ASDC AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

JANUARY-FEBRUARY 1987

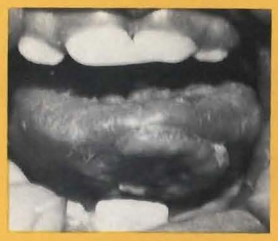
JOURNAL OF DENTISTRY FOR CHILDREN

A COMMONPLACE OF POLITICAL RHETORIC HAS IT THAT THE QUALITY OF A CIVILIZATION MAY BE MEASURED BY HOW IT CARES FOR ITS ELDERLY. JUST AS SURELY, THE FUTURE OF A SOCIETY MAY BE FORECAST BY HOW IT CARES FOR ITS young.

Daniel Patrick Moynihan

ON A CLOUD I SAW A CHILD
—William Blake

60 YEARS OF SERVICE



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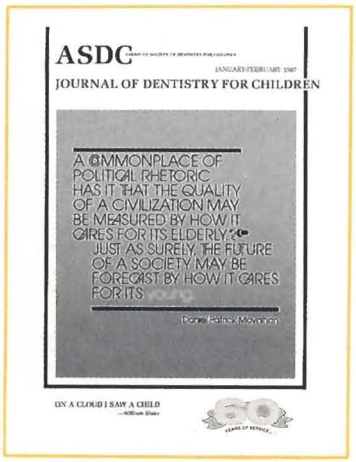
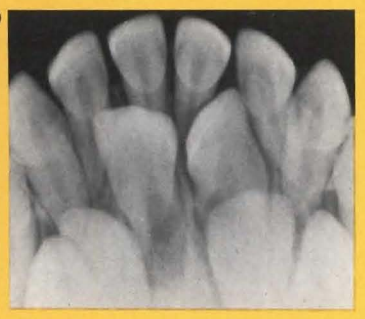
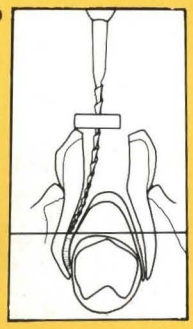
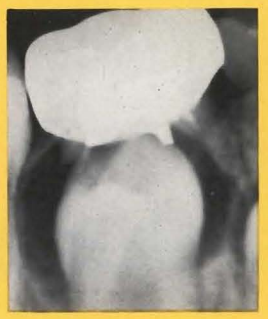


The cover implies the importance of children to the future of a society. Children are a nation's major resource and should be treated as such. Design and art by Sharlene Novak.







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For the busy reader

Fluoride retention by human enamel after *in vitro* application of nicomethanol hydrofluoride—page 9

Fluoridated dentifrices generally contain sodium monofluorophosphate or sodium fluoride—alone or in combination—or a group of organic fluorides, the amine fluorides. The purpose of this study was to determine the fluoride and retention *in vitro* by human enamel, after brushing with a dentifrice containing the amine fluoride, nicomethanol hydrofluoride, the fluoride salt of nicotinyl alcohol.

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

***In vitro* fluoride uptake by lased and unlased ground human enamel—page 15**

Laser, light amplification by the stimulated emission, has been applied in experimental dentistry for the last two decades. Its effect on the particular type of tissue depends on the wavelength and power of the laser beam, duration of application, tissue distance, and the color and interval structure of the tissue. Acquired fluoride was measured in ground enamel.

Requests for reprints should be directed to Dr. Faiez N. Hattab, Department of Children's Dentistry and Orthodontics, Prince Philip Dental Hospital, University of Hong Kong, Hong Kong.

The effect of fluoridated chocolate-flavored milk on caries incidence in elementary school children—page 18

Because chocolate is the strongly preferred milk flavoring agent in the USA and because neither food selection nor ingestion is monitored in most schools, it seemed appropriate to study the effects of chocolate-flavored, sweetened, low-fat, fluoridated milk on the caries incidence of elementary school children.

Requests for reprints should be directed to Dr. Benjamin J. Legett, Jr., Head, Department of Community and Preventive Dentistry, LSU School of Dentistry, 1100 Florida Avenue, Box 221, New Orleans, LA 70119.

Management of the refractory young child with chloral hydrate: dosage selection—page 22

Chloral hydrate is often selected for its wide range of safety, yet concerns are increasingly raised about its frequent failure to provide adequate levels of sedation while using the recommended hypnotic dosage. This paper discusses the implications of these weaknesses in a pedodontic context.

Requests for reprints should be directed to Dr. John E. Nathan, 183 South Bloomingdale Road, Bloomingdale, IL 60108.

Evaluation of an iodoform paste in root canal therapy for infected primary teeth—page 30

The treatment guidelines for success were if clinically the tooth was painless, without pathologic mobility, and the gingiva was healthy. No clinical or radiographic signs or symptoms of failure were observed in forty-three of forty-five teeth.

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

Consequences of endodontic treatment of primary teeth-Part II. A clinical investigation into the influence of formocresol pulpotomy on the permanent successor—page 35.

These 278 teeth were assessed by two observers, who always differentiated between opacities and hypoplasias when finding enamel lesions. Their degree of agreement was 96 percent, and there was no significant difference in the number of teeth with enamel lesions, between test side and control side.

Requests for reprints should be directed to Dr. W.E. van Amerongen, Head, Division of Pediatric Dentistry, Free University, Amsterdam, The Netherlands.

Assessment of the systemic distribution and toxicity of formaldehyde following pulpotomy treatment: part two—page 40

The active constituents of formocresol, formaldehyde and cresol, are known toxic agents. The authors administered increments of formaldehyde until systemic morbidity was demonstrated so that tissue damage could be equated with the number of concurrent pulpotomies required to achieve a toxic body load.

Requests for reprints should be directed to Dr. Don M. Ranly, Department of Pediatric Dentistry, The University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78284.

Dimensional stability of alginate impressions immersed in disinfecting solutions—page 45

The stability of full-arch alginate impressions that had been immersed in one of two disinfecting solutions was assessed by measuring stone casts poured from the alginate impressions. Statistically significant dimensional changes occurred, when compared with master casts, but only by 1 mm or less, a clinically insignificant amount.

Requests for reprints should be directed to Dr. David P. Durr, Department of Pediatric Dentistry, Eastman Dental Center, 625 Elmwood Avenue, Rochester, NY 14620.

Pediatric dentistry in the early and mid-1980s: a review of personnel and use of dental services—page 49

Because children are treated by both the general population of practitioners and by pediatric dentists, any discussion of the evolving use patterns should also consider changes in the overall number of practitioners. Recent changes have been favorable from the viewpoint of practitioners.

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.

Unusual case of green teeth resulting from neonatal hyperbilirubinemia—page 54

Requests for reprints should be directed to Dr. Frank L. Herbert, LSUSD Box 127, 1100 Florida Avenue, New Orleans, LA 70119.

Familial dysautonomia with Riga-Fede's disease: report of case—page 57

Requests for reprints should be directed to Dr. Meir Rakocz, Pediatric Dentistry, The Chaim Sheba Medical Center, Tel Hashomer, 52621, Israel.

Hyperdontia in the primary dentition: report of case—page 60

Requests for reprints should be directed to Dr. Edna L. Pashley, Departments of Community Dentistry and Pediatrics, Medical College of Georgia, Augusta, GA 30912.

Goldenhar's syndrome and hypodontia: report of case—page 62

Requests for reprints should be directed to Dr. R.R. Welbury, Research Associate, Department of Child Dental Health, The Dental School, Framlington Place, Newcastle-Upon-Tyne, NE2 4BW, United Kingdom.

Fluoride studies

Fluoride retention by human enamel after *in vitro* application of nicomethanol hydrofluoride

Fred Barbakow, BDS, HDD, MSc
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Caries incidence is reduced collectively by water and salt fluoridation and individually by using fluoridated dentifrices, mouth rinses and topical applications, although the fluoride concentrations used in the above methods vary considerably. Fluoridated dentifrices usually contain sodium monofluorophosphate or sodium fluoride, alone or in combination; or a group of organic fluorides called the amine fluorides. These amine fluorides are hydrofluoric acid salts of long-chain amines with cationic surface- active properties that provide antimicrobial activity; some can induce high levels of fluoride in enamel after topical application.[1-4] A fluoridated dentifrice containing the amine fluorides 297 (N,N',N'-(2-hydroxyethyl)N-octadecyl- 1,3-diaminopropandihydrofluoride; 0.1% F^-) and 242 (hexadecylamine hydrofluoride, 0.025% F^-) has been used clinically in Switzerland and other countries since 1963. Results of *in vivo* and *in vitro* tests of this dentifrice have been previously reported.[5,6]

The details of the mechanisms whereby fluorides reduce caries incidence are unknown, but well-founded theories have been documented.[7] The remineralization of previously demineralized enamel is, however, considered a major factor in this mechanism.[8] The con-

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centration of fluoride in the enamel may play a role in caries reduction by slowing down the rate at which initial enamel lesions form. Once demineralization of fluoride- rich enamel occurs, however, the additional available fluoride ions may enhance remineralization of the initial lesions, when oral conditions become favorable. The purpose of this study was to determine the fluoride uptake and retention *in vitro* by human enamel, after brushing with a dentifrice containing the amine fluoride, nicomethanol hydrofluoride, the fluoride salt of nicotinyl alcohol (C_6H_8ONF; mw = 129).

MATERIALS AND METHODS

Partially erupted or impacted human maxillary and mandibular third molars, extracted from patients attending a dental practice in a nonfluoridated area in the USA were used in this study. The teeth were stored in 0.1 percent thymol solution at 4°C until used. The 720 enamel specimens were randomly assigned to one of four main groups and were to be brushed with one of the three dentifrices listed in Table 1, using distilled water as the negative control. The fluoride ion concentrations and pH values of the test dentifrices were determined in 10 percent aqueous solutions. The surfaces of the enamel specimens were cleaned, using a flour of pumice-distilled water slurry and a rotating brush to remove pellicle. They were stored in humid conditions (0.1 percent thymol) during the entire experimental period, to prevent bacterial and fungal accumulations and to minimize brittleness of the enamel.

