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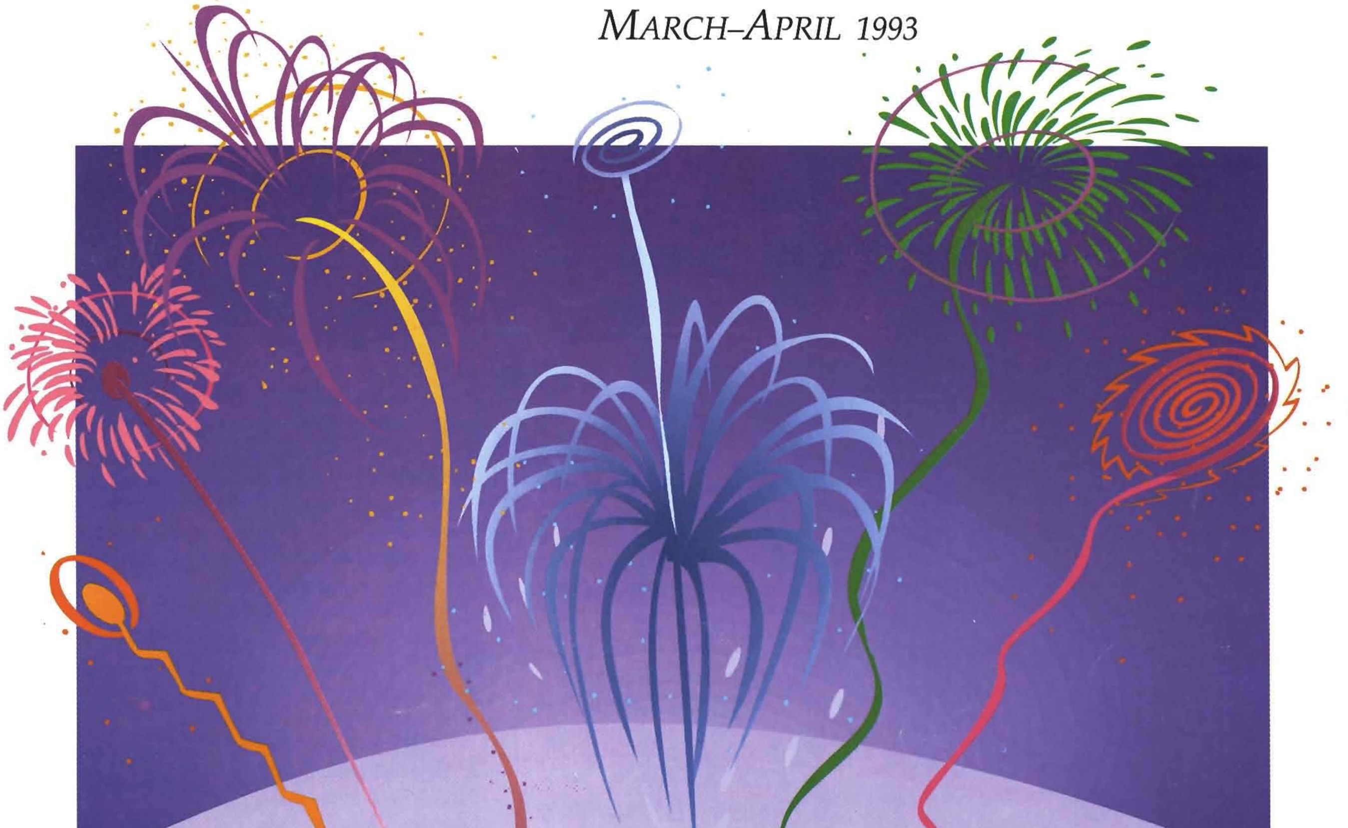
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

JOURNAL OF DENTISTRY FOR CHILDREN

MARCH–APRIL 1993



The nature of the early adolescent process is to experience the world and to react to it in forms that tend to be extreme. The youngster is head over heels in ***love***, suffused with ***rage***, covered with ***confusion***, drowned in ***embarrassment***, filled with maudlin ***sentimentality***, or overwhelmed with ***concern***—the only common factor among these states being the totality of the emotion. Nuances will come later, as adolescence advances; now the state is close to all or none. This emotional style gives rather a specific coloring to the interactions of the period, a mixture of intensity and childishness that speaks for the transitional character of the time. It also makes the moments of rebellion particularly difficult to bear.

–Joseph D. Noshpitz, M.D.

REBELIOUS HELL,
IF THOU CANST MUTINE IN A MATRON'S BONES,
TO FLAMING YOUTH LET VIRTUE BE AS WAX,
AND MELT IN HER OWN FIRE.

—Shakespare, in Hamlet

STEREOTYPE • Sexism • Children in Crisis • Handicaps

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MARCH-APRIL 1993 VOLUME 60 NUMBER 2

 USPS #279-480. Issued bimonthly—in January-February, March-April, May-June, July-August, September-October, and November-December—at 211 E. Chicago Avenue, Suite 1430, Chicago, IL, (312) 943-1244. Second class postage paid at Chicago, IL and additional mailing office. Subscription prices: within U.S.A., individuals $65.00 per volume, institution $95.00, single copies $20.00; Foreign (including Canada and Mexico) individuals $75.00 per volume, institution $105.00, single copies $25.00. Thirty-five dollars and fifty cents ($35.50) of the full membership dues are allocated to the Journal. Member—American Association of Dental Editors.

All copy and manuscripts for the journal should be sent directly to the Editorial Office, 211 E. Chicago Ave., Suite 1430, Chicago, Illinois 60611, (312) 943-1244.

Prospective authors should consult "Information for Authors." Reprints of this document may be obtained from the Editorial Office.

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The adolescent often reacts to the affairs around him in ways that are extreme, with totality of emotion as the common factor.

Art and design by Sharlene Nowak-Stellmach

CLINIC

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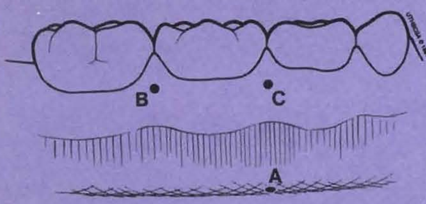

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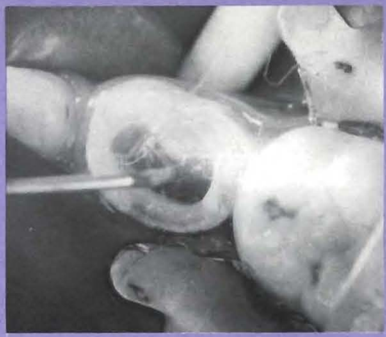

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REVIEW

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REPORTS

For the busy reader

A restorative dentistry renaissance for children: Light-hardened glass ionomer/resin cement—page 89

By the addition of the light-cured resin component, resistance to wear and fracture has been greatly improved. The new formulations with some improvements should provide us with a worthy alternative to silver amalgam for the restoration of primary teeth and for certain applications in permanent teeth.

Requests for reprints should be directed to Dr. Theodore P. Croll, Georgetown Commons, Suite 2, 708 Shady Retreat Road, Doylestown, Pennsylvania 18901-3897.

Prevalence of postoperative sensitivity with indirect class II resin composite inlays—page 95

A study was made of postoperative complaints after the placement of indirect class II resin composite inlays. Influencing factors were considered. Amalgam restorations were used as a reference. The patients were asked about the presence or absence of postoperative complaints at follow-up visits. No difference in complaints was found between teeth with inlays and those with amalgam restorations. It was also found that the patient seemed to be a major factor in whether postoperative sensitivity occurs.

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

The relationship between alveolar bone loss and proximal caries in children: prevalence and microbiology—page 99

In this study, the authors discuss the prevalence of alveolar bone loss as it is related to caries, loss of proximal contact, and loss of space. This was accomplished by means of radiographic evaluation. Microbiological aspects of the sites studied are also presented.

Requests for reprints should be directed to Professor Enrique Bimstein, Department of Pediatric Dentistry, Hadassah Faculty of Dentistry, P.O. Box 1172, Jerusalem, Israel.

Evaluation of mandibular infiltration versus block anesthesia in pediatric dentistry—page 104

The clinical effectiveness of mandibular infiltration was compared with that of mandibular block anesthesia for treatment of mandibular primary molars. The authors concluded that satisfactory anesthesia could be obtained with the infiltration technique.

Requests for reprints should be directed to Dr. Franklin Garcia-Godoy, Department of Pediatric Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78284-7888.

Electrosurgical pulpotomy: A retrospective human study—page 107

A retrospective study of 101 patients, including 164 pulpotomies, was made to determine the success of the electrosurgical technique. Patients as young as eighteen months and as old as ten years, six months (mean: five years, eleven months) were in the group. Only one case of the 164 was considered to have failed.

Requests for reprints should be directed to Dr. Ronald B. Mack, 632 Taraval Street, San Francisco, California 94116-2512.

A review of fluoride intake from fluoride dentifrice—page 115

Studies with younger preschool children are presented in the greatest detail, because these children are at greatest risk of fluorosis (20 to 28 months). These young children, in substantial proportion ingest significant amounts of dentifrice. The efficacy of dentifrices with lower concentrations of fluoride than currently available should be studied. In addition, small amounts of fluoridated dentifrice should be encouraged and used under parental direction and supervision.

Requests for reprints should be directed to Dr. Steven Levy, N330, DSB, The University of Iowa, Iowa City, IA 52242.

Dental management of the child and adolescent with major depression— page 125

It is recognized today that a sizeable number of children are afflicted with major depression, a psychiatric disorder in which mood, thought content, and behavioral patterns are disturbed for extended periods of time. Children with major depression are likely to present with conditions that lead to advanced dental disease.

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

The poisoning of our children—page 132

There were more than 2.2 million poison exposures reported to all U.S. poison centers in 1990. Almost a half of all cases of poisoning for the entire population covered in the report of the poison control centers were children under three years of age.

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

Is your next pediatric patient an addict?—page 136

In this paper the author reviews some of the results of the latest national study on drug abuse among children. It may seem unreasonable that dentists add to their problems by assuming that their next twelve-year-old patients may be drug "pushers" or users; but the reality is that your next patient could be an addict.

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

Gender trends among specialists in pediatric dentistry—page 140

The authors examined the gender and practice trends among dentists who specialize in the care of children. The results are based on 1266 anonymously completed questionnaires (1053 male, and 213 female). The number of pediatric dentists being trained each year has remained relatively constant for the past twenty years. The number of women entering the specialty has grown consistently in the last few years.

Requests for reprints should be directed to Dr. Michael W. Roberts, Department of Pediatric Dentistry, School of Dentistry, CB #7450, University of North Carolina, Chapel Hill, North Carolina 27599-7450.

Adverse reaction to a fissure sealant: Report of case—page 143

This report describes the adverse reaction of a six-year-old girl to a fissure sealant. The author describes the procedures followed to return the patient to normal.

Requests for reprints should be directed to Dr. Ulla Hallström, Special Clinic of Children's Dentistry, Tandvåardscentralen, Box 1166, s-221 05 Lund, Sweden.

CLINIC

A restorative dentistry renaissance for children: Light-hardened glass ionomer/resin cement

Theodore P. Croll, DDS
Constance M. Killian, DMD
Mark L. Helpin, DMD

Light-hardened glass ionomer/resin material was developed and introduced in the late 1980s.[1,2] Although Vitrebond (3M Dental Products Division, St. Paul, MN) was designed to serve as a light-hardened base/liner, it was found to be useful for replacement of dentin and enamel on nonstressbearing surfaces of primary teeth.[3] It logically followed that a light-hardened glass ionomer/resin material with better wear resistance and more fracture toughness than the base/liner would be a very desirable product.[3,4]

In 1992, light-hardened glass ionomer/resin dental restorative material was introduced.[5-7] The formulation consists of about 80 percent glass ionomer material combined with a 20 percent visible light polymerized resin component. This new type of restorative material has the important properties of all glass ionomers, including:

- ☐ Biocompatibility.
- ☐ Coefficient of thermal expansion similar to that of tooth structure.
- ☐ Good compressive strength.
- ☐ Chemical bonding to tooth structure.
- ☐ Fluoride ion leaching without degradation of the cement, or diminution of physical properties.
- ☐ Injectability for easy application.
- ☐ The cement is tooth-colored and available in multiple shades.
- ☐ Insolubility in oral fluids.

Unlike the self-hardening glass ionomer cements that take seven to ten minutes for initial hardening, the new formulations have dual hardening mechanisms. The resin component cures in thirty to sixty seconds by visible light exposure, giving the cement substantial initial hardness. Then, the glass ionomer hardening reaction continues within the hard resin matrix, and when completed, the physical properties of the cement are optimal.

After using several light-hardened glass ionomer/resin cements for one year, we have found the new formulations quite useful for children. Encouraged by the long-term success of glass ionomer silver cermet restorations in primary teeth and up to five-year results of using light-hardened glass ionomer/resin base/liner material for dentin and enamel restorations, we have performed over 1,700 class I, class III, class V, and class II restorations in primary teeth, using the new light-hardened cements.[3,8,9]

The authors gratefully acknowledge the ESPE-Premier Sales Corporation for their grant to support the color photographic reproduction in this work.

Dr. Croll is in private practice, pediatric dentistry, Doylestown, PA; Clinical Associate Professor, Department of Pediatric Dentistry, University of Pennsylvania School of Dental Medicine; Adjunct Professor, Department of Pediatric Dentistry, University of Texas Health Science Center at Houston (Dental Branch).

Dr. Killian is in private practice, pediatric dentistry, Doylestown, PA; Clinical Assistant Professor, Department of Pediatric Dentistry, University of Pennsylvania School of Dental Medicine.

Dr. Helpin is Chairman, Department of Pediatric Dentistry, University of Pennsylvania School of Dental Medicine; Chief, Dental Division, Children's Hospital of Philadelphia.

This paper demonstrates step-by-step restoration of a primary molar with class I caries, using Photac-Fil, a new encapsulated, light-hardened glass ionomer/resin cement. A class II restoration of a primary molar, using another brand of cement, is also pictured, along with a class III restoration of a primary canine tooth. Proper mixing of the cement and careful placement technique to avoid air entrapment are emphasized, along with the final sealing step, used to improve the trimmed cement surface and seal the cavosurface interface.

TECHNIQUE

Examples of light-hardened glass ionomer/resin cement restorations of primary teeth are shown in Figures 1-20.

Figure 1. This maxillary primary second molar had occlusal and occlusolingual caries.

Figure 2. After the tooth was completely debrided, traditional amalgam-type preparation was cut. Note the mechanical interlocking retention form.

Figure 3. Twenty-five percent polyacrylic acid is placed for five to ten seconds for removal of smear layer.

Figure 4. The premeasured capsule mixing container is activated. The handle should be compressed for at least three seconds, to force the acid solution into the mixing chamber with the powder.

Figure 5. After mixing the cement in an electric amalgamator, the material is injected into an AccuDose syringe tip (Centrix, Inc., Shelton, CT), for direct injection into the cavity preparation.

Figure 6. The thin tip of the syringe is inserted into the cavity preparation, and the cement is slowly injected. Much care is used to assure that no air is entrapped. The preparation is purposely over-filled.

Figure 7. A ball burnisher dipped in isopropyl alcohol can be used to spread the excess cement over cavosurface margins.

Figure 8. The visible light beam is applied from the occlusal direction for thirty seconds. An additional thirty-second application of light was directed from the lingual aspect.

Figure 9. A slow-speed No. 8 round bur is used to remove excess cement and roughly sculpt anatomical form.

Figure 10. After additional trimming with smaller round burs, 40 percent phosphoric acid gel is applied to the cement and peripheral enamel for twenty seconds, in preparation for final application of the sealant.

Figure 11. The clear resin sealant is applied and permitted to saturate the glass ionomer surface and surrounding enamel for ten seconds. The light beam is then applied for twenty seconds.

Figure 12. After removal of the rubber dam, occlusal relationships in all mandibular excursions are evaluated. Note that centric holding stops and functional contacts are not evident on the restorative surface.

Figure 13. The occlusal and occlusolingual restorations shown six months after placement.

Figure 14. This mandibular molar was prepared for mesioclusodistal restoration. Notice the relatively wide and deep penetration to maximize cement bulk, and the tightly wedged matrix segments.

Figure 15. The light-hardened glass ionomer/resin cement is carefully injected and the cement excess covers all cavosurface margins.

Figure 16. The light beam is applied for forty seconds in this case, because of the size and depth of the restoration.

Figure 17. After initial hardening, general occlusal anatomical form is sculpted and the restoration sealed, as described above. Marginal ridges are carved slightly out of occlusion to minimize occlusal stresses.

Figure 18A. Nine-months after placement the restoration is shown.

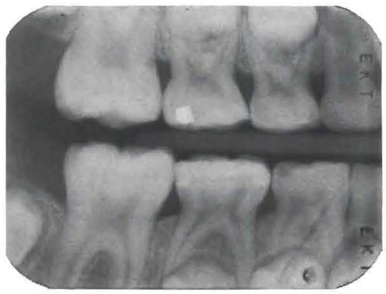

Figure 18B. Nine-months postoperative radiograph of the restoration shown in 18A.

Figure 19. Light-hardened glass ionomer/resin cement was used to restore a distal class III lesion in this primary canine tooth.

Figure 20. Nine-months after treatment, the distal restoration was intact and the margins undetectable when probed with an explorer tip.

DISCUSSION

The new light-hardened glass ionomer/resin formulations represent a great improvement over the self-hardening glass ionomers. The self-hardening cements take seven to ten minutes for initial hardening and are very sensitive to desiccation and over-hydration during placement and immediately afterwards.[10] Furthermore, they have low fracture toughness, and poor wear resistance. Addition of the 20 percent light-hardened resin component to the glass ionomer formula gives the material an initial setting time under sixty seconds, and substantially improves wear resistance and fracture toughness. Dentists using the new materials are eager to observe how they hold up in comparison to bonded composite resin restorations, glass ionomer silver-cermet restorations, and silver amalgam restorations.

Powder/liquid ratio of light-hardened glass ionomer/resin cement is important. Excess liquid makes for quick mixing and a cement consistency that is easily injected into a cavity preparation; such a mixture substantially diminishes, however, the physical properties of the hardened material. Premeasured capsules obviate uncertainties in powder/liquid proportions and make for consistent mixes of cement.

We consider the orange, fine tipped AccuDose syringing system (Centrix, Inc., Shelton, CT) essential in the procedure (Figures 4,5,6,15 and 19).[7,8] The small lumen of the syringe tip allows accurate placement of the cement, deep within the confines of even small preparations. If not injected slowly and carefully, light-hardened glass ionomer/resin cement restorations can have air voids and be significantly weakened. One is not able to compress the cement into a preparation, in the same manner as silver amalgam is condensed. The cement sticks to hand instruments, so the best way to assure complete cavity fill is by careful injection, and the fine orange Centrix tip is ideal for that purpose.

Excess cement is intentionally left, overlapping cavosurface margins (Figure 9). Unlike marginal "flash" of silver amalgam material, such excess of the adhesive glass ionomer/resin material serves as a "sealant" of cavosurface margins.[7] We perform a final etching of the cement and enamel surfaces followed by clear sealant application (Figures 10 and 11) for the same reasons that one seals posterior composite resin restorations. We speculate that the clear sealant saturates and fills cavosurface microgaps, which occur due to material shrinkage during the hardening process, and in addition repairs cement surface damage resulting from the finishing process.[11,12] At six-month reevaluation of sealed versus unsealed occlusal restorations, placed in paired contralateral primary molars, the unsealed cement appears to wear slightly more. This clinical observation needs to be verified and the full value of final resin sealant application should be investigated.

We have found that completely hardened glass ionomer/resin restorative cements have some improved physical properties compared to pure glass ionomers. Due to inclusion of the light-cured resin component, wear resistance is enhanced and fracture toughness improved. Furthermore, we have observed that the hardened cement appears to flex and absorb forces without fracturing, and rebounds once the force is withdrawn. Studies of the modulus of elasticity and the reasons for such resilience should be investigated. Fracture toughness studies comparing the new cements with composite resin and silver amalgam would also be very valuable. For the record, since January 1992, we have observed only two fractured class II light-hardened glass ionomer/resin restorations in primary molars, of over 170 placed. One of those was in a patient who habit-

Accurate measurements of ingredients and consistent mixes are essential to success of the restorations.

ually chewed ice cubes, and the other was in a patient who chewed ice cubes and uncooked pasta noodles. Long-term documentation of all our restorations will continue.

Manufacturers of light-hardened glass ionomer/resin cements should now be concentrating on developing a material with wear resistance, fracture toughness, and radiopacity, equal to that of silver amalgam. In addition, continuing studies should be performed on the full implications of fluoride ion leaching, material shrinkage during the hardening processes, long-term dimensional stability of the cement *in vivo*, and the nature and effect of marginal leakage of such restorations. If any of the new formulations pass the tests of intraoral use over a five to eight-year period, dentists will have a worthy alternative to silver amalgam for restoration of primary teeth and for certain applications in permanent teeth.

REFERENCES

1. Mitra, S.B. and Creo, A.L.: Fluoride release from light-cure and self-cure glass ionomers. J Dent Res, 68(Spec Iss): abst. #739, February 1989.
2. Mitra, S.B.: Property comparisons of a light-cure and a self-cure glass ionomer liner. J Dent Res, 68(Spec Iss): abst. #740, February 1989.
3. Croll, T.P.: Visible light-hardened glass-ionomer cement base/liner as an interim restorative material. Quintessence Int, 22:137-141, February 1991.
4. Croll, T.P.: Glass ionomers for infants, children and adolescents. J Am Dent Assoc, 120:65-68, January 1990.
5. Christensen, G.J.: Glass ionomer-resin restorations. Clin Res Assoc Newsletter, 16:1-2, February 1992.
6. Croll, T.P.: Glass ionomers and esthetic dentistry. J Am Dent Assoc, 123:51-54, May 1992.
7. Croll, T.P. and Killian, C.M.: Visible light-hardened glass-ionomer-resin cement restorations for primary teeth: new developments. Quintessence Int, 10:679-682, October 1992.
8. Croll, T.P. and Phillips, R.W.: Glass ionomer-silver cermet restorations for primary teeth. Quintessence Int, 17:607-615, October 1986.
9. Croll, T.P. and Phillips, R.W.: Six years experience with glass ionomer-silver cermet cement. Quintessence Int, 22:783-793, October 1991.
10. Wilson, A.D. and McLean, J.W.: The setting reaction and its clinical consequences. Chapter 3 in: *Glass-Ionomer Cement*. Berlin and Chicago: Quintessence Publishing Co., Inc., 1988, pp 43-56.
11. McCourt, J.W. and Eick J.D.: Penetration of fissure sealants into contraction gaps of bulk packed auto-cured composite resin. J Pedod, 12:167-175, Winter 1988.
12. Dickinson, G.L., Leinfelder, K.F., Mazer, R.B. *et al*: Effect of surface penetrating sealant on wear rate of posterior composite resins. J Am Dent Assoc, 121:251-255, August 1990.

DETERMINING SEX IN CHILDREN DURING FORENSIC INVESTIGATIONS

Determining sex in children can be even more elusive. Most of the skeletal differences, even in the pelvis, that distinguish the sexes don't fully define until early adulthood, and the differences that do exist in children are often not of the magnitude that permit a confident estimate.

One of the best indicators of sex in a child is the teeth. In determining gender, the indicator is not in how dissimilar they are, but in how alike. It is well known that in general males tend to be a year or two slower than females in their overall body development. But although girls' long bones grow earlier and faster than boys' do, for some reason that same advantage is not as extensive in the development of the teeth. Accordingly, it is possible to estimate the sex of a child's skeleton by comparing the extent of skeletal development with the level of dental maturation. The older the child, the more accurate the technique. However, we usually do not attempt to estimate the sex of immature skeletons because the rate of accuracy reaches only about 80 percent even in older children. In a forensic case, 80 percent is not good enough; we can estimate with 50 percent reliability just by guessing.

Ubelaker, D. and Scammell, H.: *Bones: A forensic detective's casebook*. New York: Harper Collins Publishers, 1992, pp. 87, 88.

Prevalence of postoperative sensitivity with indirect class II resin composite inlays

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Pulp reactions resulting from the placing of dental restorations have been described by various authors and, based on data appearing in the literature, possible causes of these reactions following the direct placement of posterior resin composites were discussed in a previous paper.[1] Measures for limiting postoperative sensitivity were also discussed. As the percentage of complaints reported in that study was 23 percent, and one of the measures was the reduction of shrinkage from polymerization in the resin composite restorations, it is worthwhile to study the effect of an extended reduction in shrinkage.

The indirect fabrication of resin composite restorations allows the material to polymerize outside the mouth. In this way, in-situ shrinkage might be reduced, which could positively affect the reduction of postoperative sensitivity.[2,3] Yet when the inlay is being placed, the resin composite luting cement has almost no free surfaces, and considerable stress from shrinkage may develop within this cement during polymerization.[4] This effect may increase when the thickness of the cement layer is reduced.[5]

This article describes the findings of a study of postoperative complaints after the placement of indirect Class II resin composite inlays. Possible influencing factors were considered. Amalgam restorations were used as a reference in both the present study and in the study previously described; this permits comparison of data from both studies with this standard.

MATERIALS AND METHODS

In this study 240 "standard-size" Class II restorations were applied to fifty-eight patients. The design of the study and the selection criteria used have been described elsewhere, and the methods were the same as those used in the study of direct resin composite restorations.[1,6] Each patient was given one or two series of four restorations: three indirect inlays, each made of one of three resin composite restoratives,→ and one amalgam● control (the four materials were randomly

≈ Porcelite Dual Cure (Kerr) CR Inlay Cement (Cavex Holland/Kuraray) Tulux-Cem (ESPE).

The authors would like to thank Professor C.L. Davidson and J.W. Hagen for their comments and suggestions.

This study has been supported by Kerr and Cavex Holland/Kuraray.

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

● Tytin (Kerr)

allocated). Fifty-six patients received one series of four restorations, and two received two series. Three dentists each made twenty series.

The procedure followed in making the indirectly applied resin composite inlays can be summarized as follows: After making an inlay preparation of "standard" size (that is, a standard conservative Class II cavity preparation, without cuspal replacement, of which the cervical outline should not exceed the cementoenamel junction) followed by removal of the remaining caries, calcium hydroxide★ was applied at the deepest places in the cavity where necessary, and the dentin removed was replaced by a glass ionomer cement†.[6] The inlay preparation was finished, and impressions were taken. A silicone material- was used for the purpose of a temporary restoration. The inlay was produced in a dental laboratory, and was polymerized with the aid of a light-heat oven[f]. The inlay was then placed using a dual-cure luting cement≈. Final finishing of the marginal adaptation was carried out during a separate appointment.

The data collected refer to the type of restorative material used, the type of tooth (premolar/molar), the type of restoration (two-surface/threesurface), the patient, and the dentist who carried out the treatment. The patients were asked about the presence or absence of postoperative complaints at follow-up visits, and half a year after the restorations had been placed. Sensitivity was recorded for each tooth, using the following scale:[1]

0 = not sensitive.

1 = sensitive, diminishing, lasting no longer than a week.

2 = sensitive, diminishing, lasting between one and two weeks.

3 = sensitive, diminishing, lasting between two weeks and three months.

4 = occasionally sensitive, not diminishing.

5 = constantly sensitive, not diminishing.

Frequency distributions are presented for the results, and Chi-square tests were conducted (SPSS/PC).

