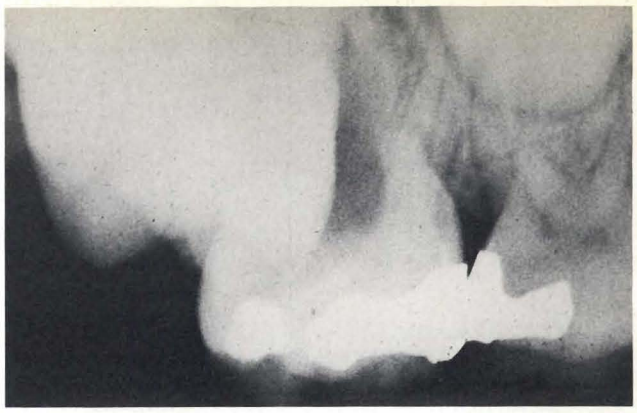

Figure 1. Noncorrected ectopic eruption of maxillary permanent first molar. Note that the permanent first molar is locked into the primary second molar.

DISCUSSION

Several authors (Chapman, 1923; Cheyne and Wessels, 1947; O'Meara, 1962; Pulver, 1968) suggested that a deficiency of maxillary arch length was a contributing factor in the etiology of ectopic eruption.[1-4] Ross (1969) indicated, furthermore, that surgery to correct palatal clefts inhibited the forward movement of the maxilla and contributed to a condition that was termed maxillary ankylosis.[8] This phenomenon could partially explain the higher frequency (26.3 percent) of ectopic eruption of maxillary permanent first molars found in this study of children with clefts, when compared to frequencies reported previously in the following noncleft samples: 2.0 percent (Cheyne and Wessels, 1947); 3.2 percent (Young, 1957); 4.3 percent (Kurol and Bjerklin, 1982).[3,5,6]

The 26.3 percent rate of ectopic eruption of the maxillary permanent first molars found in the current study of cleft children corresponds favorably with the results of Carr and Mink (1965), who reported a prevalence of 25 percent for their cleft sample.[7] The current findings of ectopic eruption by sex demonstrate, however, that the

Table 1 ☐ Sex and racial distribution of cleft sample.

	Males	Females	Total
White	64	44	108
Black	7	3	10
Total	71	47	118

Table 2 ☐ Distribution of ectopic eruption by sex of cleft sample.

	N Cleft patients	N Cleft patients with ectopic eruption	Percent Ectopic eruption cleft patients
Males	71	20	28.2
Females	47	11	23.4
Total	118	31	26.3

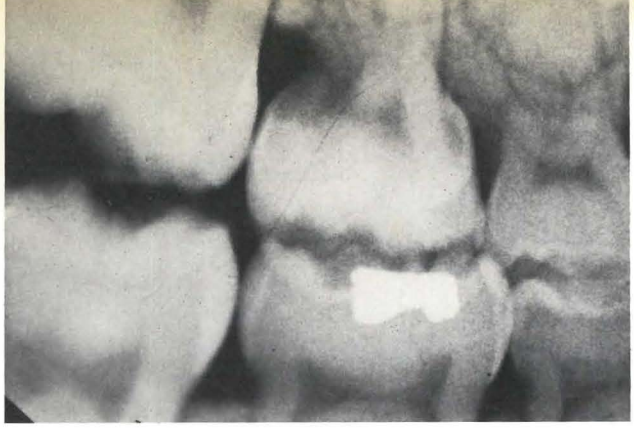

Figure 2. Self-corrected ectopically erupted maxillary permanent first molar. Note the abnormal resorption of the primary second molar, but that the permanent first molar is erupting into position.

Table 3 ☐ Distribution of ectopic eruption of maxillary permanent first molars by cleft type.

Cleft type	N Patients	N Teeth	Left		Right		Bilateral		No Ectopic eruption
			Self-corrected	Non-corrected	Self-corrected	Non-corrected	Self-corrected	Non-corrected	
Cleft lip—left side	9	18					2		16
Cleft lip—right side	5	10	1				2		7
Cleft lip & palate—left side	28	56	3		1	1	6		45
Cleft lip & palate—right side	16	32			1		2	2	27
Cleft lip & palate—bilateral	20	40	3			2	2	2	31
Cleft hard & soft palate	10	20			1		4	2	13
Cleft soft palate only	11	22	1		1		4		16
Submucous cleft	13	26	1		1		2	2	20
VPI	6	12							12
Totals	118	236	9	0	5	3	24	8	187

prevalence in males is higher (28.2 percent) than in females (23.4 percent). This is in reverse order of the findings of a previous study (Carr and Mink, 1965).[7]

Additionally, the current findings indicate that 77.6 percent of ectopic eruptions are self-corrective, while 22.4 percent are noncorrected. This is in contradistinction to the study of Carr and Mink (1965), who reported self- correction in 22.2 percent and noncorrected as 77.8 percent.[7] The current total sample of 118 cleft patients representing forty-nine individuals with ectopic eruption is higher, however, than the total sample of seventy-two cleft patients, representing eighteen individuals with ectopic eruption as reported by Carr and Mink (1965), which could account in part for these variations.[7] Consideration should also be given to the possibility that improved plastic surgical techniques contributed to the variations, during the last two decades.

The prevalence of ectopic eruption among specifically defined cleft types demonstrates that the presence of ectopic eruption is most frequently noted in left side cleft lip and palate (22.5 percent); and diagnosed less frequently in right side cleft lip, 6.1 percent; left side cleft lip, 4.1 percent; and velopharyngeal insufficiency, 0 percent. These results are consistent with the observation that left unilateral clefts of the lip and palate occur most frequently in this sample, as well as in the overall cleft population. Additionally, the fact that isolated clefts of the lip do not disrupt alveolar segments or dental units supports the contention that occlusal disharmonies should not be anticipated in these instances.

CONCLUSIONS

- ☐ The presence of ectopic eruption of the maxillary permanent first molars in children with clefts is higher than in noncleft children.
- ☐ The majority of ectopically erupting maxillary permanent first molars in children with clefts correct spontaneously.
- ☐ There are no statistically significant differences for ectopic eruption among the various cleft types, when analyzed by Chi- Square.

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Prenatal factors and tooth eruption in children with oral clefts

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Studies of healthy normal children reveal that there is some association, though ill-defined, between certain prenatal factors and dental development.[1] Crown and root development and the eruption of the permanent teeth may be delayed as much as six months, in children with oral clefts as compared to children without such aberrations. This is attributable to the same factors that caused the cleft itself.[2-4] In some children with clefts, however, there appears to be no delay in the eruption of the primary teeth except for the upper central incisor and canine on the cleft side.[5]

The purpose of this study was to ascertain the eruption ages of the primary central incisors in children with oral clefts, and to investigate the relationship between certain prenatal factors and the time of eruption of the first primary tooth in these children.

SUBJECTS AND METHODS

All children with cleft lip and palate, born in Finland, are treated in the Cleft Center of Helsinki University Central Hospital. Nearly all are registered in the Center soon after birth, and their parents are sent a booklet and other pieces of information about oral clefts and the treatment of their newborn. For one year, for the purposes of this study, a questionnaire was also mailed asking the parents to write down the eruption dates of the two first teeth in the maxilla and mandible, respectively, as well as on which side of the maxilla the first tooth erupted. A return envelope with prepaid postage was enclosed. The letter was mailed to the parents of eighty-seven children, born in the years 1982- 1983. Seventy-nine families responded, but for three children, the data were defective. Thus, there were seventy-six subjects in all, forty-three (57 percent) girls, and thirty-three (43 percent) boys.

Cleft types and other data relating to the children were assessed, using case histories. Thirty-five of them had unilateral cleft lip, with or without cleft palate (UCL(P)), six had bilateral cleft lip, with or without cleft palate (BCL(P)), and thirty-five had cleft palate (CP).

The mean eruption ages and the age-ranges of eruption of the maxillary primary central incisors on the cleft and noncleft side and of mandibular primary central incisors were calculated. The chronological eruption age and the postconceptual age of the first primary tooth were investigated in relation to four prenatal variables, using the quartiles of their distribution. Significance of the differences was calculated with one-way variance analysis. Moreover, the mean eruption ages of the first primary tooth within five other prenatal variables were compared. Significance of the differences was calculated with a t- test. Finally, the correlation coefficients between the mean eruption age of the first primary tooth and some of the variables were calculated, and partial correlations with maternal age were held constant.

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Table 1 ☐ The mean eruption ages (months) of primary central incisors in relation to sex and type of cleft. The maxillary values of BCL(P) and CP groups indicate the eruption age of the first central incisor in the maxilla.

		Maxillary				Mandibular			
		Side of jaw				Order of eruption			
		Cleft		Non-cleft		First		Second	
Girls	N	$\bar{x}$	range	$\bar{x}$	range	$\bar{x}$	range	$\bar{x}$	range
UCL(P)	16	8.9	6.3-14.0	8.8	6.9-11.8	7.1	5.0-10.9	7.7	5.2-12.1
BCL(P)	1	10.8				10.3		12.1	
CP	26			8.9	5.5-14.0	6.9	2.8-13.1	7.5	2.9-13.3
Boys									
UCL(P)	19	9.1	5.7-11.8	8.5	5.7-11.4	6.4	1.6-11.2	7.0	4.1-11.7
BCL(P)	5	7.7	6.2- 8.9			6.6	5.3- 8.5	6.9	5.8- 8.7
CP	9			10.4	7.4-13.4	8.2	4.8-11.8	8.5	4.9-12.7

UCL(P) = unilateral cleft lip with or without cleft palate
BCL(P) = bilateral cleft lip with or without cleft palate
CP = cleft palate

Table 2 ☐ The mean eruption ages (months) of the first primary tooth in quartiles of some variables. In parentheses the same ages given in postconceptual timing.

	Quartiles		
	25	25 - 75	75
Length of gestation N = 74	under 39 weeks 7.2 (16.1)	39 - 40 weeks 6.7 (15.9)	over 40 weeks 6.6 (16.1)
Birth weight N = 74	under 3090 grams 7.1 (15.8)	3090-3770 grams 6.9 (16.0)	over 3770 grams 7.0 (16.2)
Placental weight N = 67	under 500 grams 6.5 (15.6)	500-700 grams 6.7 (15.8)	over 700 grams 7.1 (16.4)
Maternal age N = 75	under 25 years 7.0 (16.2)	25 - 32 years 6.6 (15.7)	over 32 years 8.0 (16.8)

RESULTS

The mean eruption ages of primary central incisors in relation to sex and type of cleft are presented in Table 1. The differences between the cleft groups were slight. In the nine boys with isolated cleft palate, however, the first maxillary tooth and the two first mandibular teeth erupted more than one month later than in the girls with isolated cleft palate and in the girls or boys with unilateral cleft lip and palate. The first tooth in these seventy-six children erupted at the age of 1.6 months, and the last primary central incisor, at the age of fourteen months.