The enamel crowns were manually brushed horizontally, twice daily, with the assigned test dentifrice, each time for 1 min, using standardized toothbrushes, five days per week over a ten-week period. The amount of dentifrice used was not weighed, but was standardized by dispensing similar amounts along the heads of the toothbrushes. The pressure exerted during brushing was not controlled. In the control group, the crowns were brushed with toothbrushes that were first moistened in distilled water. In this way, all the enamel specimens were brushed 100 times. After each brushing the enamel specimens were washed for 10 sec, using deionized water to remove excess dentifrice and unreacted fluoride. At the end of the ten-week brushing period, a third of the specimens in each of the four main groups were not treated further; another third were washed for an additional 24 h in fast-flowing deionized water, after the last brushing; and a third were similarly water-washed for 50 h, after the last brushing. At the end of the washing periods, the enamel specimens were

Table 1 ☐ Fluoride ion concentration and pH of 10 percent aqueous solutions of the test dentifrices.

	Dentifrice		
	A Amine fluorides 297 + 242	B Nicomthanol hydrofluoride	C NaF
N (of tubes used)	44	40	45
Mean F^- ± SD (ppm)	1088 ± 71	946 ± 42	1166 ± 50
Max. F^- (ppm)	1226	1051	1308
Min. F^- (ppm)	945	880	1083
Mean pH ± SD	5.07 ± 0.2	4.98 ± 0.1	7.22 ± 0.1
Max. (pH)	5.39	5.22	7.35
Min. (pH)	4.74	4.73	7.09

A = Elmex®: P.F. Medicament, Castres, France (Positive Control)
B = PP 117: P.F. Medicament, Castres, France (Test agent)
C = Crest®: Purchased in drugstores in UK (Test agent)

Table 2 ☐ Statistical analysis of the enamel fluoride concentrations (ppm) in the first etched layer and the cumulative fluoride concentrations in the three subsequent layers of the four groups after water-washing for 10 sec only, following each brushing.

		Layer			
		1	1+2	1+2+3	1+2+3+4
	N	Mean (±SD)	Mean (±SD)	Mean (±SD)	Mean (±SD)
Amine fluoride 297 & 242	60	1417 (480)	1038 (228)	855 (176)	749 (159)
Nicomethanol hydrofluoride	60	1445 (424)	967 (216)	727 (175)	588 (159)
Sodium fluoride	60	978 (285)	588 (144)	393 (104)	298 (92)
Water	60	677 (324)	399 (203)	257 (138)	191 (107)
$S\bar{x}$ (Std. error)		49.31	25.84	19.49	17.13
F		55.11	139.47	205.91	225.16
P		<0.001	<0.001	<0.001	<0.001
*LSD < 0.05		138	72	54	48
*LSD < 0.01		181	95	72	63
*LSD < 0.001		233	122	92	81

*Least Significant Difference between two means

Table 3 ☐ Statistical analysis of the depths (μm) of the first enamel layers etched and the cumulative depths of the three subsequent layers of the four groups after water-washing for 10 sec only, after each brushing.

		Layer			
		1	1+2	1+2+3	1+2+3+4
	N	Mean (±SD)	Mean (±SD)	Mean (±SD)	Mean (±SD)
Amine fluoride 297 & 242	60	4.47 (0.94)	17.97 (2.36)	39.59 (4.48)	61.63 (8.17)
Nicomethanol hydrofluoride	60	3.78 (1.33)	16.20 (3.91)	36.34 (8.24)	58.95 (12.31)
Sodium fluoride	60	4.90 (1.06)	19.56 (3.03)	43.31 (5.57)	68.38 (8.13)
Water	60	5.39 (0.88)	20.31 (2.35)	44.03 (4.40)	68.52 (6.69)
$S\bar{x}$ (Std. error)		0.14	0.38	0.76	1.17
F		24.69	22.42	19.57	17.07
P		<0.001	<0.001	<0.001	<0.001
*LSD < 0.05		0.38	1.07	2.12	3.27
*LSD < 0.01		0.51	1.42	2.79	4.31
*LSD < 0.001		0.65	1.82	3.59	5.53

*Least Significant Difference between two means

Table 4 ☐ Statistical analysis of the enamel fluoride concentrations (ppm F) in the first etched layer and the cumulative fluoride concentrations in the three subsequent layers of the four groups after water-washing for 10 sec after each brushing and for another 24 h after the last brushing.

		Layer			
		1	1+2	1+2+3	1+2+3+4
	N	Mean (±SD)	Mean (±SD)	Mean (±SD)	Mean (±SD)
Amine fluoride 297 & 242	60	1467 (427)	1092 (229)	915 (164)	816 (144)
Nicomethanol hydrofluoride	60	1310 (465)	942 (299)	720 (199)	605 (187)
Sodium fluoride	60	811 (200)	515 (133)	341 (103)	263 (82)
Water	60	743 (334)	459 (199)	303 (140)	227 (112)
$S\bar{x}$ (Std. error)		47.88	28.82	20.05	17.69
F		56.52	117.84	219.28	255.27
P		<0.001	<0.001	<0.001	<0.001
*LSD < 0.05		134	80	56	49
*LSD < 0.01		176	106	74	65
*LSD < 0.001		226	136	95	84

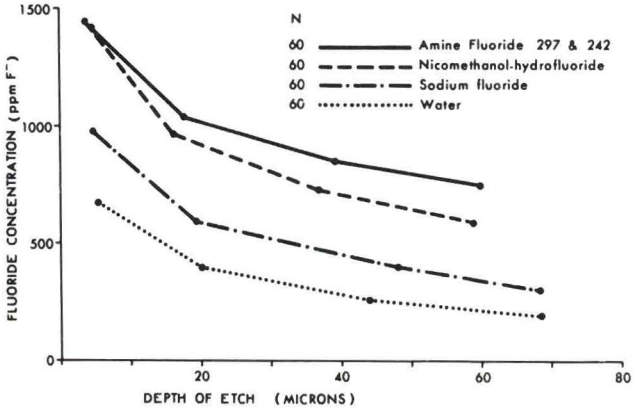
*Least Significant Difference between two means

separated from their roots, using water-cooled diamond discs and washed for 10 sec, using distilled water to remove any enamel and dentin dust from the enamel surfaces.

Fluoride concentrations and corresponding depths of etch were determined from the enamel specimens, using an *in vitro* enamel acid-etch technique in a complete block design. Enamel windows, each 3 mm in diameter, were selected by applying prepared annular Scotch-tape discs (3M Co., No. 471, St. Paul, MN, USA) along the middle- third of the buccal and palatal surfaces, avoiding macroscopic hypoplastic and cracked enamel areas. The rest of the specimen was covered with sticky wax, leaving only the enamel window exposed (Ruscher Co., Zurich, Switzerland). Each enamel window was sequentially etched four times, in four separate polyethylene beakers, each containing 5 ml of 2N HCl (Merck, Darmstadt, West Germany) for 5 sec, 10 sec, 15 sec and 15 sec, respectively. The total etching time was 45 sec, during which the specimens were regularly agitated.

The fluoride concentrations were determined in 0.5 ml aliquots of the etching solution, to which 2 ml of TISAB solution (pH 5.1) was added, using a specific fluoride ion electrode (906900, Orion Co., Boston, USA).[9] The corresponding phosphate concentrations were determined colorimetrically in 1 ml aliquots of the etching solutions.[10] With these results the initial fluoride concentrations and depths of etch after the 5 sec acid-etch and the subsequent cumulative fluoride concentrations and the cumulative depths of etch for the 3

Figure 1. Profiles of the enamel fluoride concentrations and depths of etch (cumulative) of the specimens water-washed for only 10 sec after each brushing with dentifrices containing either amine fluorides 297 & 242 (Elmex®); nicomethanol hydrofluoride; sodium fluoride (Crest®); or water.



additional acid-etchings (10 sec, 15 sec, 15 sec, respectively) were calculated, using the following formula:

A constant density of enamel of 3 g/ml, a calcium content of 38 percent and a phosphorus content of 17.5 percent throughout the etched area were assumed.[11] The mean fluoride concentrations and depths of etch of the control and the three other test groups were determined and analyzed statistically, using the one-way analysis of variance, accepting $p < 0.01$ as being statistically significant.[12]

RESULTS

The results of the fluoride ion concentrations and the pH values of the test dentifrices used in this study are listed in Table 1.

The fluoride concentrations in the first etched enamel layers and the cumulative fluoride concentrations for the subsequent three etched enamel layers of the specimens that had been water-washed for 10 sec only, after each brushing, are listed in Table 2. The corresponding initial and cumulative depths of etch of these specimens are listed in Table 3. The fluoride concentration to depth of etch profiles within this group are shown in Figure 1.

The fluoride concentrations in the first etched enamel layers and the cumulative fluoride concentrations for the subsequent three etched enamel layers of the specimens that had been water-washed for 10 sec, after each brushing and then for the additional 24 h,after the last brushing, are listed in Table 4. The corresponding initial and cumulative depths of etch of these specimens are listed in Table 5. The fluoride concentration to depth of etch profiles within this group are shown in Figure 2.

The fluoride concentrations in the first etched enamel

layers and the cumulative fluoride concentrations for the subsequent three etched enamel layers of the specimens that had been water-washed for 10 sec, after each brushing, and then for the additional 50 h, after the last brushing, are listed in Table 6. The corresponding initial and cumulative depths of etch of these specimens are listed in Table 7. The fluoride concentration to depth of etch profiles within this group are shown in Figure 3.

Occasionally conflicting levels of significance occur between two means, when tested with the listed LSD (least significant differences) results and then compared to the P value. This is important, when the conflicting results vary between being "not significant" and "significant". In this study, the level of significance derived from the P value was always given preference over the LSD results.

Table 5 ☐ Statistical analysis of the depths (μm) of the first enamel layers etched and the cumulative depths of etch of the three subsequent layers of the four groups after water-washing for 10 sec after each brushing and for another 24 h after the last brushing.