★ Life (Kerr)

† Fuji lining cement (GC)

" Fermit (Vivadent)

[f] According to the instructions of the manufacturers of the resin composites used.

≈ Porcelite Dual Cure (Kerr) CR Inlay Cement (Cavex Holland/Kuraray) Tulux-Cem (ESPE).

RESULTS

It can be seen from Table 1 that postoperative sensitivity was found in thirty-five teeth (15 percent). In nine teeth (4 percent) this sensitivity lasted between two weeks and three months. After half a year, one tooth was still sensitive, although to a diminishing extent. Of the intermittent sensitive teeth, one was occasionally sensitive after six months; the balance of the complaints had disappeared after that period of time.

For statistics, a two-way classification in sensitivity has been applied to the data of Table 1 (being sensitive and non-sensitive teeth). In this way, the restorations of Herculite and Clearfil cause a comparable degree of sensitivity, 13 percent and 12 percent respectively, whereas the amalgam restorations show less sensitivity (10 percent). The Visiomolar restorations appear to cause the most postoperative sensitivity (23 percent), but the differences observed are not significant ($p<0.05$).

The frequencies of premolars and molars with, and without, postoperative sensitivity are presented in Table 2. In the case of resin composite restorations, molars significantly show more postoperative sensitivity compared with premolars (31 percent versus 11 percent, $p<0.01$). When amalgam restorations are included, these figures are 25 percent and 10 percent,

Table 1 ☐ Number of teeth with or without symptoms of postoperative sensitivity, classified by the type of restorative material and duration of the sensitivity.

	Restorative material				
Sensitivity score	Herculite	Clearfil CR Inlay	Visiomolar	Tytin	Totals
No sensitivity	52	53	46	54	205
< 1 week	3	2	4	2	11
1 - 2 weeks	1	1	1	2	5
2 weeks - 3 months	2	1	4	2	9
Intermittent	1	3	5	0	9
Constant	1	0	0	0	1
Totals	60	60	60	60	240

Table 2 ☐ Premolars and molars with and without sensitivity.

	Restorative material				
	Herculite	Clearfil CR Inlay	Visiomolar	Tytin	Totals
Premolars					
No sensitivity	42	42	36	32	152
Sensitivity	6	4	5	2	17
Molars					
No sensitivity	10	11	10	22	53
Sensitivity	2	3	9	4	18
Totals	60	60	60	60	240

respectively ($p < 0.01$). Furthermore, 15 out of 140 two-surface restorations (11 percent) showed postoperative sensitivity. With three-surface restorations, this was 20 out of 100 (20 percent). Compared to two-surface restorations this appears not to be significantly different ($p > 0.05$).

Table 3 shows the number of patients for whom no teeth, one tooth, or more than one tooth show postoperative sensitivity. A distinction has been made between patients with one and with two series of four restorations. Thirty-six (62 percent) of the fifty-eight patients had no complaints. To obtain a better chance composition, patients with two series of four restorations are excluded, and thus seven of fifty-six patients (13 percent) appear to have twenty sensitive teeth (that is 61 percent of a total of thirty-three sensitive teeth). For three of the patients, all of the teeth in the study cause postoperative sensitivity; this includes both the resin composite inlays and the amalgam restorations.

Table 4 shows the distribution of the sensitive teeth found per dentist. The difference between the dentists appears to be significant ($p < 0.001$). Three of Dentist 3's patients reported that all of their restored teeth had shown postoperative sensitivity.

DISCUSSION

Whether extraoral polymerization of the bulk of resin composite restorations limits postoperative complaints may be studied by comparison with the results of sensitivity in our study on direct resin composite restorations (referred to as Study 1).[1] Equal measures to limit sensitivity were applied; the extra-oral polymerization was additional. In the present study (Study 2), the influence of the factors investigated appeared to be for the greater part in accordance with the findings of the previous study (Study 1). No significant influences were found for the materials, although in both studies, one resin composite material (Visiomolar) appeared to give rise to more pain complaints (not significantly different, however). The tooth type was a substantial factor. The patient also appeared to affect the prevalence of complaints; from both studies it appears that a small percentage of the patients accounts for more than half of the total complaints (in Study 1 this figure is 27 percent of the patients; in Study 2 it is 13 percent of the patients). The patient-effect is an influencing (or disturbing) factor upon the difference found between the dentists: the three patients, of which all restorations showed sensitivity, were within the group of patients of one of the dentists.

In the present study, fewer complaints were recorded when compared to the results of Study 1. This could support the hypothesis that a reduction in the in-situ shrinkage from polymerization leads to a reduction in the number of complaints. It is not only the sensitivity with the resin composites, however, that is reduced, the amalgams also showed less sensitivity. Comparison of the two studies concerning postoperative sensitivity, therefore, might be affected due to a difference in the two populations regarding the experience of sensitivity. An additional factor is the replacement of one of the three dentists, when the two studies are compared. If the results are reduced to those of the two dentists involved in both Studies 1 and 2, the prevalence of sensitivity for composites is 28 percent (Study 1) and 20 percent (Study 2). The amalgam restorations made by these two dentists show corresponding percentages (15 percent) between the two studies (data not shown). Thus, the described influences interfere with a clear comparison of the results of both studies. Furthermore, in Study 2 more premolars were restored than in Study 1 and, given the fact that premolars show less sensitivity than molars in both studies, this may also be a disturbing factor in a comparison of results.

An explanation of the difference in the number of complaints can also be sought in the accuracy of recording. The objective of this study was to record pain complaints starting from the moment when the inlays were placed. Because there is a period here between the preparation of the cavity and placement of the inlays, it is possible that a number of complaints may already have been diminished before recording begins.

Table 3 □ Number of patients with 0, 1, 2, 3 or 4 sensitive teeth, classified by the total number of restorations studied per patient.

		Number of teeth with postoperative sensitivity					
		0	1	2	3	4	Totals
Restorations studied per patient	4	36	13	4	0	3	56
	8	0	2	0	0	0	2
Totals		36	15	4	0	3	58

Table 4 □ Number of teeth with postoperative sensitivity per dentist.

	Dentist			
	1	2	3	Totals
Restorations studied	80	80	80	240
Teeth showing postoperative sensitivity	8	5	22	35

It is doubtful, however, whether the patient can still remember accurately whether the complaints developed before the placement of the inlay (thus directly after preparation) or after placement; especially if the complaints were of short duration. Although the subjectivity of research into patients' experiences of pain cannot be ignored completely, the findings with regard to the selectivity of the group studied appear to be confirmed in the literature: there is little consensus about the degree of postoperative sensitivity after placement of resin composite restorations, and the findings vary from 0-40 percent.[1]

It is still discussable whether reduction of shrinkage from polymerization by the indirect technique does have any influence upon the appearance of postoperative sensitivity. The hypothesis that preparation and restoration (and thus the amount of tooth tissue removed) in general have more unfavorable effects upon the pulp than the restorative materials chosen is not negated by the results of this study. The fact that more complaints occur in molars than in premolars, and a tendency of three-surface restorations to show more sensitivity than two-surface restorations, may lend support to this hypothesis.

CONCLUSIONS

- ☐ In this study, no difference in postoperative complaints was found between teeth with resin composite inlays and those with amalgam restorations.
- ☐ Although resin composite inlays produced fewer complaints than direct resin composite restorations, no decision can be made on the quality of the difference, particularly when reference is made to the difference in sensitivity with amalgam controls between the two populations.
- ☐ The patient seems to be a major factor in whether postoperative sensitivity occurs or not.

REFERENCES

1. Borgmeijer, P.J.; Kreulen, C.M.; van Amerongen, W.E. *et al*: The prevalence of postoperative sensitivity in teeth restored with Class II composite resin restorations. J Dent Child, 58:378-383, September-October 1991.
2. Jensen, M.E. and Chan, D.C.N.: Polymerization shrinkage and microleakage. In: Vanherle, G. and Smith, D.C. (eds.): *Posterior composite resin dental restorative materials.* The Netherlands: P. Szulc, 1985, pp 243-262.
3. McCabe, J.F. and Kagi, S.: Mechanical properties of a composite inlay material following post-curing. Br Dent J, 171:246-248, October 1991.
4. Feilzer, A.J.; De Gee, A.J.; Davidson, C.L.: Increased wall-to-wall curing contraction in thin bonded resin layers. J Dent Res, 68:48-50, January 1989.
5. Davidson, C.L.; Van Zeghbroeck, L.; Feilzer, A.J.: Destructive stresses in adhesive luting cements. J Dent Res, 70:880-882, May 1991.
6. Kreulen, C.M.; van Amerongen, W.E.; Akerboom, H.B.M. *et al*: A clinical study on direct and indirect Class II posterior composite resin restorations: Design of the investigation. J Dent Child, 58:281-288, July-August 1991.

EXTENT OF CARIES UNDER SMALL OCCLUSAL CAVITIES

Many clinicians take for granted that occlusal caries which is just visible as a cavity is extended (deep) into the dentine. This study was aimed at quantifying this opinion. The study was carried out with 60 molars containing small but visible occlusal cavities. After taking bite-wing radiographs the crowns were separated from the roots and embedded. 700-μm sections were cut and glued on plastic sheets. X-ray pictures were taken of the sections, which were scored by independent examiners. The bitewings provided inaccurate estimates of the extent of caries. Radiographs of the sections revealed that about 25 percent of the molars had caries that reached just to the dentino-enamel junction, while the remaining 75 percent showed caries extending far into the dentine.

J.P. van Amerongen *et al*: An in vitro assessment of the extent of caries under small occlusal cavities.
Caries Res, 26: 89-93, March-April 1992.

The relationship between alveolar bone loss and proximal caries in children: prevalence and microbiology

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The main etiologic factors for periodontal diseases are bacteria or their products.[1,2] In addition, recent studies have emphasized that periodontal diseases may be related to individual and/or familial susceptibilities; and that there may be a connection between periodontal diseases in childhood and adulthood.[3-5] Alveolar bone loss (ABL) in children and adolescents may also be related to local factors, such as proximal caries, that allow for food impaction and plaque retention.[6] It has been noted, however, that not in every site with extensive proximal caries, alveolar bone loss takes place and that, in an unspecified percentage of the children in which ABL takes place, it appears in more than one site.[6] These findings indicate that some children may have an increased susceptibility to periodontal diseases and that the prevention, early diagnosis, and treatment of periodontal diseases in children and adolescents should be considered a major goal by the dental profession. The purpose of the present study, therefore, was to describe, by means of radiographic evaluation, the prevalence of alveolar bone loss in children in relation to caries, as well as loss of proximal contact and space. In addition microbiological aspects of sites with/without bone loss, with/without caries or probing depths smaller/equal or larger than 2.5 mm are presented.

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MATERIALS AND METHODS

Five hundred records with bite-wing radiographs of children who had no systemic disease and attended the Hadassah School of Dental Medicine in Jerusalem during the years of 1989-1990 were included in the study. Bite-wing radiographs were chosen on the basis of having minimal distortion, minimal overlapping of the proximal surfaces, and with a clear image of the alveolar bone. In the selected radiographs, the following variables were recorded:

- ☐ Presence of proximal caries.
- ☐ Loss of proximal contact due to caries.
- ☐ Loss of space due to loss of proximal contact.

Loss of space was noted in quadrants in which obvious overlap was evident between an imaginary line or lines which was/were drawn to complete missing tooth surface(s) with the adjacent tooth/teeth.

- ☐ The condition of the alveolar bone crest in the interdental areas.

Normal: The distance from the cementoenamel junction to the alveolar bone crest (CEJ-ABC distance) was <2 mm and the lamina dura was complete.

Questionable: The CEJ-ABC distance was between 2 to 3 mm and/or with partial loss of the lamina dura.

Bone loss: The CEJ-ABC distance was >3 mm and the lamina dura was completely absent.

These measurements were chosen, because previous studies indicate CEJ-ABC distances of >2 mm as indications of alveolar bone loss.[7-9] Furthermore, due to the limitations inherent in radiographic evaluation, ABL was considered only in cases where the CEJ-ABC distances were >3 mm and the lamina dura was completely absent.

With the purpose of examining the reliability of the radiographic examination, the radiographs of forty patients were examined on two different occasions. In addition, all the radiographs were first examined by one author and those in which the alveolar bone was rated as *questionable* or *with bone loss* were subjected to a second examination, by two authors, and only sites on which unanimous agreement was achieved were recorded as having alveolar bone loss.

Five children in which ABL was evident and who had not yet received dental treatment at the time of the study were available for microbial examination of the subgingival plaque. An attempt was made to obtain equal numbers of test and control sites; however, despite the fact that four samples were obtained from each child, because of the distribution of caries, probing depths and ABL, the samples included: ten adjacent to teeth with caries and ten adjacent to teeth with no caries; nine adjacent to probing depths of ≥2.5mm, and eleven with probing depths of <2.5 mm; thirteen adjacent to ABL and seven adjacent to normal alveolar bone. For the microbial examination, the supragingival plaque was removed and the teeth carefully dried. Three sterile paper points were then inserted for ten seconds to the base of the pocket, removed, and immediately inserted into a vial containing 1 ml of reduced transfer fluid (RTF).[10] The vials were then transferred to an anaerobic chamber with an atmosphere of 85 percent N_2, 10 percent H_2 and 5 percent CO_2, for further processing.[11] The samples were vortexed for sixty seconds, and then serially diluted in RTF (1:10, 1:100, 1:1000). Samples from each dilution were plated on enriched trypticase soy agar (ETSA) by means of an automatic spiral platter.[12] Duplicate plates from each sample were then incubated: for the assessment of the number of facultative organisms in an atmosphere of H_2 and CO_2 in a Gas Pack Jar (Gas Pack BBL Microbiology Systems, Becton Dickinson and Co., U.S.A.) at 37° C for forty-eight hours; and for the assessment of the anaerobic organisms in the anaerobic chamber for seven to ten days. Following incubation, the total number of colony forming units (CFU) were counted and the mean value from three plates for each bacterium calculated. In addition, colonies of black pigmented Bacteroides (BPB), and Actinobacillus actinomycetemcomitans (A.a.) were identified by means of colony morphology, biochemistry and selective media and counted.[13-15]

RESULTS

The examined population included 232 females and 268 males, no statistically significant difference was found in age between males and females (Student's t-test pт0.05) (Table 1). Comparison of the findings from the radiographs which were examined twice indicated that the classification of the alveolar bone was correct in 97 percent of the sites.

Table 1 □ Distribution of children by age and sex.

Age	3	4	5	6	7	8	9	10	11	12	All	mean	SD
Female	1	8	18	31	42	43	48	34	4	3	232	7.7	1.8*
Male	0	12	30	42	41	45	44	43	10	1	268	7.6	1.9*
Totals	1	20	48	73	83	88	92	77	14	4	500		

* No statistically significant difference (T = 0.8, p = 0.4)

Five years of age was the earliest at which alveolar bone loss was detected.

The prevalence of ABL by age is presented in Figure; the earliest age in which ABL was evident was five years, in three children (6.3 percent of the five-year-olds). A nonsignificant difference was found between the prevalences of ABL in males (n = 36, 13.4 percent) and females (n = 24, 10.3 percent) (Chi square, p = 0.3). In thirty-four children the ABL was evident only in the maxilla; in seventeen only in the mandible; and in nine children, ABL was evident in the maxilla and the mandible. Twenty seven children had ABL in more than one site (5.4 percent of the total population or 45 percent of those with ABL).

Alveolar bone loss was evident in ninety-nine sites; thirty-three between the primary canines and first primary molars (thirty-one in the maxilla); fifty-seven between both primary molars (twenty-six in the maxilla); and nine between the second primary and first permanent molars (three in the maxilla). Among the ninety-nine sites with ABL, 37.4 percent (n = 37) were adjacent to teeth with no proximal caries, 62.6 percent (n = 62) adjacent to proximal caries, 49.5 percent (n = 49) adjacent to contact loss due to proximal caries, and 40.4 percent (n = 40) adjacent to teeth with mesial drift due to contact loss. When all the sites examined are taken in consideration, ABL was evident in 4.9 percent of those with proximal caries (62 out of 1257), in 15.8 percent of those with contact loss (45 out of 284) and in 20.5 percent of those with mesial drift (49 out of 238).

The distribution of sites with ABL according to the location in the mouth and the presence of proximal caries is presented in Table 2; only between the maxillary canine and first primary molar was the prevalence higher in sites with no caries.

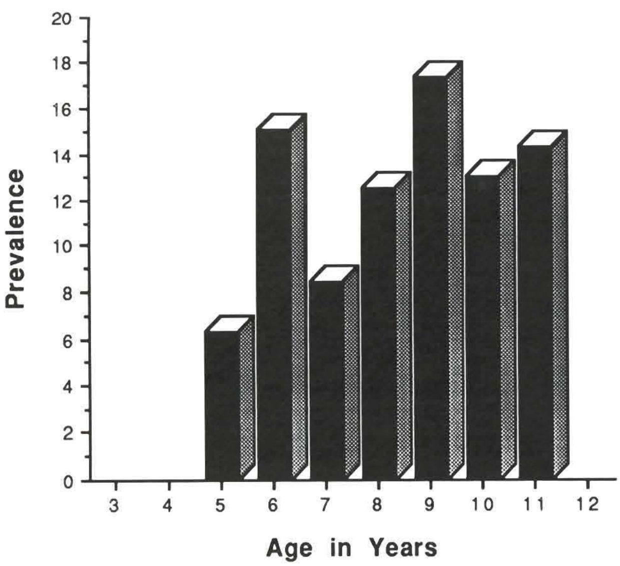

Figure. Prevalence of alveolar bone loss by age.

Anaerobic bacteria were found in every site examined. Furthermore, A.a. was found in seventeen sites and BPB in eight out of the twenty sites examined (Table 3). The differences between the mean values of CFU of A.a. or BPB percentages for sites with and without caries, or sites with and without ABL were not significant (Unpaired t-test, p > 0.05).

DISCUSSION

The prevalences by age of alveolar bone loss adjacent to the primary molars in the present study are similar to those previously reported for a different population.[6] From both studies it can be concluded that alveolar bone loss may be evident in early childhood and that

Table 2 ☐ Distribution of interproximal sites with alveolar bone loss by arch, presence or absence of proximal caries and type of teeth adjacent to them.

		Caries			
		With		Without	
Arch	Teeth	N	%	N	%
Maxilla	primary canine and first primary molar	10	32.3	21	67.7
	first and second primary molars	17	65.4	9	34.6
	second primary and first permanent molars	2	66.6	1	33.3
Mandible	primary canine and first primary molar	1	50.0	1	50.0
	first and secondary primary molars	29	93.5	2	6.5
	second primary and first permanent molars	3	50.0	3	50.0
	Totals	62	62.6	37	67.4

Table 3 ☐ Percentages of colony forming units of Actinobacillus actinomycetemcomitans (%Aa) and black pigmented Bacteroides (%BPB) from subgingival plaque adjacent to sites without (1) and with (2) proximal caries, probing depths of <2.5 mm (1) or ≤2.5 mm (2), normal alveolar bone (1) or alveolar bone loss (2).

Site	Patient	Caries	Probing	Bone	% Aa	% BPB
1	1	2	2	2	0.94	0.40
2	1	2	2	2	0.15	0.76
3	1	1	1	1	0	1.00
4	1	1	1	1	0	0
5	2	2	2	2	0.25	0.01
6	2	2	2	2	2.86	0
7	2	2	2	2	1.18	0
8	2	2	2	2	1.00	0
9	3	1	1	1	3.00	0.02
10	3	1	1	1	1.60	0.11
11	3	1	2	2	1.25	0.10
12	3	1	1	1	28.90	0
13	4	2	2	2	0	0
14	4	2	2	2	0.07	0
15	4	2	1	2	2.90	0
16	4	2	1	2	0.79	0.75
17	5	1	1	1	15.09	0
18	5	1	1	1	1.54	0
19	5	1	1	2	10.08	0
20	5	1	1	2	38.28	0

at certain ages, it may affect almost every fifth child attending dental clinics. It should be noted, however, that in both studies the majority of the sites with alveolar bone loss were adjacent to primary molars with proximal caries and, therefore, in the general population or in other populations with a lower caries rate, the prevalences of alveolar bone loss may be smaller.

Previous studies report prevalences of periodontitis in the first permanent molars of fourteen-to-fifteen-year-olds to be from 4.5 percent to 51.3 percent.[8,9,16,17] In the present study, among the 358 children, ages between seven and twelve years, only nine (2.5 percent) had radiographic evidence of alveolar bone loss between the second primary molars and the first permanent molars. The differences in the studies are probably related to the different diagnostic criteria and population characteristics, such as age.

The present study confirms previous data that in every site with proximal caries that facilitates food impaction, ABL does not necessarily occur and that, on the other hand, in a significant proportion of the children with ABL, the bone loss is evident in more than one site.[6] The etiology of these phenomena may be related to host characteristics and/or the microbial composition of dental plaque.[1-4] With the purpose to investigate the possible influence of the microbial composition of the subgingival plaque on the alveolar bone of the primary and mixed dentition, the present study examined the microbial composition of subgingival plaque from twenty sites adjacent to areas with or without proximal caries, probing depths of <2.5mm or ≥ 2.5 mm and normal or abnormal alveolar bone. A.a. and BPB were selected for examination, because these microorganisms have been incriminated as major pathogens in cases of prepubertal and juvenile periodontitis.[2-4,18-20]

Alaluusua and Aisikainen in a population of fifty-five healthy four to seven-year-old children were able to isolate A.a. in >1 site, but not in every site from the primary dentition of seven children (12.7 percent).[21] In most of the positive samples A.a. proportions were <1 percent and none exceeded 10 percent of the flora. In addition, Aisikainen *et al* in a two-year follow-up of eighty-seven teenagers recovered A.a. in four subjects (4.7 percent) at baseline and in five subjects (5.7 percent) two years later, three of them (3.4 percent) at both examinations.[22] In the present study, A.a. was found in every child or, in seventeen sites, regardless of the presence or absence of proximal caries, bone loss or probing depths. Its percent of CFU being 21 percent in eleven sites and ≥ 10 percent in four sites. One should take in consideration, however, that the present sample included only children who had at least one site affected with bone loss and that once A.a. is established, the bone loss site or the dorsum of the tongue may function as a nidus for these bacteria, which may spread to additional sites.[21] The fact that A.a. may be found both in areas with normal clinical appearance or areas with bone loss may be related to the pathogenic potential of A.a., which may increase due to phage infection, and or host related factors.[3,4,23]

Bailit *et al* found that 40 percent of the cultures from five-year-old children were positive to Bacteroides melaninogenicus and, that the presence of this microorganism increases with age were almost universal occurrences among adolescents.[24] On the other hand, Kelstrup did not find B. melaninogenicus in the sulci from primary or early mixed dentition.[25] In the present study, BPB were found in four out of the five children examined; in very small percentages, however, and in only eight of the twenty teeth. The differences be-

The majority of sites with alveolar bone loss were adjacent to primary molars with proximal caries.

tween the studies being probably the result of the different sampling methods and/or population characteristics.

The present study emphasizes the facts that alveolar bone loss may reach high prevalences in children and adolescents and, that some children may have an increased susceptibility to alveolar bone loss. In addition, this study confirms the presence of anaerobic bacteria in the subgingival plaque of children and adolescents; it failed to demonstrate, however, a relation between the values of CFU of A.a. and BPB and clinical factors, due probably to the small number of children and/or microbial samples.

REFERENCES

1. Schluger, S.; Youdelis, R.; Page, R.C. *et al*: Etiology of inflammatory periodontal disease. In *Periodontal disease*, 2nd edition. Philadelphia: Lea Febiger,1990, p 89.
2. Lindhe, J. and Slots, J.: Periodontal disease in children and young adults. In Lindhe J., ed. *Textbook of Clinical Periodontology*, 2nd edition. Copenhagen: Munksgaard, 1989, p 193.
3. Gunsolley, J.C.; Ranney, R.R.; Zambon, J.J. *et al*: Actinobacillus actinomycetemcomitans in families affected with periodontitis. J Periodontol, 61:643-648, October 1990.
4. Nishimura, F.; Nagai, A.; Kerimoto, K. *et al*: A family study of a mother and daughter with increased susceptibility to early-onset periodontitis: microbial immunological, host defensive and genetic analyses. J Periodontol, 61:755-765, December 1990.
5. Sjödin, B.; Crossner, C-G.; Unell, L. *et al*: A retrospective radiographic study of alveolar bone loss in the primary dentition in patients with localized juvenile periodontitis. J Clin Periodontol, 16:124-127, February 1989.
6. Bimstein, E.; Delaney, J.E.; Sweeney, E.A.: Radiographic assessment of the alveolar bone in children and adolescents. Pediatr Dent, 10:199-204, May-June 1988.
7. Bimstein, E. and Soskolne, A.W.: A radiographic study of interproximal alveolar bone crest between the primary molars in children. J Dent Child, 55:348-350, September-October 1988.
8. Gjermo, P.; Bellini, H.T.; Pereira Santos, V. *et al*: Prevalence of bone loss in a group of Brazilian teenagers assessed on bite wing radiographs. J Clin Periodontol, 11:104-113, February 1984.
9. Aass, A.M.; Albandar, J.; Aasenden, R. *et al*: Variation in prevalence of radiographic alveolar bone loss in subgroups of 14-year-old schoolchildren in Oslo. J Clin Periodontol, 15:130-133, February 1988.
10. Syed, S.A. and Loesche, W.J.: Survival of human dental plaque flora in various transport media. Appl Microbiol, 24:638-644, October 1972.
11. Aranki, A.; Syed, S.A., Kenney, E.B. *et al*: Isolation of anaerobic bacteria from gingiva and mouse caecum by means of a simplified glove box procedure. Appl Microbiol, 17:568-576, April 1969.
12. Jarvis, B.; Lach, V.H. and Wood, J.K.M.: Evaluation of the spiral plate marker for the enumeration of microorganisms in foods. J Appl Bacteriol, 43:149-157, 1977.
13. Laughon, B.E.; Syed, S.A.; Loesche, W.J.: Rapid identification of bacteroides gingivalis. J Clin Microbiol, 15:345-346, February 1982.
14. Slots, J.: Selective medium for isolation of Actinobacillus actinomycetemcomitans. J Clin Microbiol, 15:606-609, April 1982.
15. Hunt, D.E.; Jones, J.V.; Dowell, V.R.: Selective medium of the isolation of Bacteroides gingivalis. J Clin Microbiol, 23:441-445, 1986.
16. Hull, P.S.; Hillam, D.G.; Beal, J.F.: A radiographic study of the prevalence of chronic periodontitis in 14-year-old English schoolchildren. J Clin Periodontol, 2:203-210, April 1975.
17. Latcham, N.L.; Powell, R.N.; Jago, J.D. *et al*: A radiographic study of chronic periodontitis in 15 year old Queensland children. J Clin Periodontol, 10:37-45, January 1983.
18. Crossner, C.G., Carlsson, J.; Sjödin, B. *et al*: Periodontitis in the primary dentition associated with Actinobacillus actinomycetemcomitans infection and leukocyte dysfunction. A 3 1/2 year follow up. J Clin Periodontol, 17:264-267, April 1990.
19. Wanatabe, K.: Prepubertal periodontitis: a review of diagnostic criteria, pathogenesis and differential diagnosis. J Periodont Res, 25:31-48, January 1990.
20. Wilton, J.M.A.; Johnson, N.W.; Curtis, M.A. *et al*: Specific antibody responses to subgingival plaque bacteria aids to the diagnosis and prognosis of destructive periodontitis. J Clin Periodontol, 18:1-15, January 1991.
21. Alaluusua, S. and Asikainen, S.: Detection and distribution of Actinobacillus actinomycetemcomitans in the primary dentition. J Periodontol, 59:504-507, August 1988.
22. Asikainen, S.; Alaluusua, S.; Kleemola-Kujala, E.: A 2 year follow up on the clinical and microbiological conditions of periodontium in teenagers. J Clin Periodontol, 18:16-19, January 1991.
23. Preus, H.R.; Olsen, I.; Namork, E.: The presence of phage-infected Actinobacillus actinomycetemcomitans in localized juvenile periodontitis patients. J Clin Periodontol, 14: 605-609, November 1987.
25. Bailit, H.; Baldwin, D.C.; Hunt, E.E.: The increasing prevalence of gingival Bacteroides melaninogenicus with age in children. Arch Oral Biol, 9:435-438, July-August 1964.
26. Kelstrup, J.: The incidence of Bacteroides melaninogenicus in human gingival sulci, and its prevalence in the oral cavity at different ages. Periodontics, 4:14-18, January-February 1966.