Length of gestation had no influence on the eruption age of the first primary tooth, when the postconceptual age was used. Birth weight explained none of the variations in the eruption ages, but placental weight and maternal age seemed to do so; children with higher placental weight and with an older mother were slightly delayed in this respect (Table 2). The differences within the maternal age variable were statistically significant (one-way variance analysis, $p<0.05$). Although there were no statistically significant differences within the variables "birth order", "place of birth", "additional anomalies", "anomalies in relatives" and in "mother's use of drugs during pregnancy", it can be noted that in children from the eastern provinces of Finland, a possible tendency to delayed tooth eruption was found (Table 3).

The calculated correlations between the eruption age of the first primary tooth and some prenatal factors were weak, and even weaker when the maternal age was held constant (Table 4).

DISCUSSION

The parents of cleft-affected infants are interested in their child's development, in the mouth area in particular. This was confirmed by the high response percentage (91 percent) of the questionnaire. Previously, there was no information available about the mean eruption ages of the first primary teeth in children with oral clefts.

Table 3 ☐ The mean eruption ages (months) of the first primary tooth in some variables.

Birth order	First child (N = 32) 6.7	Other children (N = 44) 7.1	
Additional anomalies	No (N = 57) 7.0	Yes (N = 19) 6.7	
Anomalies in relatives	No (N = 48) 7.0	Yes (N = 25) 6.9	
Mother's use of drugs during pregnancy	No (N = 50) 7.1	Yes (N = 26) 6.7	
Place of birth in Finland	South-west (N = 45) 6.8	East (N = 11) 7.8	North (N = 20) 7.0

Table 4 ☐ Correlations between the eruption age of the first primary tooth and five prenatal variables, and partial correlations between the eruption age and the variables, with maternal age held constant.

	Correlation coefficient	Partial correlation coefficient (maternal age held constant)
Birth weight	−0.10	−0.08
Placental weight	0.10	0.07
Length of gestation	−0.12	−0.15
Birth order	0.07	0.07
Maternal age	0.14	

In a study of dental casts of 473 Finnish children with oral clefts, the eruption ages of some primary teeth were found to be very close to those of healthy children.[5] This was also established in the present clinical study. The mean eruption ages of the studied teeth do not differ markedly from the results of healthy Finnish children or Icelandic children.[6-8]

Nearly all the children with cleft lip, with or without cleft palate, had undergone surgical treatment, once or twice before the first teeth erupted. Nevertheless, the possible effect on eruption is slight; only the maxillary cleft side central incisor is slightly delayed. Because other teeth on the cleft side not situated adjacent to the cleft have been found to develop asymmetrically to their contralaterals, the reason for asymmetrical eruption of maxillary central incisors is probably not a surgical one.[5,9] No marked differences were found between the cleft groups. The delay in both jaws of the boys with isolated cleft palate confirmed previous results in permanent tooth development; the delay in the cleft palate group is longer than what could be expected on the basis of the external severity of the cleft.[10]

Differences between the sexes in the eruption of the first primary teeth are small and the published results are somewhat contradictory.[6-8] In the present study, no directional difference was found between girls and boys, except the delay, in the nine boys in the cleft palate group.

In the category of eruption age, individual variations are substantial. It was interesting to clarify, therefore, whether these variations can be explained by the prenatal variables. Only the eruption age of the first primary tooth was established in this respect, because the data concerning it were considered to be the most reliable.

The effect of hereditary factors on the eruption time of primary teeth has been estimated at 78 percent.[11] In studies attempting to determine which prenatal factors are linked with tooth development and eruption, some tentative associations have been found. Low birth weight was shown to delay eruption in both dentitions.[1,7] Short gestation and young maternal age are also linked with developmental delays.[1,7]

In the present study with cleft-affected infants, birth weight did not influence the eruption time of the first primary tooth, but high placental weight appeared to have a slight influence toward delay. Older maternal age acted contrary to what could be expected, in that it appeared slightly linked with the delay. The eruption age of the first primary tooth was found to vary inversely with the gestational age, which supports previous results.[12]

Somewhat stronger correlations have been found between birth weight and the number of erupted permanent teeth, as well as between some other prenatal factors and tooth formation, than between these variables and the eruption time of the first primary tooth, in the present study. Thus, six to ten years later, prenatal factors seem to affect the permanent dentition more than the primary dentition, during the first year of life.

This study was supported by the Finnish Dental Association.

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Comparison of oral microflora between well-nourished and malnourished Nigerian children

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Most of the host defenses that protect oral tissues from infection are to some degree compromised by nutritional deficiencies. For example, nutritional deficiencies have been shown to increase permeability of the oral mucosal epithelium, while nutritional supplementation has been shown to cause a decrease in the gingival fluid flow.[1,2] As a result of nutritional deficiency of vitamin C and nicotinic acid, severe fusospirochetal infections of the oral cavity as well as of the vagina may occur. In other instances, certain tissues contain nutritional substances that favor the growth of certain types of microorganisms. *B. melaninogenicus* requires hemin and *T. microdentium* requires alpha-2-macroglobulin. Because the gingival crevicular fluid contains both of these nutrients, these organisms favor the gingival crevice over other areas of the oral cavity. With host defenses depressed as the result of nutritional deficiencies, the oral tissues are more susceptible to infection by these and other microorganisms. The incidence of acute necrotizing ulcerative gingivitis (ANUG) and cancrum oris (noma) in nutritionally deprived children from Nigeria is a graphic example of this relationship of nutrition, oral health and disease.[3,4] The cancrum oris is

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apparently being caused by an uncontrolled proliferation of normal oral microorganisms in patients whose resistance has been seriously compromised.

Because of this intimate interrelationship of nutrition, oral flora and oral disease such as ANUG, noma, and periodontal disease, it was decided to study the oral microflora in a group of well-nourished and a group of malnourished children in Nigeria. After some of the children in the malnourished group had received a high-protein and balanced diet for six months, an attempt was made to reexamine the children to observe any changes that had occurred in their oral microflora and oral health. The following report gives the results of our observations.

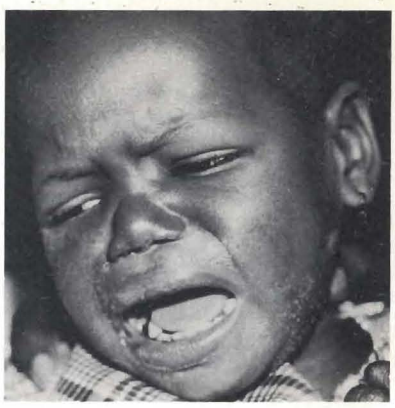

Figure 1. This four-year-old female shows hair color change, hair loss and a scaly dermatitis. The tissues of this malnourished child were easily traumatized as noted by bleeding around the right mandibular molars, following examination.

MATERIALS AND METHODS

Subjects

Twenty-two children, ranging in age from one to five years, were used in this study. Children were ethnically from the Yoruba tribe and lived in the Oyo State of Nigeria. Grouping of subjects into malnourished vs well-nourished children was made on the basis of clinical findings and laboratory data. Subjects were clinically evaluated by the medical staff at the Baptist Hospital and Kearsey Nutritional Center at Ogbomosho. Factors taken into consideration included age, weight, edema, hair changes, and extended abdomen (Figures 1,2). In addition to strong clinical evidence of malnutrition in this subject group, serum protein levels were taken for confirmation. As can be seen in Table 1, statistically significant differences were found between well-nourished and malnourished subjects when considering total protein ($P < 0.05$), albumin ($P < 0.001$), and albumin/globulin ratio ($P < 0.005$). All well-nourished and fifteen of the seventeen malnourished group had blood drawn for this purpose.

Once the experimental groups had been determined, oral examinations were made and samples were taken for microbiologic analysis as described below. In addition, three subjects (two malnourished and one well-nourished), were observed for a period of six months during which time they were placed on a high-protein and balanced diet. At the end of the six months, these subjects were reexamined and again cultured for oral microbial flora.

Specimen collection

Samples for microbiologic analysis were taken with sterile alumina-coated cotton wool swabs (Exogen Ltd., Dumbarton Road, Glasgow). Swabs were passed carefully over the gingiva at the dentinogingival margin and broken into Amies Transport Medium to discard the contaminated stem. A transport medium was necessary due to the distance some samples needed to be taken.

Culturing techniques

Each specimen was plated by the technique of Gilles and Dodds on the following media: two plates Blood agar (Oxoid Columbia Agar Base, CM 331, with 7 percent horse blood, Wellcome); Chocolate Agar; MacConkey Agar (Oxoid); Sabourauds Agar (Oxoid, code CM41a); Neomycin Blood Agar (Blood agar to which 75 μg/ml neomycin was added; BM-Kanamycin Agar (Holbrook, Ogston and Ross, 1978); and Mitis-Salivarius Agar (Oxoid).[5] After inoculation, one Blood Agar, Mitis-Salivarius Agar, and Sabourauds Agar were incubated aerobically at 37°C. Chocolate agar was incubated in air plus 10 percent CO_2 at 37°C. The other Blood Agar, BM-K Agar,

Table 1 □ Serum proteins in well-nourished children and malnourished children.

Group	Age	Number examined	Total protein (g/100 ml) Mean	S.D.	Albumin (g/100 ml) Mean	S.D.	Globulin (g/100 ml) Mean	S.D.	A/G ratio Ratio	S.D.
Well-nourished	2-5*	5	7.45	0.25	4.80	0.38	2.65	0.32	1.86	0.33
Malnourished	1-5	15	6.76	0.98	3.27	0.73	3.27	0.73	1.01	0.43

*Age in years

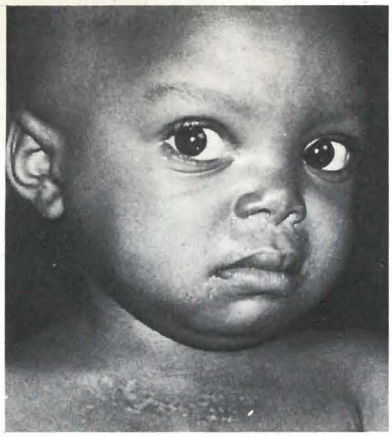

Figure 2. This Nigerian child, although on the road to recovery, still manifests hair loss and color changes, as well as a healing dermatitis.

and Neomycin Blood agar were incubated anaerobically, using a gas generating system (Oxoid). Aerobically incubated plates were inspected at 24 h and 48 h, and anaerobically incubated plates at 48 h.

Identification of microorganisms

Before identification, representative colonies of each type were subcultured on fresh agar plates to insure purity. Aerobic microorganisms were identified using the techniques of Cowan and Steel.[6] Anaerobes were identified, using the comprehensive identification scheme of Duerden *et al* and Rotimi *et al.*[7,8] Presence of spirochetes was determined by microscopic observation of smears made from sampled material.