		Layer			
		1	1+2	1+2+3	1+2+3+4
	N	Mean (±SD)	Mean (±SD)	Mean (±SD)	Mean (±SD)
Amine fluoride 297 & 242	60	4.21 (0.39)	16.50 (3.08)	36.26 (6.00)	57.50 (9.4)
Nicomethanol hydrofluoride	60	4.54 (1.45)	17.33 (4.47)	37.87 (8.73)	59.77 (13.02)
Sodium fluoride	60	5.32 (1.19)	19.79 (3.07)	43.08 (5.60)	67.33 (8.07)
Water	60	3.99 (1.02)	17.06 (3.58)	39.20 (5.97)	63.25 (8.60)
$S\bar{x}$ (Std. error)		0.15	0.46	0.86	1.29
F		15.03	9.83	11.35	11.12
P		<0.001	<0.001	<0.001	<0.001
*LSD < 0.05		0.42	1.29	2.41	3.59
*LSD < 0.01		0.55	1.71	3.18	4.73
*LSD < 0.001		0.71	2.19	4.08	6.08

*Least Significant Difference between two means

DISCUSSION

Enamel topically treated *in vitro* with fluorides can be washed with water, artificial saliva or KOH, to remove the so-called fluoride "on the enamel" and thereby leave the fluoride "in the enamel".[13] The major portion of the acquired topical fluoride "on the enamel" is removed within the first 24 hours of washing.[14-16] The 50 h water-washing in this study was thus long enough to ensure removal of most of the acquired "fluoride on" the test specimens. Comparing the *in vitro* fluoride concentration "on" and "in" enamel, reported in different studies, is, however, complicated by variations in the manner and durations of the dentifrice applications and by the washing procedures. Within each study, compositions of the test dentifrices also influence the results. In the present study, the differing pH values between the amine fluoride and the NaF-containing dentifrices were important factors; these dentifrices, however, are sold as such to the public.

The enamel was brushed with the dentifrices to mimic the *in vivo* situation as closely as possible. In contrast, enamel in other studies was incubated with slurries of the dentifrices, while Kirkegaard (1977) brushed enamel with rotating brushes for 64 min.[17-19] Repeated brushing of enamel might abrade some of the outer fluoride-rich enamel, but abrading does not occur, when enamel is incubated in dentifrice slurries.[20] One could, therefore, have expected higher enamel fluoride concentrations, if the specimens in this study had been incubated in dentifrice slurries and not brushed. The control specimens were brushed with water only and not with a fluoride-free dentifrice, to obtain the true natural fluoride concentration of enamel.

Although the preextraction fluoride histories of the teeth used were unknown, their posteruptive exposure to concentrated fluoride agents was minimal, as the third molars used were unerupted or partially erupted. Care was taken to use only the middle third of the buccal or lingual surface to avoid variations in the fluoride concentrations over the entire surface as previously described.[21]

The depth of etch of the standardized area of enamel is difficult to control in the acid-etch technique. When

Figure 2. Profiles of the enamel fluoride concentrations and the depths of etch (cumulative) of the specimens water-washed for 10 sec after each brushing plus an additional 24 h after the last brushing with dentifrices containing either amine fluorides 297 & 242 (Elmex®); nicomethanol hydrofluoride; sodium fluoride (Crest®); or water.

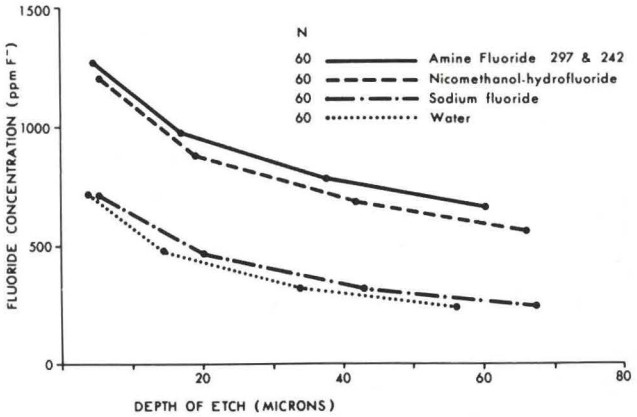


Table 6 ☐ Statistical analysis of the enamel fluoride concentrations (ppm) in the first etched layer and the cumulative fluoride concentrations in the three subsequent layers of the four groups after water-washing for 10 sec after each brushing and for another 50 h after the last brushing.

		Layer			
		1	1+2	1+2+3	1+2+3+4
	N	Mean (±SD)	Mean (±SD)	Mean (±SD)	Mean (±SD)
Amine fluoride 297 & 242	60	1271 (357)	967 (187)	775 (136)	660 (125)
Nicomethanol hydrofluoride	60	1204 (381)	877 (202)	680 (145)	557 (132)
Sodium fluoride	60	710 (251)	461 (176)	316 (128)	242 (104)
Water	60	716 (313)	475 (205)	317 (145)	232 (105)
$S\bar{x}$ (Std. error)		42.52	24.86	17.88	15.07
F		51.09	113.28	181.14	210.21
P		<0.001	<0.001	<0.001	<0.001
*LSD < 0.05		119	69	50	42
*LSD < 0.01		156	91	66	56
*LSD < 0.001		201	117	85	71

*Least Significant Difference between two means

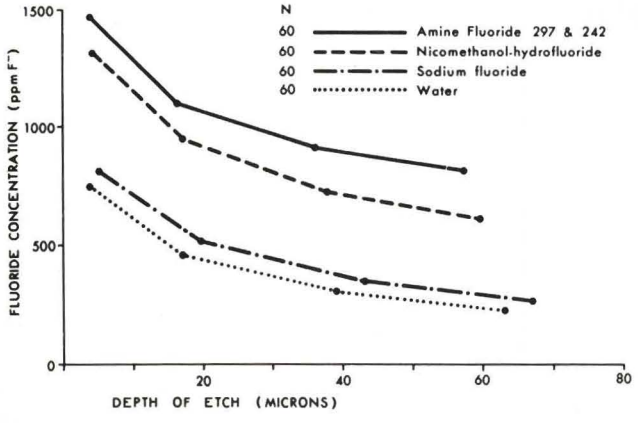
Table 7 ☐ Statistical analysis of the depths (μm) of the first enamel layers etched and the cumulative depths of etch of the three subsequent layers of the four groups after water-washing for 10 sec after each brushing and for another 50 h after the last brushing.

		Layer			
		1	1+2	1+2+3	1+2+3+4
	N	Mean (±SD)	Mean (±SD)	Mean (±SD)	Mean (±SD)
Amine fluoride 297 & 242	60	4.47 (1.35)	17.07 (3.61)	37.75 (7.40)	60.56 (12.71)
Nicomethanol hydrofluoride	60	5.21 (1.22)	19.22 (3.18)	41.99 (5.05)	66.28 (7.27)
Sodium fluoride	60	5.31 (0.86)	20.03 (2.48)	43.06 (4.35)	67.53 (6.18)
Water	60	3.80 (0.87)	14.48 (3.47)	33.98 (6.48)	56.07 (9.10)
$S\bar{x}$ (Std. error)		0.14	0.42	0.77	1.18
F		25.04	35.79	29.50	20.21
P		<0.001	<0.001	<0.001	<0.001
*LSD < 0.05		0.39	1.16	2.14	3.30
*LSD < 0.01		0.52	1.53	2.82	4.35
*LSD < 0.001		0.67	1.96	3.62	5.58

*Least Significant Difference between two means

comparing enamel fluoride concentrations, ideally, the depths of etch of the outer 10 μm at least, should be similar, because of the typical fluoride distribution in enamel. Retief *et al* (1983) compared the fluoride concentrations after statistically adjusting to standard depths, but this was not done in the present study.[22] Recently, abrasive biopsy techniques were described, but one must question the fluoride concentration and depths obtained, considering that some material is lost in this technique.[23]

Figure 3. Profiles of the enamel fluoride concentrations and the depths of etch (cumulative) of the specimens water-washed for 10 sec after each brushing plus an additional 50 h after the last brushing with dentifrices containing either amine fluorides 297 & 242 (Elmex®); nicomethanol hydrofluoride; sodium fluoride (Crest®); or water.



The reproducibility of the acid-etch technique is good and can be verified by comparing the results of the present control specimens with those of previous similar studies.[24] It is difficult, however, to explain the differences between the depths of etch of the control specimens, which were washed for 10 sec only (Table 3), and the control specimens, which were additionally washed for 24 h and 50 h (Tables 5 and 7, respectively). There were some significantly different depths of etch, particularly in the first layer, between the specimens brushed with the various test dentifrices. The general pattern, however, indicated significantly higher enamel fluoride concentrations ("fluoride in the enamel") in all four layers, in the specimens brushed with both amine fluoride-containing dentifrices compared to those brushed with the NaF-containing dentifrice (Crest ®), after the water-washing procedures. This confirms the findings of previous *in vitro* studies testing different dentifrices on human enamel.[17,19] There were no differences in the enamel fluoride concentrations of the specimens brushed with the amine fluoride, nicomethanol hydrofluoride, and the dentifrice containing amine fluorides 297 and 242.

The clinical importance of an increased enamel fluoride concentration *in vitro* is controversial. Reports have both confirmed and questioned the inverse relationship between surface enamel fluoride concentration and caries incidence.[25-28] One might speculate that enamel with a higher concentration of fluoride would be more resistant to an *in vivo* acid demineralization, and once such

enamel has been demineralized, a higher fluoride concentration would be available for subsequent remineralization, when suitable conditions develop at the plaque-enamel interface. If this holds true, it is logical to use, from early age on, a dentifrice that ensures a higher enamel fluoride concentration than a NaF dentifrice. Amine fluoride dentifrices generally induced higher enamel fluoride concentrations than sodium fluoride dentifrices (Crest ®) in rat caries and clinical studies.[6,29,30] It is also possible that enamel with a high fluoride concentration will enable a slow continuous loss of the fluoride into the oral fluids after brushing, and could mimic the "slow fluoride releasing devices" being studied to facilitate remineralization.[31]

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In vitro fluoride uptake by lased and unlased ground human enamel

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Laser, light amplification by the stimulated emission of radiation, has been applied in experimental dentistry for the last two decades. Among the types of laser used are: solid-state ruby and neodymium-YAG (yttrium-aluminum- garnet) and gaseous, as helium-neon, nitrogen, carbon- dioxide, krypton and argon. Earlier studies were concerned with the effect of carbon-dioxide laser on enamel structure and chemical changes incurred. Microradiographic findings indicate that carbon-dioxide irradiation imparts to the enamel surface a degree of impermeability, and zonal changes in the mineral distribution.[1-3] Using two cell chambers separated by bovine enamel, Borggreven *et al* found that carbon-dioxide laser increased the permeability of enamel rather than decreased it, as suggested by others.[1,2,4] Ultraviolet or visible laser lights were proposed as a method for early detection of caries lesions in human subjects and in research animals.[5-7] Data on whether laser can affect the uptake and penetration of fluoride (F) by and in enamel are unavailable.

This study was designed, therefore, to investigate the influence of continuous-wave argon laser on enamel F-uptake.