Evaluation of mandibular infiltration versus block anesthesia in pediatric dentistry

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Historically, the anesthesia technique of choice for mandibular primary and permanent molars has been the inferior alveolar nerve block (mandibular block).[1,2] Infiltration anesthesia for the mandibular primary molars has been suggested based on personal experience or in studies evaluating the technique.[2-5] This latter study suggested that the infiltration technique should be investigated as a possible alternative to mandibular block anesthesia in young children.

The purpose of this study was to compare the clinical effectiveness of the mandibular infiltration versus the mandibular block techniques for the operative and surgical treatments of primary molars.

MATERIALS AND METHODS

Eighteen children ages six to nine years old who required bilateral identical dental treatment on mandibular primary molars were selected for this study. The bilaterally selected teeth for treatment had no more than one third root resorption evident. Informed consent was obtained from the parent or guardian for the child's participation in the study. The same operator performed all procedures without the parent or guardian present and was assisted by a trained dental assistant. Nitrous oxide was gradually introduced and when the desired effect of the nitrous oxide was achieved, anesthetic solution was administered to the patient using inferior alveolar nerve block or a supraperiosteal infiltration technique. A random number table was used to determine which side of the mouth was to receive the block or the infiltration technique and a coin toss determined which would be administered first. A standard technique for the inferior alveolar nerve block was used.[1]

For both the mandibular block and infiltration techniques, a short 27 gauge needle was used to inject a 2 percent lidocaine solution with 1:100,000 epinephrine. Topical anesthetic was applied before injection in both techniques. The lip and/or cheek was shaken to provide distraction. In the mandibular block technique, approximately 1.3 ml of anesthetic was placed near the inferior alveolar nerve, 0.5 ml as a long buccal infiltration distal to the permanent first molar or well distal to the second primary molar. In the infiltration technique, 0.4 ml of the anesthetic solution was placed in the bottom of the sulcus and 0.2 ml. in the mesial and distal papillas of the primary tooth being treated (Figure) until blanching of the lingual tissue was observed. This made a total of 0.8 ml injected. The patient was

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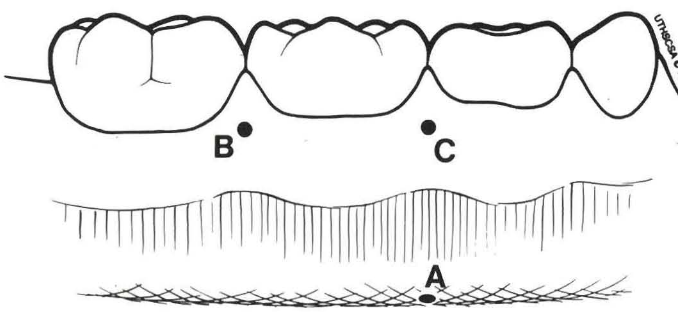

Figure. Infiltration sites. A. Bottom of sulcus; B. Distal papilla; C. Mesial papilla.

Table ☐ Comparison of the mandibular block *versus* the infiltration technique.

Factor evaluated	Mean (S.D.)	
Response to injection	Dentist	Patient
Block	2.61 (2.38)	3.72 (3.21)
Infiltration	3.72 (3.39)	3.17 (2.77)
P value	0.191*	0.525*
Response to procedure	Dentist	Patient
Block	1.67 (2.20)	3.39 (3.68)
Infiltration	1.33 (2.09)	2.44 (3.01)
P value	0.547*	0.101*

* Not significant

re-oxygenated with 100 percent oxygen for five minutes. Bilateral identical procedures were performed on each patient in the study during the same appointment. These procedures included amalgam restorations, stainless steel crowns, pulp therapy, and extractions. A rubber dam and Molt bite block were used for restorative procedures.

Upon completion of treatment and prior to dismissal, each patient was asked to rate the level of discomfort (pain) for the injection as well as for the procedure performed. The questionnaire consisted of a numbered scale and instructions were explained to the patient by the dental assistant. The operator rated the patient's exhibited level of discomfort for the injection and the procedure, independent of the patient. The operator based his ratings on patient cooperation, facial and body language, vocalization of the patient during the injection and procedure, and tearing of the patient's eyes. All patients were age six to nine years old with the cognitive skills to understand what was asked of them. Patients exhibiting obvious anxiety or uncooperative behavior were not included in this study.

The following factors were evaluated:

☐ Dentist's response to injection.
☐ Dentist's response to clinical procedure.
☐ Patient's response to injection.
☐ Patient's response to work.

Statistical evaluation of the data was performed using a paired t-test.

RESULTS

The paired t-test indicated there was no significant difference between the block and infiltration techniques for any of the factors evaluated (Table).

DISCUSSION

This study confirms a previous one by Garcia-Godoy demonstrating that satisfactory anesthesia for the primary mandibular teeth of children can be obtained with the infiltration technique.[5]

Satisfactory anesthesia is obtainable using the infiltration technique, with fewer risks.

The anesthesia obtained in children with the mandibular infiltration technique could be due to the greater porosity of the cortical layer of bone in young children permitting diffusion of the anesthetic solution through the bone.[3]

The infiltration technique described in this study is not a mental nerve block, because in small children the mental foramen is located near the apex of the mesial root of the first primary molar and the infiltration technique injects between the two primary molars and the mesial and distal papillas. The infiltration of the papillas to the lingual area would anesthetize the lingual nerve filaments. With the mandibular block, many times the lingual nerve is not anesthetized and a lingual infiltration is required to supplement the block.[3]

The infiltration technique could also be considered useful in hemophiliacs without replacement of deficient factor.[6] In these patients, cervical hematomas and death have occurred after mandibular block anesthesia.[7]

One of the most common complications following mandibular block anesthesia is trauma to the soft tissue caused by biting the anesthetized lip, tongue and/or inner surface of the cheek.[1] With the mandibular infiltration technique, the lip is not anesthetized and fewer incidences of self-inflicted soft tissue trauma, therefore, should be expected. Because of the localized effect of the infiltration technique, bilateral dental work may be completed in one appointment, reducing the amount of anesthetic solution, and a decreased risk of self-inflicted injuries.

REFERENCES

1. McDonald, R.E. and Avery, D.R.: *Dentistry for the child and adolescent.* 5th ed. St. Louis: C.V. Mosby Co., 1985, pp 305-306.
2. Kisby, L.: Pain control in pedodontics. In White, G.E. *Clinical oral pediatrics.* Chicago: Quintessence Pub. Co., 1981, p 207.
3. Northrop, P.M.: Practical techniques in administration of local anesthetic agents: II. Questions and answers. J Am Dent Assoc, 38:444-448, April 1949.
4. Shiere, F.R.: Oral anesthesia for children. J Am Dent Assoc, 41:414-418, October 1950.
5. Garcia-Godoy, F.M.: A simplified local anesthetic technique for mandibular primary molars. Acta Odontol Pediatr, 3:53-56, December 1982.
6. Steinle, C.J. and Kisker, C.T.: Pediatric dentistry for the child with hemophilia. New Eng J Med, 283:1325-1326, December 1970.
7. Archer, W.H. and Zubrow, H.J.: Fatal hemorrhage following regional anesthesia for operative dentistry in hemophiliac. Oral Surg, 7:464-470, April 1954.

UPTAKE AND CLEARANCE OF FLUORIDE FROM THE BUCCAL MUCOSA

It is well established that the salivary clearance of topically applied fluoride follows a multiexponential curve. The initial rapid decline in fluoride concentration has been attributed to swallowing and dilution by freshly secreted saliva [Dawes, 1983; Dawes and Weatherell, 1990], but those factors which influence the other components of the curve remain obscure. It has been suggested that one important aspect may be the existence of some form of intraoral fluoride store [Duckworth et al., 1987; White and Nancollas, 1990] which is replenished when the fluoride concentration in saliva is high and depleted as the concentration falls. It has also been postulated that the relatively high fluoride levels found in plaque may fulfil this function or, alternatively, that CaF_2 associated with the dental hard tissues may release fluoride into saliva when the conditions are favourable [Øgaard et al., 1983]. The possibility also exists, however, that fluoride is absorbed/adsorbed by the oral soft tissues, particularly the non-keratinized mucosa, and subsequently released over an extended period.

Jacobson, A.P.M. *et al*: Fluoride uptake and clearance from the buccal mucosa following mouthrinsing.
Caries Res, 26:56-58, January-February 1992.

Electrosurgical pulpotomy: A retrospective human study

Ronald B. Mack, DDS
Jeffrey A. Dean, DDS, MSD

Maintenance of almost all primary teeth infected by caries or subjected to traumatic injury is possible through the use of pulp therapy. The primary indication for the pulpotomy procedure in primary teeth is when the infected coronal tissue can be amputated and the remaining radicular tissue is judged to be vital, or affected but still vital. The 1991 American Academy of Pediatric Dentistry (AAPD) Guidelines for Pulp Therapy for Primary and Young Permanent Teeth describe the pulpotomy procedure in primary teeth as the amputation of the coronal portion of the affected or infected dental pulp, preserving the vitality and function of all or part of the remaining radicular portion of the pulp.[1] Evidence of success in therapy includes:

- ☐ Vitality of the majority of the radicular pulp.
- ☐ No prolonged adverse clinical signs or symptoms such as prolonged sensitivity, pain, or swelling.
- ☐ No radiographic evidence of internal resorption or abnormal canal calcification.
- ☐ No breakdown of periradicular supporting tissues.
- ☐ No harm to succedaneous teeth.

Many pharmacotherapeutic agents have been employed to achieve the above criteria, when performing pulpotomies on primary teeth. Formocresol has been a popular agent of choice for use in the pulpotomy procedure since it was advocated by Sweet in 1930, mainly because of its ease in use and excellent clinical success.[2] Yet, despite its excellent clinical success rate, the formocresol pulpotomy technique has come under close scrutiny because of safety considerations.[3-7] Concerns regarding the systemic distribution (i.e. liver and kidney) of this agent and its potential for toxicity, allergenicity, carcinogenicity, and mutagenicity have led to additional research investigations into possible alternatives. Other medicaments, such as glutaraldehyde, calcium hydroxide, collagen, and ferric sulfate have been suggested as possible replacements for formocresol. Success rates have varied with the agent used and with the particular study; several medicaments contributed to favorable results. Concerns regarding the safety of these materials, however, continue. It is clear that additional research on the use of these and other pharmacotherapeutic agents is required.

In addition to the techniques mentioned above, nonpharmacologic hemostatic techniques have been suggested for the pulpotomy procedure, such as electrosurgery and laser therapy.[8] Research on both of these techniques is sparse. Laws, in 1957, described the use of electro-coagulation on the pulps of permanent teeth.[9] In 1975, Yakushiji suggested that the elec-

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The authors would like to acknowledge the assistance of Rosario H. Potter, DMD, MSD, MS for her help in the statistical analysis of the data. Dr. Potter is Director of Dental Biometry and Professor of Oral Facial Genetics, Indiana University School of Dentistry, Indianapolis, Indiana.

trosurgical pulpotomy can be valuable, particularly when the current of the "electrotome" is at a low output current and of brief duration.[10] In 1982, Anderman describes the electrosurgical pulpotomy in primary teeth as a smooth and time-efficient method that is relatively free from postoperative complications.[11]

In 1983, Ruemping *et al*, compared the results of electrosurgical versus formocresol pulpotomies in a primate study.[12] The results of the study demonstrated that histologically the electrosurgically treated teeth responded as favorably as the teeth treated with conventional formocresol during the two-month postoperative period. In contrast, Shulman *et al* found that the electrosurgical technique used in their study produced pathologic root resorption and periapical/furcal morbidity.[13] Perhaps the main difference between the two studies was that Ruemping *et al* amputated the coronal pulp mechanically and then treated the remaining pulpal stumps electrosurgically. Shulman *et al* used electrosurgery to remove the coronal pulp tissue and to treat the pulpal stumps. Problems with excessive production of heat and electricity probably hampered Shulman's results.

Sheller and Morton (1987), used eleven human caries-free primary canine teeth to study the effects of the electrosurgery pulpotomy technique.[14] Clinical and radiographic success 100 days postoperatively was obtained with ten teeth, while only seven teeth exhibited histological success. The results showed a variable pulpal response based apparently upon the amount of normal exfoliative apical root resorption. Also in 1987, Shaw *et al* published a study comparing the conventional formocresol pulpotomy technique with the electrosurgical pulpotomy technique in the primary teeth of five monkeys. Histologic results indicated that the techniques produced comparable tissue responses. Although the results did not show the electrosurgical technique to be superior to the formocresol technique, the primary advantages of electrosurgery are that it can be performed quickly and that there are no pharmacotherapeutic agents involved that may produce undesirable local and systemic effects. Shaw *et al* suggested that well-documented human studies should be the next step in evaluating the electrosurgical technique.

PURPOSE

The purpose of this study was to observe retrospectively the clinical and radiographic results of the electrosurgical pulpotomy technique used on human primary molar teeth requiring pulp therapy secondary to carious involvement, in a private practice setting. The hypothesis of this study was that long-term clinical and radiographic success rates for the electrosurgical pulpotomy technique in human primary molars are comparable to the clinical and radiographic success rates previously reported for the formocresol pulpotomy technique.

METHODS AND MATERIALS

In order to perform this retrospective study, a private pediatric dental practice engaged in the use of the electrosurgical (specifically, electrofulguration) pulpotomy procedures on primary teeth for over twenty-five years was used as the source of patient records. The criteria for selecting the study sample were based on the following:

- ☐ All cases were primary molars that required a pulpotomy, because of pulpal exposure to caries, and were treated by the same operator (the first author of this study).
- ☐ All case reports included bitewing and/or periradicular radiographs of the treated tooth taken at least a month postoperatively (to avoid evaluating teeth with only an immediate postoperative radiograph). The bitewing radiographs must have shown at least a furcal view of the treated tooth.
- ☐ The cases used were patients in the 1 to 10-year age-group, focusing on the younger patients to allow for longer periods of evaluation of the pulpotomized primary molars.
- ☐ Otherwise, these charts were selected on an "as come basis, starting with patients seen ten years ago to the present.

While the exact electrosurgical technique used by the operator varied slightly over the period of time that this study covers, the basic technique, using the Hyfrecator® Instrument* electrosurgical unit is described as follows (Figure 1): Following profound local anesthesia, quadrant rubber dam isolation, and occlusal reduction with a high-speed bur (#37), caries was removed with a large slow-speed round bur (#6). If pulpal exposure occurred, the roof of the coronal pulp chamber was removed with a high-speed bur (#1558). Following this, the coronal pulp was amputated with hand instruments and/or with the previously mentioned large slow-speed round bur. A series of large, sterile cotton pellets were placed into the chamber with pressure to obtain

*Birtcher Medical Systems, 50 Technology Drive, Irvine, California 92718. 1-800-888-1771.

Figure 1. Mirror image clinical photographs of the electrosurgical pulpotomy technique on a mandibular left first primary molar (#L).

Figure 1a. The anesthetized and isolated tooth.

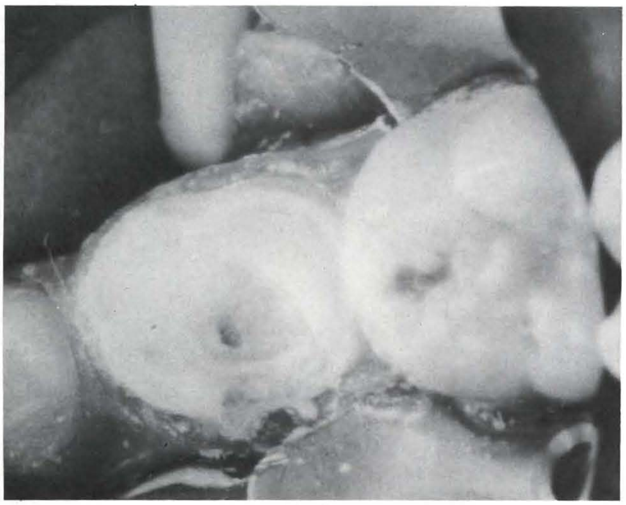

Figure 1b. Following caries removal, the carious pulp exposure is noted.

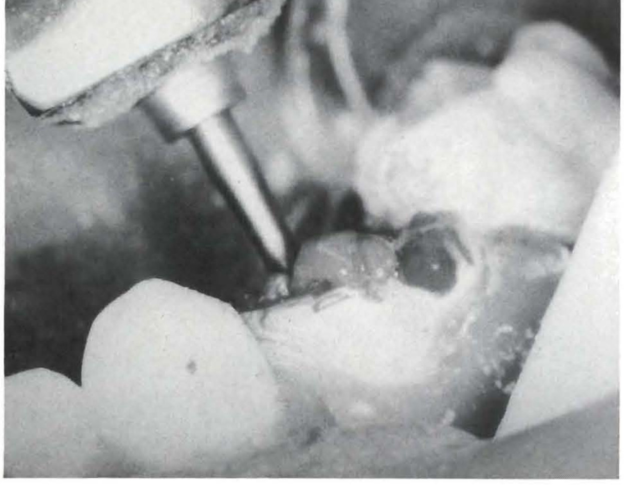

Figure 1c. The coronal pulp tissue is mechanically removed.

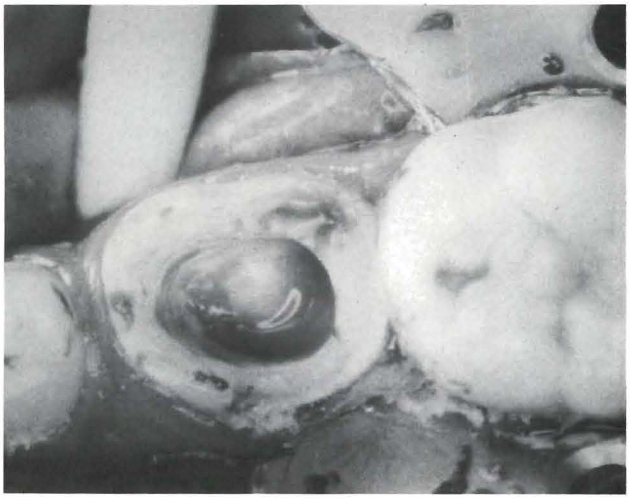

Figure 1d. Note fluids in coronal pulp chamber following tissue removal.

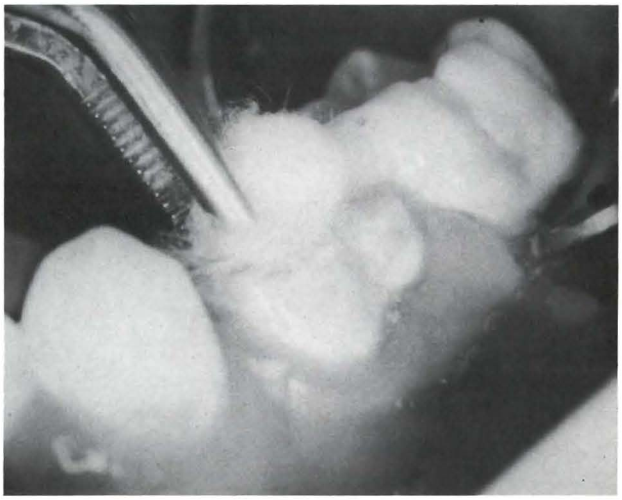

Figure 1e. Cotton pellets are used to dry the chamber.

Figure 1f. The dry chamber immediately prior to electrosurgery.

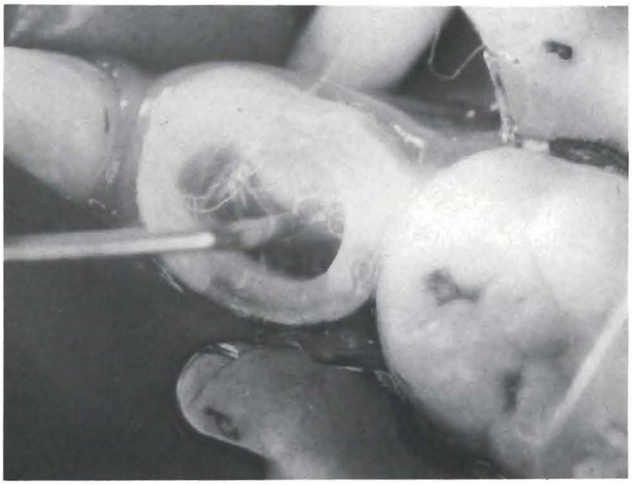

Figure 1g. The electrode positioned over the distal pulpal stump.

Figure 1h. Extraoral photograph demonstrating the electrical arc typically seen in this pulpotomy technique.

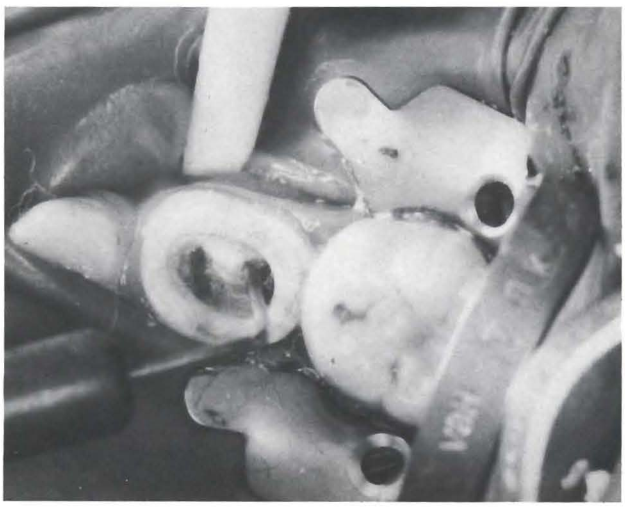

Figure 1i. Second application of electrosurgical energy to the pulp stumps (only when necessary).

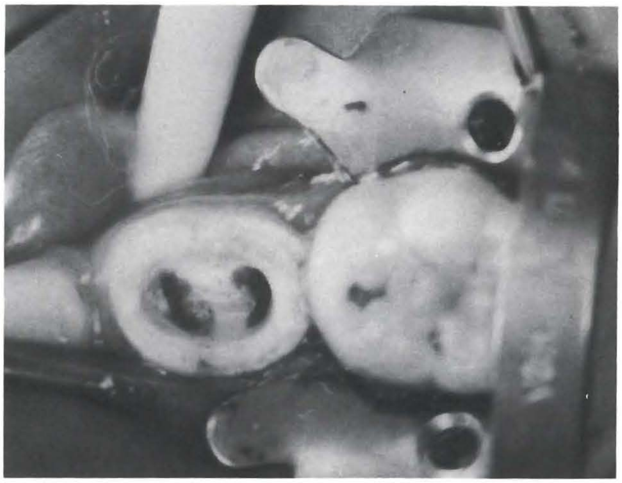

Figure 1j. Pulp stumps after the electrosurgical technique is completed.

Figure 1k. Zinc oxide-eugenol placement.

Figure 1l. Stainless steel crown cemented.

temporary hemostasis. The cotton pellets were then quickly removed and the Hyfrecator® 705A* electrofulguration dental electrode was immediately placed slightly above the tissue (i.e. 1-2 mm). The Hyfrecator® was set at 40 percent power (e.g. 12 watts) using the "high" port only. The electrical arc was allowed to bridge the gap to the first pulpal stump for 1 second followed by a cool-down period of 5 seconds. Heat and electrical transfer were thus minimized by keeping the electrode as far away from the pulpal stumps and tooth structure and still allow electrical arcing to occur. This procedure was generally only repeated up to a maximum of three times at each pulpal orifice. Single current applications of 1 second each were made to each orifice in a rotational sequence to avoid heat build-up in any one area of the tooth. After each and every current application, a new, large sterile pellet was placed with pressure on the next pulpal orifice to be electrosurgically treated, to absorb any blood or tissue fluids before the next current application (e.g. pellet-electrode-pellet-electrode, etc.) When properly completed the pulpal stumps appeared dry and completely blackened. A reinforced zinc oxide-eugenol dressing (i.e. IRM® **) was then placed directly on the radicular pulpal stumps and in the coronal pulp space. The interproximal surfaces were then prepared and the tooth was beveled and rounded with a high-speed bur (#699). The tooth was then restored with a stainless steel crown.

Clinical and radiographic evaluation of the success or failure of the electrosurgical pulpotomy procedure was based upon the following:

Success:

- ☐ Maintenance of the treated teeth until normal, physiologic exfoliation of the tooth as judged by the patient' s age and impending eruption of the succedaneous tooth.

and/or

- ☐ Lack of radiographically evident pathologic internal or external root resorption, periapical or furcal radiolucency.

Failure:

- ☐ Premature loss or extraction of the treated tooth, before the normal exfoliation time, secondary to pathologic root and/or bone resorption.

and/or

- ☐ Radiographic evidence of pathologic internal or external root resorption, periapical or furcal radiolucency.

**The LD Caulk Division, Dentsply International, Inc., Milford, Delaware 19963.

RESULTS

Over 4,000 files of patients seen in this practice within the last ten years were screened. Charts of 101 patients having received one or more primary molar pulpotomies, for a total of 164 pulpotomies, were reviewed for success of the treatment. The youngest patient at the time of treatment was eighteen months and the oldest was ten years, six months, with a mean treatment age of five years, eleven months. The postoperative observation time ranged from one month to five years, ten months with a mean postoperative observation time of two years, three months. Fifty-four of the 164 teeth had a postoperative observation time of thirty-six months or longer (Table 1).

Of the 164 teeth studied, 127 were normal at the last observation visit, thirty-two had undergone normal exfoliation, four had an abnormality associated with the pulpotomized tooth, (either pre- or posttreatment), but not considered a failure, and one was considered a failure. One failure out of 164 pulpotomized teeth represents a clinical and radiographic success rate of 99.4 percent (Table 2). Radiographs of a "normal" case at the last visit are shown in Figure 2 and a case having undergone normal exfoliation is shown in Figure 3.

Table 1 ☐ Age at time of treatment and post-op observation time.

Mean age at time of treatment	70.8	months
Age range at time of treatment	18 to 126	months
Mean post-op observation time	26.5	months
Range of post-op observation time	1 to 70	months
Number of teeth with 3 years or more post observation time	54	teeth

Table 2 ☐ Clinical and radiographic result categories.