RESULTS

Microbiologic analysis of malnourished and well-nourished subjects

As seen in Table 2, percentage of isolates from malnourished patients containing facultative and aerobic microflora was not different from that found in well-nourished subjects. The number of isolates containing anaerobic microflora, however, was significantly higher in the malnourished patients. This pattern held true for all groups of anaerobes, including the spirochetes. In fact, anaerobes were rarely isolated from well-nourished subjects, when sampled by our methods, whereas malnourished subjects were culture-positive for anaerobic microflora most of the time.

A detailed analysis of isolated microorganisms and frequency of isolation may be seen in Tables 3 through 5. As might be expected from the sampling procedures, all patients, regardless of nutritional status, had facultative gram-positive cocci isolated from their oral cavities (Table 3). In contrast, *Peptostreptococcus* or *Peptococcus* could not be isolated from well-nourished subjects, but was isolated occasionally from the malnourished individuals. Table 4 shows the gram-positive rods isolated. Our sampling procedure did not result in the recovery of any facultative or aerobic gram-positive rods. *Actinomyces israelii* was isolated 76 percent of the time, however, in poorly nourished subjects, while never from those deemed well-nourished. As seen in Table 5, facultative gram-negative rods were isolated with approximately the same frequency in all patients. As seen before, great differences existed in isolation rates in the group of anaerobic gram-negative rods. Specifically, the *Bacteroides* were always isolated from malnourished pa-

Table 2 ☐ General distribution of microorganisms in malnourished and well-nourished children.

Group	Percent positive samples	
	malnourished	well-nourished
Facultative and aerobic flora		
gram-negative rods	77	80
gram-positive cocci	100	100
Nonsporing anaerobes		
gram-negative rods	100	20
gram-positive rods	77	0
gram-positive cocci	47	0
Spirochetes	88	0

Table 3 ☐ Relative proportion of specimens from which gram positive bacteria were isolated.

Species or group	Percent positive isolates	
	malnourished	well-nourished
Aerobic cocci		
S. mutans	77	60
S. mitis	88	60
S. salivarius	65	60
S. sanguis	24	20
S. faecalis	59	80
Staph. aureus	18	20
Staph. epidermidis	6	40
Anaerobic cocci		
Peptostreptococcus	41	0
Peptococcus	6	0
Aerobic rods		
none isolated		
Anaerobic rods		
Actinomyces israelii	76	0

Table 4 ☐ Relative proportion of specimens from which gram-negative rods and spirochetes were isolated.

Species or group	Percent positive isolates	
	malnourished	well-nourished
Aerobic		
Pseudomonas aeruginosa	41	40
E. coli	59	80
Enterobacter cloacae	6	0
Klebsiella pneumoniae	35	40
Anaerobic		
Bacteroides melaninogenicus	100	0
Bacteroides asaccharolyticus	76	0
Bacteroides oralis	88	0
Bacteroides ruminicola	18	0
Bacteroides fragilis	12	0
Fusobacterium sp.	71	20
Spirochetes	88	0

tients, but never from those well-nourished. Likewise, spirochetes were never found in the well-nourished group (Table 5).

Comparison of oral microflora in subjects before and following a regimen of high protein and balanced diet

After being instructed on basic oral hygiene practices and placed on a high-protein, well-balanced diet for six months, three patients were reexamined. General health, oral health, and results of another microbiologic analysis were recorded. Both general and oral health improved over that period. Both of the malnourished subjects, who entered the study with severe to moderate gingivitis, had improved to a state of mild gingivitis after six months. In Table 5 the typal shifts that occurred in microbial flora before and after improved diet are described. As seen previously on first culture, the malnourished patients possessed not only facultative flora, but also spirochetes, anaerobic gram- negative rods and cocci. In contrast, we did not culture any anaerobic flora of this type from our well-nourished controls. After the six-month interval, the healthy control did not change, relative to his lack of anaerobic microflora. In contrast, the two originally malnourished subjects shifted from a predominantly anaerobic microflora to one from which anaerobes were not isolated. One exception was that in subject 1, *Bacteroides oralis* could still be isolated, although all other *Bacteroides* species disappeared.

Table 5 ☐ General distribution of microorganisms in well-nourished and malnourished Nigerian children before and following a high-protein and balanced diet.

Group	Percent positive samples					
	malnourished				well-nourished	
	Subj. 1 / Subj. 2					
	B	A	B	A	B	A
Facultative and aerobic flora						
gram-negative rods	+	+	+	+	−	+
gram-positive cocci	+	+	+	+	+	+
Nonsporing anaerobes						
gram-negative rods	+	+	+	−	−	−
gram-positive rods	+	−	+	−	−	−
gram-positive cocci	+	−	+	−	−	−
Spirochetes	+	−	+	−	−	−

*Before initiation of high-protein and balanced diet
**After six months of high-protein and balanced diet

DISCUSSION

Oral disease is no stranger to Nigeria. In fact, Nigeria has been shown to have one of the highest prevalences of severe periodontal lesions in the world.[9] Individuals with such serious disease are usually malnourished and subject to several endemic diseases.[10] We set out to determine the specific oral microflora of a malnourished group of children and compare these findings with a similar group who were deemed well-nourished. When cultured by techniques described above, it was found that malnourished subjects had a high prevalence of anaerobic microflora, when compared to well-nourished controls. This is not surprising since it was shown that there is a serious compromise in host defenses in the malnourished individual.[11,12] In fact, it is generally believed that this compromise can modulate progression of, or later susceptibility to, periodontal disease.[13,14] Protein- calorie malnutrition has been correlated with several destructive processes in the periodontium, such as degeneration of periodontal collagen fibers, thinning of the periodontal ligament, and delayed gingival wound healing, all of which would promote infection by the sulcular bacteria.[15-18]

The anaerobic microorganisms most frequently isolated from the malnourished subjects were the *Bacteroides* group (species *melaninogenicus*, *asaccharolyticus*, *oralis*, *ruminicola*, and *fragilis*), *Actinomyces israelii*, and *Fusobacterium* sp., and the spirochetes (observed in smears). Although some of these microorganisms may often be isolated as normal flora, they are only found deep in the sulcus, where anaerobic conditions exist.[19] For them to be cultured from samples taken in the manner described above is indicative of serious disease processes taking place in the oral cavity.

The most frequently isolated anaerobic genus from our study was *Bacteroides*. *B. melaninogenicus*, *B. asaccharolyticus*, and *B. oralis* were all isolated from more than 75 percent of the malnourished subjects, but never from the well-nourished group. The genus *Bacteroides* is well known to be associated with various types of periodontal disease and the black-pigmenting *Bacteroides* are considered to be some of the major pathogens involved.[20,22] In fact, there is even some suggestion that different groups of black-pigmenting *Bacteroides* are associated with different isolation sites and different forms of the disease.[20] White and Mayrand demonstrated a direct correlation between presence of high numbers of *B. asaccharolyticus* and severe gingival inflammation, and Spiegel *et al* have shown this organism to be the predominant species in advanced periodontitis.[23,24]

It has been shown that in gingivitis, *Actinomyces* was among the predominant, isolated subgingival and su-

pragingival microflora.[25,26] In the periodontal pocket, however, numbers of *Actinomyces* species was very low.[27] This is in keeping with our findings (Table 3) in that *Actinomyces* was not isolated from our well-nourished subjects, but was found in 76 percent of the malnourished individuals.

Another striking correlation is the finding of spirochetes in 88 percent of the malnourished group, but never in the well-nourished individuals. This is in agreement with many investigators who have found increased proportions of spirochetes in experimentally induced gingivitis.[28] Armitage *et al* reported that through microscopic examination of plaque samples, 0.39 percent of flora was spirochetes in the healthy state, 7.08 percent of the flora was spirochetes in patients with moderate gingivitis, and 11.52 percent of the flora was spirochetes in those with severe periodontitis.[29]

Certainly it is clear from the data above that the malnourished subjects possessed a microflora strongly indicative of deteriorating oral health or existing oral disease. The importance of oral health, however, is not only related to existence of oral disease, but also to the oral cavity as a site for seeding potential pathogens elsewhere. If these microorganisms should enter the system of an individual possessing impaired defense mechanisms, serious infection or death may result.

From the information described above, it is seen that bacteria isolated from the malnourished subjects were strongly indicative of disease processes and could be explained by the malnourished state of these individuals. Likewise, from the few follow-up patients available, it appears as though an improvement in nutrition, along with presumably better oral care, caused a shift in microbial flora to one indicative of oral health. Information provided by this investigation adds further evidence as to the importance of proper nutrition in maintaining normal oral microflora, and oral as well as general health.

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Physiological role of dietary fiber: a ten-year review

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GROWTH OF INTEREST IN DIETARY FIBER

The past ten years have shown much growth of interest in dietary fiber in both nutritional and medical communities. This article reviews the fundamental metabolic aspects of fiber ingestion and the relationship of fiber to many diet-related diseases. Reviews of these complex topics are covered in other publications.[1-4]

In 1980, the British Royal College of Physicians recommended an increased consumption of fiber.[5] A few years later, the (British) Health Education Council advised that dietary fiber intake should increase 50 percent.[6] By 1985, the National Cancer Institute (NCI) in the U.S. stated that an optimum intake of fiber is about 25-35 grams per day.[7] The NCI recently modified their recommendations to state that Americans should try to double the amount of fiber they eat.[8]

Recently, the British Medical Association reported low fiber intakes were associated with constipation, diverticular disease, cancer of the large bowel and coronary heart disease.[9] It noted that fiber intakes had been reduced the past century by about one-third, that bread consumption had fallen, but that more whole grain bread and fiber-rich breakfast cereals were being eaten.

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DEFINITIONS AND CONSTITUENTS

Until the mid 1970s, crude fiber (cellulose and lignin) was the form of fiber measured in foods. In 1976, Trowell defined dietary fiber as the "remnants of plant cells resistant to hydrolysis by the alimentary enzymes of man. It is composed of celluloses, hemicelluloses, oligosaccharides, pectins, gums, waxes and lignin."[10] This definition of dietary fiber by Trowell, *et al*, has been used both in British Food Tables and in the U.S. by the Association of Official Analytical Chemists (AOAC) analysts.[11-12]

The main components of dietary fiber are complex polysaccharides present in plant cell walls. Non-starch undigested storage polysaccharides are also included. Large amounts are present in leguminous seeds of beans and peas; these are recommended in new high fiber diets for diabetes. Dietary fiber is a very complex group of substances which influence the digestion and absorption of food in the upper portion of the alimentary track and the formation of feces in the lower portion, i.e., in the colon and rectum. Cereal fiber differs from vegetable and fruit fiber in composition and action in the body.