MATERIALS AND METHODS

Twelve impacted mandibular third molars were extracted. After making certain that the enamel of these teeth was free of cracks or other defects, they were stored in deionized water containing thymol at 4°C, until used. The teeth were hemisectioned buccolingually, and a half from each tooth was randomly selected to serve as experimental (lased F-treated) sample, while its corresponding half served as a control (unlased F-treated). The enamel buccal surface of each tooth-half was ground flat with a water-cooled abrasive disc, to a depth of about 300 μm from the surface. The ground surface was thoroughly washed with water under pressure, followed with deionized water, and then dried with tissue paper. A circular adhesive disc (4 mm in diameter) was placed on the buccal surface and burnished to ensure a good marginal adaptation of the disc to the surface. The tooth-half was covered with nail varnish and allowed to dry; the disc was then removed, leaving behind a well defined area (12.6 mm^2) for acid-etch enamel biopsy. The experimental tooth-halves were exposed to a continuous wave of argon-ion laser (Model CR-2SG)* working at a wavelength of 488 nm and power density of 35 mW/cm^2. The laser beam was adjusted to illuminate the entire enamel area. The tooth-half was placed 1-cm-distance from the laser apparatus (i.e. beam's point of application) and illuminated for 1 min. Immediately after laser exposure, the enamel was treated with the F agent. The experimental (lased) and control (unlased) groups were treated for 1 min with 2 percent NaF solution (pH 7.2), using a cotton applicator soaked in F solution. After F treatment, the tooth halves were washed for 15 sec under running deionized water, to remove residual solution

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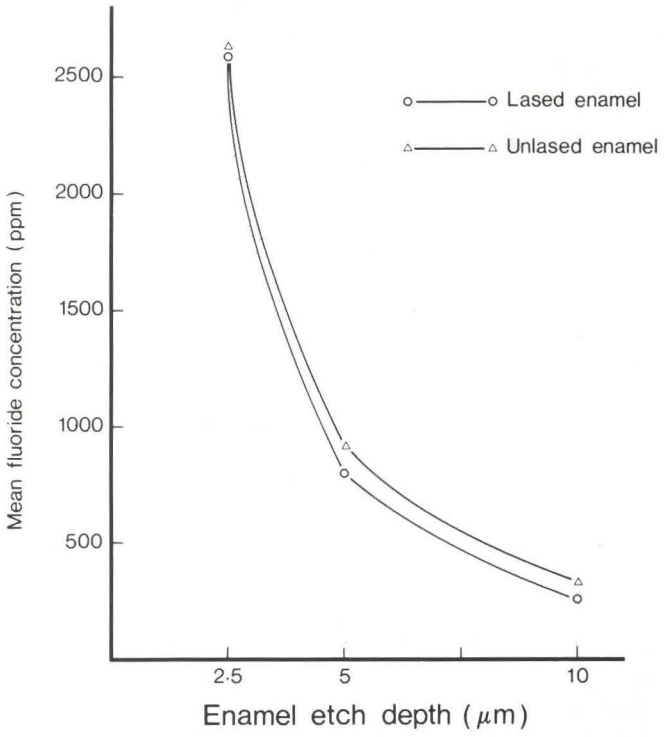
*Coherent, Palo Alto, CA

and unreacted F, and then dried with tissue paper. Three successive enamel layers were biopsied by means of F-free cotton pellets saturated with 0.1 ml of 0.5 M $HC10_4$, for periods of 15, 30 and 30 sec, respectively. The etching solutions that contained the biopsied samples were analyzed for F, calcium and phosphate. Fluoride was determined with a combination F^-selective electrode (Model 96-09)* coupled with a Model 901 ionalyzer. Calcium was determined by an atomic absorption spectrophotometer (Model SP 190).† Phosphate was determined, in duplicate, by a double-beam spectrophotometer (Model 24)‡ using the one-step malachite green method.[8] From calcium and phosphate determinations, the weight of enamel removed was calculated assuming enamel to contain 37.4 percent calcium and 17.5 percent phosphorus; 2.90 was taken as an average density of enamel at its middepth.[9] From the relationship between the mass of F and enamel dissolved in each etching period, the F concentration (ppm) at the corresponding biopsied enamel layer was calculated as F (ppm) = F (μg) x 10^6 / enamel (μg), details of which were described elsewhere.[10] Because the biopsy depth is an uncontrollable variable, and in order to make valid comparisons of the enamel F concentration between the experimental and control groups, the F concentrations were adjusted to a standardized depth of 2.5, 5.0 and 10 μm. This was done by fitting the absolute values of F concentrations and depths for each tooth-half to the power curve formula: $y = ax^{-b}$ where (y) is the F concentration in ppm, (x) is the depth in μm, (a) is the intercept, (b) is the slope, i.e. a measure of F gradient with depth.

The rectified data yielded an average correlation coefficient of 0.99 ± 0.004 ($\overline{X}$ ± SD, number 24). The generated fitting line was used to predict the F concentration by using the standardized depth as a predictor.

RESULTS

The average rate of lased enamel removed as a function of the total etching period was 8.76 μg/sec which corresponds to a thickness of 0.24 μm/sec. The corresponding data for unlased enamel were 8.57 μg/sec and 0.23 μm/sec, respectively. The Ca/P weight ratio was 2.3 ± 0.3 (mean ± SD). The mean concentrations of F found at standardized depths in lased and unlased enamel are presented in the Table and in the Figure. No statistically significant differences are found between the F concentrations in the experimental and control groups ($P > 0.5$, t-test).



Mean fluoride concentration at standardized enamel depth. Each curve represents the mean of 12 tooth- halves.

DISCUSSION

The effect of laser on particular type of tissue depends on the wavelength and power of the laser beam, duration of application, tissue distance from the point of application, and the color and internal structure of the tissue. Earlier studies on the effect of carbon-dioxide laser on enamel indicates fusion of surface enamel defects and pores, and, therefore, renders the lased enamel less permeable than the unlased enamel.[1,2] This finding was not confirmed by a diffusion study, probably due to the differences in the laser densities used and to the method of assessment.[4]

Under the present experiment ground enamel was used, which is known to contain a low and consistent F concentration. Thus, it provides a background against which small amounts of acquired F can be measured[11]. In a preliminary study, the natural F levels in ground enamel (≈ 300 μn depth from the outer surface) were determined and found to be in a range between 225 to 240 ppm with a relative standard deviation of 12.4 per-

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†Pye Unicam Ltd., Cambridge, U.K.
‡Beckman Instruments, Inc., Fullerton, CA

Table ☐ Mean fluoride concentration (ppm ± SD) in three enamel layers of the experimental (lased) and control (unlased) groups after 1-min treatment with 2 percent NaF solution.

Group	Number of tooth halves	Standard depths		
		2.5 μm	5 μm	10 μm
Lased	12	2590 ± 523	813 ± 134	253 ± 70
Unlased	12	2639 ± 547	932 ± 272	316 ± 111

cent, indicating uniform F distribution. When an agent with a high F concentration is applied to enamel, the principal chemical product of the reaction is CaF_2. It should be pointed out that prolonged washing (24 h or longer) in water, artificial saliva or KOH is required to remove CaF_2 and other reaction products from the surface of F-treated enamel.[12-15] The brief washing used in the present study, however, was intended not to interfere or to mask any possible effect of laser on the deposition of F on enamel. The selected wavelength and power density were based on previous reports, which showed that 488 nm was the most suitable wavelength for early detection of caries lesions.[7] Moreover, the relatively low intensity of laser was considered to be safe for patient and operator.[6]

In a study on the effect of argon laser on powdered enamel mixed with NaF salt, it was found that 5-min irradiation at a wavelength of 514 nm resulted in significant increase in F content of powdered enamel.[16] The discrepancy between the previously and present findings could be explained on the basis that the enamel powder reacts differently with F than enamel surface.[17] Moreover, the longer wavelength, higher power output, and prolonged irradiation period used in the previous study may account for the increase in F content of powdered enamel.

Several workers have provided evidence that the mechanism of enamel F-uptake is a diffusion process accompanied by simultaneous chemical reaction.[18,19] The insignificant differences in F concentrations between lased and unlased enamel reported here indicates that argon laser does not affect the enamel permeability to, nor its reactivity with F agent. This information is required since argon laser has recently been suggested as a useful clinical tool in the diagnosis of initial caries lesions.

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The effect of fluoridated chocolate-flavored milk on caries incidence in elementary school children: two and three-year studies

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For economical, political, geographical or technological reasons, large segments of the world's population still are without benefit of fluoridated water. Alternate methods of providing fluoride have been used successfully in the United States and elsewhere. Fluoride tablets and mouthrinses, and topically applied preparations have reduced caries significantly, when the prescribed regimens are followed closely.[1-3]

The use of fluoridated milk has recently gained interest through studies completed in the USA, Israel, Scotland, and Hungary.[4] Milk would appear to be a good vehicle, because it is often provided at school on a daily basis. The addition of small quantities of fluoride does not adversely affect milk quality or dairy technology, and no adverse physiological effects have been reported with the fluoride-ion concentrations used in the fluoridated milk studies.[5] Caries incidence rates have been reduced from 31 percent to 76 percent, in the studies completed thus far.[4,6-9]

The variables of compliance with the daily intake regimen and the effect of the flavoring agents on caries, however, have not been considered. Banoczy reported using "cocoa milk", but did not attempt to determine the effect of this variable in her report.[10] Further, most clinical trials have used institutionalized or other populations for which the question of compliance was not a major issue.

Because chocolate is the strongly preferred milk flavoring agent in the USA and since neither food selection nor ingestion is monitored in most schools, it seemed appropriate to study the effects of chocolate flavored fluoridated milk in a voluntary school setting.

The objective of this study was to examine the effect of chocolate flavored, sweetened, low fat, fluoridated milk on the caries incidence of elementary school children.

METHODS

Students in grades K-4 from five demographically similar elementary schools, in a nonfluoridated (<0.4 ppm) community were invited to participate in this study. Forty-three percent of the combined schools' enrollment consented to participate. A descriptive study was first conducted to determine

- ☐ Whether the study population was similar in caries experience to its regional counterparts.
- ☐ Whether selection differences existed between tests and controls.

Calibrated examiners recorded all data for the study in

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the initial and second pretest examinations and a single examiner was used for the subsequent examinations. American Dental Association Class II screening criteria were used.