Normal up to exfoliation	32	Success rate = 99.4%
Normal at last observation	127	
Radiographic abnormality	4	
Failure	1	Failure rate = 0.6%
Total teeth evaluated	164	

DISCUSSION

Many different studies have attempted to quantify the success rates of formocresol pulpotomies. The 1986 retrospective study by Hicks *et al*, is used here to compare statistically the success rates of the formocresol pulpotomy technique to the results of this electrosurgical pulpotomy study.[16] It was selected because of several similarities between the studies and its relatively high rate of success and length of postoperative obser-

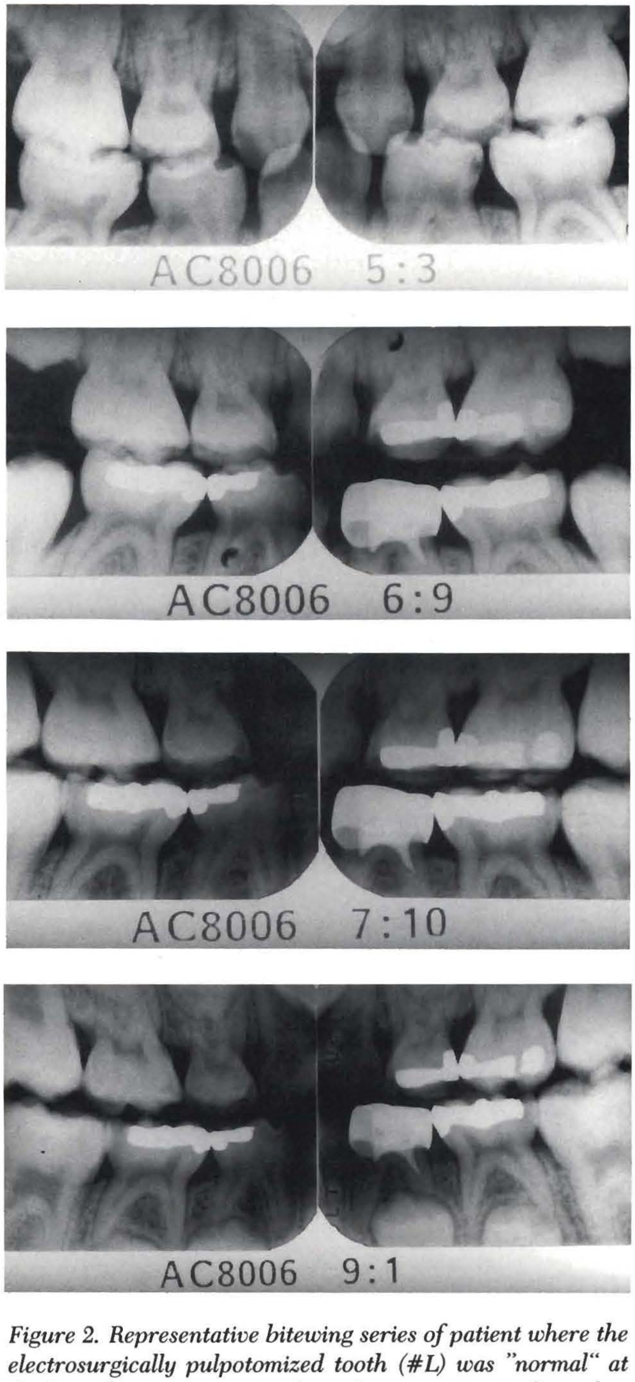

Figure 2. Representative bitewing series of patient where the electrosurgically pulpotomized tooth (#L) was "normal" at the last observation visit. The pulpotomy was performed at age 5 years, 3 months (a); and postoperative radiographs were taken at age 6 years, 9 months (b); 7 years, 10 months (c); and 9 years, 1 month (d).

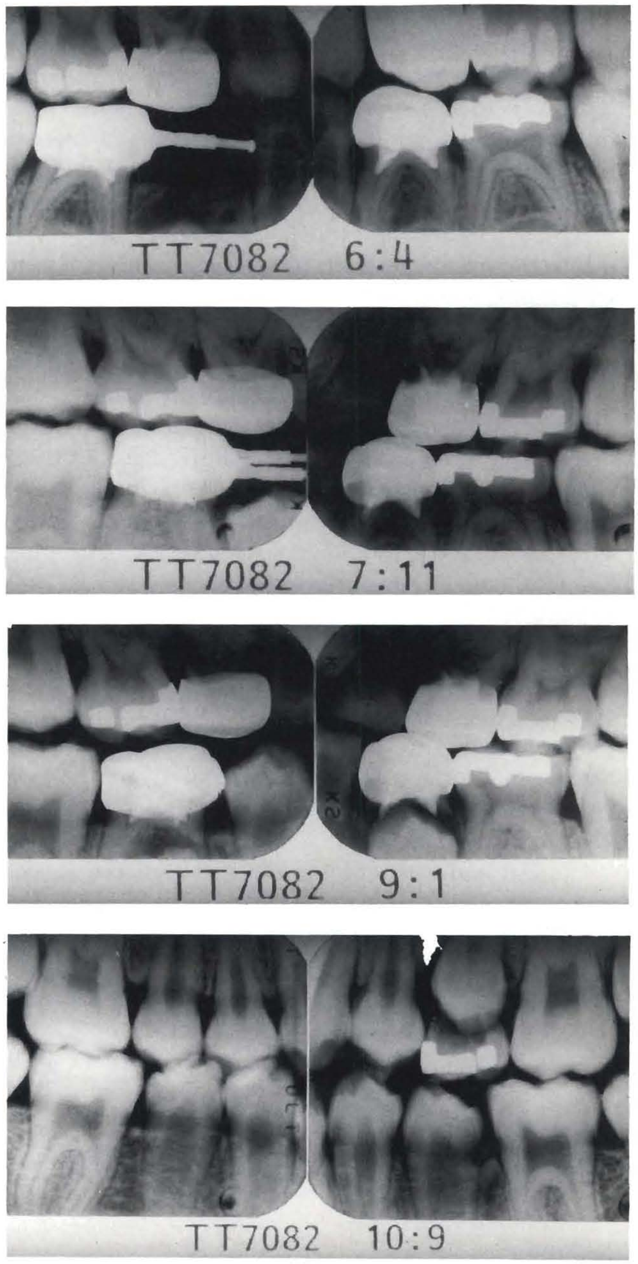

Figure 3. Representative bitewing series of patient where electrosurgically pulpotomized teeth (#I, L and T) were followed to exfoliation. All 3 teeth were treated with a pulpotomy at age 5 years, 8 months. Postoperative radiographs were taken at age 6 years, 4 months (a); 7 years, 11 months (b); 9 years, 1 month (c); and 10 years, 9 months (d).

Figure 4a. Tooth (#T) with pre-treatment furcal bone rarefaction.

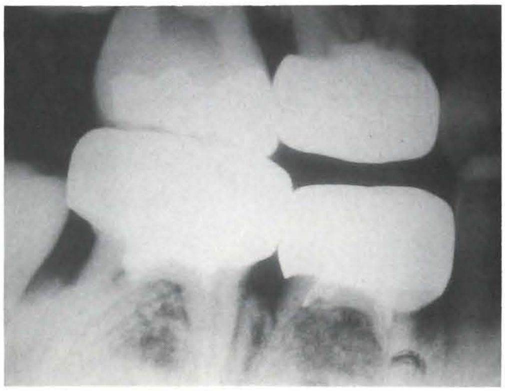

Figure 4c. Tooth (#S) with apical root replacement with bone.

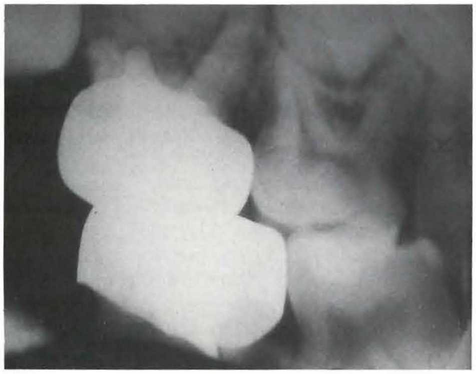

Figure 4b. Tooth (#A) with resorption of the distal root.

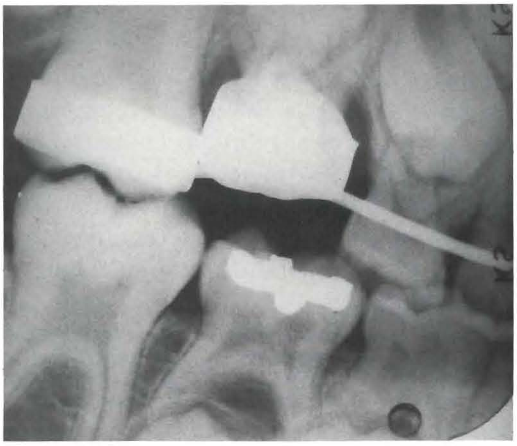

Figure 4d. Tooth (#A) with ankylosis.

Figure 4. Radiographs of the four electrosurgically pulpotomized teeth with an associated abnormality.

vation time. While there are remarkable similarities between Hicks' study and this study, it is important to note that there was no collaboration between the two groups. Their retrospective study of 164 formocresol pulpotomized primary molars showed a success rate of 93.9 percent (154 out of 164) as compared to this retrospective study of 164 electrosurgically pulpotomized teeth with a success rate of 99.4 percent. This is a statistically significant difference between the two studies at the $p < 0.01$ level. The Binomial Test and Fisher's Exact Probability Test were used in this analysis.

Acceleration of the exfoliative process following formocresol pulpotomy treatment has been noted previously and speculated to be a result of the formocresol. This pattern of early exfoliation was found, however, in this present study as well. Some other mechanism other than the action of formocresol must play a role in accelerating this process.

Radiographs of the four teeth with an abnormality associated with the pulpotomized tooth are shown in Figure 4. One of the four teeth with an associated radiographic abnormality had pretreatment furcal bone rarefaction. Despite the fact that the traditional treatment choice for this tooth was a pulpectomy, it was nonetheless treated with a pulpotomy and responded well. The remaining three teeth exhibited distal root resorption (possibly secondary to ectopic eruption of a maxillary first permanent molar), apical root replacement with bone, and ankylosis. Despite these radiographic findings, none of the four teeth exhibited any

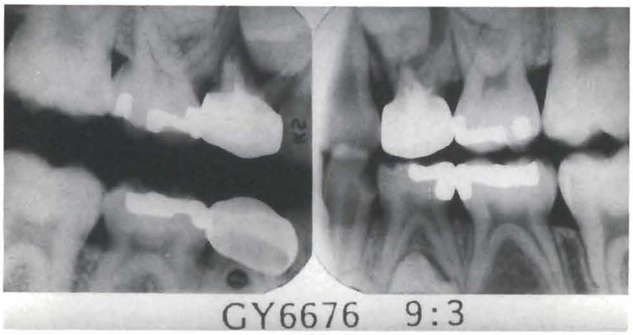

Figure 5. Radiograph of the electrosurgically pulpotomized tooth (#B) considered a failure secondary to the periradicular radiolucency.

clinical signs or symptoms of morbidity that could be ascertained from the patient file. These four teeth were singled out because of the difficulty in categorizing them as either a success or a failure for the purposes of this study. Certainly, they cannot be described as simply as the 159 healthy pulpotomized teeth previously discussed; however, they are not failures either. Based on the above, it was decided to consider these pulpotomies successful.

Radiographs of the one case considered a failure are shown in Figure 5. This case did not exhibit any clinical signs or symptoms of morbidity and the bone resorption could have been associated with the eruption of the succedaneous premolar. The contralateral premolar does not exhibit, however, this extent of eruption and bone resorption. Based on this and since only one radiograph was available for review of this tooth, the pulpotomy was considered a failure.

As with all retrospective studies, this study has its limitations. For instance, minor alterations of the electrosurgical pulpotomy technique probably occurred over the long time period that these teeth were treated. A plastic radiographic positioning instrument was occasionally used in taking the bitewings. Unfortunately, this decreased the view of the furcal area in some instances. In addition, the selection criteria for each tooth that was chosen to receive a pulpotomy may have varied. The total number of teeth treated, the lengthy observation periods, and the high clinical and radiographic success rates, however, make the results of this study noteworthy. The relatively benign nature of electrosurgical pulpotomy treatment as compared to pharmacotherapeutic pulpotomy procedures is encouraging. Long-term, prospective human clinical studies are now required to confirm the optimistic results of this study.

CONCLUSIONS

The following conclusions can be drawn from this study:

- ☐ For this retrospective study, a 99.4 percent (163 out of 164) clinical and radiographic success rate was observed for primary molars undergoing electrosurgical pulpotomy.
- ☐ When compared to a formocresol pulpotomy study of similar design, the clinical and radiographic success rate for the electrosurgical pulpotomy procedure in this study is significantly higher ($p < 0.01$).

REFERENCES

1. Guidelines for Pulp Therapy for Primary and Young Permanent Teeth. American Academy of Pediatric Dentistry Reference Manual 53-56, 1991-1992.
2. Sweet, C.A.: Procedure for treatment of exposed and pulpless deciduous teeth. J Am Dent Assoc, 17:1150-1153, June 1930.
3. Ketley, and Goodman, Jr., C.E.: Formocresol toxicity: is there a suitable alternative for pulpotomy of primary molars? Int J of Paediatric Dent, 2:67-72, August 1991.
4. Judd, P.L. and Kenny, D.J.: Formocresol concerns, a review. J Cana Dent Assoc, 5:401-404, May 1987.
5. Sipes, R. and Binkley, C.J.: The use of formocresol in dentistry: a review of the literature. Quint Internation, 17:415-417, July 1986.
6. Ranly, D.M. and Garcia-Godoy, F.: Reviewing pulp treatment for primary teeth. J Am Dent Assoc, 122:83-85, September 1991.
7. Avram, D.C. and Pulver, F.: Pulpotomy medicaments for vital primary teeth. J Dent Child, 56:426-434, November-December 1989.
8. Udin, R.D.: The formocresol pulpotomy revisited: looking at alternatives. Calif Dent Assoc J, 19:27-34, September 1991.
9. Laws, A.J.: Pulpotomy by electro-coagulation. New Zealand Dent J, 53:68-70, April 1957.
10. Yakushiji, J.: Pulpotomy of primary teeth by means of electrosurgery. Shoni Shikagaku Zasshi, 13:213-219, 1975.
11 Anderman, I.: Indications for use of electrosurgery in pedodontics, Symposium on Electrosurgery, Dent Clin N Am, 26:711-728, October 1982.
12. Ruemping, D.R.; Morton, T.H.; Anderson, M.W.: Electrosurgical pulpotomy in primates - a comparison with formocresol pulpotomy. Pediatr Dent, 5:14-18, March 1983.
13. Shulman, E.R.; McIver, F.T.l; Burkes, E.J.: Comparison of electrosurgery and formocresol as pulpotomy techniques in monkey primary teeth. Pediatr Dent, 9:189-194, September 1987.
14. Sheller, B. and Morton, T.H.: Electrosurgical pulpotomy: a pilot study in humans. J Endo, 13:69-76, February 1987.
15. Shaw, D.W.; Sheller, B.; Barrus, B.D. *et al*: Electrosurgical pulpotomy - a 6-month study in primates. J Endo, 13:500-505, October 1987.
16. Hicks, M.J.; Barr, E.S.; Flaitz, C.M.: Formocresol pulpotomies in primary molars: a radiographic study in a pediatric dentistry practice. J Pedodont 10:331-339, Summer 1986.

REVIEW

A review of fluoride intake from fluoride dentifrice

Steven M. Levy, DDS, MPH

There has been substantial professional discussion in recent years about the appropriate use of fluorides, with emphasis on achieving substantial dental caries prevention while minimizing risks of dental fluorosis. Important aspects of the subject were addressed at several recent international meetings:

- A joint IADR/ORCA International Symposium on Fluorides held in Georgia in 1989.[1]
- An American Association of Public Health Dentistry Symposium on Appropriate Use of Fluorides held in Boston in 1990[2]
- A National Institute of Dental Research sponsored Workshop on Changing Patterns of Fluoride Intake, in Chapel Hill, North Carolina in 1991.[3]
- A Canadian conference for Evaluation of Current Recommendations Concerning Fluorides, in Toronto in 1992.[4]

The U.S. Department of Health and Human Services' 1991 *Review of Fluoride: Benefits and Risks* also reviewed this subject and emphasized the need for increased attention regarding the "prudent health practice of using no more (fluoride) than the amount necessary to achieve a desired effect" and the need to "avoid excessive and inappropriate fluoride exposure."[5] It also recommended that: "Parents should educate young children to minimize swallowing of fluoridated toothpaste and to use only small amounts of toothpaste on the brush."

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The purpose of this paper is to review in detail the existing literature concerning fluoride intake from fluoride dentifrice among young children, in order to provide the practitioner with a better understanding of the importance of avoiding excessive ingestion of toothpaste by young children.

With use of fluoride dentifrice almost universal in the United States and Canada, fluoride ingestion from dentifrice is an important consideration.[6] Several studies have explored usage and ingestion of fluoride from dentifrice among diverse age-groups, but with various methodologies, often under artificial conditions.[7] Despite these limitations, the studies established that ingestion of fluoride from toothpaste is common and often substantial.[7] Most mothers start brushing their children's teeth with toothpaste at a young age. Because fluoride dentifrices available in the United States and Canada generally have 1000-1100 ppm fluoride, about 1.0 mg of fluoride is being used (or ingested) with each g of dentifrice used (or ingested).[7] Extra-Strength Aim® contains 1500 ppm fluoride and is available in the United States. A full strip of dentifrice covering an adult-sized toothbrush is often more than 1.0 g of dentifrice, while a strip covering a child-sized brush is approximately 0.75 to 1.0 g.

Relatively few studies have reported toothbrushing habits and toothpaste use among preschool children. The studies with younger preschool children are presented here in the most detail, because they are most important in our consideration of risks of fluorosis. Although there is increasing evidence for the importance of the post-secretory or maturational state of enamel formation, the most critical period of fluorosis risk for the esthetically important maxillary central incisors is believed to be about 20 to 28 months.[8-14]

REVIEW OF THE LITERATURE

Palmer and Prothero reported that 72 percent of British seventeen-month-olds and 98 percent of three-year-olds used fluoridated toothpaste with the majority starting by twelve months of age.[15] Among those using toothpaste, 29 percent of seventeen-month-old children and 55 percent of the three-year-old children reportedly brushed at least twice daily. Twenty-three percent of the parents of three-year-olds reported that their children swallowed most of the toothpaste and 24 percent reported that their children ate toothpaste at other times.

Dowell interviewed 115 British mothers concerning initiation of use of toothpaste with their three-year-olds.[16] All children had begun toothbrushing and 96.5 percent were reportedly brushing at least once per day, with about half of the children brushing twice per day or more. Forty-one percent reported starting to use toothpaste by twelve months of age and 74 percent had begun by eighteen months. Eighty-eight percent of mothers reported that children liked the taste of the toothpaste and 8 percent made unsolicited comments about the children sometimes eating toothpaste directly from the tube.

Several other recent studies also showed substantial toothbrushing and use of fluoride dentifrice among those younger than age three. In a Norwegian study, 83 percent of mothers of children eighteen to thirty-six months old reported regularly brushing the children's teeth.[17] In a retrospective study in the Netherlands, 70 percent of eighty-three children were reported to have used fluoride toothpaste at least twice daily.[18] Szpunar and Burt found that more than 50 percent of six- to twelve-year-old Michigan children reportedly had their teeth brushed before age two.[19] In their case-control study of fluorosis in Toronto, Osuji *et al* reported that 53 percent of 139 eight- to ten-year-old children (27 percent of controls vs 82 percent of cases) had begun brushing with fluoride toothpaste before the age of twenty-five months.[20]

The Table summarizes results from many of the studies of use and ingestion of the dentifrice. To facilitate comparison, all results are presented in grams (g) of dentifrice used or ingested. To convert to milligrams (mg) of fluoride for an individual patient, one must know which dentifrice is being used. For 1000 -1100 ppm fluoride dentifrices, the number of mg fluoride approximately equals the number of grams of dentifrice. For 1500 ppm dentifrices, the number of mg fluoride equals 1.5 times the grams of dentifrice. (In several places in the table, the mean proportion (percentage) of dentifrice ingested does not equal the mean quantity ingested divided by the mean quantity used. The reasons for this are due to round-off to two decimal places and because the mean proportions ingested are calculated directly from the individuals' proportions ingested and not from the group totals.)

Ericsson and Forsman supervised toothbrushing by ten Swedish six-year-olds and performed the brushing for ten four-year-olds. All rinsings and toothpaste on the lips were collected and the fluoride level determined by a fluoride ion electrode. In five cases, the fluoride left on the toothbrush after rinsing was analyzed and found to be less than 2 μg (0.002 mg), so this source was neglected in future calculations. Two different toothpastes were used several days apart, with generally similar results. The younger children used an average of about 0.45 g of paste and retained an average of 0.13 g (range of 0.04 to 0.30 g) for an average of 30 percent ingested. Among six-year-olds, mean use was 0.45 g with mean retention of 0.12 g (range of 0.06 to 0.19 g) for an average of 26 percent ingested.

Hargreaves *et al* conducted excretion studies on toothpaste ingestion among 105 British children age three to six from a low fluoride area.[22] Fluoride ingestion was determined by using urinary excretion as a marker, with urine samples drawn after using placebo pastes and monofluorophosphate (MFP) paste for comparison purposes. Assuming constant fluoride ingestion from sources other than dentifrice, ingestion from dentifrice was calculated as the difference when using the MFP paste vs placebo. Also, a subset of twenty-seven children later received 2 mg of fluoride per day on sugar lumps and excretion was determined to average about 20 percent. This 20 percent factor and the fluoride concentration in the toothpaste were used to calculate ingested quantity from excreted quantity among the 105 children. Sixty-eight percent of children were estimated to ingest up to 0.25 g paste/day, 22 percent more up to 0.50 g, and 10 percent greater than 0.50 g. (Note that this study is not summarized in the table because means and ranges were not included in the article.)

Hargreaves *et al* reported on the use and ingestion of toothpaste among forty-four British children age three to six years.[23] In contrast with their previous excretion marker study where "failure to recover any part of the ingested marker will tend to underestimate the amount

Table ☐ Dentifrice use and ingestion.

Study	Age group	Number	Dentifrice used per brushing (g)			Dentifrice ingested per brushing (g)			Mean percentage ingested
			Mean	Range	90%	Mean	Range	90%	
Ericsson and Forsman[1] (1969)	4	10	0.45	—	—	0.13	0.04–0.30	—	30%
	6	10	0.45	—	—	0.12	0.06–0.19	—	26%
Hargreaves *et al.* (1972)	3–6	44	1.38	0.18–5.14	2.04	0.38	0–1.69	0.80	28%
Barnhart *et al.* (1974)	2–4	62	0.86	0.19–2.41	—	0.30	—	0.73	35%
	5–7	56	0.94	0.15–2.08	—	0.13	—	0.27	14%
	11–13	73	1.10	0.31–2.00	—	0.07	—	0.12	6%
	20–35	60	1.39	0.42–3.29	—	0.04	—	0.12	3%
Glass *et al.* (1975)	8–10	67	1.04	0.23–2.57	1.57	0.12	0–0.41	0.23	12%
Baxter (1980)	5–16	85	—	—	—	0.19	up to 0.75	0.47	—
	5–6 only	8	—	—	—	0.27	—	—	—
Dowell (1981)	3	115	0.55	0.07–1.97	—	—	—	—	—
Bruun and Thylstrup[2] (1988)	3	63	1.1	0.17–3.0	1.6[3]	—	—	—	—
	7	31	1.5	0.20–3.7	2.3[3]	—	—	—	—
	9	27	2.3	1.20–4.3	3.1[3]	—	—	—	—
	9[4]	24[4]	1.6[4]	0.54–2.5[4]	1.8[3,4]	—	—	—	—
	16	9	3.4	2.10–4.9	4.3[3]	—	—	—	—
	16[4]	25[4]	2.1[4]	0.72–4.9[4]	2.7[3,4]	—	—	—	—
Salama *et al.* (1989)[5]	3–10	19	1.0[5]	1.0–1.0[5]	1.0[5]	0.36	0.08–0.82	—	36%
Simard *et al.* (1989)	2–3	5	0.46	—	—	0.28	—	—	59%
	4	9	0.78	—	—	0.39	—	—	48%
	5	9	0.65	—	—	0.22	—	—	34%
Naccache *et al.* (1990)	3	23	0.50	—	—	0.18	—	—	41%
	5	25	0.47	—	—	0.11	—	—	30%
Simard *et al.* (1991)	1	15	0.16	0.03–0.51	0.40	—	—	—	—
Naccache *et al.* (1992)	2	36	0.62	—	—	0.33	—	—	65%
	4	81	0.45	—	—	0.22	—	—	49%
	7	77	0.50	—	—	0.16	—	—	34%
Maurice *et al.* (in press)	1–4	59	0.43[6]	0.01–2.39[6]	0.89[6]	—	—	—	—
			0.47[7]	0.03–1.27[7]	0.92[7]	—	—	—	—

1. Average of results obtained with two different dentifrices
2. Results from two-week usage and diary period
3. For this study, these are 75th percentiles, not 90th
4. Dentifrice tube orifice was 21% smaller than for other subgroups
5. All subjects used 1.0 g
6. Single observation
7. Weekly use and diary period

Note: The quantity of fluoride in mg can be calculated from the quantity of dentifrice in g as follows: if 1000 ppm fluoride, then number of mg fluoride = number of g of dentifrice; if 1100 ppm fluoride, then number of mg fluoride = 1.1 times the number of g of dentifrice; and if 1500 ppm fluoride, then number of mg fluoride = 1.5 times the number of g of dentifrice.

ingested", this study used a difference method (quantity used minus recovered equals ingested) which has the "disadvantage that any accidental loss will be recorded as ingestion."[22] On three different days, all children placed the paste and did all brushing, and all spitting and rinsings, etc. were placed into the beakers provided. Recovery of the abrasive polishing agent was used for determination of the quantity of dentifrice recovered. The amount of toothpaste used per brushing ranged from 0.18 to 5.14 g with a mean of 1.38 g. The individuals' averages for the three days ranged from 0.34 to 2.94 g. The amount not recovered and presumed ingested ranged from 0 to 1.69 g with a mean of 0.38 g, with individuals' three-day averages from 0 to 1.16 g. Thirty-six percent of children ingested a mean of less than 0.25 g, 34 percent ingested from 0.25 to 0.50 g, and 30 percent ingested greater than 0.50 g. The mean proportion of toothpaste ingested was 28 percent. Overall, the patterns were similar to those from their previous study, although the quantities estimated to be ingested were somewhat higher. The authors noted that analysis-of-variance showed that the variation among children was substantially greater than that within children.

Barnhart *et al* used a one-way mirror to observe Ohio individuals' directed to follow their normal toothbrushing habits.[24] A number of subjects who dropped dentifrice were excluded from the results. As seen in the Table, subjects were from four age-groups. Ninety-five percent of the two- to four-year-olds had their mothers accompany them and apply the dentifrice to the toothbrush and mothers brushed the teeth in 40 percent of the cases. Double deionized water was used, all rinsings were collected and non-ingested quantities were determined by atomic absorption spectroscopic analyses for LiCl which was included in the dentifrice as a chemical tracer. The youngest children brushed once each, while the other age-groups conducted duplicate brushings. Most subjects used substantial quantities of dentifrice, although older subjects used somewhat more. The age-related differences in ingestion were dramatic with mean ingestion of 0.30 g per brushing among two- to four-year-olds (35 percent of that used) and 0.13 g (14 percent) among those age five to seven. Ten percent of those age two to four ingested 0.73 g of paste or more per brushing. Barnhart *et al* reported that within-patient variation in ingestion from duplicate brushings for those age five to seven or older was greater than between-patient variation.[24]

Glass *et al* reported on sixty-seven children age eight to ten years who conducted three replicate trials with the quantity not recovered from the toothbrush or rinsings being considered as ingested.[25] Brushing and rinsing were to be carried out as normally done by the children. A fluoride specific electrode was used for determination of the fluoride content. The range of toothpaste used was from 0.23 to 2.57 g with a mean of 1.04 g. The mean weight ingested was 0.12 g with a range of 0 to 0.41 g. The proportion of dentifrice ingested ranged from 0 to 32 percent, with a mean of 12 percent. All of these children were old enough to be beyond the age of risk of fluorosis to the majority of the permanent teeth.