INTAKES

In countries more economically developed, adult intakes of dietary fiber are estimated to average about 20g/day. In a typical Western diet, the main sources of fiber are vegetables (45 percent), cereals and legumes (35

percent) and fruits (20 percent).[13] Consumption of bread and, therefore, cereal fiber has decreased considerably during the present century. In Britain, consumption of wheat flour fell from 280 lbs/person/year in 1880 to 156 lbs/person/year in 1964.[14] In the U.S., cereal fiber intakes have decreased 55 percent from 1909 to 1975.[13] On the other hand, African peasants consume large amounts of home-grown lightly processed plant foods, such as corn, millet, potatoes and plantains, so that adults often consume dietary fiber at 130g/day.[15]

ANALYSIS

A number of methods of analysis of dietary fiber have been proposed and have been recently reviewed.[16] AOAC collaborative trials have been used to verify the total dietary fiber method as the preferred method of choice in the U.S. Values for total dietary fiber in certain cereal products are whole wheat flour (12.8 percent), white flour (3.3 percent), wheat bran (42.4 percent), corn bran (88.8 percent) and white rice (4.1 percent).[17] British food tables have reported the dietary fiber content of British common foods; examples are whole wheat bread (8.5 percent), brown bread (5.1 percent), white bread (2.7 percent) and white rice (2.4 percent).[11]

DIETARY FIBER AND THE LARGE BOWEL

Since fiber is not digested by the alimentary enzymes present in the small intestine, it passes into the large bowel, profoundly affecting the large bowel functions. Fiber increases fecal bulk and stool frequency, shortens transit time and alters the microflora.

Constipation is difficult to define precisely and, therefore, impossible to determine its incidence exactly. The best definition is that constipation is present in any patient who strains to defecate and does not pass easily at least one soft stool daily.[18] Feeding experiments report that for every extra gram of cereal fiber, stool weight increases by 3-9 g, compared to only 1.9 g when a comparable amount of fiber is derived from fruit and vegetables.[19] Cereal fiber is only slightly degraded by colonic microflora and provides energy for bacterial growth; this increases fecal bulk.[18] Constipation is often due to low intake of cereal dietary fiber. It is, therefore, very common in Western communities where bread consumption has fallen substantially in the last 100 years and has shifted from whole grain to low fiber white bread.

Diverticular disease is due to a low intake of fiber, especially cereal fiber. It was a rare disease in Western communities in the last century, but incidence has risen rapidly in the present century until now it is present in one in three people over 60 years.[20] Diverticular disease is a rare disease in African blacks but is now appearing in urban Africans who have adopted Western eating habits. Low fiber diets produce firm fecal material within the large bowel, which has difficulty in propelling this hardened material along its lumen. Many small pouches (diverticula) form in the wall of the bowel; eventually some of these may become inflamed or may burst. Treatment now relies largely on a high fiber diet supplemented by pharmaceutical preparations of fiber. This relieves the abdominal pain and reduces the risk of the serious complications, such as perforation.[21]

Hemorrhoids are caused by severe constipation and straining to pass hard small fecal masses. The anus is normally closed by highly vascular cushions. Straining to defecate causes venous engorgement of these vascular cushions and passage of firm fecal masses exerts a shearing stress on these engorged cushions. This can cause bleeding and may rupture the attachment of the cushions to the surrounding sphincter muscle leading to prolapse. It is estimated that one in two Americans over age 50 has suffered at one time from hemorrhoids, but the disease is quite uncommon in African blacks.[22]

CANCER OF THE LARGE BOWEL

Burkitt described in detail the epidemiology of large bowel cancer and its association with a low dietary fiber intake; he suggested that fiber regulated the speed of transit, bulk and consistency of the stool to dilute carcinogens and alter microbial metabolism.[23] The International Association for Research in Cancer (IARC) has coordinated two detailed studies to test the fiber hypothesis in Finland and Denmark.[19] Bowel cancer incidence was four times higher in Copenhagen (Denmark) than in Kuopio (Finland). It was concluded that higher intakes of dietary fiber in Finland have offered protection against large bowel cancer. Cummings concluded his review stating, "It is unrealistic to believe that dietary fiber is the only important dominant determinant of this disease...many dietary, genetic and environmental factors contribute to the eventual experience of a community of this cancer, then in the context of Europe, America and possibly other industrialized countries, a case can be made that dietary fiber is protective.[24] Nevertheless, we are a long way from being able to determine which sort of fiber and how much."

DIABETES MELLITUS

Clinical use of high fiber, high carbohydrate diets in the treatment of diabetes is now widely recognized. It has occurred only recently and is a reversal of the previous teaching which recommended low carbohydrate, high fat diets. The change was initiated by Anderson and Ward in the U.S.[25] At first, new high fiber diets for diabetes relied on cereal fiber but soon the potential value of gel-forming fibers, present in leguminous seeds, were found to lower both basal and post-prandial blood glucose levels.

Mann has also revealed recent developments in the investigation of nutritional etiological factors in type II diabetes, non-insulin-dependent diabetes mellitus (NIDDM).[26] He has stated that "there is more evidence for, and less against, the hypothesis that dietary fiber and the starchy foods are important nutritional factors in the etiology and treatment of type II diabetes than for the sugar hypothesis."[27]

Mechanisms of action of high fiber diets in treatment of both type I, insulin-dependent diabetes mellitus (IDDM), and type II (NIDDM) remain obscure. The result appears to depend by no means exclusively on the nature and amount of fiber, but also on the physical structure of the food, especially its carbohydrate. Thus removal and disruption of the fiber can alter the blood glucose levels. For instance, when apples have been given either whole, pureed or as juice, it was found that increased removal or disruption of the fiber resulted in higher blood glucose levels.[28] The physical structure of food, such as grinding or cooking, also affects the blood glucose levels.

The efficacy and practicality of the high fiber diets for diabetics, employing ordinary foods, now appears confirmed. There is agreement that the blood sugar levels are more easily controlled. It is justifiable to hope that this will lead to a reduction in the occurrence of long-term complications.

Jenkins developed the concept of the glycemic index of foods in order to elucidate the question of which foods should be recommended in the treatment of diabetes.[29] Foods have been graded according to their glycemic index, i.e., the area under the blood glucose curve after eating 50g of a specified carbohydrate. It is expressed as the percentage of the response to a reference carbohydrate of white bread or glucose. Leguminous beans have the lowest glycemic response, only 20 percent to 40 percent of that in white bread. Some index figures appear confusing: thus whole wheat bread and white bread have a similar glycemic index. In view of all these uncertainties, the choice of the most appropriate foods for diabetics should rely on prolonged feeding studies rather than on short-term response to individual foods and on the glycemic index.

When the foods for diabetics were reviewed, the question of etiological factors that produce diabetes has been reopened after many years of little discussion. Inheritance is certainly an important factor in type II (NIDDM), but less so in type I (IDDM). Obesity also is an important etiological factor; this has been known since the pioneer studies by Joslin in the U.S. A high intake of fat (common in diabetic diets) is believed to be the major factor in producing overweight in susceptible phenotypes.[30] Excessive energy intakes can be a factor, but probably only if they produce obesity.

CORONARY HEART DISEASE

Coronary heart disease (CHD) is a multifactorial disease. Major risk factors are elevated blood pressure, cigarette smoking and high serum cholesterol levels. Trowell put forward the hypothesis that a "high consumption of starchy carbohydrates, with fiber, is protective against hyperlipidemia and CHD.[31] Vegetarians and Seventh Day Adventists generally consume high fiber diets but have low incidence of CHD. Morris *et al*, observed British civil servants and found that those who had more exercise and ate more food developed less coronary heart disease; they also ate more cereal dietary fiber.[32] Kromhou *et al*, observed a large group of men for 10 years and reported that those who ate a high fiber diet developed less atherosclerosis.[33]

After many experiments in man and animals, a picture begins to emerge of the effect of dietary fiber and fiber components on plasma lipids.[34] Some types of dietary fiber, such as oat bran and corn bran, lower cholesterol levels, but wheat bran does not. Some fruits, vegetables, legumes and viscous polysaccharides, such as pectin and guar, all lower plasma lipid levels.[34]

The U.S. Lipid Research Clinic's coronary prevention trial showed that a modest reduction in blood cholesterol levels was associated with a significant reduction (19 percent) of coronary heart disease deaths after 7years.[34] Coronary death rates in the U.S., although falling, are still unacceptably high. The mean plasma cholesterol level in middle-age U.S. adults averaged 223 mg/dl in 1971-1974.[35] The U.S. Health and Human Services goals for 1990 are to decrease the proportion of U.S. adults with mean serum cholesterols above 230 mg/dl by at least 50 percent.[35] The hypocholesterolemic effect of dietary fiber in fiber-rich foods may prove to be helpful in achieving this objective.[34]

SUMMARY

It is accepted nowadays that dietary fiber is an important constituent of the diet. There is growing evidence that the low fiber Western diets and the low consumption of whole grain products are important factors in several common diseases of the large bowel.

Cereal fiber differs from that present in vegetables and fruit. A low intake of cereal fiber has been implicated in cancer of the large bowel, diverticular disease of the colon and coronary heart disease. High fiber diets are often prescribed for diabetes. Although fiber consumption by British and American consumers has decreased over the past century, consumption of whole wheat breads and fiber-rich breakfast cereals has received new attention during the past ten years.

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Clinic

Undetected errors in cavity preparations from a preclinical course and their possible clinical implications

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Although cavity preparations should be carefully evaluated, in order to assure successful restorations, evaluation techniques have a limited inherent reliability.[1,2] In preclinical courses, in order to increase the efficiency of the evaluation process, the use of a systematic approach, based on detailed evaluation forms that include lists and drawings of the different criteria requested, has been recommended.[3,4] Further improvement in the effectiveness of the evaluation process is made by comparing the student's evaluation of his own cavity preparations with the instructor's evaluation.[5]

Even though a meticulous evaluation is performed, mistakes in cavity preparation may remain undetected in preclinical courses that simulate clinical conditions; it may be assumed, furthermore, that the same mistakes may remain undetected under clinical conditions. The purposes of this study were to disclose undetected errors in acceptable class II cavity preparations, from a preclinical course in pediatric dentistry; to analyze their plausible etiological factors; and to discuss their probable clinical implications.

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MATERIALS AND METHODS

The Preclinical Pediatric Dentistry Course in the School of Dentistry, University of the Pacific, includes the use of a manual with detailed explanations and drawings of the criteria for evaluation of the various kinds of cavity preparations. Video tapes and/or sound-slide modules, followed by a discussion of the procedures, precede each laboratory session. The criteria for the cavity preparations are based on the fundamentals established by Black, as well as on modern cavity design for primary teeth.[6,7]

The instructors were board-eligible or certified pedodontists, with the exception of two University of the Pacific graduates, involved in the practice of pediatric dentistry. Before beginning the course, manuals were distributed and an orientation meeting with the instructors was held.

Pediatric typodonts* mounted on a post were used. Students were requested to work in a 10 to 12 o'clock sitting position, to simulate clinical conditions. Individual laboratory lights were available and all maxillary procedures were done by indirect vision, using a dental mirror. Carbide burs and hand instruments were used for the cavity preparations.