Necessary approvals were obtained from the University Institutional Review Board, State Health Agency, Food and Drug Control Unit, State Health Agency Milk Sanitation Unit, State Board of Pharmacy, Federal Food & Drug Administration, the public school board, school administrations, parent groups and local dentists. To facilitate state approval it was necessary to use the Louisiana State University (LSU) Creamery for production and delivery of the test milk. Training conferences with school teachers and food service personnel were conducted, to minimize error in the distribution of milk to the children.

Because test milk could be offered on only 170 of the 180 school days per year, and the natural fluoride concentration in the community water averaged less than 0.4 ppm, each 236 ml carton was formulated to contain 1.0 mg sodium fluoride. This was done to approximate the recommended 1.0 mg daily intake over the year. Quantities sufficient for a one-week supply of chocolate flavored, low-fat, homogenized, pasteurized milk were manufactured in the LSU Creamery and tested in that laboratory for standard coliform counts, milk-fat percentages, antibiotic contaminants, and fluoride concentration in ppm, using an Orion specific ion electrode. Additionally, samples from each production batch were tested in the LSU School of Dentistry Preventive Dentistry Laboratory, and randomly by the State Health Department. The average concentration of all samples tested over the period was 3.9 ppm, approximating the 4.0 ppm desired standard. No batches exceeded the upper acceptable limit of 5.5 ppm. Fresh whole milk with 1 percent fat, and vitamins A and D added, was used in the formulation. To each 100 gallons (860 lbs.) of milk, 12 lbs. (1.4 percent) of a standard basic cocoa mix, for flavor; and 50 lbs. (5.8 percent) of cane sugar were added. It was then agitated before pasteurization.

Predosed units of sodium fluoride prepared in the project laboratory were added by trained creamery technicians, to achieve the 4 ppm desired and to achieve other quality control criteria. The test milk was delivered to the schools on a weekly basis in a refrigerated truck.

Each school day, children who were to receive test milk with their lunches were identified by prelunch teachers with a colored sticker placed on their collars. Food service workers removed the stickers from the child and provided a package of the test milk. Regular, nonfluoridated milk was provided, if a child requested a second serving. The sticker process was monitored on a regular basis by the school nurse employed by the milk program. There was no problem with study children receiving the proper milk at any school. In the public school system, however, there is no monitoring, to determine whether the children actually consume the food provided. The only measure of consumption was for the group rather than the individual subjects. Consumption was determined by the number of containers ordered per week as compared to the number of test subjects.

Table 1 ☐ Test Milk Distribution

	1982	1983	1984	1985
January		EB	X	X
February	X	EB	X	X
March	X	EB	X	X
April	X	EB	X	X
May	X	EB	X	X
June	SR	EB	SR	
July	SR	EB	SR	
August	SR	EB	SR	
September	X	X	X	
October	X	X	X	
November	X	X	X	
December	EB	X	X	

X Distribution
SR Summer recess
EB Equipment breakdown

The test milk was actually available 451 days over a thirty-nine month period between February, 1982 and May, 1985. Because no back-up source of an identical milk formulation was available, there was a ten-month hiatus (December, 1982 to September, 1983), during which the milk production plant was inoperable due to renovation. Table 1 depicts the pattern of milk distribution throughout the study. Because of a high early attrition rate, due to academic promotions and family transfers, and because of the ten-month hiatus, a second cohort was added, one year after the trials began. Thus, there were two trials, a two-year group and a three-year group.

RESULTS

Of the 706 children originally examined, pretest and posttest data were available for 157 of the three-year group and 187 of the two-year group. Independent t-tests were used to compare baseline DMFT and DMFS scores, between the tests and controls. The statistical analyses were performed, using a program from the "Statistical Analysis System User's Guide: Basics."[11] There were no significant differences in Baseline DMFT or DMFS scores between test subjects and control sub-

jects, for either the two-year or the three-year cohorts.

Table 2 shows differences in DMFT scores for the prepost measures in the two-year and three-year cohorts. Independent t-tests were used to compare the prepost DMFT and DMFS score differences for the control group to the differences for the test group. There is a significant difference in DMFT scores for the two-year cohort ($t = 2.17$, $df = 185$, $p \leq 0.05$) representing 77.2 percent fewer decayed teeth for the test subjects. Analysis of data for the three-year cohort did not show significant differences between test subjects and control subjects.

Table 3 shows differences in DMFS scores for the two-year and three-year cohorts. The two-year group showed a 76.6 percent reduction in decayed surfaces for the test group ($t = 1.98$, $df = 185$, $p \leq 0.05$). The three-year cohort had a DMFS reduction of 21.8 percent, but this was not statistically significant.

Table 2
A. Mean DMFT increment by group after two years.

Group	Number of children	DMFT increment	Difference from control (percent)
C (control)	111	0.2883	—
T (test)	76	0.0658	77.2

B. Mean DMFT increment by group after three years

C (control)	88	0.6818	—
T (test)	69	0.6667	2.2

Table 3
A. Mean DMFS increment by group after two years.

Group	Number of children	DMFT increment	Difference from control (percent)
C (control)	111	0.4505	—
T (test)	76	0.1053	76.6

B. Mean DMFS increment by group after three years

C (control)	88	1.0568	—
T (test)	69	0.8261	21.8

Table 4 ☐ Percent of time fluoridated milk offered.

Two-year cohort		Three-year cohort	
Days in 17 month period	645	Days in 39 month period	1170
School days milk offered	357	School days milk offered	451
Percent of days offered	55	Percent of days offered	38

DISCUSSION

Despite strong statements of support from the community, there was an early and larger-than-expected attrition rate. Some subjects withdrew, because of parental concern with a national drug tampering scare, some because of an untimely publication of antifluoride articles in local newspapers; but most of the attrition was directly due to student transfers to middle school and families moving out of the area. This led to a decision to add a new cohort in the second year and provide a second study group.

The three-year group was offered test milk 451 school days and the two-year group 357 days. Table 1 displays the distribution pattern over the entire study period. It can be seen that for the three-year group, in addition to the planned three-month summer recess, there was an unavoidable hiatus in the study. Although consideration was given to substitution of another fluoridated milk product, this was rejected, since it would not have complied with the original research protocol. There was a considerable difference in caries reduction between the two-year cohort and the three-year cohort. Possible explanations for the difference were considered. Were the two cohorts different from each other? Did the differences in the milk distribution patterns have an effect? Were the students monitored differently? Was compliance a factor? Did the ratio of days-milk-offered to total days in the period make a difference?

Although the three-year cohort began the study with a slightly lower mean DMFT and DMFS than the two-year cohort, these differences were not significant. Also the three-year group had fewer erupted permanent molars at baseline. These facts do not appear to have affected the results.

A likely explanation for the difference centers around the observations of frequent noncompliance in drinking the milk during the first year of the three-year cohort. Some of the noncompliance was due to the unavoidable gap in the program. Once the students became accustomed to drinking the commercial nonfluoridated milk, some continued to express a preference for it, even when the study milk was again made available. Ingestion was not mandatory and the schools' policy was that children did not have to eat their lunches or drink their beverages. As a consequence, both food and milk were often thrown away and drinks from home substituted.

An attempt to encourage compliance was instituted with the beginning of the two-year cohort. Small tokens of recognition were given to those who drank their milk regularly; and based on feedback from teachers and food service personnel, compliance did improve.

The most interesting questions are raised, when one looks at the percentage of time milk was available in relation to the total time of the study (Table 4), in light of

recent findings related to the nature of the topical benefits of fluoride and the dynamics of the remineralization/demineralization process. The frequency of ingestion and length of time a fluoride ion reservoir is available on the tooth may be the critical factors in explaining the poor performance of the three-year cohort. The better compliance, and higher percentage of time the test milk was available to the two-year cohort may have provided better tooth protection. The ten-month lapse in delivery of milk to the three-year cohort could reasonably affect the potential caries reduction, but it is not known how much of an effect the interruption may have had.

CONCLUSIONS

Although there was a 77 percent caries reduction in a two-year study of chocolate-flavored, fluoridated milk, where ingestion was encouraged through a reward system, there is no simple explanation for the poorer results seen in a less tightly controlled three-year study.

The experience of the LSUSD Fluoridated Milk Study shows that caries reduction is possible with this vehicle, when compliance levels are maintained. This condition is most likely to be satisfied in an environment where the fluoridated milk is needed for its nutritional value as well as its anticaries value, and where children are expected or required to drink it, when it is provided.

Further work should be done on the nature of the topical effect of fluoride in milk, to address the dynamics of the ion exchange at the tooth/plaque interface and to determine optimum standards for milk fluoride, concentration and ingestion patterns.

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A SOCIETY FREE OF SMOKING

It is imperative that government agencies and private organizations work together at international level to provide the public with information concerning the adverse consequences of smoking. I feel that we can reduce the number of smokers worldwide through public health education, media presentation, legislation, and other activities.

The pathway to a society free of smoking may have many barriers thrown across it and may seem to be going uphill, but every civilized society should follow it. Otherwise, there is scant hope of reducing the numbers of premature deaths from cancer, heart disease, stroke, and chronic obstructive lung disease.

C. Everett Koop: A society free of smoking by the year 2000?
World Health Forum, 7:225-29, 1986.

Behavior

Management of the refractory young child with chloral hydrate: dosage selection

John E. Nathan, DDS, M Dent Sc

Despite the widespread use of chloral hydrate, there is little agreement among pediatric dentists regarding its therapeutic dosage for management of very uncooperative young children. Often selected for its wide range of safety, increasing concern has been raised with respect to its frequent failure to provide adequate levels of sedation, when using the manufacturer's recommended hypnotic dosage. To date, few studies have been conducted in an attempt to establish a therapeutic dosage range of chloral hydrate (alone or in combinations) for management of the refractory young child needing extensive treatment. While some pediatric dental texts have advocated dosages exceeding manufacturer's recommendations, none has offered controlled data to substantiate claims of safety and efficacy.

Several patient and dentist factors contribute toward a declining selection of more potent and predictable modalities, such as general anesthesia or parenteral sedation in the management of refractory children. These include increased public concern regarding the appropriateness and safety of sedation and general anesthesia by dentists, prohibitive costs associated with treatment performed in the hospital, anesthetic risks, increased liability costs, and the need for extensive armamentariums in an office setting.

This paper has two objectives. The first will focus on a discussion of the problematic issues associated with the use of chloral hydrate and will include a detailed analysis

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of the studies which have appeared in the literature. The second aim will be to discuss the implications of the weaknesses in our understanding of this agent in a pedodontic context. Several considerations regarding the design of future research to circumvent these difficulties will be discussed.