Baxter studied supervised toothbrushings on three occasions by eighty-five British schoolchildren age five to sixteen.[26] By use of atomic absorption spectroscopy, the total calcium content of the toothpaste expelled from the mouth or remaining on the toothbrush was determined and the weight of toothpaste ingested was estimated. The overall mean quantity ingested was 0.19 g, with 70 percent averaging less than 0.25 g ingested and about 95 percent averaging less than 0.5 g ingested per brushing. Ingestion values were substantially higher, however, for the eight children age five to six with a mean of 0.27 g ingested. Five of the eight young children had at least one reading greater than 0.3 g ingested and rinsing with water did not lead to lower ingestion among five- to six-year-olds.

Dowell asked 115 mothers of three-year-olds to place their child's usual amount of toothpaste on a pre-weighed child's toothbrush.[16] Reweighing of the brush allowed the amount of toothpaste used to be calculated. Toothpaste use ranged from 0.07 g to 1.97 g with a mean of 0.54 g. All mothers reported that their children swallowed some toothpaste and 64 percent thought that about half or more was ingested. The author commented on the substantial variation among individuals, ranging from "a smear of paste to virtually the maximum quantity which can be loaded on to a child's small toothbrush." Although acknowledging the artificial conditions of dispensing the toothpaste, Dowell concluded that "some children are exposed to fluoride in toothpaste young enough and in large enough quantities for there to be a systemic effect during the formation of not only the permanent dentition but also some of the posterior deciduous teeth." Concern was also expressed over some small children having access to the toothpaste and eating it freely.

Bruun and Thylstrup assessed toothpaste usage during a two-week period among 179 Danish children age three, seven, nine, or sixteen.[27] There were sixty-three children age three who were all given the same widely used commercial dentifrice. The total amount of dentifrice used was determined as the difference between the mean weight of the full tubes and that of each child's returned tube. Parents (for the younger children) and older children were instructed to use the toothpaste "in exactly the normal way, and that nobody else should use the tube." Those age three and seven averaged 2.0 brushings per day while nine-and-sixteen-

Non-rinsers ingested 75 percent more toothpaste than rinsers.

year-olds averaged 2.1 to 2.2. For 97 percent of the three-year-olds, the toothpaste was reportedly dispensed by an adult and 79 percent reported a water rinse in connection with the brushing. Fifty-four percent of seven-year-olds reportedly dispensed their own toothpaste and 77 percent reportedly rinsed. The quantity of dentifrice used daily varied substantially. For three-year-olds, the range of daily dentifrice use was 0.17-3.0 g, the mean was 1.1 g, the median was 1.0 g, and 25 percent of children used 1.6 g or more. With the same brand of dentifrice, daily amounts used "increased at a fairly constant rate" with increasing age, to means for seven-year-olds of 1.5 g, for nine-year-olds of 2.3 g, and for sixteen-year-olds of 3.4 g. The authors did not assess quantity of toothpaste actually ingested. However, assuming that three-year-olds swallowed 30 percent and seven-year-olds 15 percent of the toothpaste they used, it was determined that, depending on water fluoride level (<0.2 ppm vs 0.2-0.7 ppm), 35 percent of seven-year-olds would be exceeding from dentifrice alone the recommendation for systemic fluoride supplementation in Denmark.

Salama *et al* determined the amount of fluoride not recovered from the mouth and presumed ingested after toothbrushing by nineteen children age three to ten.[28] Each child brushed with 1.0 g of 0.1 percent fluoride dentifrice and rinsed, if it was their habit to do so. The fluoride in the expectorate and remaining on the toothbrush was analyzed with a fluoride electrode. Brushing time averaged fifty-nine seconds and was not correlated with age. Seven of nineteen children (37 percent) did not expectorate. The quantity of fluoride not recovered ranged from 0.08 to 0.82 mg with a mean of 0.36 mg and, therefore, the mean proportion ingested was 36 percent. Older children retained less fluoride. The authors concluded that the average amount of ingested fluoride from a single brushing exceeded the average dietary fluoride intake of young children in non-fluoridated areas and was about 75 percent of that in fluoridated areas and that toothpaste is an important source of ingested fluoride.

Simard *et al* reported pilot study findings concerning twenty-three Quebec children age two to five.[29] They first gathered information from the parents by questionnaire and then, depending on the responses, either the child or a dental hygienist (in lieu of the mother) put the toothpaste on the toothbrush and brushed the child's teeth. Children either rinsed or not as was their habit. Brushing lasted five minutes on average. All fluoride not ingested, and thus rejected, was collected and analyzed by direct determination of fluoride with a fluoride ion-specific electrode. The quantity ingested was the difference between quantity used and rejected. The authors acknowledged that this method tends to overestimate the quantity ingested since all loss is considered ingested, but close attention to recovery of dentifrice was believed to have held this overestimation to a minimum. On the questionnaire, parents reported putting toothpaste on for 77 percent of the children. In 41 percent of the cases, the quantity of toothpaste put on the toothbrush reportedly covered a third or less of the brush, in 50 percent of cases it was from one-third to two-thirds, and in 9 percent it was two-thirds or more. Seventy-eight percent reported that children rinsed their mouths after brushing. From the brushing observations and calculations, it was determined that the overall mean quantity of dentifrice used was 0.66 g, with a mean of 48 percent of it ingested for a mean quantity ingested of 0.30 g. Children not rinsing their mouths ingested approximately 75 percent more paste than did those rinsing (0.45 g vs. 0.25 g). Those age two to three ingested an average of 59 percent of the toothpaste compared with 48 percent for four-year-olds and 34 percent for five-year-olds. By considering frequency of toothbrushing, mean daily quantity of toothpaste ingested was calculated to be

Unrelated to age, the mean quantity used per brushing was 0.50 g.

about 0.67 g. Almost a third of the children were ingesting (from toothpaste alone) more than their age-specific recommended daily total quantity of fluoride.[30] The excess ranged from 0.17 mg to 0.76 mg.

Naccache *et al* assessed the amount of dentifrice used and ingested by forty-eight children age three or five years old.[31] All selected children were ones who brushed their teeth themselves, used dentifrice, and placed the toothpaste on the brush themselves. The major purpose of the study was to investigate the variability in the amount of dentifrice used and ingested. Each child had three brushing sessions a week apart with the quantity of fluoride rejected being determined by fluoride ion-specific electrode, and quantity ingested then calculated by subtraction as in their earlier study.[29] The mean quantity of dentifrice used by the twenty-three three-year-olds was 0.50 g with 0.18 g ingested compared with means of 0.47 g used and 0.11 g ingested for twenty-five five-year-olds. The average of the proportions ingested were 41 percent for three-year-olds and 30 percent for five-year-olds. Repeated measures analysis of variance was employed to investigate differences in the amounts of toothpaste used, amounts ingested, and proportions ingested. There were no statistically significant differences in the amounts of dentifrice used or ingested across the three brushings, but differences among children were statistically significant. For proportion of dentifrice ingested, differences among children were significant, as was the tendency to ingest a higher proportion the first brushing vs. the second or third, but "the variability was not of an important magnitude." The amounts used and ingested were comparable with those found by Ericsson and Forsman with four- to six-year-olds, but lower than those found by Barnhart *et al* and Hargreaves *et al* whose ingestion figures may have been inflated due to loss of dentifrice other than from swallowing.[21,23,24] In summary, Naccache *et al* reported that children used and ingested different amounts each brushing, but the differences were minor and largely random.[31] Thus, the variation within a given child was probably due to chance and not to the order of brushings. Variability among children was clearly greater than that within children. They recommended that future studies examine larger numbers of children, therefore, instead of smaller numbers repetitively.

Simard *et al* surveyed fifty-nine parents of Quebec children age twelve to twenty-four months concerning the children's toothbrushing habits and use of fluoride toothpaste.[32] Forty-two percent reported using toothpaste and, among those, 48 percent reported beginning before twelve months. Sixty percent reported brushing once per day vs 40 percent more than once per day. Seventeen percent of those less than eighteen months of age used fluoride toothpaste compared with 67 percent of those eighteen months of age or greater. For fifteen of the twenty-five children using a dentifrice, a dental hygienist met with the parent to observe and quantify the amount of toothpaste used by weighing the toothpaste tube before and after. Fifty-three percent used less than 0.1 g, 33 percent used 0.1 g-0.3 g, and 14 percent used more than 0.3 g per brushing. The authors suggested that "with the widespread use of fluoride dentifrice at an early age and the risk of developing dental fluorosis, parents should be advised to delay the use of fluoride dentifrice until the child is older than twenty-four months...." This is in contrast with current recommendations to begin using fluoride dentifrice when the first primary tooth erupts.[33]

Dentifrice use was investigated retrospectively in exposures from birth to age six among sixty-nine school-aged children in Iowa.[34] Sixty-two percent reported that their children's teeth were first brushed before age two, 24 percent reported beginning at age two, and 14 percent reported beginning at age three or older. Fifty-four percent reported beginning use of fluoride dentifrice by age two, 39 percent at age two to three, and 7 percent age four or later. Six percent reported that their children began brushing their own teeth before age two and an additional 57 percent brushed their own teeth by age two or three. Parents reported that 36 percent of their children typically brushed twice daily from age two to six, 54 percent reported once daily, and 10 percent reported less than daily brushing from age two to six. Parents used a photograph with pictures of 8 toothbrushes with various quantities of dentifrice to report their children's typical use of dentifrice separately up to age two, age two to three, and age four to five. Prior to age two, 8 percent reportedly used no paste, 27 percent used less than one-quarter of a toothbrush strip (less than about 0.25 g), 26 percent used about 0.25 g, 30 percent used about 0.5 to 0.75 g, and 9 percent used about 1.0 g. At age two and three, 2 percent typically used none, 20 percent used less than 0.25 g, 19 percent used about 0.25 g, 44 percent used about 0.50 to 0.75 g, and 15 percent used about 1.0 g. When age four or five, 59 percent reportedly used about 0.5 to 0.75 g, 29 percent used about 1.0 g, and 12 percent used about 0.25 g or less. These data supported the conclusions of Beltran and Szpunar that young children could ingest enough fluoride from toothpaste alone to be at risk of dental fluorosis.[35]

Naccache *et al* recently explored the effects of age, amount of dentifrice used, and rinsing after brushing on the ingestion of fluoride dentifrice among 405 Quebec children age two to seven, with eligibility limited to those brushing their own teeth with toothpaste.[36] In the 23 percent of the children who did not usually place the toothpaste on the brush themselves, a trained dental hygienist watched the mother at home place the toothpaste, measured the quantity by weighing before and after, and reproduced the quantity for the child at school where a single brushing by each child with a standard toothbrush and toothpaste was observed. As in their previous studies, fluoride contents of the rejected quantities of toothpaste were determined with a fluoride ion-specific electrode and the quantity ingested was derived as the difference between the quantity used and rejected.

The overall mean quantity used per brushing was 0.50 g with the amount used not significantly related to age.[36] The variability among the two-year-olds was much greater than in the other ages. There was a significant age-related pattern of rinsing, with few of those under age six rinsing their mouths. Quantity ingested declined significantly with increasing age, from a mean of 0.36 mg F (0.33 g of 1100 ppm fluoride toothpaste) for two-year-olds to 0.24 mg F (0.22 g of toothpaste) for four-year-olds and 0.18 mg F (0.16 g of paste) for seven-year-olds. The proportion of toothpaste ingested was significantly related to age, with the mean proportion ingested declining from 65 percent among two-year-olds to 49 percent among three- and four-year-olds to 34 percent among six- and seven-year-olds. The overall mean quantity of fluoride ingested was 0.23 mg F from 0.21 g of 1100 ppm fluoride paste ingested. Among four- and five-year-olds, rinsing was associated with significantly less ingestion of dentifrice (0.17 vs. 0.26 mg F). Younger children swallowed similar quantities of dentifrice regardless of rinsing behavior, probably due to inadequate control of swallowing reflexes. The older children apparently "had learned to expectorate properly by that age, regardless of their rinsing habits," and ingested less dentifrice whether rinsing or not.

In a longitudinal study of approximately eighty Iowa children from birth to eighteen months in which parents completed questionnaires every three months, parents did the majority of the toothbrushing of the children's teeth at each age.[37]. Sixty-six percent reportedly had teeth brushed regularly at twelve months, 81 percent at fifteen months, and 92 percent at eighteen months. Pictures of toothbrushes with various toothpaste quantities were viewed by the parents. About 25 percent used at least a quarter of a toothbrush strip (about 0.25 g or more) of toothpaste per brushing at twelve, fifteen, and eighteen months. Approximately a third of those used about 0.25 g and two thirds used about 0.5 g or more.

Nowak recently studied toothpaste use among 384 Iowa preschool children up to age five.[38] Parents were asked to select from pictures of four toothbrushes with various quantities of dentifrice (0.10, 0.25, 0.50, 1.0 g) the one closest to matching their child's typical use. There was a clear trend toward increasing quantity of dentifrice used with increasing age. Overall, 5 percent used about 1.0 g, 36 percent used 0.50 g, 33 percent used 0.25 g, and 25 percent used 0.10 g. Twenty-eight percent of two- to three-year-olds used about 0.5 g or more of dentifrice compared with 44 percent of four- to five-year-olds.

In a recent study of toothpaste use and habits among fifty-nine Iowa children age one to four, parents and children simulated their normal toothbrushing routine during a single observation at the college of dentistry.[39] They were provided with a preweighed toothpaste tube of the brand and flavor they typically used. Quantity used ranged from 0.01 to 2.39 g with a mean of 0.43

In policy-making discussions, the mean and the distribution of results should be considered.

g. Older children tended to use more toothpaste. No precise measurement of quantity ingested was made, but 54 percent of children ingested most of the toothpaste based on their lack of any rinsing or expectorating. Furthermore, the majority of those who rinsed with water, ingested all or most of the rinse instead of expectorating. Younger children tended to rinse and expectorate less frequently and, therefore, appeared to ingest larger proportions of dentifrice used. Only 5 percent of those less than age two-and-one-half spit out toothpaste vs 32 percent among those age two-and-one-half to four. Children's toothbrushing patterns and toothpaste use at home were then assessed for a one-week period with a diary. By comparison of toothpaste tube weight before and after the week, total weekly use was determined. Mean toothpaste use per brushing was 0.47 g with a range of 0.03 to 1.27 g and mean use per day was 0.77 g (range of 0.03 to 3.21 g). Again, the older children tended to use more toothpaste than did the younger children. Patterns of toothpaste use during observation and from the diary correlated well. In a substudy with twenty-nine of the children, use of dentifrice specially flavored for children was associated with the use of larger quantities of dentifrice than when using regular flavor dentifrice, both across groups of children and for individual children using both products with a crossover design.[40] The preschoolers used a daily average of 0.81 g of dentifrice flavored for children vs 0.66 g of regular flavor dentifrice.

DISCUSSION

These studies of dentifrice ingestion were conducted by different investigators using different methods in different nations and settings.[7] Nevertheless, all investigators reported widespread use of fluoride dentifrice, with most children using dentifrice by age eighteen to twenty-four months.[7] There was substantial variation among subjects in their use and estimated ingestion of fluoride dentifrice.[7] The variation within subjects on repeated brushings sometimes was substantial as well. Overall, however, variation among subjects was generally much greater than that within subjects, and variation within subjects was believed to be due to chance with the differences being relatively minor.[31]

Within the age range of two to seven, younger children generally used quantities of toothpaste fairly similar to those used by older children, but ingested larger proportions and quantities.[7] The mean quantities ingested by two- to three-year-olds were approximately 0.3 g per brushing and the ingested portion of toothpaste used averaged up to 59 percent to 65 percent for these children, who were at the age of maximal risk of developing dental fluorosis of the maxillary incisors.[7,12,13] In Ripa's review of dentifrice use and ingestion, he discounted the data of Simard *et al* because of the apparently long mean brushing time of five minutes and he did not have access to the more recent supportive data.[29,32,36,39,41] Ripa's calculated mean proportion of toothpaste ingested (27 percent) by children under age six and mean quantity of fluoride ingested (0.134 mg) are probably underestimates, therefore, especially among the youngest children. Only small percentages of young children have been found to rinse their mouths and many who do rinse with water may, due to inadequate control of their swallowing reflexes and lack of training, still ingest the majority of dentifrice used.[42] Although dentifrice use and ingestion are quite variable among infants and one-year-olds, substantial proportions of these youngest children also ingest significant quantities of dentifrice.

Several reports provide data showing details of the frequency distribution of dentifrice use and ingestion and generally they show about 20 percent of the children's quantities used and ingested to be well beyond the mean results most often quoted in the literature.[7] The use of mean figures only can be misleading, and consideration should be given to both the mean and the distribution of results during policy-making discussions.[7] Most authors have acknowledged this variation in toothpaste ingestion and recognized that some children could ingest more fluoride from dentifrice alone than the recommended daily total.[7] "When combined with fluoride from other regular sources, such as beverages and food and dietary fluoride supplements, the total quantity of fluoride ingested increases and larger percentages of children are beyond the "optimal range" of fluoride intake."[7] Ripa concluded that fluoride dentifrices were not the primary cause of the increase in the prevalence of fluorosis.[41] It has been concluded, however, that ingestion of fluoride dentifrice is an "important part of total daily fluoride intake and some young children may be ingesting enough fluoride from dentifrice to cause dental fluorosis."[7,35,43] Burt concluded that the use of a fluoride dentifrice by young children "has to be recognized as a risk factor for dental fluorosis".[43]

Although Ripa pointed out that the ingestion of fluoride toothpaste has generally not been identified as a risk factor for fluorosis in analytical studies, Pendrys and Rozier emphasized that the majority of the studies of fluorosis risk were not well designed for assessment of fluoride dentifrice as a risk factor, because of insufficient statistical power to draw such a conclusion.[41,44,45] For example, most only asked whether fluoride toothpaste was used at an early age (yes/no) or when brushing was initiated, without regard to frequency of brushing, quantity of paste used and ingested, rinsing patterns, etc. The studies with more powerful designs, such as that by Osuji *et al* were more likely to report statistically significant effects of the early use of fluoride toothpaste on the prevalence of fluorosis.[20] Several other recent studies have linked the early use of fluoride dentifrice with fluorosis.[46-48] A recently concluded case-control study among 108 Iowa pediatric dental patients found that the number of years of use of fluoridated water and estimated total quantity of fluoride dentifrice used from birth to age eight (considering years of use, estimated amount used per brushing, and frequency of brushing) were independently associated with the risk of fluorosis.[49,50] Future studies of the use and ingestion of dentifrice and risk factors for fluorosis must consider the details of fluoride ingestion from dentifrice and attempt to measure it.

Also, as suggested by others, "use of higher concentration fluoride toothpastes by preschool children must be avoided."[3,4,7,35,41,43] Further development and testing of lower concentration fluoride toothpastes are encouraged, and small quantities of dentifrice should be used with parental direction and supervision so that inappropriate "eating" of fluoride dentifrice is avoided.[7] In these ways, use of fluoride dentifrice will continue to be an important caries preventive tool, while minimizing its role in the etiology of dental fluorosis.

REFERENCES

1. International Association for Dental Research: International symposium on fluorides: mechanisms of action and recommendations for use. J Dent Res, 69:505-831, February 1990.
2. American Association of Public Health Dentistry: Symposium on appropriate uses of fluoride - considerations for the '90s. J Public Health Dent, 51:20-63, Winter 1991.
3. Bawden, J.W. ed: Proceedings of the workshop: changing patterns in systemic fluoride intake. J Dent Res, 71:1212-1265, May 1992.
4. Clark, D.C., ed.: Report of the working group of the Canadian workshop on the evaluation of current recommendations concerning fluorides. Submitted to Community Dent Oral Epidemiol.
5. U.S. Public Health Service: Review of fluoride benefits and risks. Report of the Ad Hoc Subcommittee on Fluoride. Washington, DC: U.S. Government Printing Office, February 1991.
6. Graves, R.C. and Stamm, J.W.: Oral health status in the United States: Prevalence of dental caries. J Dent Ed, 49:341-351, June 1985.
7. Levy, S.M.: A review of fluoride exposures and ingestion. Paper presented at the Canadian workshop "Evaluation of Current Recommendations Concerning Fluorides", Toronto, April 9, 1992. (Manuscript submitted to Community Dent Oral Epidemiol)
8. Fejerskov, 0.; Thylstrup, A.; Larsen, M.J.: Clinical and structural features and possible pathogenic mechanisms of dental fluorosis. Scand J Dent Res, 85:510-534, November 1977.
9. Larsen, M.J.; Richards, A.; Fejerskov, 0.: Development of dental fluorosis according to age at start of fluoride administration. Caries Res, 19:519-527, November-December 1985.
10. Richards, A.; Kragstrup, J.; Josephsen, K. *et al*: Dental fluorosls developed in post-secretory enamel. J Dent Res, 65:1406-1409, December 1986.
11. Evans, R.W.: Changes in dental fluorosis following an adjustment to the fluoride concentration of Hong Kong's water supplies. Adv Dent Res, 3:154-160, September 1989.
12. Evans, R.W. and Stamm, J.W.: Dental fluorosis following downward adjustment to fluoride in drinking water. J Public Health Dent, 51:91-98, Spring 1991.
13. Evans, R.W. and Stamm, J.W.: An epidemiologic estimate of the critical period during which human maxillary central incisors are most susceptible to fluorosis. J Public Health Dent, 51:251-259, Fall 1991.
14. Pendrys, D.G. and Stamm, J.W.: Relationship of total fluoride intake to beneficial effects and enamel fluorosis. J Dent Res, 69(Special Issue) 529-538, February 1990.
15. Palmer, J.D. and Prothero, D.L.: Young children and toothpaste. Br Dent J, 150:338-339 (letter), June 16, 1981.
16. Dowell, T.B.: The use of toothpaste in infancy. Brit Dent J, 150:247249, May 5, 1981.
17. Grytten, J.; Rossow, I.; Holst, D. *et al*: Longitudinal study of dental health behaviors and other caries predictors in early childhood. Community Dent Oral Epidemiol, 16:356-359, December 1988.
18. Woltgens, J.H.; Etty, E.J.; Nieuwland, W.M. *et al*: Use of fluoride by young children and prevalence of mottled enamel. Adv Dent Res, 3:177-182, September 1989.
19. Szpunar, S.M. and Burt, B.A.: Fluoride exposure in Michigan schoolchildren. J Public Health Dent, 50:18-23, Winter 1990.
20. Osuji, O.O.; Leake, J.L.; Chipman, M.L. *et al*: Risk factors for dental fluorosis in a fluoridated community. J Dent Res, 67:1488-1492, December 1988.
21. Ericsson, Y. and Forsman, B.: Fluoride retained from mouthrinsing and dentifrices in preschool children. Caries Res, 3:290-299, 1969.
22. Hargreaves, J.A.; Ingram, G.S.; Wagg, B.J.: Excretion studies of the ingestion of a monofluorophosphate toothpaste by children. Caries Res, 4:256-268, Third quarter 1970.
23. Hargreaves, J.A.; Ingram, G.S.; Wagg, B.J.: A gravimetric study of the ingestion of toothpaste by children. Caries Res, 6:237-243, Third quarter 1972.
24. Barnhart, W.E.; Hiller, L.K.; Leonard, G.J. *et al*: Dentifrice usage and ingestion among four age groups. J Dent Res, 53:1317-1322, November-December 1974.
25. Glass, R.L.; Peterson, J.K.; Zuckerberg, D.A. *et al*: Fluoride ingestion resulting from the use of a monofluorophosphate dentifrice by children. Br Dent J, 138:423-426, June 3, 1975.
26. Baxter, P.M.: Toothpaste ingestion during toothbrushing by school children. Br Dent J, 148:125-128, March 4, 1980.
27. Bruun, C. and Thylstrup, A.: Dentifrice usage among danish children. J Dent Res, 67:1114-1117, August 1988.
28. Salama, F.; Whitford, G.M.; Barenie, J.T.: Fluoride retention by children from toothbrushing (Abstract #1227). J Dent Res, 68(Special Issue - AADR Abstracts):335, March 1989.
29. Simard, P.L.; Lachapelle, D.; Trahan, L. *et al*: The ingestion of fluoride dentifrice by young children. J Dent Child, 56:177-181, May-June 1989.
30. Council on Dental Therapeutics, American Dental Association. *Accepted Dental Therapeutics*, 40th Ed. Chicago: American Dental Association, 1984.
31. Naccache, H.; Simard, P.L.; Trahan, L. *et al*: Variability in the ingestion of toothpaste by preschool children. Caries Res, 24:359-363, September-October 1990.

32. Simard, P.L., Naccache, H.; Lachapelle, D. *et al*: Ingestion of fluoride from dentifrices by children aged 12 to 24 months. Clinical Pediatrics, 30:614-617, November 1991.
33. American Dental Association: A guide to the use of fluorides for the prevention of dental caries. J Am Dent Assoc, 113:503-565, September 1986.
34. Levy, S.M. and Zarei-M, Z.: Retrospective evaluation of fluoride exposures in children. J Dent Child, 58:467-473, November-December 1991.
35. Beltran, E.D. and Szpunar, S.M.: Fluoride in toothpastes for children: Suggestions for change. Pediatric Dent, 3:185-188, September 1988.
36. Naccache, H.; Simard, P.L.; Trahan, L. *et al*: Factors affecting the ingestion of fluoride dentifrice by children. J Pub Health Dent, 52:222-226, Summer 1992.
37. Levy, S.M.; Maurice, T.J.; Jakobsen, J.R.: Feeding patterns, water sources, and fluoride exposures of infants and l-year-olds. J Am Dent Assoc, in press.
38. Nowak, A.J.: Toothbrush selection for children 1-5 years old (Abstract #1232). J Dent Res, 71(Special Issue-IADR Abstracts):669, July 1992.
39. Maurice, T.J. and Levy, S.M.: Dentifrice use among preschool children. J Am Dent Assoc, in press.
40. Levy, S.M.; Maurice, T.J.; Jakobsen, J.J.: A pilot study of preschoolers' use of regular flavored dentifrices and those flavored for children. Pediatr Dent, 14:388-391, November-December 1992.
41. Ripa, L.W.: A critique of topical fluoride methods (dentifrices, mouthrinses, operator-, and self-applied gels) in an era of decreased caries and increased fluorosis prevalence. J Public Health Dent, 51:23-41, Winter 1991.
42. Whitford, G.M.; Allmann, D.W.; Shahed, A.R.: Topical fluorides: Effects on physiologic and biochemical process. J Dent Res, 66:1072-1078, May 1987.
43. Burt, B.A.: The changing patterns of systemic fluoride intake. J Dent Res, 72:1228-1237, May 1992.
44. Pendrys, D.G.: Reaction paper: Some perspectives on the appropriate uses of fluoride for the 1990s and beyond. J Public Health Dent, 51:53-55, Winter 1991.
45. Rozier, R.G.: Reaction paper: Appropriate uses of fluoride - consideration for the '90s. J Public Health Dent, 51:56-59, Winter 1991.
46. Pendrys, D.G. and Katz, R.V.: Risk of enamel fluorosis associated with fluoride supplementation, infant formula, and fluoride dentifrice use. Am J Epidemiol, 130:1199-1208, December 1989.
47. Milsom, K. and Mitropoulos, C.M.: Enamel defects in 8-year-old children in fluoridated and non-fluoridated parts of Cheshire. Caries Res, 24:286-289, July-August 1990.
48. Lalumandier, J.A.: The prevalence and risk factors of fluorosis among children in a pediatric practice in Asheville, North Carolina. J Public Health Dent, 52:188-189 (abstract), Spring 1992.
49. Skotowski, M.C.: Risks factors associated with dental fluorosis. Master's Thesis, The University of Iowa, 1991.
50. Skotowski, M.C.; Hunt, R.J.; Levy, S.M.: Risk factors for dental fluorosis in pediatric patients (abstract). J Public Health Dent, in press (Manuscript submitted to Pediatr Dent).
51. Winter, G.B.; Holt, R.D.; Williams, B.F.: Clinical trial of a low-fluoride toothpaste for young children. Int Dent J, 39:227-235, December 1989.
52. Horowitz, H.S.: The need for toothpastes with lower than conventional fluoride concentrations for preschool-aged children. J Public Health Dent, 52:216-221, Summer 1992.