Evaluation forms with detailed descriptions of the criteria and drawings were used. The evaluation process involved two evaluations, the first one by the student and the second by the instructor; the two were compared, and in cases of disagreement, the cavity was reexamined and a consensus reached. All evaluations were made from the same position and from the same level of visibility as that used during the cavity preparations. Periodontal probes, scored in one millimeter increments, were used for depth measurements.

Preparation criteria were judged to be *acceptable*, *improvable*, or *not acceptable*. In cases in which the cavity preparation had one or more *not acceptable* criteria, they were discarded and a new preparation was requested. When preparation criteria were graded either *acceptable* or *improvable*, the student attempted to modify those that were considered improvable to an acceptable level and, an additional complete evaluation was done in order to determine whether the required improvements were achieved, without creating new unacceptable areas. Only cavities in which all the criteria were considered to be *acceptable* were credited.

Among the projects of the course, two class II cavity preparations were included, one to be filled with dental amalgam. After the course was finished, the typodonts were collected and 127 accepted, unfilled, mesioclusal cavity preparations, on the maxillary left second primary molars, were available for a postcourse evaluation.

Table 1 ☐ Prevalence of statistically significant undetected errors in 127 mesioclusal cavities in second primary molars, $p<0.01$.

Error	N	Percent
Deep occlusal floor, (>1.5 mm.).	7	5.5
Lingual wall of proximal box placed in an axial direction or diverging occlusally.	7	5.5
Gingival floor in contact with adjacent tooth.	7	5.5
Rough cavity walls.	10	7.9
Overextended lingual wall of proximal box.	10	7.9
Flat or concave axial wall in the proximal box.	13	10.2
Underextended occlusal outline.	15	11.8
Undermined enamel on the buccal surface of the box.	15	11.8
Sharp pulpoaxial line angle.	16	12.6
Deep axial wall in proximal box (>1.25 mm).	20	15.7
Shallow occlusal cavity (<1.25 mm).	21	16.5
Ocelusal outline, not fluid.	21	16.5
Occlusal outline, not centered.	25	19.7
Occlusal isthmus, too narrow (<1.25 mm.).	28	22.0
Undermined enamel on the lingual surface of the box.	37	29.1

Utilizing the same evaluation form that was used in the course, the course director (E.B.), holding the typodonts in the hand, examined each preparation by direct vision. By this method, the cavity preparations were more visible than on the typodont stand, facilitating the detection of incorrectly evaluated criteria. Each criterion was first evaluated as *met* or *not met* and those judged *not met* were classified either *underprepared* or *overprepared*.

Statistical analysis for the significance of the difference between proportions was made, to test for the

- ☐ Prevalence of each undetected error in 127 cavity preparations.
- ☐ Difference between underprepared and overprepared scores for the same criterion.
- ☐ Difference of the same error in the buccal and lingual surfaces.[8]

RESULTS

Nineteen preparations were found to meet all the required criteria. Of the remaining preparations, the statistical analysis of the prevalence of undetected errors proved to be significant for fifteen of twenty-eight criteria employed ($p<0.01$) (Table 1).

The difference between proportions of underpreparation and overpreparation of the same criterion was significant for the values for deep and shallow occlusal cavities (5.5 percent and 16.5 percent, respectively,

* Columbia Dentoforms

$p<0.01$), deep and shallow axial wall in the proximal box (15.7 percent and 0.8 percent, respectively, $p<0.01$), and between underextended and overextended lingual proximal box wall (0.8 percent and 7.9 percent, respectively, $p<0.01$).

The difference between the proportions of the same error in the buccal or lingual surfaces was found to be statistically significant for the overextension of the proximal box walls (2.4 percent for the buccal and 7.9 percent for the lingual, $p<0.01$) and for the presence of unsupported enamel on the proximal box walls (11.8 percent for the buccal and 29.1 percent for the lingual, $p<0.01$).

DISCUSSION

The simulation of clinical conditions in preclinical courses is of great importance, since it is expected to prepare the student to perform with adequate working habits. Indirect vision through a dental mirror causes limitations in the approach to, and visibility of, cavity preparations. This may affect the student's performance as well as the evaluations made by the student and the instructor.

During the postcourse evaluation, the simulation of clinical conditions was not relevant. Examination of the cavities by direct vision, facilitated the finding of errors which were not previously disclosed, either by the student or the instructor.

A review of the factors published by Mackenzie *et al*, on the *Analysis of disagreement in the evaluation of clinical products*, was conducted by the three authors, who found that the following factors had a significant influence on the students' performances and the students' and instructors' evaluations:

Visual acuity

Working in a 10 to 12 o'clock position with indirect vision on a maxillary molar, limits visibility and approach. This factor had a greater effect on the lingual than on the buccal walls of the preparations examined in this study. These differences proved to be statistically significant for the overextension and presence of undermined enamel on the buccal and lingual walls of the proximal box (Table 1). It is assumed that for a left-handed operator working in a 12 to 2 o'clock position, the errors would be mirror images to those described previously.

Table 2 □ Undetected errors in order of frequency and the nature of their contribution to possible clinical failure.

Undetected error	Clinical implications
Occlusal depth more than 1.5 mm	Pulp exposure
Lingual wall of occlusal cavity not retentive	Lack in retention
Gingival floor not retentive	Incomplete caries removal
Rough cavity walls	Amalgam fracture
Over-extended lingual wall in proximal box	Amalgam failure
Axial wall not parallel to the dentinoenamel junction	Amalgam failure, pulp exposure
Pits or fissures not included	Incomplete caries removal
Undermined enamel in buccal surface of proximal box	Tooth fracture
Acute axioclusal angle	Amalgam fracture
Deep axial wall	Pulp exposure
Shallow occlusal preparation	Amalgam fracture, incomplete caries removal
Occlusal outline not fluid	Amalgam fracture
Occlusal preparation not centered	Tooth fracture, pulp exposure
Narrow isthmus	Amalgam fracture
Undermined enamel on lingual wall of proximal box	Tooth fracture

Estimation of size

The acceptability of the criteria for cavity preparations in primary teeth is graded in fractions of millimeters. Periodontal probes graded in millimeter increments did not allow for adequate accuracy.

Unspecified methods of observing

Review of the instructor's orientation revealed that evaluation methods for several criteria were not established.

Incomplete operational definition

For some criteria the evaluation method was established, but not enough detail was given on how to conduct it.[2]

The influence of other factors mentioned by Mackenzie *et al* was minimal, probably due to the review of the manuals by the instructors, the orientation meeting, and the mandatory use of evaluation forms.[2] It was also considered that the limited anatomy and morphology of typodont teeth had a definitive negative influence on both the students' performances and the students' and instructors' evaluations. In a clinical patient, the presence of enamel and dentin facilitates the determination of cavity depth.

During the course, the conditions of the study were performed under simulated clinical conditions. It may

be assumed, then, that the same errors that remained undetected in the present course, could also remain undetected in clinical practice. In Table 2, the significantly prevalent undetected errors are listed and related to the failure that could occur in clinical practice.

CONCLUSIONS

The information gathered in this study proved to be helpful in indicating which criteria, in a class II cavity preparation, in a primary maxillary molar, are most prone to incorrect evaluation. Careful attention should be paid to these criteria, to prevent failure because of lack of retention, tooth fracture, amalgam fracture, pulp exposure or incomplete removal of caries.

The authors express their appreciation to Dr. Gunnar Ryge and Dr. David Chambers for their suggestions and review of the manuscript.

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TWAIN OR BALZAC

I reasoned that when the time for locking up the dean or harassing the police passed and these no-longer-young persons turned to matters such as who would become dean or make the list for sergeant, the effect of numbers would be quite different: it would dampen spirits rather than raise them. The lecture was entitled "Peace." I proposed that the campus disorders of the preceding period would now end, but I tried to look further ahead than that. I observed that had I been asked ten years earlier who to read to find out what America was like, or was going to be like, I would unhesitatingly have said, "Read Mark Twin. Twain mostly told the truth, as Huck Finn testified, and perfectly conveyed a sense of the ebullience, the growth, the prospects, the limitless energy and potential, of our great and far, far reaching land." On the other hand, I continued, if asked then about the future, I would have recommended Balzac. "Find out what it's like to live in a society where if you want to be a professor, you wait until the man who is professor dies. Then the fifteen of you who want the job compete. . .One of you gets it. The rest hope for the best for their sons."

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Anterior space maintainer and regainer

Ruben E. Bayardo, DDS, MS

Frequently overlooked are the essential functions of the primary anterior teeth: masticatory, cosmetic, guidance for the eruption of the succedaneous teeth, a role in speech development, and a role in the avoidance of undesirable oral habits.

Caries and trauma are frequent causes of tooth loss. The premature loss of the primary incisors is frequently ignored. This attitude may prevail because the emotions of the child are misinterpreted and misunderstood by parents and clinicians. There is a lack of objective research, furthermore, to support a more positive attitude toward this problem.

REVIEW OF LITERATURE

It is generally agreed that only the very early loss of primary incisors will encourage drifting in the primary dentition.[1,2] Space closure in this segment is commonly the result of the mesiolingual inclinations of the teeth adjacent to the space, and assisted by pressures of the surrounding musculature. The degree of closure, however, varies from child to child, and the extent of closure is very difficult to predict.

Barber stated that it is not always necessary to place a space maintainer, whenever a primary tooth is lost prematurely.[3] The specific tooth, the arch, and the dental age of the child when the tooth is lost should be the determining factors. Thurow says that space maintenance is not required, because, in his opinion, premature loss of anterior teeth has no serious effect on the adjacent teeth. Loss of space or arch length occur when the lateral or anterior segments of the arch become shortened. The maintenance of maxillary arch form is dependent on the integrity of the mandibular arch. The premature loss of a mandibular incisor, however, is usually ignored, in the expectation that this segment will widen with growth. Crowding of the permanent incisors may be the result of this misconception.[3]

The normal primary arches are either in a spaced or closed relationship, a condition that is congenitally determined and is not developmental. Most children of preschool age have no anterior spacing of the primary dentition. In this condition, the intercanine space development is greater than that found in children who have spacing.[5]

Two of the most conspicuous changes of the primary dentition are attrition and spacing and their developmental sequelae. Thurow stresses the fact that the permanent incisors are considerably wider than their primary predecessors, thus requiring a considerable increase in arch length to accommodate them.[4] The alveolar processes have been reported to increase rapidly in height and width during these early years, so that by the time the primary incisors are shed, the permanent incisors can be accommodated.

Traditional anterior space management techniques consist of partial dentures or fixed appliances, most of which have been placed merely for cosmetic purposes.

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There are some disadvantages, however, associated with those appliances: the removable appliance is often lost, broken or irregularly used, whereas the fixed maintainer requires close cooperation of the patient, to control cleanliness and retention. Most types of appliances are rigid and static.[1,3-5]

Although the direct bonding technique offers clinicians several advantages over traditional types of maintainers, the retention that the technique provides has not been completely satisfactory.[6,7]

The objective of placing the anterior space maintainer described in this paper is to prevent drifting of the remaining teeth, restore function, and mainly, to permit spacing to occur, when feasible. Also the design for a space regainer will be presented, using basically the same appliance.