PATIENT AND DRUG CONSIDERATIONS IN ORAL PEDIATRIC DENTAL SEDATION

Patient considerations

An immediate objective of the pediatric dentist is to accomplish treatment in the least stressful manner possible. For many young children below the age of reason, neurologically handicapped and lacking in cooperative ability, conventional communication and nonpharmacologic approaches may be inadequate or even inappropriate. The selection of a management strategy must take into account factors such as safety, feasibility, patient and parent acceptance, risk and cost considerations. The oral route of administration offers distinct advantages with respect to patient acceptance and diminished risk, when compared to more potent parenteral routes. Limitations include variable absorption and no opportunity for titration. Despite disadvantages, its safety record and practicality maintain the oral route as an attractive initial alternative to a general anesthetic or parenteral technique.

Parent considerations

Preparation of the parent regarding the sedation visit and adequate informed consent are essential. It is imperative the parent clearly understands the rationale by which the modality suggested has been selected, the NPO instructions, onset, treatment, and recovery expectations.

While some practitioners prefer to administer medications upon arrival in the office, others permit parental home administration (less threatening environment) 30-45 minutes before the appointment time. This decision is generally based upon anticipated parental compliance and patient considerations. All oral instructions should be reinforced in written form.

Parents must be apprised of the long duration of onset, action and recovery. To minimize or prevent alarm, it would be helpful to identify potential sources of alarms

- ☐ Occasional agitation or disorientation while the drug is being absorbed (not uncommon).
- ☐ Sedation adequate to permit treatment may last 1-1 1/2 hours, after which drug effects will dissipate slowly over the next several hours. Although easily arousable when spoken to or by physical stimulation, many children may be seen to return to a somnolent state, during the recovery period. Discharge from the dental office, however, should not occur until the patient is stable, awake and ambulatory.

Drug considerations

Critical to the successful use of pedodontic oral sedation, is recognition that the child is not a "small adult", when selecting drugs or dosages.[1] Pharmacokinetic factors that reflect differences in surface area, organ size, cardiac rate and output, basal metabolic rate, distribution and glomerular filtration support the contention that children often require greater doses per body weight than adults.[2] As such, it is acknowledged that earlier dosage rules established for children, (e.g. Young's Clark's, Fried's, Cowling), representing small fractions of adult dosages based on age and weight, are of limited value. Unfortunately, dosages for sedative- hypnotics, on the basis of surface area, have not been presented.

Further, it must be understood that manufacturer's dosage recommendations for sedative-hypnotics are calculated to provide sedation for an essentially cooperative individual and can, at best, be expected to serve as minimum baseline starting dosage to overcome highly resistive child behaviors. Musselman *et al* suggested that additional factors such as physical activity level, emotional status, degree of cooperation, stomach contents, and time of day likely contribute to the need to surpass baseline dosages.[2]

If there appears to be some mystique about oral dosage selection for children, it is probably in large part a skill acquired from years of experience witnessing children's reactions to oral sedation. On the basis of over twenty-five years, Kopel suggested dosages in excess of manufacturer's recommendations, yet consistent with current pedodontic textbook recommendations.[3,4]

The initial assessment of patient response and clinical judgment to alter dosages above or below manufacturer's recommendations are of critical importance.

An important concept regarding pedodontic oral sedative-hypnotics is the differentiation between paradoxical excitement and overdosage. The novice is likely to misinterpret agitation of the child as a sign of overdosage either during or shortly after the latent period. This is a common phenomenon observed before drug absorption

is maximal and an adequate level of sedation is achieved. Under circumstances where agitation persists, underdosage, rather than overdosage is usually the correct assessment. Also characteristic of a slight underdosage is a child who may become somnolent during the latent period, yet becomes fully aroused and unmanageable upon the introduction of noxious stimulation (insertion of a mouth prop, injection, clamp placement, or cavity preparation). This state generally persists until distribution and dissipation of the drug effects occur. Often it is necessary to terminate the procedure and make adjustments in either dosages or modality for the next visit. Occasionally, additional drug(s) can be administered and treatment resumed following an additional 15-45 minute latency, if patient and office schedules permit. Alternatively, the use of a titrable agent such as N_2O may be added to achieve adequate sedation and avoid postponement. This is not easily accomplished, however, since a degree of patient compliance is required for this technique to be applied.

Table 1 ☐ Chloral hydrate (manufacturer's recommendations).[7]

Orally:	Sedative dose: 25 mg/kg Hypnotic dose: 50 mg/kg
Rectally:	Hypnotic dose
Maximum single dose:	1000 mg.
Toxicity:	10 grams (one death reported with 4 grams)

Table 2 ☐ Chloral hydrate (pediatric dental texts recommendations).

Oral	Rectal	Source
25-50 mg/kg (for children > 60 lb less for sedation	Same	McDonald & Avery[5] (1984)
50-70 mg/kg	Same	Trapp, L.[9] (1982) (in Stewart *et al*)
500 mg (2-3 yr; 25-30 lb) 750 mg (3-4 yr; 30-35 lb) 850 mg (4-5 yr; 35-40 lb) 1000 mg (5-10 yr; 40-65 lb)	60-900 mg	Malamed[10] (in Braham & Morris) (1980)
500 - 700 mg (2-4 yr) 750 - 900 mg (4-7 yr) 1000 - 1500 mg (> 7)	Same	Sim[11] (in Wright, G.) (1975)

DEPTH OF SEDATION: CONSCIOUS SEDATION OR DEEP SEDATION

The response to increased public concern regarding the appropriateness and safety of sedation in the dental office has been substantial both within and outside the profession. On state and federal levels and through increased initiatives by dental specialty organizations, guidelines and legislation have been proposed for the use of conscious sedation, deep sedation and general anesthesia.[5,6] Inherent in the safe utilization of sedative modalities is appropriate dentist training and familiarity with drugs and techniques, proper monitoring of vital functions, ability to recognize when health is compromised, and the proper availability and use of emergency drugs and equipment.

Continuous monitoring and assessment of the depth of sedation of the patient receiving hypnotic dosages of chloral hydrate is essential. An expectation commonly associated with this agent is an onset of somnolence. Although it could be argued that this level of depressed consciousness does not differ from the unmedicated child patient who falls asleep during treatment, the induction of somnolence precludes classification of the depth of sedation as falling within the realm of conscious sedation. Although believed to be easily arousable by physical or voice stimulation, be definition, classification when accompanied by somnolence, most appropriately should be considered "deep sedation" (controlled state of depressed consciousness which may be accompanied by a partial loss of protective reflexes, including inability to respond purposefully to voice command). Interpretations regarding minimum precautions, monitoring requirements, etc. may vary, therefore, according to individual state regulations.

CHLORAL HYDRATE IN PEDODONTIC PRACTICE

Tables 1 and 2 list the manufacturer's and pedodontic text dosage recommendations for chloral hydrate. The ensuing section reviews the pedodontic trials which attempted to assess the efficacy and safety of various dosage schedules. Tables 3 and 4 summarize the pedodontic and medical trials. It should be noted that the medical trials make use of higher doses of chloral hydrate, and none represents a controlled investigation.[24-28]

Czarnecki and Binns premedicated 100 "difficult-to-manage" children (majority under nine years of age) in a span of 422 visits, with chloral hydrate using dosages of 500 mg for children up to six years and 1000 mg for those over six.[12] Excellent and good results were reported in 21 percent and 60 percent respectively; treatment was completed on another 17 percent, although not without difficulty while only 2 percent were unmanageable. The high frequency of success (81 percent), which differs markedly from the later findings of Smith and Evans *et al*, who examined similar dosages, strongly questions the extent to which subjects were difficult to manage without drugs.[13,14]

Table 3 ☐ Pedodontic trials using chloral hydrate alone or in combination.

Drug dosage ranges and experimental conditions	Therapeutic success	N =	Adverse reactions reported	Source
50 mg/kg + 50% N_2O vs. 75 mg/kg + 40% N_2O (2 Hr. NPO)	18% 75%	17 17	vomiting 4/34	Houpt *et al*[23] (1985)
50 or 70 mg/kg + 25 mg hydroxyzine ± 20-30 mg meperidine ± 10-50% N_2O (min. 6 Hr. NPO)	83% (meperdine) 43% (w/o meperdine)	63 79	vomiting 4/142	Nathan and West[22] (1985)
75 mg/kg + 50% N_2O vs. 50 mg/kg + 25 mg promethazine + 50% N_2O	79% 90%	21 21	45% vomiting 14% vomiting	Houpt *et al*[20] (1984)
75 mg/kg + 50% N_2O vs. 50 mg/kg + 25 mg promethazine + 50% N_2O vs. 50 mg/kg + 50% N_2O	72% 89%	19 19	48% vomiting 28% vomiting	Koenigsberg *et al*[19] (1984)
60 mg/kg + 40% N_2O vs. 40 mg/kg + 40% N_2O vs. 20 mg/kg + 50% N_2O vs. Placebo (3 Hr NPO)	79% 27% 40% 46%	15 15 15 15	4/15 (27%) airway —— obstr. —— ——	Moore, *et al*[21] (1984)
75 mg/kg + 40-50% N_2O vs. 50 mg/kg + 40-50% N_2O	65% 6%	17 17	4/34 vomiting	Sheskin *et al*[18] (1983)
40 mg/kg vs. Placebo (30 min. latency) no NPO restrict.)	No stat diff	21 21	no episodes of vom.; one 219 lb. pt. rec'd 3,984 mg without problem	Barr *et al*[17] (1977)
1000 mg + 50-200 mg hydroxyzine vs. 1500 mg + 50-200 mg hydroxyzine	62% 56%	29 9	——	Tobias *et al*[16] (1975)
15 grains vs. 7.5 grains + 25 mg promethazine	60% 68%	58 pts. 142 visits	10% vomiting	Robbins[15] (1967)
500 mg < 6 yrs. 1000 mg > 6 yrs.	81% Overall	100 pts. 422 visits	—	Czarnecki and Binns[12] (1963)

Evans *et al* acknowledged the need to study pedodontic premedication agents under controlled conditions.[14] Using rating scales to score patient manageability and emotionality quantitatively, they compared 12 and 15 mg/lb doses of chloral hydrate under blind conditions with a placebo. An occlusal restoration was placed, on each of two visits, in seventy-five children (3-8 years of age) who "appeared apprehensive and difficult" to manage. The authors reported surprise that no significant differences were found at that dosage and attributed the failure to find drug effects, to a failure of drug action. The fact that parents were instructed to feed children thirty minutes prior to drug administration and treatment commenced following only a thirty-minute latent period likely minimized drug absorption. Despite these factors, the low dosage and probable low levels of pretreatment anxiety, this study was among the first to draw attention to the need to evaluate oral premedication agents, more objectively.