FLUORIDE CONCENTRATIONS OF INFANT FOODS AND DRINKS

Fluoride analyses of baby foods were carried out using a microdiffusion technique, which was found to be reproducible and accurate with less than 8 percent error. Analysis of 113 baby foods and drinks showed a wide range of fluoride concentrations: 0.01–0.31 mg F/kg for baby milk products; 0.04–0.72 mg F/kg for meat products; 0.04–0.70 mg F/kg for cereals; 0.03–0.48 mg F/kg for vegetable products; 0.03–0.07 mg F/kg for fruits; 0.02 – 0.28 mg F/kg for desserts, and 0.01–0.51 mg F/1 for baby drinks. None of the baby foods and drinks contained fluoride of a sufficiently high concentration to be of concern or likely to contribute to enamel mottling, when used in the normal way.

A. Vlachou *et al*: Fluoride concentrations of infant foods and drinks in the United Kingdom.
Caries Res, 26:29-32, January-February 1992.

BEHAVIOR

Dental management of the child and adolescent with major depression

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Until the 1970s many mental health professionals held that a juvenile's cognitive and psychological state of development was insufficient to permit the child to experience profound and persistent depression. It is now recognized that the disease afflicts numerous youngsters and that its rate is on the rise. Major depression is a psychiatric disorder in which mood, thought content, and behavioral patterns are impaired for an extended period of time. It is to be distinguished from transient sadness precipitated by disappointment and the unhappiness that represents normal fluctuations of mood. Without proper treatment major depression will adversely affect the child's social, educational, and emotional functioning. Dentists must be familiar with the disorder, because of its frequent association with extensive dental pathology and with the possible need to modify standard plans of care.

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The authors acknowledge the editorial assistance of Ms. W. Emerson.

MAJOR DEPRESSION

The hallmark symptoms of major depression are an intense and persistently sad or irritable mood and a loss of interest or pleasure in most daily activities. Impairment in cognition, social relations, and somatic function usually accompany these symptoms.[1] The ability to identify and verbalize feelings of depression is influenced by the child's stage of physiological and psychological maturation.

Characteristics in preschoolers

Preschoolers (ages three to five years) do not usually complain of depression. At this phase of development, they are just beginning to formulate concepts of time and self and to differentiate between their internal mood and external events. These young children generally lack the necessary language skills to express their feelings in words. They do tend to exhibit behaviors that are consistent, however, with their moods. They look sad most of the time, rarely smile, and are frequently tearful. They may also be angry, uncooperative, and irritable. They seem to lose interest in or fail to perform newly acquired skills such as running, climbing, buttoning clothes, putting on shoes, riding a bicycle or counting. There may even be a regressive return of thumbsucking, babytalk and bedwetting and/or soiling in a previously well-toilet-trained child. Often, depressed children refuse to go to nursery school, stop playing with friends, appear preoccupied and become more isolated. Cognitive functions, including language ability, decline. Sleep is disturbed by frequent nightmares thematically related to death, danger, and hopelessness. Some depressed children will speak of their wish to disappear or die, while others demonstrate self-destructive behavior in the form of head-banging, biting and scratching themselves, swallowing sharp objects or becoming accident prone.

Characteristics in school-age children

School-age children (ages six to twelve years) are more verbal than preschoolers and can directly communicate their depressed feelings to others. They may not do so spontaneously, but if questioned they are capable of talking about their affect and behavior. They will admit to feeling sad, or unhappy and will describe restrictions in their current activities (e.g., decreased participation in scouting, or organized sports) and loss of interest in future events (e.g., indifference about a planned trip to Disneyland). Depressed children are frequently self-absorbed and may be obsessed with their own worries and thoughts. They may withdraw from previously enjoyed group activities and refuse to play with friends, claiming that other kids are "mean" or "stupid" or "don't like me." They may also abandon hobbies and favorite solo activities, such as stamp collecting, drawing, and watching television. Instead, they may prefer to sit for hours staring at the floor and answering questions in a monotone voice, using one or two-word sentences. These behaviors are generally accompanied by vegetative signs and symptoms, including anorexia, weight loss, insomnia, early morning awakening and fatigue. Dreams are characteristically morbid, centering on loss and abandonment, personal injury and death. School work suffers as the child demonstrates impairments in attention and concentration, has difficulty following directions, is careless, and may be unable to finish assignments. Fatigue and psychomotor retardation result in tardiness and an oppositional stance in completing chores and in accomplishing tasks of personal hygiene. Absence from school is frequent, often caused by psychosomatic illness and hypochondriasis manifesting as stomach aches, leg pains, and chronic headaches.[2] These symptoms combine to exacerbate the low self-esteem, worthlessness, hopelessness, and suicidal ideation that are intrinsic to a major depressive episode.

Characteristics in adolescents

Adolescents (ages twelve to eighteen years) suffering from major depression are usually profoundly sad, but may also have intense mood swings and a proclivity toward rage. The affective disorder interferes with the phase-specific tasks of adolescence. For example, menstruation may trigger revulsion and tearfulness in teenage girls, while depressed boys might react with intense guilt and self-loathing over the sexual nature of their dreams and associated nocturnal emissions. Both male and female adolescents frequently isolate themselves

At times, tobacco, alcohol, and drugs are used as an escape from psychic pain.

in their bedrooms and sleep for prolonged periods of time as a means of coping with their illness. As with their younger counterparts, adolescents will encounter school problems as their cognitive ability declines. Procrastination in finishing assignments, skipping school, and a lack of concern about achievement and future vocation are common. Depressed adolescents may brood over having failed themselves and others. At times they try to defend against low self-esteem by denial, by omnipotent fantasies or by escaping their psychic pain through the use of tobacco, alcohol, and illicit drugs. These adolescents are not usually interested in dating or heterosexual interactions. Females may engage in self-deprecating promiscuity, however, without the precautions necessary to prevent pregnancy and venereal disease.[3] Depressed adolescents, especially males, may act out their depressed, angry mood-state through such antisocial behaviors as stealing, violence, running away, and causing motor vehicle accidents.

Prevalence of major depression increases with age.

Suicide Risk

Severely depressed children and adolescents despair about themselves and their future, and some contemplate suicide.[4] Although young children between the ages of three and five years may openly verbalize suicidal ideas ("I feel like dying"), they rarely make overt attempts at suicide.[5] Older depressed females are more likely than depressed males to attempt suicide, but the majority of completed suicides are accomplished by males. In 1985, 255 children under the age of fifteen, and almost 5000 adolescents between the ages of fifteen and eighteen years are reported to have committed suicide in the United States.[6]

Epidemiology

The prevalence of major depression among children and adolescents in the general population increases with age. Surveys have found an approximate 0.5 percent prevalence among preschoolers, 2 percent among preadolescent/prepubertal children, and 5 percent among adolescents ages fourteen to sixteen years.[7-9] These rates are notably higher than in past years, and there are indications that they will continue to rise.[10] Before puberty, boys are five times more likely to suffer from major depression than girls, but after puberty the ratio is reversed with a 5:1 female preponderance.[11] Despite numerous investigations, there are no definitive explanations for these sex differences in prevalence rates.[12] An increased rate of the disorder occurs in the child and adolescent offspring of adults suffering major depression and bipolar disorder. The younger the age of the child at the onset of the disorder, the more likely it is that one or both parents are similarly afflicted.[13,14] Recent demographic studies have found that both poverty and minority ethnic status place teenagers at a higher risk for developing the disorder.[15] Untreated major depressive episodes last approximately eight months, and there is a greater than 70 percent likelihood of recurrence within five years.[16,17] These relapses often recur through adulthood.[18]

Etiology

Historically, stress from life events such as death of a parent, parental divorce, and serious illness were thought to be the most significant etiologic factors in the development of childhood depression. More recent studies indicate that these stressful events promote and sustain depression rather than cause it.[19] Most authorities now agree that major depression is related to neuroendocrine abnormalities.[20] Mood and emotions are presumed to be associated with the limbic system of the brain. The limbic system, in concert with the hypothalamus and other portions of the brain, regulates the pituitary gland, which in turn controls adrenal, thyroid, and gonadal hormone release. Dysfunction of the hypothalamus is suggested by the alterations in sleep, appetite, and sexual behavior. Aberrant concentrations of cortisol and growth hormone in peripheral blood and melatonin in urine confirm endocrine dysfunction.[21-23]

It was initially believed that depressive symptoms were due to a decrease in concentrations of norepinephrine and/or serotonin at specific synaptic receptor sites in the brain.[24] More recent studies have shown that there is a complex deregulation of multiple neurotransmitters implicated in the pathogenesis of major depression.[25]

MEDICAL MANAGEMENT OF MAJOR DEPRESSION

Depression can, on occasion, occur as a response to certain medical disorders (thyroid disease, rheumatoid arthritis, Sjogren's disease, etc.) or as an adverse reaction to particular medications (e.g., reserpine, indomethacin and prednisone). To exclude these causes a comprehensive medical history, complete physical examination, and routine blood and urine tests are performed.

The traditional tricyclic antidepressants (TCAs), monamine oxidase inhibitors (MAOIs), or the more recently introduced compounds—trazodone (Desyrel), bupropion (Wellbutrin), sertraline (Zoloft) and fluoxetine (Prozac)—are usually the agents of choice in treating the disorder.[26,27] These medications are believed to relieve symptoms of depression (usually in fourteen to twenty-eight days) by modulating noradrenergic and serotonergic activity within the central nervous system. TCAs have been the best studied antidepressants in children. Cardiovascular side effects are relatively common with the TCAs and include hypertension, tachycardia, hypotension, and orthostatic hypotension. TCAs, especially imipramine (Tofranil), have recently been implicated as the cause of sudden death among a number of children. These reports speculate that youngsters medicated with TCAs are at unique risk for fatal cardiac arrhythmias.[28,29] Anticholinergic side effects are variable in children, but when they occur they include dry mouth, constipation, and urinary hesitancy.[30,31]

Fluoxetine, a bicyclic agent, is a selective serotonin-uptake inhibitor. It provides considerable symptomatic improvement when administered to depressed adolescents.[32] The most commonly reported adverse effects are headache, nervousness, insomnia, nausea, and diarrhea.

Monoamine oxidase inhibitors (MAOIs) are often effective for the approximately 25 percent of patients who have not responded to TCAs or fluoxetine.[33] They relieve symptoms of depression by inhibiting the breakdown of norepinephrine and serotonin in presynaptic nerves by the enzyme monoamine oxidase.[34] MAOIs do not cause adverse anticholinergic side-effects, but they may interact with tyramine containing foods (cheese, aged meats, red wine) or medications with tyramine-like actions (e.g., amphetamine and ephedrine) to produce a life-threatening hypertensive crisis.

Psychotherapy is a recommended component of care for youngsters with major depression. Even when medication is efficacious and affective symptoms abate, almost always there remains a deficit in interpersonal skills and intrapsychic conflicts about the experience of a depressive illness. In the privacy of the therapist's office, the child may verbalize taboo feelings and ideas or express through play issues too painful to describe. Working through the hidden messages in feelings and behaviors and assisting the child and parent respond appropriately to life stresses are the therapists initial goals.[35]

Dental Findings

Children with major depression are likely to present with a unique set of factors that lead to the development of advanced dental disease. Depressive illness is often associated with a disinterest in performing appropriate preventive oral hygiene techniques. It is also

Advanced dental disease in children may be linked with major depression.

associated with impaired taste perception and ability to appreciate moderately sweet foods. As a consequence these youngsters tend to consume a very sweet, highly cariogenic diet.[36,37]

A diminution in whole-mouth resting salivary flow and parotid salivary output has been noted in untreated, depressed patients. The magnitude of the decrease in salivary flow is directly related to the severity of depression.[38] The tricyclic and bicyclic antidepressant medications may magnify the problem of xerostomia by blocking, through anticholinergic action, parasympathetic stimulation of the salivary glands.[39] Hyposalivation results in an intensification of periodontal disease and in rapid caries progression because of adverse changes in the oral environment.[40]

The prevalence of regular cigarette smoking is much higher among depressed adolescent boys and girls than among their well-adjusted peers.[41] Smoking causes hypoxia of oral tissues, inhibition of neutrophil function, and reduced antibody production. Clinically this results in an increased prevalence of acute necrotizing ulcerative gingivitis, ulcerative gingivitis, periodontitis, delayed healing, localized osteitis ("dry socket"), and infection.[42]

During periods of severe depression, patients may complain of atypical facial pain or temporomandibular joint pain-dysfunction syndrome. Resolution almost always occurs as the patient responds to antidepressant medications.[43]

DENTAL MANAGEMENT OF DEPRESSED CHILDREN

Before starting dental therapy, the child's psychiatrist should be consulted. After obtaining consent, the information requested should include the patient's current psychological status and current psychotropic medication regime. The findings of the medical history for both patient and familial cardiac disease, and the analysis of the electrocardiogram for arrhythmias are especially pertinent for children receiving TCA therapy. The psychiatrist must also be questioned concerning the patient's history of alcohol and illicit drug use.

Youngsters receiving tricyclic antidepressant therapy and having cardiac disease or a familial history of heart disease or sudden death should be evaluated by a pediatrician or pediatric cardiologist before extensive dental treatment is begun. Those with a history of alcohol abuse should have liver function tests (blood serum levels of hepatic enzymes, albumin and total proteins), a complete blood count, and a coagulation profile (prothrombin time and partial thromboplastin time) performed.[44]

Preventive dental education for this group of patients and their family members is paramount. They should receive instruction in proper tooth brushing and flossing methods that maximize removal of dental plaque.

Profound local anesthesia is mandatory in order to perform dental treatment procedures for depressed and anxious youngsters adequately. Numerous factors must be considered, when choosing the appropriate local anesthetic, especially for the young, low-weight child concurrently receiving a TCA. The TCAs potentiate the cardiovascular actions of adrenergic vasoconstrictors. The pressor (elevation of mean arterial and central venous pressures) and arrhythmogenic effects of levonordefrin [Neo-Cobefrin (used in local anesthetic formulations containing mepivacaine)] may be increased to potentially dangerous levels. Thus, it is best to avoid this product.[45] The interaction of the tricyclics with epinephrine is more modest in magnitude, but a reduction in maximum dosage is prudent in order to en-

Profound local anesthesia is mandatory in order to perform dental treatment procedures for depressed children adequately.

sure patient safety.[46] It is recommended that the dose of 2 percent lidocaine with 1:100,000 epinephrine be limited to one cartridge for every 10 kg or 20 lbs body weight up to a maximum of seven cartridges (in the adult-sized adolescent). Formulations without a vasoconstrictor would seen ideal; the relatively large concentrations of anesthetic agent required for acceptable efficacy (3 percent for mepivacaine, 4 percent for prilocaine), however, make it all too easy for maximum recommended doses to be exceeded in small children.[47,48] They also should be limited to one cartridge for every 10 kg or 20 lbs body weight up to a maximum of seven cartridges.

Adverse drug interactions between medications used in dentistry and the tricyclics may produce significant morbid reactions. Sedative-hypnotics, barbiturates, and narcotics may have their depressant effects potentiated by TCAs, and severe respiratory depression may ensue. The administration of medications with anticholinergic properties such as atropine or scopolamine, can cause an increase in intraocular pressure and worsen narrow angle glaucoma.[49]

The monoamine oxidase inhibitors do not potentiate the pressor or cardiac effects of exogenously administered, direct-acting catecholamines. Epinephrine and levonordefrin may be used without special reservation in patients concurrently receiving monoamine oxidase inhibitors.[50] As with TCAs, drugs with central nervous system depressant properties, however, will be potentiated, necessitating extra care when providing conscious sedation for these patients. Meperidine (Demerol) is absolutely contraindicated, because it interacts with MAOIs to produce a life-threatening mixture of central depression and stimulation.[51]

At three-month recall visits a clinical examination, oral prophylaxis and application of a fluoride gel should be performed. Correction of defects in the dentition should also be performed during these recall visits. On occasion the dentist may observe signs of declining psychosocial adjustment in a previously depressed child during a follow-up visit. Such signs may include evidence of alcohol/substance abuse, or deterioration of oral hygiene. In these cases the responsible psychiatrist should promptly be advised so that appropriate intervention can be instituted.

REFERENCES

1. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, Revised, (DSM III R). Washington D.C.: American Psychiatric Association, 1987, pp 213-233.
2. Muse, N.J.: *Depression and Suicide in Children and Adolescents: Child Guidance Mental Health Series.* Austin; Pro-Ed, Inc., 1990, pp 1-61.
3. Dolgan, J.I.: Depression in children. Pediatr Ann, 19:45-50, January 1990.
4. Ryan, N.D.; Puig-Antich, J.; Ambrosini, P. *et al*: The clinical picture of major depression in children and adolescents. Arch Gen Psychiatry, 44:854-861, October 1987.
5. Ryan, N.D.: The pharmacologic treatment of child and adolescent depression. In: Shaffer, D.: *Pediatric Psychopharmacology.* Psychiatr Clin N Amer, 15:29-40, January 1992.
6. National Center for Health Statistics: *Monthly Vital Statistics Report: Advance Report of Final Monthly Statistics, 1986.* Washington D.C.: National Center for Health Statistics, September 30, 1988, pg 30.
7. Kashani, J.H.; Holcomb, W.R.; Orvaschel, H: Depression and depressive symptomatology in preschool children from the general population. Am J Psychiatry, 143:1138-1143, September 1986.
8. Kashani, J.H. and Simonds, J.F.: The incidence of depression in children. Am J Psychiatry, 136:1203-1205, September 1979.
9. Kashani, J.H.; Carlson, G.A.; Beck, N.C. *et al*: Depression, depressive symptoms, and depressed mood among a community sample of adolescents. Am J Psychiatry, 144:931-933, July 1987.
10. Ryan, N.D.; Williamson, D.E.; Iyengar, S. *et al*: A secular increase in child and adolescent onset affective disorder. J Am Child Adolesc Psychiatry, in press.
11. Anderson, J.C.; Williams, S.; McGee, R. *et al*: DSM-III disorders in preadolescent children. Arch Gen Psychiatry, 44:69-76, January 1987.
12. Mollica, R.F.: Mood disorders: Epidemiology. In: Kaplan, H.I.; Sadock, B.J.: *Comprehensive Textbook of Psychiatry/V* (ed. 5), Baltimore: Williams and Wilkins, 1989, pp 859-867.
13. Puig-Antich, J.; Goetz, D.; Davies; M. *et al*: A controlled family history study of prepubertal major depressive disorders. Arch Gen Psychiatry, 46:406-418, May 1989.
14. Weissman, M.M.; Gammon, G.D.; John, K. *et al*: Children of depressed parents: Increased psychopathology and early onset of major depression. Arch Gen Psychiatry, 44:847-853, October 1987.
15. Garrison, C.Z.; Schlucter, M.D.; Schoenbach, V.J.: Epidemiology of depressive symptoms in young adolescents. J Am Acad Child Adolesc Psychiatry, 28:343-351, March 1989.
16. Kovacs, M.; Feinberg, T.L.; Crouse-Novak, M.A. *et al*: Depressive disorders in childhood: A longitudinal prospective study of characteristics and recovery. Arch Gen Psychiatry, 41:229-237, March 1984.
17. Kovacs, M.; Feinberg, T.L.; Crouse-Novak, M.A. *et al*: Depressive disorders in childhood: A longitudinal study of the risk for a subsequent major depression. Arch Gen Psychiatry, 41:643-649, July 1984.
18. Harrington, R.; Fudge, H.; Rutter, M. *et al*: Adult outcomes of childhood and adolescent depression: Psychiatric status. Arch Gen Psychiatry, 47:465-473, May 1990.
19. Kashani, J.H. and Schmid, L.S.: Epidemiology and etiology of depressive disorders. In: Shafii, M. and Shaffii, S.L.: *Clinical Guide to Depression in Children and Adolescents.* Washington, D.C.: American Psychiatric Press Inc., 1992, pp 43-64.
20. Yaylayan, S.; Weller, E.B.; Weller, R.G.A.: Neurobiology of depression. In: Shafii, M. and Shafii, S.L.: *Clinical Guide to Depression in Children and Adolescents.* Washington, D.C.: American Psychiatric Press Inc., 1992, pp 65-88.
21. Pfeffer, C.R.; Stokes, P.; Weiner, A. *et al*: Psychopathology and plasma cortisol responses to dexamethasone in prepubertal psychiatric inpatients. Biol Psychiatry, 26:677-689, November 1989.
22. Kutcher, S.P.; Williamson, P.; Silverberg, J. *et al*: Nocturnal growth hormone secretion in depressed older adolescents. J Am Acad Child Adolesc Psychiatry, 27:751-754, November 1988.

23. Shafii, M.; Foster, M.B.; Greenberg, R.A. *et al*: The pineal gland and depressive disorders in children and adolescents. In: Shafii, M. and Shafii, S.L.: *Biological Rhythms, Mood Disorders, Light Therapy, and the Pineal Gland.* Washington, D.C.: American Psychiatric Press Inc., 1990, pp 99-116.
24. Schildkraut, J.J. and Kety, S.S.: Biogenic amines and emotion. Science, 156:21-30, January 1990.
25. Leonard, B.E.: The amine hypothesis of depression: A reassessment. In: Tipton, K.F. and Youdim, M.B.H. (eds): *Biochemical and Pharmacological Aspects of Depression.* London: Taylor and Francis Publishing Co., 1989, pp 25-49.
26. Preskorn, S.H.; Weller, E.B.; Weller, R.A.: Depression in children: Relationship between plasma imipramine levels and response. J Clin Psychiatry, 43:450-453, November 1982.
27. Puig-Antich, J.; Perel, J.M.; Lupatkin, W. *et al*: Imipramine in prepubertal major depressive disorders. Arch Gen Psychiatry, 44:81-89, January 1987.
28. Biederman, J.: Sudden death in children treated with a tricyclic antidepressant. J Am Acad Child Adolesc Psychiatry, 30:495-498, May 1991.
29. Tingelstad, J.B.: The cardiotoxicity of the tricyclics. J Am Acad Child Adolesc Psychiatry, 30:845-846, September 1991.
30. Ryan, N.D.: Pharmacologic treatment of major depression. In: Shafii, M. and Shaffii, S.L.: *Clinical Guide to Depression in Children and Adolescents.* Washington, D.C.: American Psychiatric Press Inc., 1992, pp 219-232.
31. Geller, B.; Cooper, T.B.; Graham, D.L.: Pharmacokinetically designed double-blind placebo-controlled study of Nortriptyline in 6 to 12 year olds with major depressive disorder. J Am Acad Child Adolesc Psychiatry, 31:34-44, January 1992.
32. Simeon, J.G,; Dinicola, V.F.; Ferguson, B.H. *et al*: Adolescent depression: A placebo-controlled fluoxetine treatment study and follow-up. Prog Neuro Psycho Pharm Biolog Psychiatry, 14:791-795, 1990.
33. Ryan, N.D.; Puig-Antich, J.; Rabinovich, H. *et al*: MAOIs in adolescent major depression unresponsive to tricyclic antidepressants. J Am Acad Child Adolesc Psychiatry, 27:755-758, November 1988.
34. Murphy, D.L.; Garrick, N.A.; Aulakh, C.S. *et al*: New contributions from basic science to understanding the effects of monoamine oxidase inhibiting antidepressants. J Clin Psychiatry, 45(7, Pt 2):37-43, July 1984.
35. Shaw, J.A.: Childhood depression. Med Clin North Am, 72:831-845, July 1988.
36. Harris, B.; Young, J.; Hughes, B.: Appetite and weight changes in patients presenting with depressive illness. J Affect Dis, 6:331-339, June 1984.
37. Amsterdam, J.D.; Settle, G.; Doty, R.L. *et al*: Taste and smell perception in depression. Biol Psychiatry, 22:1481-1485, December 1987.
38. Friedlander, A.H. and West, L.J.: Dental management of the patient with major depression. Oral Surg Oral Med Oral Pathol, 71:573-578, May 1991.
39. Clemmesen, L.: Anticholinergic side-effects of antidepressants: Studies of the inhibition of salivation. Acta Psychiatr Scand Suppl, 345:90-93, 1988.
40. Grant, D.A.; Stern, I.B.; Listgarten, M.A.: *Periodontics*. St. Louis: C.V. Mosby Co., 1988, pp 135-146.
41. Covey, L.S. and Tam, D.: Depressive mood, the single parent home, and adolescent cigarette smoking. Am J Public Health, 80:1330-1333, November 1990.
42. Jones, J.K. and Triplett, R.G.: The relationship of cigarette smoking to impaired intraoral wound healing: A review of evidence. J Oral Maxillofac Surg, 50:237-293, March 1992.
43. Tversky, J.; Reade, P.C.; Gerschman, J.A.: Role of depressive illness in the outcome of treatment of temporomandibular joint pain-dysfunction syndrome. Oral Surg Oral Med Oral Pathol, 71:696-699, June 1991.
44. Friedlander, A.H.; Mills, M.J.; Gorelick, D.A.: Alcoholism and dental management. Oral Surg Oral Med Oral Pathol, 63:42-46, January 1987.
45. Yagiela, J.A.: Local anesthetics. In: Neidle, E.A. and Yagiela, J.A. eds: *Pharmacology and Therapeutics for Dentistry*, 3rd ed. St. Louis: C.V. Mosby Co., 1989, pp 230-248.
46. Yagiela, J.A.; Duffin, S.R.; Hunt, L.M.: Drug interaction and vasoconstrictors used in local anesthetic solutions. Oral Surg Oral Med Oral Path, 59:565-571, June 1985.
47. Malamed, S.F.: *Handbook of local anesthesia*, ed. 2. St. Louis: C.V. Mosby Co., 1986.
48. Hersh, E.V.; Helpin, M.L.; Evans, O.B.: Local anesthetic mortality: Report of a case. J Dent Child, 58:489-491, November-December 1991.
49. Baldessarini, R.J.: Drugs and the treatment of psychiatric disorders. In: Gilman, A.G.; Rall, T.W.; Nies, A.S. (eds.) *et al: Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th ed. New York: Pergamon Press, 1990, pp 383-435.
50. Jastak, J.T. and Yagiela, J.A.: Vasoconstrictors and local anesthesia: A review and rationale for use. JADA, 107:623-630, October 1983.
51. Meyer, D. and Halfin, V.: Toxity secondary to meperidine in patients on monoamine oxidase inhibitors: a case report and critical review. Psychopharmacol, 1:319-321, February 1981.