CASE PRESENTATION

Case 1

A three-year-old boy was seen nine days after the avulsion of the maxillary left primary incisor (Figure 1). The edentulous area was almost completely healed, the remaining teeth were asymptomatic, periapical radiographs showed no other anomalies, and the occlusion was in class I relationship with a slightly deep overbite.

An alginate impression was taken of the maxillary arch and poured in stone. Two .018" x .025" Edgewise standard buccal tubes were adapted to the mesiodistal dimension of the right central and left lateral incisors. A stainless steel mesh was spot welded to the tubes and trimmed at the base. A piece of rectangular .018" x .025" standard wire was bent U shaped, making sure that the wire touched the mesial surfaces of the abutment teeth (Figure 2). A polycarbonate crown was used as a pontic, and the palatal surface was grooved, to conform to the wire. The crown and wire were fixed with composite (Figure 3).

The space maintainer was tried in the patient's mouth, to check the area of buccal retention, the alignment of the pontic, the levels of the two tubes and the relationship of the distal end of the tube with the distal end of the wire.

After the teeth in the area were cleaned, the buccal surfaces of the two abutment teeth were lightly ground with a sandpaper disc, to remove the prismless layer of enamel present in primary teeth.[8] The teeth were isolated with a lip spanner, dried with air, and acid etched, using 35 percent phosphoric acid for one minute. The teeth were washed and dried and a thin coat of enamel bond was applied on the buccal surfaces of the abutment teeth. The space maintainer was bonded using an orthodontic self-polymerizing composite* (Figure 4).

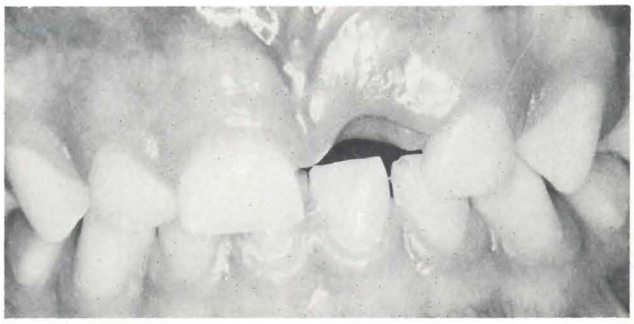

Figure 1. Early loss of a primary incisor due to avulsion.

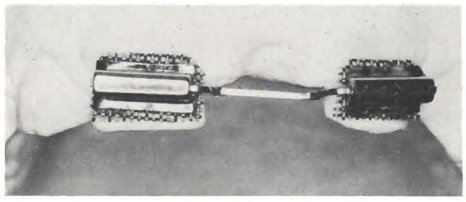

Figure 2. Simple orthodontic devices prepared on the working model.

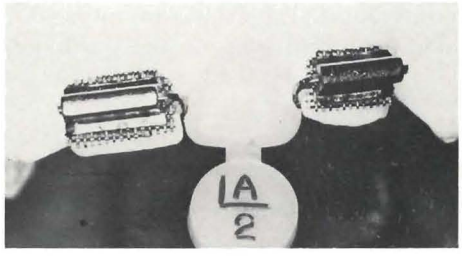

Figure 3. An esthetic and functional crown is selected as a pontic.

Figure 4. Direct bonding technique is used in the construction of the functional space maintainer appliance.

*Concise, 3M

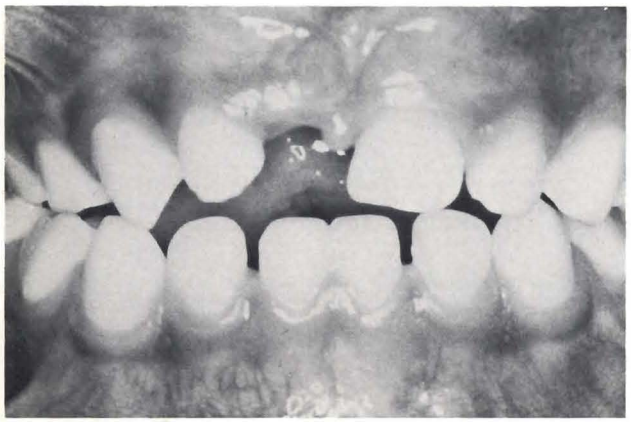

Figure 5. Space loss is aided by the premature loss of the primary incisor.

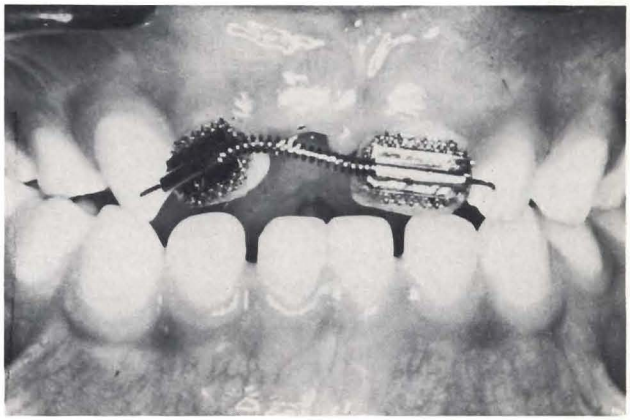

Figure 6. Direct bonding is used in the space regaining procedure.

After checking the occlusion, the patient and his parents were instructed to avoid hard foods or masticatory stress on the first day, to brush thoroughly, and to prevent improper use of the teeth.

The patient was checked after one week, also at three and six months after the appliance was inserted. At the last control visit, the space maintainer was functioning properly.

Case 2

A four-year-old boy presented, with a maxillary primary right central incisor missing, extracted four months earlier. The space was partially lost and the anterior teeth had drifted to the space. The patient showed a right posterior crossbite, possibly related to sporadic respiratory difficulty (Figure 5).

The patient received a prophylaxis, and two .018" x .025" standard labial tubes were adapted in the mouth. A stainless steel mesh was spot welded and trimmed to the tubes.

The enamel of the labial surfaces of the left central and right lateral incisors was treated as in case 1. Each labial tube was individually bonded to each abutment tooth. When the composite polymerized, a piece of .014" standard round wire was introduced into the lateral incisor tube. The wire was then inserted in a .036" x .009" open coil spring previously selected, and passed through the labial tube of the central incisor. A distal bend was made 2 mm from the distal ends of the tubes (Figure 6).

After three weeks, the coil spring was activated and after the space was slightly overwidened, a .016" round wire was inserted with the same coil spring. Three weeks later the wire was changed to a .018" and finally to a .018" x .025" wire, leaving the coil spring only for retention (Figure 7).

Five weeks later, an acrylic pontic was fixed over the wire and coil spring, using the same type of composite already in the patient's mouth (Figure 8).

The posterior crossbite decreased slightly with the regaining procedure alone, but complete treatment was postponed until the respiratory anomaly is corrected.

DISCUSSION

Despite the limited number of cases treated with this technique, the results are satisfactory. The greatest quality of this versatile appliance is that the clinician can control movement with it. When used as a space maintainer, therefore, it does not permit the teeth to move toward the space.

Special care should be given to very young children whose primary dentitions show no spacing of the teeth. Because the total width of the permanent incisors in each arch is about 6 to 7 mm greater than in the primary incisor teeth, they will require more than 1 mm of space on either side of each incisor.[4] Also, nonspaced arches are more prone to space closure after premature loss of the primary incisors than spaced arches.

Direct bonded appliances require of the clinician complete knowledge of enamel conditions and characteristics, concentration of the etching solution, length of etching period, isolation techniques, and proper use of the different composites. All play significant roles in the success rate of these procedures.

Rigid removable or fixed anterior space maintainers should be carefully used, since the geometry of occlusal function is affected by the changes in the size of the face and jaws.

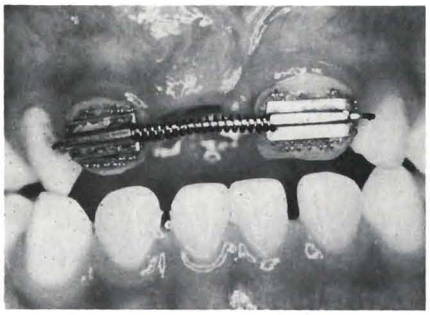

Figure 7. Space slightly overcorrected and stabilized.

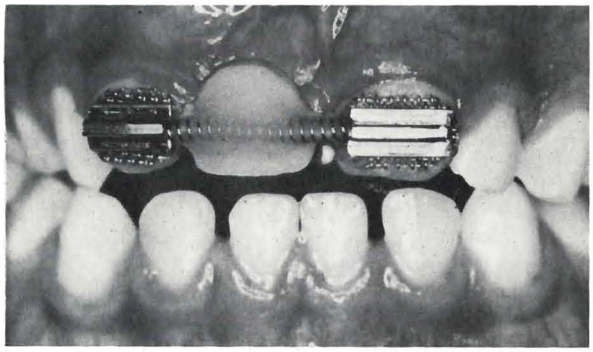

Figure 8. Function is restored with an acrylic pontic, fixed on the regainer with composite resin.

Some of the advantages of the space maintainer proposed in this paper are that it maintains arch symmetry, thus permitting spacing of the anterior teeth; function to near normal levels; encourages normal habits in lip and tongue posture; does not interfere with speech; avoids high laboratory costs; does not cause the patient discomfort; can be made directly in the mouth or on a model; and avoids occlusal impairment. The abutment on the upper teeth, however, is not esthetically accepted by all parents and occasionally not by the patients, who are usually more interested in cosmetic effects than in function.

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FIRST IMPRESSIONS

If the child's first impression of the dentist is a favorable one, and the dentist does not frighten the child patient or inflict pain, the child will become a good patient naturally. Happiness is a child's normal state of mind, and if he is happy in the dental chair his inclination is to do as the dentist tells him, to have no fear and to sit relaxed. It is possible for a child to become as fond of his dentist as he is of his parents, and particularly is this reaction true up to the ages of five or six. But the child must have the same confidence in his dentist as he has in his parents. If the future adults are to have good teeth, dentists and parents must create in children a good mental attitude toward dentistry, stress the benefits of dental health and not implant dental-office inhibitions in children.