Acknowledging the frequent failure rate, when following the manufacturer's recommended dose of chloral hydrate, Sheskin *et al*, in a two visit cross-over design, compared the effectiveness of 50 mg/kg with a 75 mg/kg dose on seventeen children (18-46 mos.).[18] Successful

Table 4 ☐ Medical trials involving chloral hydrate.

Drug dosage ranges and experimental conditions	Therapeutic success	N =	Adverse reactions reported	Source
80 mg/kg oral C.H.	85%	231	1 episode of respiratory distress associated with excess secretions & enlarged tonsilar & adenoid tissues	Thompson, *et al*[28] (1982)
50 mg/kg for < 4 wks. 100 mg/kg for > 4 wks.	98%	300	15% nausea/vomit 5% excitement	Judisch *et al*[27] (1980)
75 mg/kg	——	No data presented	——	Houser *et al*[26] (1975)
50-100 mg/kg rectal 500 mg p.o. (1-2 yrs.) 750-1000 mg (> 2 yrs.)	——	No data presented	——	Davis[25] (1973)
25 mg/ (month of age)	90%	No data		Carabelle[24] (1961)

sedation as evidenced by a lack of movement and/or crying which interfered with treatment was found in 6 percent and 65 percent of the visits with the low and high doses, respectively. Four episodes of vomiting were reported. Had N_2O in concentrations of 40 - 50 percent not been administered to all subjects, interpretations with respect to the effectiveness of the oral medications alone may have been enhanced. In view of the wide individual variation among children as well as adults with respect to therapeutic concentrations of N_2O, it is conceivable that some children may have shown agitation (stage II) by N_2O alone. On the other hand, considerable differences were reported in the control of interfering behaviors between the high and low doses with a relatively low incidence of vomiting. The behavioral selection criteria was not defined other than to indicate that subjects were nonexperienced and required at least two premedication visits. The fact that such low success rates were achieved, particularly with the 50 mg/kg dose, however, strongly suggests subjects were severely anxious to warrant medication. The impact of N_2O nevertheless confounds this issue. Houpt *et al* (1985), described later, amplifies on this design.

In preliminary presentations, Koenigsberg *et al* and Houpt *et al*, adding a few more subjects, compared the effectiveness and safety of chloral hydrate with and without promethazine.[19,20] Using a cross over design and blind conditions in two visits, they rated the behavioral (degree of sleep, crying, and body movements) and physiologic responses (blood pressure, pulse and respiratory rates, and pupil size) before, during, and after dental treatment of twenty-one children, (15-45 mos; mean 32 mos.). Subjects received randomly 75 mg/kg chloral hydrate or 50 mg/kg chloral hydrate plus 25 mg promethazine. All subjects received 50 percent N_2O and were restrained in a papoose board® with head holder. While Koenigsberg *et al* reported an incidence of nausea/vomiting of 48 percent and 28 percent respectively (in 19 subjects), Houpt *et al* reported 45 percent and 14 percent (in twenty-one patients) receiving the chloral hydrate without and with promethazine, respectively.[19,20] The increase in nausea/vomiting of subjects receiving 75 mg/kg chloral hydrate differs markedly from those receiving the identical regimen in Sheskin's study.

Successful sedations (lack of crying and/or interrupting movements) were found in 90 percent of the cases using the antiemetic compared to 79 percent with the high dose chloral hydrate without the antiemetic.

On the basis of this data, it would appear that the addition of 25 mg promethazine dramatically increased the success rate of the 50 mg/kg dosage of chloral hydrate from 6 percent to 90 percent; while success of the 75 mg/kg dose supplemented with 50 percent N_2O in this study (72 percent) did not differ greatly from Sheskin's findings (65 percent).

In view of the upsetting nature of chloral hydrate on the gastrointestinal functions and the high level of N_2O administered, it was not surprising to observe a 48 percent incidence of vomiting. The authors acknowledged that both factors may have contributed to the high incidence of vomiting and agree that future study should reduce or eliminate the confounding nature of N_2O. As a result, the increased success achieved by adding 25 mg of promethazine was not easily addressed.

Noteworthy, however, were the use and description of rating scales for the assessment of patient alertness, movement, quality of crying, and overall behavior. The use of pupillary size as a fine index of depth of sedation and the noncontinuous monitoring of vital signs, however, seem inadequate from the perspective of being able to differentiate depth of sedation, particularly in the somnolent patient. Greater opportunity for comparison may have been possible had promethazine been administered with both chloral hydrate dosages.

Moore *et al* compared the responses of sixty children, aged two to five years, considered uncooperative, under blind conditions for one dental visit. Subjects received either a placebo, 20 mg/kg, 40 mg/kg, or 60 mg/kg chloral hydrate, with and without 40 percent N_2O. Using a Frankl rating system, statistically significant improvements were observed only for subjects receiving 60 mg/kg chloral hydrate compared to placebo (without N_2O-O_2). It was particularly interesting to note that positive behaviors among the placebo group ranged from 46-67 percent when rated across all aspects of a visit, particularly during the injection with N_2O. This finding strongly suggests their behavioral selection criteria included a large percentage of nonanxious subjects, or at least subjects who were highly responsive to standard nonpharmacologic management approaches. Similar high percentages of positive behaviors (40 percent and 27 percent) observed with the 20 mg/kg and 40 mg/kg groups, respectively, during injections with N_2O confound the ability to draw conclusions regarding the safety (or lack of) of using 60 mg/kg dosage with 40 percent N_2O.

These authors reported airway obstruction in four of fifteen subjects for the 60 mg/kg group, when 40 percent N_2O was administered. It should not be surprising to find this dosage to be too high, if used for nonanxious or minimally anxious subjects. It seems warranted that

further study utilizing an adequate behavioral selection criteria upon which to evaluate the efficacy and safety of various dosages of chloral hydrate should be made.

Nathan and West retrospectively compared the efficacy and safety of a chloral hydrate-hydroxyzine combination with and without low doses of oral meperidine on 135 unmanageable (requiring harsh physical restraint) patients, ages eighteen to sixty months (mean: 34 mos.).[22] Treated over a span of 142 visits, subjects received either 50 or 70 mg/kg doses of chloral hydrate with 25 mg hydroxyzine with and without 20-30 mg meperidine. Success of sedations were defined on the basis of the ability to complete treatment and the extent to which physical restraint was needed to overcome persistent interfering behaviors.

Conducted in a private practice setting, Robbins compared the responses of fifty-eight children (22 mos - 6 years of age) over a span of 142 visits to placebo, 15 grains chloral hydrate, and 7.5 grains of chloral hydrate plus 25 mg promethazine.[15] The author indicated that subjects demonstrated strongly apprehensive behavior during an initial visit for examination and radiographs. Statistically significant differences were reported for both drug conditions compared to the placebo, with less nausea/vomiting resulting from the combination using promethazine. Of importance was the observation that the addition of an anti-emetic enabled using half the chloral hydrate dose without reducing therapeutic success. This benefit was later amplified in studies of Houpt and Koenigsberg described below.

Tobias *et al* assessed the effectiveness of a chloral hydrate-hydroxyzine combination on thirty-nine previously unmanageable children (ranging from 1.75-10.5 years of age; mean 3.9 years).[16] Subjects received either 1000 or 1500 mg chloral hydrate, and 50 mg hydroxyzine one hour before appointments. In addition, some subjects received up to 150 mg (in 50 mg divided doses) of hydroxyzine starting the day, evening and/or morning before the appointment doses. Two three-year old subjects received two additional 1000 mg doses of chloral hydrate upon rising and in the morning before the preoperative combination. Fifty-one percent of the cases were reported as effective, 13 percent were semieffective and 28 percent showed fair-to-poor results. The arbitrary selection (on the basis of age) of chloral hydrate dosages and a failure to utilize a specific mg/kg dosage no doubt minimized meaningful assessment of the efficacy of this regimen. No attempts were made to examine the potentiating effects of the multiple dosage administrations of hydroxyzine of chloral hydrate.

Barr *et al* subjectively assessed the responses of twenty-one patients, ranging in age from one to seventeen years, using cross-over blind conditions across two visits.[17] Subjects received a placebo at one visit and 40 mg/kg chloral hydrate for the other visit. No statistically significant differences were found at this dosage, and the authors concluded that chloral hydrate is not recommended for the very young, the mentally retarded, or the emotionally disturbed pediatric dental patient. No description was made of the behavioral selection criteria and the authors acknowledged that several subjects manifested improved behavior throughout a series of visits. On the basis of the high incidence of positive behavior from the placebo, one must question whether subjects were sufficiently difficult to warrant medication at the outset. Other design limitations included small sample size, wide distribution of age, inclusion of neurologically normal and handicapped patients, low chloral hydrate dosage, no definitive NPO instructions, and an insufficient latency period (30 minutes).

Smith evaluated the behavioral (Frankl Scale) and kinesic/vocalization responses of fourteen neurologically handicapped patients, ranging in age from four to sixteen years (mean: 10 years) in two dental visits.[13] Using a cross-over and blind conditions, subjects received chloral hydrate or a placebo. Dosages of chloral hydrate ranged from approximately 400 mg to 1500 mg. Children weighing 5-10 kg received 75 mg/kg with dosage decreasing to 30 mg/kg for subjects weighing up to 50 kg. No differences between placebo and chloral hydrate were found for either behavior or kinesics. Despite the appropriate use of the blind cross-over, design flaws which included small sample size, wide distribution of age, and variable dosage no doubt minimized the opportunity to detect group differences.

Although treatment objectives were completed in 71 percent and 97 percent of the nonmeperidine and meperidine visits, rigid restraint was needed in 57 percent and 17 percent of these cases, respectively. Successful sedations (no need for persistent application of restraints) occurred, therefore, in 43 percent and 83 percent of the cases involving nonmeperidine and meperidine, respectively. N_2O (in concentrations ranging from 10- 50 percent) was used only after premedications were judged inadequate to produce adequate levels of sedation. No significant improvements in success were attributable to either the higher dose of chloral hydrate or the addition of N_2O. Limitations of this preliminary study included the lack of control conditions and relatively small sample size (N= and 9) of those receiving the high dose of chloral hydrate with meperidine, with and without N_2O, respectively. En-

hanced success by the addition of low doses of meperidine, while enabling significant reduction of chloral hydrate dosage warrants further study.