SUICIDE: RISK FACTORS

The most common of [such] presuicide diagnoses are alcohol abuse, depression, an anxiety disorder, and aggressive behavior. Alcohol abuse seems especially important. In one study of adults, alcohol abuse was second only to depression as a risk factor for suicide; in a study of adolescents, approximately two thirds of 17- to 19-year-old male suicides were reported to drink excessively. Psychological autopsies have been complemented by a number of small-scale biological autopsies that have consistently identified systematic differences between suicides and control subjects in their markers of central nervous system serotonin function. Similar differences have been observed between highly aggressive and impulsive individuals and control subjects.

Editorial. Am J Public Health, 83:171, February, 1993.

EPIDEMIOLOGY

The poisoning of our children

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

In 1991, more than 1.8 million poisonings were reported to the American Association of Poison Control Centers, including 1.1 million poisonings of children under five years of age and forty-four deaths of children under six years of age. "Extrapolating from reported cases to actual poisonings allows us to estimate that 2.75 million children under five years, and 3.16 million children under 18 years, were actually poisoned accidently in 1989."[1]

Almost all situations were accidental. Almost all incidents resulted in minor damage to the child. A series of reports from the Association of Poison Control Centers for the period since the mid-1980s provide an opportunity for all health practitioners who provide services to youngsters to alert parents and guardians on the nature and extent of the dangers that threaten children of all ages (and even oldsters*).[2-9]

SOURCE OF INFORMATION

A national data collection system was established in 1983 by the American Association of Poison Control Centers. Since its inception there has been a progressive increase in the number of participating centers, populations served by these centers, and the number of reported human poison exposures (increasing from a quarter of a million cases in 1983 to 1.7 million in 1990).** In 1990, there were seventy-two reporting centers serving most of the population in the nation (191.7 million}.*** Case volumes per poison control center ranged from 606 to 71,515 reports, with a mean of 8.9 reported exposures per thousand population. Extrapolating from the covered population and the number of cases, it is estimated that there were more than 2.2 million poison exposures reported to all U.S. poison centers.[6]

NUMBER OF CASES

There were almost eight hundred thousand cases of poisoning reported for children less than three years of age. Almost a half (46 percent) of all cases of poisoning for the entire population covered in the report were reported for these young children (Figure). Two-year-old children had the highest number of reported poisonings (a third of a million cases). There were more reported cases of poisoning in males less than thirteen years of age than their female counterparts. The relationship was reversed for the population thirteen years

* As I began to write this material, I bent down to pick up a "piece of dirt" from the rug in my office. The "piece of dirt" (i.e. a bee) stung me on the palm of my hand. As it can be imagined, there was a slight delay before continuing with this presentation while I dealt with my own case of poisoning (one of the millions of cases of poisoning that were not reported to poison control centers).

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** Unless otherwise stated, data in this report are for 1990, the last year for which extensive published data are available.

*** Data do not include information from the states of AL, CT, DE, HI, IL, LA ME, ND, OK, SC, VT; some areas of AR,IA, MS, NC, NV, TX; and minor sections of CA, NY, OH, TN, WI.

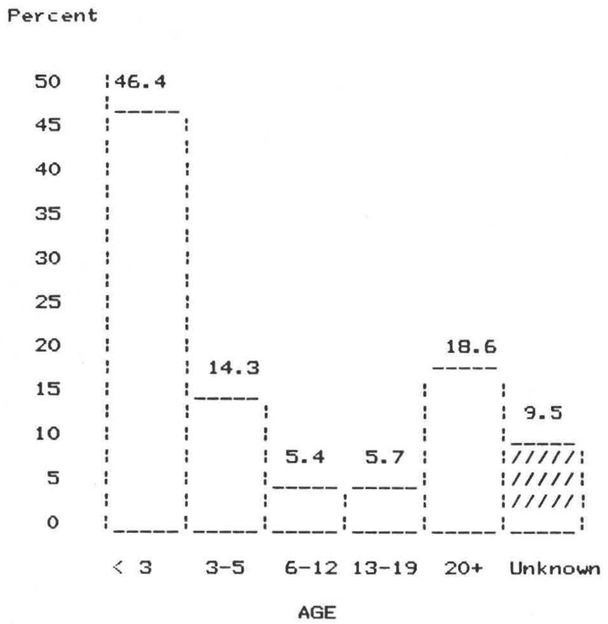

Figure. Distribution of poison exposure cases by age: 1990[6]

Table 1 □ Distribution of poison exposure cases by age and gender: 1990.(6)

Age	Male	Female	Totals*	Percent	Cumulative percent
< 3 yrs.	419,436	369,773	794,783	46.4%	46.4%
3-5 yrs.	134,141	110,626	246,473	14.3	60.8
6-12 yrs.	51,797	39,437	91,911	5.4	66.1
13-19 yrs.	40,752	57,074	98,243	5.7	71.9
Subtotal	646,126	576,910	1,231,410		71.9
20 + yrs.	137,733	180,284	319,030	18.6	90.5
Unknown	66,050	88,727	163,022	9.5	100%
Totals*	848,909	845,921	1,713,462	100%	100%

* Includes unknown gender or age.

Table 2 □ Distribution of poison exposure cases by age and cause: 1990.(6)

	Less than 6 years		6-12 years		13-17 years	18 + years	Totals*
Cause	No.	Perc.	No.	Perc.	Percent	Percent	Percent
Accidental	1,032,959	60.3%	85,499	5.0%	2.2%	13.4%	88.1%
Intentional	2,010	0.1	5,423	0.3	2.2	5.9	10.0
Adverse reaction	2,924	0.2	1,563	0.1	0.1	.8	1.5
Unknown	1,274	0.1	535	<0.1	<0.1	.2	0.5
Totals	1,039,167	60.6%	93,020	5.4%	4.5%	20.3%	100%

* Includes unknown age.

and older. In terms of the total population, there were minor differences in the rates of reported cases by gender (Table 1).

In 1985 through 1989, there were 3.8 million poison exposures involving children less than six years of age that were reported to poison control centers. Of this number, 2,117 patients experienced a major outcome (life threatening effect or residual disability) and an additional 111 fatalities occurred.[7]

CAUSE AND OUTCOME

The overwhelming number of the more than a million reported poisoning cases of children less than six years of age resulted from accidental exposure, 2,000 resulted from intentional actions and 3,000 resulted from adverse reactions. Similar findings were reported by older children and adults. Overall, 88 percent of poisonings were accidental (Table 2).

The vast majority of poisoning cases resulted in "no effects" or "minor effects" in children less than thirteen years of age. Less than a tenth of one percent of cases resulted in the death of the child (Table 3).

Fatalities

Twenty-five of the twenty-seven fatalities resulting from poisoning of children less than thirteen years of age were as a result of an accidental exposure. Twenty-five

The list of substances involved in the poisoning of children appears to be endless.

Table 3 □ Outcome of poison exposure by age: 1990.[6]

Outcome	< 6 yrs.	6-12 yrs.
No effect	38.9%	24.7%
Minor effect	13.2	29.1
Moderate effect	.4	1.7
Major effect	< .1	.1
Death	< .1	< .1
Other*	47.4	44.4
Totals	100%	100%

* Includes no follow-up and unknown.

Table 4 □ Cause of fatalities from poisoning by age: 1990.[6]

Age	Accident	Intentional	Adverse reaction	Unknown	Totals
< 6 yrs.	24		1		25
6-12 yrs.	1	1			2
13-17 yrs.		33			33
17 + yrs.	67	428	9	48	552
Totals	92	462	10		612

Table 5 □ Poison exposure by age and substance: 1990.[6]

Substance	Less than 6 years	6-11 years
Nonpharmaceuticals	(5,000 or more cases)	(1,000 or more cases)
Adhesives/glues	x	x
Alcohols	x	x
Arts/crafts office supplies	x	x
Batteries		x
Bites and venoms	x	x
Chemicals	x	x
Household cleaners	x	x
Cosmetic/personal care products	x	x
Deodorizers	x	
Essential oils	x	
Fertilizers	x	
Food products and poisoning	x	x
Foreign bodies/toys	x	x
Fumes/gases/vapors		x
Hydrocarbons	x	x
Insecticides	x	x
Lacrimators		x
Moth repellants	x	
Mushrooms	x	
Paints and stripping agents	x	x
Plants	x	x
Polishes and waxes	x	
Rodenticides	x	
Tobacco	x	
Pharmaceuticals		
Analgesics	x	x
Antihistamines	x	x
Antimicrobials	x	x
Asthma therapies	x	x
Cardiocvascular drugs	x	
Cough/cold preparations	x	x
Electrolytes/minerals	x	
Eye/ear/nose/throat preparations	x	
Gastrointestinal preparations	x	x
Hormones	x	
Sedatives/hypnotics/antipsychotics	x	x
Stimulants/street drugs		x
Topicals	x	x
Vitamins	x	x

of the twenty-seven fatalities involved children less than six years of age (Table 4).

TYPES OF AGENTS

The list of pharmaceutical and nonpharmaceutical substances involved in the poisoning of children appears to be endless. Seemingly anything in, or around the place of residence, recreation, or work, can and has resulted in the poisoning of youngsters and adults. The highest incidence of poisoning of children less than six years of age resulted from the misuse of cosmetics (121,000 reported cases) and cleaning substances (113,000 reported cases). The highest incidence of poisoning of children age six to eleven was reported for bites (13,000 reported cases) and cleaning substances (10,000 reported cases) (Table 5). From 1985 through 1989, cosmetics and personal care products, cleaning substances and plants accounted for 30 percent of reported poisonings of children.[7]

The enumeration of substances most frequently implicated in poison exposures is one of the most com-

For children under six years of age, cosmetics was the most frequent offender.

monly used reporting techniques. As previously noted, most instances of poisoning, however, produce "minor effects." Thus, "...frequent exposure does not imply toxicity."[7] A more "... sound approach... (would be to) concentrate efforts on those household products that are likely to truly harm a small child who is unintentionally exposed." For example,

- ☐ Poison control centers receive more than a hundred thousand calls each year about children who have eaten some type of outdoor plant. In a vast majority of those cases, children suffered minor stomach problems or no ill effects.[8]
- ☐ Almost half of the nation's children will ingest something by the age of six that a parent believes may have been poisonous. In a vast majority of these instances, no major illness or fatality occurs.[8]
- ☐ In the second half of the 1980s, iron supplements were the single most frequent cause (30 percent) of pediatric unintentional pharmaceutical ingestion fatalities. This high fatality rate is likely the result of multiple factors, including the availability of prenatal vitamins and iron supplements in homes, and the similar appearance of many brightly colored iron tablets to popular candies.[7]

THERAPY

In the majority of cases, (approximately a million reports) dilutions, irrigation and washing were the therapies used in poison exposure cases. Activated charcoal, ipecac syrup, and cathartics were the next most frequently used form of therapy (Table 6).

THE ROLE OF PEDIATRIC DENTISTS

The network of regional poison control centers was established to reduce poisoning, educate people about hazardous exposures, and provide expert information to professionals and the public. It was estimated that by handling nontoxic and mildly toxic poisoning exposures by telephone consultations, $35 million are saved annually in New York State.[9]

Pediatric practitioners (both medical and dental) can play a critical role in alerting parents to the dangers of poisoning and the availability of professional help by telephone consultation. Pediatric dentists increasingly are making efforts to provide services to younger patients. "...(the American Academy of Pediatric Dentistry) recommend(s) a dental health objective urging early parental counseling and dental/oral examination for all infants, prior to 12 months of age."[10] If we advocate counseling and preventive services, then we cannot overlook "the poisoning of our children."

Is the telephone number of your regional poison control center listed in the office (and your home, too) for you, your staff and your family?

Table 6 ☐ Therapy provided in poison exposure cases: 1990.[6]

Therapy	Number of cases
Initial decontamination	
Dilution	654,620
Irrigation/washing	332,964
Activated charcoal	114,324
Ipecac syrup	104,731
Cathartic	94,838
Gastric lavage	49,510
Other emetics	4,101
Measures to enhance elimination	6,600
Specific antidotes	16,537

REFERENCES

1. T.L. Litovitz, President, American Association of Poison Control Centers; in Dear Abby, Newsday, June 5, 1992, p104.
2. Veltri, J.C. and Litovitz, T.L.: 1983 annual report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med, 2:420-443, September 1984.
3. Litovitz, T.L.; Normann, S.A.; Veltri, J.C.: 1985 annual report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med, 4:427-458, September 1986.
4. Litovitz, T.L.; Schmitz, B.F.; Matyunas, N. *et al*: 1987 annual report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med, 6:479-515, September 1988.
5. Litovitz, T.L.; Schmitz, B.F.; Bailey, K.M.: 1989 annual report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med, 8394-8442, September 1990.
6. Litovitz, T.L.; Bailey, K.M.; Schmitz, Holm, K.C. *et al*: 1990 annual report of the American Association of Poison Control Centers National Data Collection System. Amer J Emerg Med, 9:461-509, September 1991.
7. Litovitz, T.L. and Manoguerra, A.: Comparison of pediatric poisoning hazards: an analysis of 3.8 million exposure incidents. Pediatrics, 89:999-1006, June 1992.
8. Iron pills lead list in killing the young. NY Times, June 9, 1992. pC9.
9. Anonymous: Poison control. NY State J Med, 90:474-475, September 1990.
10. Dental health objectives for children for the year 2000: Statement of the American Academy of Pediatric Dentistry. Chicago: American Academy of Pediatric Dentistry, 1989.

Is your next pediatric patient an addict?

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The picture shows a dentist bending over an adult patient and applying cardio-pulmonary resuscitation procedures. The accompanying copy reads:

"Before giving lidocaine, ask about cocaine, or you may be pressing more than your luck."[1]

This public service message (in the Journal of the American Dental Association) stressed the relationship between the vasoconstrictor effects of cocaine and the use of local anesthetics. Surely, most dental practitioners are aware of the plague of drugs that confronts our nation. Few dentists may be aware, however, of the extent to which children use both illicit drugs and drugs that are legal, but only for older populations. For example, in 1990 more than one out of five youngsters (ages 12-17) had used illicit drugs and almost a half had used alcohol.[2] Given this reported wide use of drugs, the public service message, noted above, could just as well have pictured a dentist attempting to save the life of a child who had experienced the adverse effects of drug interactions.

The following presentation will review some of the results of the latest national study on drug abuse in an effort to alert practitioners to the realities that, "your next pediatric patient may be an addict."

SOURCE OF DATA

The 1990 National Household Survey on Drug Abuse, carried out by the National Institute on Drug Abuse, was the tenth in a series of household interviews of the U.S. population (not including Alaska and Hawaii) aged twelve and older. The sample design was a multistage area probability sample. There were 9,259 completed interviews (an interview response rate of 82 percent).[2,3] Other federal agency reports will be used to supplement the findings from the National Household Survey.

THE FINDINGS

First the good news:

- ☐ The percent of children between twelve and seventeen years of age* who reported the use of illicit drugs in the past month decreased from 17.6 percent in 1979 to 8.1 percent in 1990.[2]

And now a bit of reality:

- ☐ The number of cocaine related emergency room episodes for children more than tripled (from 255 to 801) between 1985 and 1989 (in a sample of emergency rooms in 21 metropolitan areas). Increases were reported for males, females, whites, blacks, and Hispanics (Table 1).
- ☐ While the number of actual deaths from drug abuse were not that "high" for youngsters, medical examiners continue to report drug related deaths of children in their preteen and early teen years (Table 2).

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* Unless otherwise specified, the data in this presentation refers to children ages 12-17 years.

Table 1 ☐ Cocaine (including crack) related emergency room episodes by race, ethnicity and gender for children age 12-17 in selected metropolitan areas: 1985-1989.[4]

	1985	1987	1989
Totals*	225	668	801
White			
Male	77	155	195
Female	66	118	135
Black			
Male	37	141	191
Female	26	104	99
Hispanic			
Male	21	62	70
Female	10	27	36

* Includes unknown race/ethnicity and/or gender.

Table 2 ☐ Number of drug abuse deaths in 27 metropolitan areas* as reported by medical examiners for children 10-17 years of age: 1987.[5]

Age	Male	Female	Totals
10-14 yrs.	8	4	12
15-17 yrs.	22	25	47

* Not including New York metropolitan area.

Table 3 ☐ Number of arrests of children for drug abuse violations by gender, age and place of residence: 1990.[6]

	< 10	10-12	13-14	Age 15	16	17	Totals
Gender							
Male	169	710	6,544	9,431	16,162	24,441	57,457
Female	24	155	1,156	1,357	1,931	2,660	7,283
Residence							
City	149	740	6,800	9,323	15,615	22,771	55,398
Suburban	33	92	703	1,142	1,942	3,282	7,194
Rural	11	33	197	323	536	1,048	2,148
Totals	193	865	7,700	10,788	18,093	27,101	64,740

Note: 10,206 agencies for a 1990 population of 193,507,000.

☐ In 1990 there were about 65 thousand arrests of children for drug abuse violations (as reported by 10,206 agencies for a population of 193,507,000). These arrests included more than one thousand children less than thirteen years of age and more than eight thousand children less than 15 years of age. While most of these arrests involved males and residents of city areas, thousands of females and residents of suburban and rural areas also were arrested (Table 3).**

☐ During the 1970s, there was a general increase in the percent of children reporting the use of illicit drugs and the nonmedical use of psychotherapeutic drugs. During the 1980s, however, the trend was reversed.

Nevertheless, in 1990, almost 15 percent of youngsters reported they had used marijuana (11 percent during the past year).

Inhalants (gasoline, spray paints, glue, aerosol sprays [e.g. Pam and locker room odorizers], lacquer thinners, amyl nitrites, nitrous oxide, and correction fluids) were another "popular" item for about 8 percent of youngsters (4 percent during the past year).

In addition, 10 percent of the children had used psychotherapeutic drugs for nonmedical purposes (7 percent in the past year).

☐ Almost half of the youngsters consumed alcohol (41 percent in the past year) and 40 percent had smoked cigarettes (22 percent in the past year) (Tables 4 and 5). The use of alcohol by youngsters was reported equally by males and females, almost twice as frequently by whites as by blacks, greater in large metropolitan areas than in other population areas and equally throughout all regions of the nation (Table 6).

Reports of smoking in the past year by youngsters were highest among males, whites (more than 2.5 times greater than blacks) in nonmetropolitan areas and in the North Central region of the nation (lowest in the West) (Table 6).

**For a more extensive review of the arrests of children, see a previous presentation in the Journal of Dentistry for Children.[7]

In 1990, 65 thousand children were arrested for drug abuse.

Table 4 ☐ Percent of children aged 12-17 reporting drug use during their lifetime: 1972-1990.[2,3]

	1972	1976	1979	1982	1985	1990
Use of any illicit drug	na	na	34.3%	27.6*	29.5%	22.7%
Marijuana/ hashish	14.0%	22.4%	30.9	26.7	23.6	14.8
Cocaine (including crack)	1.5	3.4	5.4	6.5	4.9	2.6
Inhalants	6.4	8.1	9.8	na	9.2	7.8
Hallucinogens	4.8	5.1	7.1	5.2	3.3	3.3
Heroin	0.6	0.5	0.5	na	na	0.7
Nonmedical use of any psychotherapeutics	na	na	7.9	10.3	12.1	10.2
Stimulants	4.0	4.4	3.4	6.7	5.6	4.5
Sedatives	3.0	2.8	3.2	5.8	4.1	3.3
Tranquilizers	3.0	3.3	4.1	4.9	4.8	2.7
Analgesics	na	na	3.2	4.2	5.8	6.5
Alcohol	na	53.6	70.3	65.2	55.5	48.2
Cigarettes	na	45.5	54.1	49.5	45.2	40.2

* Not including inhalants.

Table 5 ☐ Percent of children aged 12-17 reporting drug use in the past year: 1976-1990.[2,3]

	1976	1979	1982	1985	1990
Use of any illicit drug	na	26.0%	22.0*	23.7%	15.9%
Marijuana/ hashish	18.4%	24.1	20.6	19.7	11.3
Cocaine (including crack)	2.3	4.2	4.1	4.0	2.2
Inhalants	2.9	4.6	na	5.1	4.0
Hallucinogens	2.8	4.7	3.6	2.7	2.4
Heroin	na	na	na	na	0.6
Nonmedical use of any psychotherapeutics	na	5.6	8.3	8.5	7.0
Stimulants	2.2	2.9	5.6	4.3	3.0
Sedatives	1.2	2.2	3.7	2.9	2.2
Tranquilizers	1.8	2.7	3.3	3.4	1.5
Analgesics	na	2.2	3.7	3.8	4.8
Alcohol	49.3	53.6	52.4	51.7	41.0
Cigarettes	na	13.3	24.8	25.8	22.2

* Not including inhalants.

Table 6 ☐ Percent of children age 12-17 reporting the use of alcohol and cigarettes in the past year, by various demographic characteristics: 1990.[2]

	Alcohol	Cigarettes
Total	41.0%	22.2%
Gender		
Male	40.8	23.6
Female	41.1	20.8
Race/ethnicity		
White	45.6	25.9
Black	24.7	9.8
Hispanic	37.9	19.4
Population density		
Large metropolitan	42.6	20.4
Small metropolitan	40.4	21.5
Nonmetropolitan	38.9	26.2
Region		
Northeast	41.2	21.9
North Central	42.6	25.9
South	40.2	23.1
West	40.0	16.9

Table 7 ☐ Percent of children age 12-17 reporting use of any illicit drugs in the past year by demographic characteristics: 1988-1990.[2]

	1988	1990
Totals	16.8%	15.9%
Gender		
Male	15.8	14.7
Female	17.9	17.3
Race/Ethnicity		
White	17.8	16.9
Black	12.1	12.7
Hispanic	16.3	17.0
Population density		
Large metropolitan	18.6	14.5
Small metropolitan	15.8	18.9
Nonmetropolitan	15.5	14.5
Region		
Northeast	17.7	12.8
North Central	15.3	19.3
South	15.9	14.2
West	20.5	17.8

In addition, 11.8 percent of youngsters (21.9 percent of males and 1.2 percent of females) reported the use of smokeless tobacco.[2]

☐ In 1990 a greater percent of females than males reported use of illicit drugs. A smaller percent of black children than white or Hispanic children reported the use of illicit drugs. A greater percent of children in small metropolitan areas than in large metropolitan and nonmetropolitan areas reported the use of illicit drugs. The illicit use of drugs by children was reported to be highest in the North Central and lowest in the Northeast (Table 7).

☐ 28.4 percent of youngsters reported the use of both illicit drugs and alcohol. 1.5 percent reported the use of drugs with needles.[2]

☐ The average age when illicit drugs were first used was between twelve and fourteen years. Cigarettes were first smoked at age 11.5 and alcohol first was used before thirteen years of age.[2]

☐ As a result of the use of illicit drugs and alcohol, 17.5 percent of youngsters reported that during the past year they had one or more serious problems, including depression, general health difficulties, emergency medical help, difficulties in thinking clearly, and getting school work accomplished.[2]

☐ A quarter of youths who drank alcohol had experiences of being "unable to remember what happened." One of five took "quick drinks" when no one was looking and 13 percent had been high on alcohol at school.

Child drug users are at risk for depression, general health difficulties, emergency medical help, unclear thinking, failure to complete school work.

☐ And most worrisome, was the percent of children who thought the following activities were not of great risk:

☐Smoking marijuana occasionally, 48 percent.

☐Try cocaine once or twice, 45 percent.

☐Try phencyclidine (PCP or angel dust, an hallucinogen) once or twice, 51 percent.

☐Try heroin once or twice, 51 percent.

☐Occasional use of anabolic steroids, 47 percent.

☐One or two drinks of alcohol nearly every day, 65 percent.

☐Smoke one or two packs of cigarettes per day, 52 percent.

GENERAL THOUGHTS

During the past twenty years, I have presented a course of studies for undergraduate college students at Stony Brook: an analysis of the health care system. Each year I have asked for a show of hands on the number of students who have never consumed an illicit drug. Five, maybe ten students out of a class of 110-125 students will raise their hands. Although I realize that peer pressure, shame or embarrassment may affect the numbers, it is scary to think that 90 percent or more of these eighteen to twenty-year-old students are willing to indicate that some time in their past (or present) they consumed illicit drugs. But of equal (even more?) concern is the fact that at an average age of twelve to fourteen years, children are exploring the use of illicit drugs.

It is all too easy to be lulled into complacency with reports that indicate that since the early 1980s, the use of drugs and alcohol by youngsters has been decreasing. The reality is that 16 percent have used illicit drugs in the past year, 8 percent in the past month. In addition, 14.1 percent have used both alcohol and illicit drugs in the past year.[2]

With all the difficulties one encounters in the care of children, it seems almost beyond reason to be required to add to the problems by suggesting that you assume that your next twelve-year-old patient may be a drug "pusher" or user. Unfortunately, the reality is that your next patient could be an addict. Are you willing to take the chance and think otherwise?

REFERENCES

1. Public service announcement. J Amer Dent Assoc, 123:128, March 1992.
2. Department of Health and Human Services, National Institute on Drug Abuse. National Household Survey on Drug Abuse: Main findings, 1990. DHHS Pub. No. (ADM) 91-1788. Rockville, MD, Alcohol, Drug Abuse and Mental Health Administration, 1991.
3. Department of Health and Human Services, National Institute on Drug Abuse: Highlights 1990. National Household Survey on Drug Abuse: Main findings, 1990. DHHS Pub. No. {ADM} 91-1789. Rockville, MD, Alcohol, Drug Abuse and Mental Health Administration, 1991.
4. Department of Health and Human Services, Centers for Disease Control. Health United States 1990. DHHS Pub. No. (PHS) 91-1232. Hyattsville, MD, National Center for Health Statistics, 1991.
5. Department of Health and Human Services, Division of Disadvantaged Assistance. Health Status of the Disadvantaged: Chartbook 1990. DHHS Pub. No. (HRSA) HRS-P-DV-91. Washington, D.C. Government Printing Office, 1991.
6. Department of Justice, Federal Bureau of Investigation. Uniform Crime Reports for the United States. Washington, D.C. Government Printing Office, 1991.
7. Waldman, H.B.: Juvenile arrests - it's a crime. J Dent Child, 59:373-375, September-October 1992.

DEMOGRAPHICS

Gender trends among specialists in pediatric dentistry

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Ceib L. Phillips, PhD, MPH

In 1991, 36.3 percent of the entering dental school students were women.[1] This reflects a decrease of 3.5 percent over the previous year, but the long-term trend is for more women to enter dentistry. It has been projected that 15 percent of dentists in the United States by the year 2000 and 30 percent by 2030 will be women.[2,3] The number of women entering postdoctoral studies in pediatric dentistry surpassed 50 percent of first-year students in 1990.[4]

Studies point to gender differences in career patterns among professionals, including dentists.[5-7] Professional women tend to work part-time more often than men and have shorter professional careers.[8,9] The impact of a greater number of women entering dentistry on patient care availability has been investigated.[6,7,10] The purpose of our study was to examine the gender and practice trends among dentists who specialize in the care of children.