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Tourette's syndrome: management of oral complications

Oariona Lowe, DDS

Tourette's syndrome, once thought to be a medical curiosity, is now a well-recognized clinical disorder, which appears regularly in the practice of child psychiatry. Recognized associated features of Tourette's syndrome include obsessions, compulsions, attentional difficulties, impulsivity, and personality problems.[1]

Gilles de la Tourette's syndrome was first identified by J.M.G. Itard in 1825. Later, in 1885, George de la Tourette further described the disease, characterizing it as consisting of multiple uncoordinated movements, especially tics, and voice utterances. An essential characteristic of the diagnosis is one of fluctuation of the various symptoms over the years, some movements or utterances are disappearing and changing, while others are beginning. Coprolalia (involuntary swearing) is an infamous hallmark of this syndrome, but it is not present in all cases and is not essential for diagnosis.[2]

No intellectual deterioration accompanies the disease, as once thought. The disease has been recognized as a movement disorder rather than a psychiatric illness and treatment with Haldol (haloperidol) has represented a major breakthrough in alleviating symptoms in many patients. Golden suggested that Tourette's syndrome may be a phenotype shared by biochemically distinct disorders.[3] The disease as characterized by Woodrow involves sudden involuntary movements, including vulgar gestures (copraxia), explosive involuntary utterances—both inarticulate noises (barks, yelps, grunts, coughs), and articulate obscenities (coprolalia)—and imitative phenomena verbal (echolalia) and behavioral echopraxia.[4]

Tourette's syndrome is widely misunderstood. It develops in children and is frequently misdiagnosed as a behavior disorder, since it is often characterized by motor dysfunction and hyperactivity. The onset of Tourette's disease typically occurs in childhood between the ages of two and fifteen. The disease is seen in males and females in a ratio of 3,4:1 and it appears in all racial groups. It is a movement disorder that often begins with uncontrollable twitchings of the face that gradually becomes worse.

Almost all patients develop motor and verbal tics. The tic can involve any part of the body but most often involves the muscles of the face, neck, shoulder, and trunk. The tic may be complex and consist of coordinated motor functions including a variety of behaviors. Shapiro, *et al* detailed the pattern of symptoms in this disorder and noted that the areas most frequently involved were the face, 94 percent; head, neck, and shoulders, 92.1 percent; upper limbs, 76.5 percent; lower limbs, 55.9 percent; and torso, 52.9 percent.[5] The muscle twitchings and jerks are noted to progress in severity in the initial years but may stabilize and cease in later years. The vocal tics may consist of barking or the explosive production of a phrase or short sentence. The cause of the tic syndrome is not fully understood but there is a general agreement that it is a motor discharge of psychogenic tension.[6] Patients have indicated that the severity of symptoms increases, if they are anxious,

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tense, angry, or self-conscious, and may be reduced when they are at rest or concentrating.

The twitching is usually followed by involuntary noises such as grunting or barking, or the patient may repeatedly echo words that he has heard. The most striking type of vocalization is sudden cursing or swearing. The patients express a sense of sudden increasing inner tension that is relieved by this swearing. The patients also describe a compulsive need to produce the sudden movement or vocalization and feel a relief of tension when they have done so.[7] Some patients need to touch things repetitively or have the need to touch other people on the breasts, buttocks, or genitalia; the patient may also handle his own genitals, exposing himself. Comings has suggested that hypersensitivity of dopamine receptors is involved in the exhibitionistic behavior displayed.[8] It has also been observed that some of the repetitive, self-directed actions appear to have a self- destructive aspect. The commonest manifestation noted is nail-biting, to the extent that the fingers are often raw and bleeding. In a study by Van-Hoert and others in 1976, 43 percent of the 114 patients observed has some evidence of self-mutilation.[9] This is a probable part of the compulsive tension-release phenomenon.

There is a lot of speculation about the etiology of the disease, but recent studies by Comings and his staff at the City of Hope Medical Center indicate a strong genetic component.[1,10] The genetic predisposition to Tourette's may be related to a neurotransmitter metabolism. Drugs that release dopamine from the nerve terminals have been reported to worsen or provoke the symptoms of Tourette's.[11]

The clinical diagnosis of Tourette's syndrome has recently been defined by the American Psychiatric Association as follows:

- ☐ Age at onset, between two and fifteen years.
- ☐ Recurrent involuntary, repetitive, rapid, and "purposeless" movements of many muscle groups.
- ☐ Involuntary vocalizations (sounds, words, or profanities).
- ☐ Voluntary suppression of movements for minutes or hours.
- ☐ Variation in intensity of symptoms over weeks or months.
- ☐ Duration of symptoms for more than one year.[12]

Many other movement disorders can be distinguished from Tourette's syndrome on the basis of pattern of movements and clinical course. As mentioned earlier, Tourette's may be associated with a continuation of the disorders. The tics associated with the disease are usually sudden, brief and repetitive, stereotyped movements that occur at irregular intervals. It is unquestioned that Tourette patients have complex motor tics that may be harmful to the individual.

Figure 1. Pretreatment: Traumatic biting of the tongue.

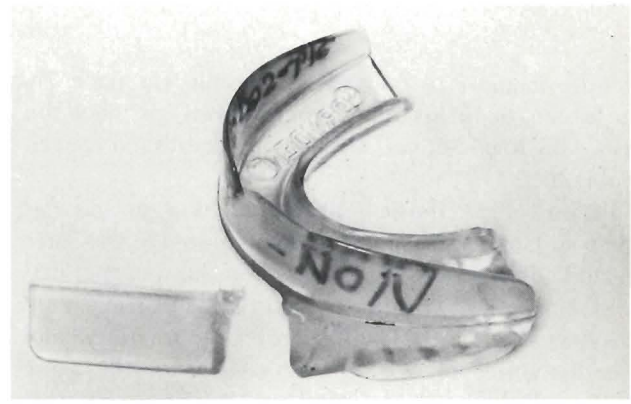

Figure 2. Plastic bite-block used in the Department of Anesthesiology.

A case of Tourette's syndrome, in which the patient's sudden movements were manifested in self-mutilization, is reported below.

M.R. was a sixteen-year-old Hispanic male, who was admitted to the city of Hope on January 8, 1985, with a diagnosis of Tourette's syndrome and compulsive biting of the tongue, secondary to Tourette's syndrome.

MEDICAL HISTORY

The medical history showed that the patient had an onset of tics at the age of five. By eight years of age, he began making weird noises, and by thirteen years of age, the onset of tongue-biting and cheek-biting was taking place. He was placed on daily doses of 5-15 mg of Haldol to control the biting. It was not until this late age

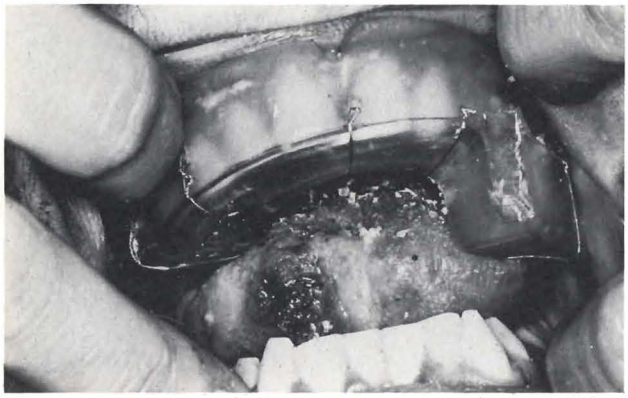

Figure 3. Plastic bite-block wired into place with orthodontic ligatures.

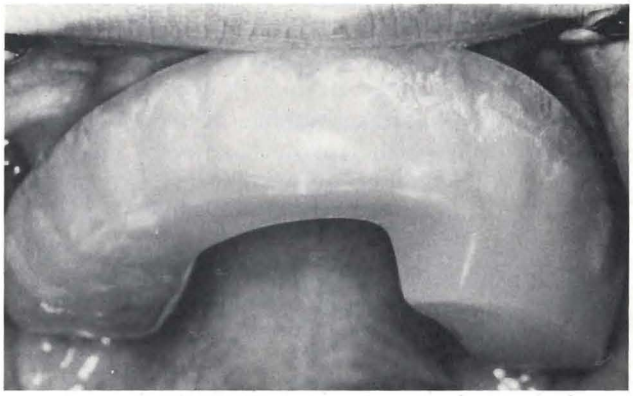

Figure 4. Upper permanent appliance.

that the diagnosis of Tourette's syndrome was made. By age fifteen, he bit his tongue continuously, twitched the eyes, was hyperactive, and had an established speech problem.

In May, 1983, the patient was hit by a car and was taken to County Hospital for treatment. He was later treated at Children's Hospital, where he was operated on six times. He was admitted to the Psychiatric Unit at Los Angeles County Hospital, for further treatment and was discharged several days later. He was still taking 10-15 mg Haldol daily. In October, 1984, his psychiatrist stopped the Haldol and the tics and tongue-biting worsened. On December 4, 1984, he was referred to the City of Hope for treatment, where he was admitted on January 8, 1985.

SOCIAL HISTORY

M.R. lives with his parents. His mother suffers from lupus and arthritis and is confined to a wheelchair. He often swears at her, but there is no coprolalia. His father is a used car salesman. M.R. has a better relationship with his father than with his mother.

PHYSICAL EXAMINATION AND REVIEW OF SYMPTOMS

On admission, the physical examination was within normal limits and the ENT examination revealed the right lateral tip of the tongue missing from repeated biting (Figure 1). The patient periodically calls out in pain from biting his tongue, but has to bite it to relieve the tension, as in a typical tic. The plan was to increase the Haldol until side effects occurred or the biting was stopped. The patient was also given Cogentin and Benadryl. It was learned from the patient that he is aware of the tic or prevailing movement disorder. He first notices a pain in the right temporomandibular joint area, sees colors, and then bites the tongue. He claims that biting relieves the tension. Valium appeared to decrease the tendency to bite. It was later suggested that an epileptic-type discharge may be involved. On the second day an EEG was ordered to rule out a seizure disorder, and Dilantin 100 mg b.i.d. was ordered. The EEG was read as normal.

His hospitalization showed an increase in the frequency of the tongue biting. Because it was considered possible that Haldol might be the cause of the increased frequency it was decreased to 1 mg b.i.d. The Dilantin was also stopped. A dental consultation was requested and recommendations for fabricating a protective appliance were suggested. Several unsuccessful attempts were made to take impressions for fabricating an appliance for M.R. By the fourth day, swelling and adenopathy of the right chin area were noted and the tongue appeared infected. Consequently, the patient was placed on antibiotics. An emergency procedure was begun, which involved overnight sedation with morphine sulphate, I.V.; and droperidol I.M. The patient was anesthetized to permit impressions for an intraoral appliance and to provide him with a temporary appliance that might protect the tongue from further trauma. A plastic bite-block, used in the Department of Anesthesiology to protect the teeth and keep the mouth open during laryngoscopy, was fitted in the patient's mouth and wired to the upper arch with orthodontic wires (Figures 2, 3). The appliance stayed in place for approximately five minutes, until the patient showed signs of regaining consciousness, when he became nauseous and pulled at the appliance, dislodging it. The

Figure 5. Lower permanent appliance.

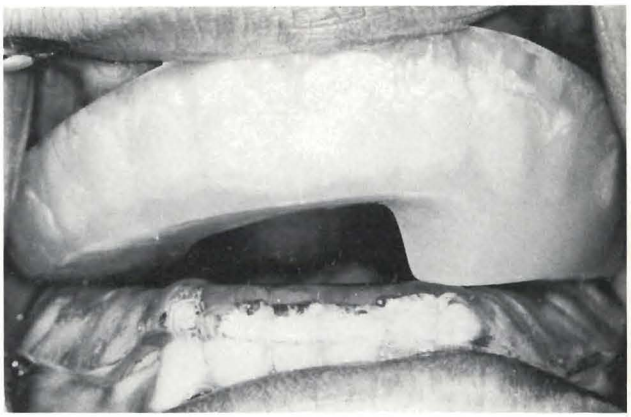

Figure 6. Upper and lower appliances in place.

next crucial step was to convince the patient that he should accept the appliance. It took a lot of positive reinforcement and feedback to get him to use it. M.R., however, kept the appliance in the mouth and removed it only when eating. He was also kept on morphine, 2 mg/hr; and droperidol, 0.5 mg q 4 hours p.r.n., for restlessness. This was continued for a day and a half and resulted in a marked decrease in biting and in some sedation.

Follow-up visits revealed that the patient had stopped biting his tongue and that the tongue was healing. The new appliance (Figures 4,5,6,7) was given to him and he was soon discharged. An eight-week check-up revealed the tongue to be healing well and the patient to have stopped biting his tongue.

DISCUSSION

Tourette's syndrome has become an important and fascinating disorder in recent years, because of the rapid growth of the neurosciences. It is a complex neuropsychiatric disorder with many clinical manifestations. It is associated not only with movement disorders, but also with impulsive and obsessive-compulsive conditions.

The cause of Tourette's syndrome is not known, but there is widespread speculation about possible precipitating factors. Organic abnormalities are suspected because of the evidence obtained from neurological testing, mental status examination, psychological testing, and the electroencephalogram. Neurological findings are inconsistent. In the past, Tourette's syndrome was commonly seen as a neurotic manifestation of an underlying psychiatric disturbance. The increased frequency of left-handedness, emotional trauma in childhood, and a family history of mental disease have supported this assumption.[13] Recent studies indicate that an imbalance exists between neurotransmitter systems, especially the central transmitters, dopamine and serotonin. Speculations concerning the response of patients with Tourette's syndrome to drug therapy can be based on a reduction of dopamine activity.[14] The decision to begin therapy is based upon the severity of symptoms and their potential for adversely affecting the psychosocial development of interrelationships. Treatment is strictly symptomatic, and, at present, drugs that act chemically to interfere with dopaminergic transmission appear to be the most effective. The literature provides documentation of the effectiveness of haloperidol. It should be noted that a good deal of variation exists in determining the maximum dose.

The theoretical significance of Gilles de la Tourette's disease lies mainly in its repetitive, purposeless movements. Some have likened the disease to schizophrenia.

CONCLUSION AND SUMMARY

Although genetic components are suggested, there seems to be no definitive evidence for a specific organic cause for Tourette's syndrome. It appears that an assumption can be made that perhaps the basic etiology of this syndrome is varied, with functional onset in some cases, and an organic or neurological onset in others.

With the exception of the initial description of Tourette's syndrome, the documentation of haloperidol's effectiveness in treating many of these patients may be the most significant contribution thus far to understanding this ill-defined disorder.

In the case presented, the following questions are raised: Why did he keep biting his tongue? What movement disorder was involved? It is speculated that the following conditions may have been responsible.

□ Spasms of the jaw muscles or the temporoman-

Figure 7. Healing tongue eight weeks after delivery of the appliance.

dibular joint, causing the jaw to shift to the right.

- ☐ Spasms of the extrinsic and intrinsic muscles of the tongue (hypoglossal nerve innervation).
- ☐ Involvement of the trigeminal or facial nerves, causing uncontrollable twitchings of the face.
- ☐ The drug and medication dosage prescribed.
- ☐ A combination of the preceding.

Whatever the symptomatology and possible etiology of the tongue-movement disorder, the patient did stop biting his tongue. The appliances helped to modify the behavior that initiated the tongue biting.

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TRANSPOSITION OF TEETH

Exchange of position of teeth most often involves the canine and the teeth adjacent to it. More rarely, transposition of premolars and molars has been noted also. In most instances, the transposed teeth are fully erupted and in normal alignment in the arch. There often is bilateral occurrence, which suggests that the anomaly has its origin in the anlage stage of development and not in a change in position in the course of eruption. An extremely rare occurrence is the transposition of a central incisor and a lateral incisor.

Jibilisco, J.A.: Stafne's Oral Radiographic Diagnosis, 5th ed. Philadelphia: W.B. Saunders Company, 1985, p 31.

Abstracts

Lee, J.G.; Moore, B.K.; Avery, D.R.; Horijitra, S.T.: Bonding strengths of etched porcelain disks and three different bonding agents. J Dent Child, 53:409-414 November-December, 1986.

Forty porcelain disks were resin bonded to etched base metal surfaces to measure tensile bond strengths. One metal surface was bonded to each porcelain surface, making each test specimen "sandwiched", with a porcelain disk between two metal surfaces. The mean bond strength values of the Conclude group was 3017 ± 869 psi. The mean for the Mar-Bond group was 3747 ± 693 psi, and the mean for the two Chameleon groups combined was 3172 ± 544 psi. No two groups were significantly different.

Bonding agents, Tensile bond strengths, Conclude, Mar-Bond, Chameleon.

Barbakow, Fred; Lutz, Felix; Scherle, Walter; Ransberger, Marinus: Solubility changes of human enamel treated with precipitate-free, aged SnF_2/amine fluoride solutions. J Dent Child, 53:415-419, November-December, 1986.

Enamel specimens were immersed in fluoride solutions (250 ppm F) containing either freshly mixed SnF_2 or aged precipitate-free aqueous solutions of SnF_2 and amine fluoride 297 (AmF 297) at molar ratios of 3:1; 1:1: or 1:3, respectively. After the specimens were treated, the enamel treated with SnF_2 in the SnF_2/AmF 297 solutions.

SnF_2, Amine fluoride 297, Enamel, Solubility

Morrow, Jack W.; Seale, N. Sue; Berry, Charles W.; Love, William D.: Incidence of temperature elevations after a full mouth dental rehabilitation under general anesthesia. J Dent Child, 53:420-424, November-December, 1986.

General anesthesia is a useful adjunct to dental treatment of certain children; postoperatively, elevated temperatures can occur. In this study, these observed elevations were compared for any association or correlation between the Oral Hygiene Index, Gingival Index Score, extraction of teeth, and degree of soft tissue trauma. The results showed no cause-and-effect relationships existed.

Temperature elevation, Tooth extraction, General anesthesia

Moody, Edward H.; Mourino, Arthur P.; Campbell, Robert L.: The therapeutic effectiveness of nitrous oxide and chloral hydrate administered orally, rectally, and combined with hydroxyzine for pediatric dentistry. J Dent Child, 53:425-429, November-December, 1986.

This study compared the effectiveness of three pediatric sedation modalities when they were used with nitrous oxide. Comparisons were based on a subjective evaluation of the overall quality of the sedation technique and data obtained from a pulse/oximeter. Oral administration of 50 mg/kg of chloral hydrate was the least effective modality.

Nitrous oxide, Chloral hydrate, Sedation

Roberts, Michael W.: Treatment of ectopically erupting maxillary permanent first molars with a distal extended stainless steel crown. J Dent Child, 53:430-432, November-December, 1986.

The exaggerated mesial eruption of the

Continued on page 462

a general practitioner in Minneapolis. Dr. Posnick for many years was active in ASDC activities and a strong promoter of dental care for children. This award is sponsored by the ASDC Foundation.

ASDC Journalism Awards. These awards are sponsored by the ASDC Foundation and are awarded for exceptional articles, published in the previous year's volume of the *Journal of Dentistry for Children*. Three awards are made and include a certificate for framing and a check for $500 for 1st prize, $300 for 2nd prize, and $200 for 3rd prize. The winners:

First prize to Ingegerd Mejàre DDS, PhD for the article "Deep bacterial penetration of early proximal caries lesions in young human premolars," which appeared in the March-April, 1985 issue of the *Journal of Dentistry for Children*. Co-author was Martin Bränström, DDS, PhD.

Second prize to Mirja Varpio, DDS for the article "Proximoclusal composite restorations in primary molars: a six-year follow-up" which appeared in the November-December, 1985 issue of the *Journal of Dentistry for Children*.

Third prize to Jimmy R. Pinkham, BS, DDS, MS for the article "Voice control: an old technique reexamined," which appeared in the May-June, 1985 issue of the *Journal of Dentistry for Children*. The co-author was John R. Paterson, DDS, MS.

NEW AID FOR NEEDY DENTAL STUDENTS

Proposed rules for a new program of financial aid for needy dental, medical and osteopathic students were published in the September 5 Federal Register. Eligibility will be limited to students from "a disadvantaged background" who have "exceptional financial need."

Under the program authorized by Public Law 99-129, the Health Professions Training Assistance Act of 1985, needy students may receive up to $10,000 a year to help pay for tuition, books and equipment, and living costs.

Only schools training the students may receive grants for the new Financial Assistance for Disadvantaged Health Professions Students.

Administrative expertise "does not exist in entities other than schools," the Department of Health and Human Services said. HHS also believes "it would be difficult for a school other than the medical or dental student's own school to assure that both the disadvantaged and the exceptional financial need criteria are met."

To be financially eligible, a student's available resources for educational costs can't exceed $5,000 or one-half the cost of attendance at the school, whichever is less. A "disadvantaged background" is one that inhibited the student from obtaining knowledge and skills to enroll in dental school or a low income family, a family of four for example with $14,300 or less annual income.

Funds will come from the HHS disadvantaged assistance program, which traditionally provided money only for recruitment, retention and placement of students. PL 99-129 directed 20 percent of these funds to the new program. About $4.9 million is available and will be awarded by the end of September, HHS said.

NIDR APPROPRIATION

The Senate Appropriations Committee recommended $116,553,000 for NIDR, but it is expected to be reduced by an amendment sponsored by Senators Pete Domenici (R-NM) and Lawton Chiles (D-FL). Their amendment increased funds for math and science education by $57 million and reduced funds for the National Institutes of Health by $11 million.

ABSTRACTS

Continued from page 407

permanent first molar can result in the tooth becoming impacted, or the premature atypical resorption and exfoliation of the primary molar, with a resultant loss of space. A stainless steel crown with a distal guide plane was placed on a primary molar to correct the aberrant eruption angle of the permanent tooth. This procedure can be used successfully for both partially and totally tissue-impacted teeth, and is described in this report.

Impaction, Ectopic eruption, Stainless steel crown

Pöyry, Matti; Prenatal factors and tooth eruption in children with oral clefts. J Dent Child, 53:436-438, November-December, 1986.

The timing of eruption for primary central incisors was studied in seventy-six children with oral clefts. The eruption timing was found to be very close to that of healthy children, and only weak correlations were found between prenatal factors and eruption times of first primary teeth.

Prenatal factors, Tooth eruption, Oral clefts