METHODS

A questionnaire was mailed to 2760 pediatric dentists in the United States who were members of the American Academy of Pediatric Dentistry (AAPD). The gender distribution of the AAPD membership was 79.5 percent males and 20.5 percent females.

We requested specific information regarding gender, age, primary practice site and number of hours per week devoted to patient care. No follow-up correspondence was sent to nonresponding individuals. The Cochran-Mantel-Haenszel row mean score test was used to determine whether the distribution of time spent was different for females and males. Time spent in patient care was considered ordinal because the participants responded to categories of time. The Chi square test was used to evaluate gender preference for practice site. Level of significance was at $P=0.05$.

RESULTS

We attempted to minimize sampling bias by including in our survey all pediatric dentists in the United States who were members of the AAPD. Respondent bias in surveys is more difficult to address. The percentage and ages of male and female responders were similar, however, to the membership profile of the AAPD.

We received 1266 anonymously completed questionnaires (1053 male responders and 213 female responders). The mean age of male responders was 45.0 years and 36.9 years for female responders. Table 1 gives the percentage of males and females in each practice site. Over 75 percent of all respondents were in private practice.

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Table 1 ☐ Practice site by gender.

	Male		Female	
	%	Number	%	Number
Private practice	90.1	949	78.9	168
University/Hospital	9.2	168	19.7	42
Institution	0.7	7	1.4	3

The number of hours practiced per week by gender and practice site are shown in Table 2. There were only ten (seven males and three females) who identified their principle site of practice to be an institution. For statistical analysis these were combined with the university/hospital group.

In our sample, females in private practice work, on an average, only slightly fewer hours per week providing patient care than do males. There was a statistically significant difference between males and females, however, in the distribution of the work-week length. Twenty-seven percent of the female respondents work thirty hours or less per week, while only 8 percent of the males reported this length of work-week.

The distribution of time spent in patient care among pediatric dentists who identified universities, hospitals, or institutions as their primary practice location is also given in Table 2. Females reported more hours per week spent in patient care than did the males in these settings. The difference in work week distribution between men and women was not significant (p=0.06), although 47 percent of females reported working thirty or more hours per week compared to only 35 percent of males. The reduced sample size for those indicating site of practice other than a private setting prohibited the detection of this 12 percent difference as being significant.

Table 2 ☐ Hours practiced.

Private practice				
Number*	Male		Female	
of hours	%	Number	%	Number
>40	13.8	131	11.9	20
30-40	78.4	744	60.7	102
20-30	6.5	62	20.2	34
10-20	0.7	7	5.4	9
<10	0.6	5	1.8	3
Mean number of hours/wk		34.7		32.1

* Males and females significantly different (p = 0.0001) in distribution of number of hours (Cochran-Mantel-Haenszel)

University/hospital/institution				
Number*	Male		Female	
of hours	%	Number	%	Number
>40	18.6	18	23.9	11
30-40	16.4	17	23.8	11
20-30	9.3	9	11.9	5
10-20	52.6	54	38.1	17
<10	3.1	6	2.4	1
Mean number of hours/wk		22.9		26.9

* Males and females are not significantly different (p = 0.06) in distribution of number of hours (Cochran-Mantel-Haenszel)

Table 3 ☐ Type of practice.

	Private practice*		Other	
	%	Number	%	Number
Male	85.0	949	15.0	168
Female	79.6	175	20.4	45

* Proportion of males and females in private practice are significantly different (p = 0.04; Chi-square)

Males reported providing patient care in a private practice setting significantly more often than did females (Table 3). The large number of respondents permitted detection of a 5 percent difference between the genders. This suggests that university/hospital/institutions are selected more often by females than males, but the difference is subtle.

In 1991, 55 percent of students enrolled in graduate pediatric dental programs were women.

DISCUSSION

The number of pediatric dentists being trained each year has remained relatively constant for the past twenty years. The number of women entering the specialty has been increasing consistently, however, in the past few years. In 1990 the number of women enrolled in pediatric dentistry graduate programs exceeded 50 percent for the first time and rose to 55 percent in 1991.[12]

There have been contradictory reports regarding the number of hours practiced per week and weeks per year by female dentists compared to males.[13] These studies did not distinguish between general dentists and specialists. What is the experience of female pediatric dentists? Do women choose a different practice profile than males? If so, does this result in reduced availability of specialty care to children? These are very relevant questions which need to be addressed.

The difference in number of hours devoted to patient care is only slightly less among female than male pediatric dentists in private practice, although the practice profile appears to be different. It has been claimed in the past that females take more, and longer, leaves of absence from practice, however, than do males.[8-14] This was due primarily to maternity leave, illness, and child rearing. The extent of difference has been challenged recently, but the issue is not resolved.[12] Potential of responder bias cannot be directly evaluated in studies such as this one. The percentage of males and females who responded and the mean ages of the respondents are quite similar to the distribution of the AAPD membership. We have no reason to believe that this sample is not representative of the AAPD membership.

The finding that a greater percentage of women practice in university/hospital/institution sites was consistent with previous reports.[6] The inverse relationship of time spent in patient care between females and males in universities, hospitals, and institutions could have been anticipated. Clinic schedules in these facilities are usually well defined and not subject to significant change by the attending staff. The most senior staff often have collateral administrative duties, while more junior staff are assigned primarily clinical responsibilities. The significant increase of women entering the specialty of pediatric dentistry has been a rather recent event. Consequently, a greater number of males hold senior staff positions than women. The number of hours dedicated to direct patient care by the two genders will probably become relatively equal in the future as women gain seniority.

The impact of a greater number of women entering the practice of pediatric dentistry and the reciprocal fewer number of men poses a number of interesting yet unanswered questions. Periodic surveys of the specialty over the next few years are indicated to provide the data necessary to determine whether there are gender-related issues that require addressing by the profession.

REFERENCES

1. Council on Dental Education, American Dental Association. Annual Report, Chicago, IL, 1991-92.
2. Inglehart, J.K.: Datawatch: trends in health personnel. Health Affairs, 5:128-137, Winter 1986.
3. Waggoner, W.F.: Women dentists: impact, trends and implications. J Am Coll Dent, 58:11, Fall 1991.
4. Personal communication: American Academy of Pediatric Dentistry, Chicago, IL, 1991.
5. Bielby, D.D. and Bielby, W.T.: Work commitment, sex-role attitudes, and women's employment. Am Soc Rev ,49:234-247, April 1984.
6. Dolan, T.A. and Lewis, C.E.: Gender trends in the career patterns of recent dental graduates. J Dent Ed, 51:639-645, November 1987.
7. Wilson, A.A.; Branch, L.G.; Niessen, L.C.: Practice patterns of male and female dentists. J Am Dent Assoc, 116:173-177, February 1988.
8. Mitchell, J.B.: Why do women physicians work fewer hours than men physicians? Inquiry, 21:361-368, Winter 1984.
9. LeBailly, S.A. and Brotherton, S.E.: Career paths of men and women in pediatrics. Descriptive findings from the survey on pediatric careers. Working paper #15. Department of Research, American Academy of Pediatrics, Elk Grove, IL, 1988.
10. Bureau of Economic and Behavioral Research, Council on Dental Practice, American Dental Association. *A comparative study of male and female dental practice patterns*. Chicago: Am Dent Assoc, May 1989.
11. Fleiss, J.L.: *Statistical methods for rates and proportions*. 2nd ed., New York: John Wiley and Sons, 1981, pp 143-147.
12. Council on Dental Education, Department of Educational Surveys, American Dental Association. *Minority Enrollment in Advanced Dental Education Programs*. Chicago: Am Dent Assoc, February 1992.
13. Dolan, T.A.: Gender trends in dental practice patterns. J Am Coll Dent, 58:12-18, Fall 1991.
14. Tillman, R.S. and Horowitz, S.L.: Practice patterns of recent female dental graduates. J Am Dent Assoc, 107:32-35, July 1983.

REPORT

Adverse reaction to a fissure sealant: Report of case

Ulla Hallström, DDS

The sealing of fissures with a resin material, such as Bowen's (Bis-GMA), has been practiced since the early seventies. The procedure has been used successfully for prophylactic treatment of permanent molars, is widely recognized, and often delegated to dental auxiliaries.[1]

ADVERSE REACTIONS

Adverse reactions to dental materials have been increasingly reported and discussed. In Sweden, interest has focused mainly on reactions to amalgam and mercury, while reactions to dental resin materials have been reported by Hensten-Pettersen; Nathansson and Lockhart; Lind; and Katz.[2-4,6]

Allergic reactions to various substances are reported to be increasing in children.[6]

CASE REPORT

The four first permanent molars of a six-year-old girl (MF) were sealed around ten o'clock on day 1 with Delton (Johnson & Johnson), a chemically cured fissure sealant material. The treatment was performed with a well-known technique and no complications were noticed.

On the night of day 1, the girl began to show signs of asthmatic reaction, identical to what she had experienced before in an allergic reaction to mites. This continued during day 2; during the night between days 2 and 3, a severe urticaria with rashes and swellings covered the entire body, including the arms and legs, followed by blistering of the face, ears, and lips.

The family consulted the Children's Clinic of the local hospital on day 3. The urticaria and a bronchial reaction were considered to be of allergic origin. A dose of a cortisone preparation was given immediately: Betapred 0.5 mg, eight tablets, supported with a bronchodilating inhalator, Ventoline.

On day 4, the rashes were, if anything, worse. A four-day medication with Betapred 0.5 mg, totally thirty tablets, was prescribed. The parents questioned whether the dental treatment, performed a few days earlier, might have caused the condition of their daughter. They consulted their daughter's dentist on day 4. She referred the girl to the Special Clinic of Children's Dentistry in Trelleborg and the girl was seen in our clinic on day 5 at 10 o'clock.

She is a six-year-old girl, in good health except for a bronchial asthmatic allergy to mites. This started about six months ago in connection with a cold. Her asthma recurs during infections. She has been free from it for the last few months, until it started again in the night of day 1. She also has blisters, swellings and rashes on her arms, legs and body, face, ears and lips (figures 1 and 2). The rashes are very itchy.

Intraorally there is a blistery lesion on the gingiva, distal of 36 and a few white striae on the buccal mucosa in the region of 26 and 36 (Figure 3).

After a careful discussion with the girl, her parents and her pediatrician, a connection between the adverse

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Figure 1. Urticaria of the forearm and palm.

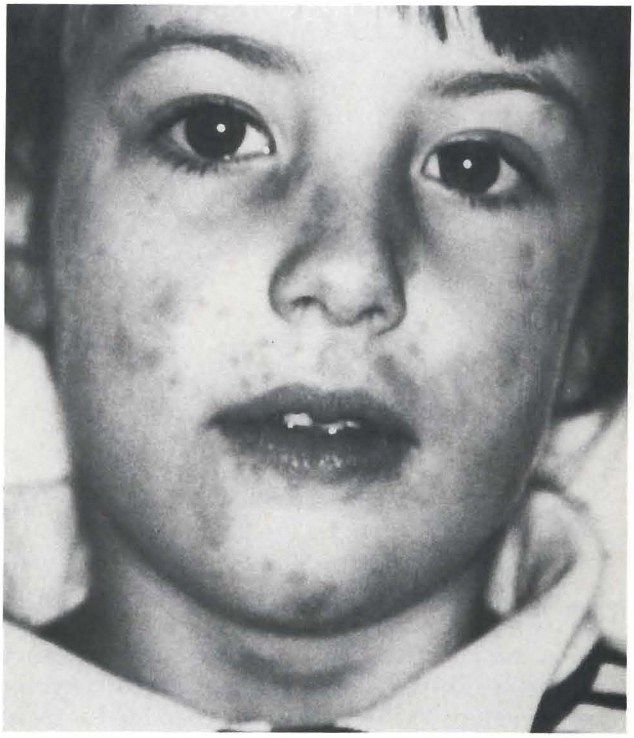

Figure 2. Allergic reaction to a fissure sealant.

reaction and the dental material is considered very probable. We decided to remove the fissure sealant. Because molars are not fully erupted, it was not possible to use a rubber dam. The resin was removed with a high speed jet drill under a strong water spray; and evacuation of water and saliva by vacuum. The girl was instructed not to swallow the water, and she was allowed to rest and to rinse her mouth frequently. She was very cooperative.

The treatment was finished in approximately an hour. The family was instructed to call the local hospital, if her condition worsened, and to complete the cortisone regimen as prescribed.

Our patient appeared normal on her return on day 9. The skin and mucosal reactions have disappeared, except for the white striae on the buccal mucosa, probably from biting the inside of the cheeks. The parents explained that the rashes and swellings began to fade and disappear approximately two hours after the removal of the resin. They reappeared after another six hours, only to vanish completely during the following twenty-four hours. The cortisone treatment was continued and finished on day 7. On day 9, the girl was free from symptoms of the skin, oral mucosa, and respiratory tracts.

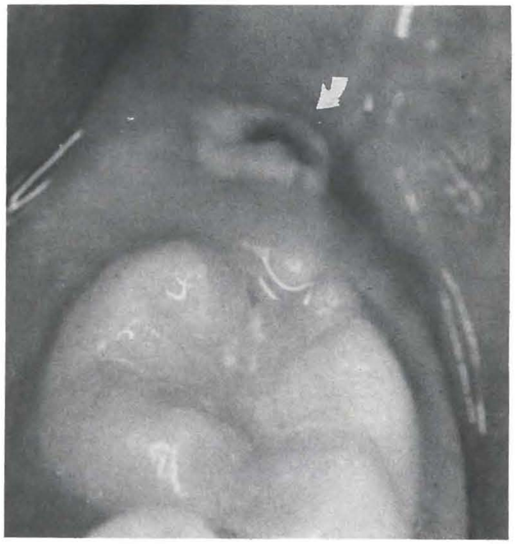

Figure 3. Reaction of mucosa to sealant.

The girl was referred to the Dermatological Clinic of the Lund Hospital for study of allergy to the fissure sealant material.

Allergological investigation

The fissure sealant material Delton contains the following components: TEGDMA, Bis-GMA and XDEA monomer systems. Formaldehyde is a potential allergen, and formation of formaldehyde has been proved to occur in the curing of the latter components. The highest concentrations of formaldehyde are observed in specimens polymerized in contact with air.[7]

Benzoic acid is known to be an elicitor of nonimmunological contact urticaria. It occurs naturally in many fruits, added as preservative (E 210) in salad dressings and other processed foods. It also occurs as a degradation product of benzoylperoxide, used in composite resins.[8] At the Dermatological Clinic, the following tests were performed: Open, closed, PRICK, and 48-hour test of formaldehyde, BHT (butylated hydroxy toluene), Bis-GMA, and benzoylperoxide. Freshly mixed resin was applied (catalyst + universal paste) as closed and 48-hour test. TEGDMA was applied as a 48-hour test.

No allergic reaction could be proved.

DISCUSSION

Allergic reactions are of various types and may have complicated etiologies. The reactions are not necessarily limited to the exposed tissue. It has been suggested that hypersensitivity to acrylic resin can occur as asthma.[9] On reexposure to the acrylic resin, the described patient had a severe exacerbation of respiratory symptoms.[9] Elimination of the prosthesis in that case was followed by relief.

The term *adverse reaction* is applied to cases of nonspecific reaction mechanisms.[8]

The lack of proof of allergic reaction in the present case was not unexpected, since there is no adequate method to chart the origin of urticaria by testing. As the patient had received no previous dental treatment, there was little reason to believe that she could have had an immunologically released urticaria from the acrylic components of the sealant. It is possible, however, that she had been sensitized to formaldehyde or BHT from a shampoo or a food product, and the latter may also have caused an exposure to benzoylperoxide.

No proof of allergic reaction to Bis-GMA has been presented, nor to the tertiary amines present in the universal paste.

The patient's reaction to the application of the fissure sealant and the fact that the symptoms disappeared after removal of the sealant indicate an adverse reaction to the material. The recurrence of the urticaria six hours after removal of the sealant can be regarded as a reaction to the inevitable reexposure to the material during the removal, since a rubber dam could not be used.

No allergic reaction could be proved in test. To go further in this case and expose the girl to the material intraorally again does not seem justified, since a very strong reaction seems likely. She should be considered hypersensitive to the material and should not be exposed to resin materials again.

Her first permanent molars later were sealed with a glass ionomer cement. No complications were observed.

Considering the increasing number of allergies among children and in the population as a whole, it seems important to observe reactions to dental materials, even in persons who have not previously been exposed to such materials. It is also important that new members of the dental staff are made aware that this kind of reaction may occur, both in patients and in staff.

REFERENCES

1. Wendt, L-K and Koch, G.: Fissure sealants in permanent first molars after 10 years. Swedish Dent J, 12:181-185, Issue 5 1988.

The symptoms began to disappear after the removal of the resin.

2. Hensten-Pettersen, A. and Mjr, I.: Plastfyllnings-materialer: Biologiske egenskaper. In: *Nordisk Klinisk Odontologi*, E. Asmussen & E. Budtz-J rgensen, eds. K benhavn: Forlaget for Faglitterature, 7B-II, 1-10.
3. Nathanson, D. and Lockhart, P.: Delayed extraoral hypersensitivity to dental composite material. Oral Surg, 47:329-333, April 1979.
4. Lind, P.O.: Oral lichenoid reactions related to composite restorations. Acta Odont Scand, 46:63-65, February 1988.
5. Katz, R.A.: Soft tissue reaction to a composite resin splint. Dent Survey, 156(6):48, 1977.
6. Tunander, J., 1990: Pers. comm.
7. Öysaed, H.; Ruyter, I.E.; Sjvik Kleven, J.J.: Formation of formaldehyde in dental composites. J Dent Res, 67:762, April 1988, Abstr. 82.
8. Hensten-Pettersen, A. and Jacobsen, N.: Toxic effects to dental materials. Internat Dent J, in press.
9. Stungis, T.E. and Fink, J.N.: Hypersensitivity to acrylic resin. J Prosth Dent, 22:425-428, October 1969.

EFFECTS OF GLASS IONOMERS ON THE METABOLISM OF *S. MUTANS*

In order to study the effects of different glass ionomers on the metabolism of *Streptococcus mutans*, test slabs of freshly mixed conventional glass ionomer (Fuji), silver glass ionomer (Ketac-Silver), composite (Silux), and 2-week-old Fuji were fitted into the bottom of a test tube. A plaque-like layer of *S. mutans* strain Ingbritt was centrifuged onto the test slabs, and the samples were incubated for 20 h in 1.7% (w/v) sucrose solution. For freshly mixed Fuji and Ketac-Silver, the pH fall was significantly smaller than for old Fuji and composite. These materials also released the largest amount of fluoride into the fluid phase. Incubation with glass ionomer materials led to an increase in the cellular concentration of fluoride in bacteria, but intracellular fluoride did not correlate with the fall in pH. The lowest pH was associated with the lowest cellular magnesium content. Ketac-Silver released large amounts of calcium in the fluid phase, and the cellular calcium content was doubled in this group. The results show that freshly mixed glass ionomers affect acid production and electrolyte metabolism of *S. mutans* in vitro. The effect of conventional glass ionomer, however, seems to disappear after a few weeks. The effects of calcium and silver released by cermet glass ionomer deserve further study.

Seppä, L. *et al*: Effect of different glass ionomers on the acid production and electrolyte metabolism of *Streptococcus mutans* Ingbritt.
Caries Res, 26:434-438, November-December 1992

ABSTRACTS

Croll, Theodore P.; Killian, C.M.; Helpin, M.L.: A restorative dentistry renaissance for children: Light-hardened glass ionomer/resin cement. J Dent Child, 60:89-94, March-April 1993.

The light-hardened glass ionomer/resin dental restorative material was introduced in 1992. Its formula includes 80 percent glass ionomer material combined with a 20 percent visible light polymerized resin component. The authors have completed over 1300 class I, class III, and class V restorations in primary teeth, using the new material. The new material is considered by the authors to be a great improvement over the self-hardening glass ionomers. The addition of the 20 percent light-hardened resin component to the glass ionomer formula gives the material an initial setting time under sixty seconds, and improves resistance to wear and fracture.

Light-hardened glass ionomer/resin cement; Resistance to wear; Resistance to fracture

C.M. Kreulen; W.E. van Amerongen; R.J.M. Gruythuysen *et al*: Prevalence of postoperative sensitivity with indirect class II resin composite inlays. J Dent Child, 60:95-98, March-April 1993.

In this clinical trial, the prevalence of postoperative sensitivity is studied in a comparison of standard size indirect class II resin composite inlays with class II amalgam restorations. Fifteen percent of the 240 restored teeth (resin composite and amalgam restorations) showed some form of sensitivity, the majority of them of a passing nature. No difference was found between amalgam and composite restorations. Molars appear to show more complaints than premolars and, rather than the restorative material applied, the patient seems to determine the results. Differences in prevalence of sensitivity between direct resin composite restorations in a previous study and the indirect restorations are discussed, using among others the differences in sensitivity that were found between the amalgam-control restorations of both studies.

Postoperative sensitivity; Amalgam restorations; Indirect class II resin composite inlays

Bimstein, Enrique; Shapira, Lior; Landau, Ela *et al*: The relationship between alveolar bone loss and proximal caries in children; prevalence and microbiology. J Dent Child, 60:99-103, March-April 1993.

The present study describes the prevalence of alveolar bone loss (ABL) in children in relation to caries, contact and space loss. In addition, the microbial composition of the subgingival plaque of 20 sites, from 5 children, is presented. Bite-wing radiographs from 500 children were examined. ABL was evident in: 99 sites from 60 children; >1 site in 27 children; the maxilla only in 34 children; the mandible only in 17 children; both arches in 9 children; 37 sites with no caries; 4.9 percent of all sites with proximal caries; 15.8 percent of all sites with contact loss; and 20.5 percent of all sites with mesial drift. Anaerobic bacteria were cultured from all 20 sites. No significant differences in the percentages of colony forming units of *Actinobacillus actinomycetemcomitans* and black pigmented Bacteroides were found among sites with/without bone loss, with/without caries or probing depths smaller/equal or larger than 2.5 mm.

Alveolar bone loss; Dental caries; Loss of tooth contacts; Loss of space; Anaerobic bacteria

Donohue, D.; Garcia-Godoy, F.; King, D.L. *et al*: Evaluation of mandibular infiltration versus block anesthesia in pediatric dentistry. J Dent Child, 60:104-106, March-April 1993.

The authors consider mandibular infiltration as a possible alternative to mandibular block anesthesia in young children. The study sample comprised eighteen children ages six to nine years old who required bilateral identical treatment on primary mandibular molars. A random number table was used to determine which side of the mouth was to receive the block or the infiltration technique and a coin toss determined which would be administered first: A short 27 gauge needle was used to inject a 2 percent lidocaine solution with 1:100,000 epinephrine, in both techniques. The paired t-test indicated no significant difference between the two techniques for any of the factors evaluated.

Infiltration anesthesia; Block anesthesia; Comparison of techniques.

Mack, Ronald B. and Dean, Jeffrey A.: Electrosurgical pulpotomy: A retrospective human study. J Dent Child, 60:107-114, March-April 1993.

While the formocresol pulpotomy has enjoyed long-term clinical use and success, concerns over its toxicity and mutagenicity have prompted research into other pulpotomy techniques. The purpose of this study was to observe retrospectively the results of the electrosurgical pulpotomy technique used on primary molar teeth requiring pulp therapy, secondary to carious involvement. The mean age at the time of treatment was 5 years, 11 months and the mean postoperative observation time was 2 years, 3 months. Of the 164 teeth studied, 127 were normal at the last observation visit; 32 had undergone exfoliation; 4 had an abnormality associated with the pulpotomized tooth, but were not considered failures; and 1 was considered a failure. This is a 99.4 percent success rate. Compared to a formocresol pulpotomy study of similar design, the success rate for the electrosurgical pulpotomy procedure in this study is higher at the statistically significant level of $p < 0.01$.

Pulpotomy; Electrosurgical technique

Levy, Steven M.: A review of fluoride intake from fluoride dentifrice. J Dent Child, 60:115-124, March-April 1993.
Ingestion of fluoride dentifrice by preschool children recently has been identified as a risk factor for dental fluorosis. There are relatively few published studies concerning patterns of fluoride dentifrice use and ingestion among preschool children at greatest risk for dental fluorosis. This paper reviews the literature on dentifrice use and ingestion in an attempt to emphasize to the practitioner the potential importance of this fluoride exposure in fluorosis etiology.
Fluorosis; Fluoride dentifrice; Ingestion

Arthur H. Friedlander, DDS; Ida Kreinik Friedlander, RN, BSN, MS John A. Yagiela, DDS, PhD *et al*: Dental management of the child and adolescent with major depression. J Dent Child, 60:125-131, March-April 1993.
Major depression is a psychiatric disorder in which mood, thought content, and behavioral patterns are impaired, often for an extended period of time. This condition appears to have an increasing prevalence among young children and adolescents. It may be associated with a disinterest in performing appropriate preventive oral hygiene techniques, a cariogenic diet, rampant dental decay, and advanced periodontal disease. Appropriate dental management necessitates a vigorous preventive dental education program and special precautions when administering local anesthetics and prescribing sedative and analgesic medications.
Major depression; Oral hygiene; Diet; Dental caries; Periodontal disease

Waldman, H. Barry: The poisoning of our children. J Dent Child, 60:132-135, March-April 1993.
In the second half of the 1980s, almost four million exposures to poison were reported for children less than six years of age. The causes, types of poisoning and treatment are reviewed.
Poisoning; Treatment

Waldman, Barry H.: Is your next pediatric patient an addict? J Dent Child, 60:136-139, March-April 1993.
There have been decreases in the use of illicit drugs by youngsters in our country. However, the reality is that great numbers of children continue to use these substances. A review is provided of the use of illicit drugs by children. In addition, the consumption of alcohol and cigarettes by youngsters also is considered.
Drugs, illicit; Alcohol; Cigarettes; Usage; Children

Roberts, Michael W.; McIver, F. Thomas; Phillips, Ceib L.: Gender trends among specialists in pediatric dentistry. J Dent Child, 60:140-142, March-April 1993.
The number of women entering the dental profession has increased over the past few years and the percentage of females enrolling in pediatric postdoctoral studies surpassed 50 percent in 1990. The impact of the shift in the gender of practicing pediatric dentists was examined in this study. A survey was designed to evaluate the relationship between the age and gender of the pediatric dentist, location of principle practice site, and the number of hours dedicated to direct patient care. Twenty-seven hundred and sixty surveys were mailed to pediatric dentists in the United States and 1246 were returned (46 percent). Significantly more males were in private practice than females, although the difference in percentages was small (85 percent versus 80 percent). Women identified universities, hospitals, and institutions as their principle site of practice significantly more often than males (20 percent versus 15 percent). On average, females in private practice work only slightly fewer hours per week providing patient care than do males (32 versus 35 hours/week). In university, hospital, and institution, settings the reverse is true: women spend more time in direct patient care than men (27 versus 23 hours/week).
Practice; Gender trends

Hallström, Ulla: Adverse reaction to a fissure sealant: Report of case. J Dent Child, 60:143-146, March-April 1993.
A six-year-old girl with a known allergy to mites had her first permanent molars sealed with Delton. On the night of the treatment day, she began to have asthmatic trouble. Urticaria appeared a few days later. Treatment with cortisone gave no relief. When the fissure sealant material was taken away, the asthma and urticaria disappeared. Possible explanations to the reaction are discussed in the report.
Allergic reactions; Fissure sealant