Houpt *et al*, using an identical design and assessment criteria to their earlier study, compared the effectiveness of 50 mg/kg vs 75 mg/kg chloral hydrate on seventeen children (21-46 mos., mean 31 mos.)[23] All subjects received N_2O in concentrations 40 - 50 percent and the papoose board without the head restraint. All parents were instructed to feed children a light meal consisting of a small glass of milk and bowl of cereal at least two hours before the appointment. While the impact of this feeding on drug absorption is unclear, the incidence of nausea was reduced to four instances.

Overall, 82 percent of the low dose administrations were rated as bad or very bad, while 75 percent of the high dose administrations were rated good or very good. The authors concluded that 75 mg/kg + 40 percent N_2O provided significantly better sedation than 50 mg/kg chloral hydrate + 50 percent N_2O. They also conceded that N_2O. They also conceded that N_2O likely had a confounding effect and suggested future studies be done without N_2O.

SUMMARY AND IMPLICATIONS FOR FUTURE RESEARCH

Considerable study of the use of chloral hydrate in pediatric dentistry was undertaken. Consistent with observations of Duncan *et al*, wide variation in dosage selection exists among pedodontic practitioners.[29] Of the clinical trials conducted within pedodontic and medical fields, rarely are dosage recommendations and claims of clinical success based upon substantive controlled data.

On the basis of these studies, several conclusions emerge which should serve as focus for future study. ☐ Despite widespread use, no criteria have been established to clarify effective and safe dosage guidelines for management of the severely resistive young child. ☐ Explanations to account for the range of success/failure reported with chloral hydrate dosages (alone or in combinations) appear multifactorial in nature. Deficiencies in research design include:

- ☐ Inadequate subject selection criteria. Ample demonstration of adequate levels of pretreatment anxiety is needed to assure the ability to detect differences attributable to a drug. Inclusion of minimally anxious subjects sensitive to standard behavior management techniques further obscures the opportunity to differentiate effects of experience and adaptation when multiple visit studies are conducted.
- ☐ Inadequate NPO enforcement and allowance for latent periods. Need exists to control factors that affect drug absorption by the oral route. An adequate period (4-6 hours) of fasting or restriction of foods and nonclear liquids as well as allowance for a sufficient latent period can be expected to facilitate drug absorption. Other pertinent variables which include "first pass" metabolism, and anxiety itself which limits gastrointestinal motility, need to be factored into dosage assessment.
- ☐ Inadequate sample size.
- ☐ Lack of blind conditions.
- ☐ Avoidance of confounding drug(s).
- ☐ Demonstrated validity of behavioral assessment scales/ratings.
- ☐ Need for continuous measurement/monitoring/recording of vital physiologic functions during operative and recovery periods.
- ☐ No assessment of recovery.

Despite a dearth of studies involving chloral hydrate in children, controlled data are needed to clarify its efficacy and criteria for dosage selection. Additional research appears warranted to guide the most effective use of this sedative agent as an alternative to general anesthesia or parenteral sedation.

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CHILDREN WITH IDDM

The majority of children who are diagnosed as having insulin-dependent diabetes mellitus, (IDDM) appear to adjust to this illness, and for those who display adjustment problems, the majority improve shortly after their diagnosis. While education about the nature and treatment of IDDM is important for parents and patients, it is not sufficient to ensure diabetic compliance. Assessment strategies—such as direct, nonjudgmental questioning, observing the child or parent perform the technique, and looking at the quality of the weekly monitor sheet—can provide information about a child's and parent's compliance to the medical regimen. For the child who does not adhere to aspects of the diabetic regimen—most likely daily blood glucose monitoring—contingency contracting between parents and child appears successful. More disturbed families need family therapy to improve compliance.

Steven J. Beck: Adjustment and compliance of the child with diabetes. Feeling, 28: 28, 1986.

Clinic

Evaluation of an iodoform paste in root canal therapy for infected primary teeth

Franklin Garcia-Godoy, DDS, MS

Root canal treatments (or pulpectomy techniques) for primary teeth have been recommended by many authors.[1-10]

The primary goals are to eliminate infection and retain the tooth in a functional state until it is normally exfoliated, without endangering the permanent dentition or the health of the child.

The most popular root canal filling material for primary teeth is zinc oxide and eugenol. Because of its hardness, however, deflection of the succedaneous tooth may occur.[5] Barker and Lockett reported that the material, when extruded from the apex, was not resorbed, and caused a mild foreign body reaction.[11] Erausquin and Muruzabal showed that zinc oxide and eugenol is irritating to the periapical tissues and may produce necrosis of bone and cementum.[12] For this reason, care should be taken not to force the material past the apex.

In recent years, Rifkin reemphasized the use of a resorbable iodoform paste instead of zinc oxide and eugenol, based on excellent clinical and radiographic findings.[7,8] Excess paste extruded from the apex was resorbed within one to two weeks. The iodoform paste he used, KRI 1 paste, is basically Walkhoff's paste.[13]

The purpose of this study was to evaluate the effectiveness of the iodoform paste in the root canal treatment of infected primary teeth.

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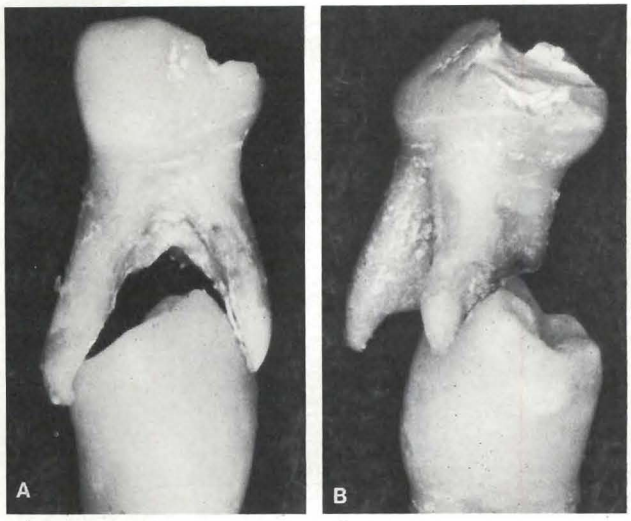

Figure 1. Radiographic root canal measurement could give unreliable values based on pathological/physical resorption damaging the permanent tooth. Even cases with the permanent tooth below the apices could give inaccurate measurements as in Figure 9.

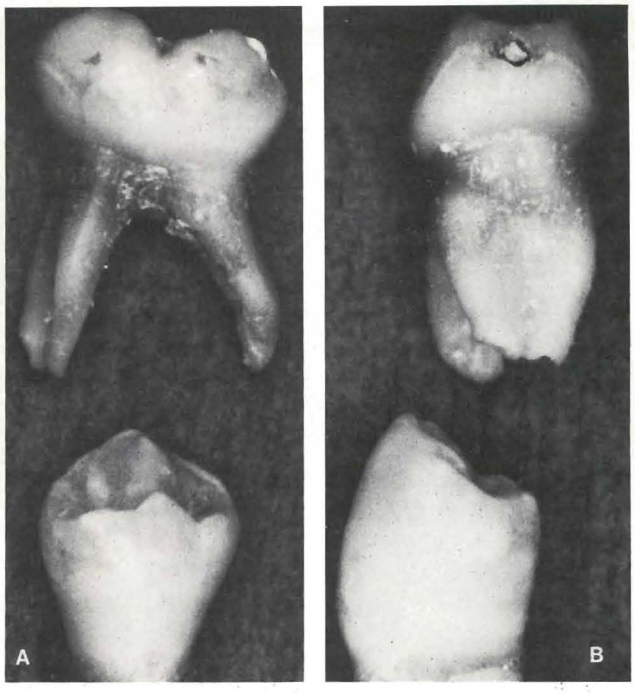

Figure 2. Radiographic root canal measurement could be more reliable and safe when the permanent tooth bud is below the apices of the primary molar.

MATERIALS AND METHODS

The sample consisted of fifty-five children, 2.5 to nine years of age, with fifty-five infected primary teeth (ten maxillary, thirty-five mandibular). The teeth were considered infected, if they had one or more of the following signs or symptoms:

- ☐ Presence of an abscess or a fistula.
- ☐ Radiographic evidence of morbidity.
- ☐ Presence of pus, excessive bleeding or little or no pulp tissue remaining when the pulp chamber was entered.

Treatment was considered contraindicated, if any of the following conditions were present:

- ☐ Perforated pulpal floor.
- ☐ Radiographic evidence of extensive internal or external root resorption.
- ☐ The tooth is not restorable.
- ☐ Extensive bone resorption over the permanent tooth.
- ☐ Extreme mobility.
- ☐ Patient has medical or behavioral problems.

Treatment regimen

Anterior teeth were isolated with cotton rolls and posterior teeth with rubber dam. Access was gained to the pulp chamber by removing the chamber roof with a bur in a high-speed handpiece. All necrotic tissue was removed with excavators and barbed broaches. The length of the root canals was determined from preoperative radiographs. Most of the canals measured 10 to 15 mm. The canals were irrigated with sodium hypochlorite and hydrogen peroxide and dried with paper points. A cotton pellet slightly dampened with KRI 3 liquid* was sealed in the pulp chamber with fast-setting zinc oxide eugenol cement for three to seven days. Fistulae were incised, compressed with sterile gauze, and swabbed with tincture of iodine in a cotton applicator. If, after cleaning the root canals, a discharge continued to exude from the apical area, drainage was established by leaving the canals open, with a dry cotton pellet in the pulp chamber to avoid food impaction in the canals. At the next appointment (usually at three days), the KRI 3 liquid was sealed as previously described.

At the second visit, the tooth was isolated as at the first visit and, if moisture was present, the treatment was repeated; if not, Hedstrom files with root canal stops at 11 mm were used in a pull-back direction, to clean and slightly enlarge the canals. In the case of primary molars, if the unerupted permanent tooth bud was within the furcation area, instrumentation of the canals was limited to a level above the occlusal plane of the permanent tooth (Figure 1). If the permanent tooth bud was below the apices of the primary tooth, the canals were cleansed and filed for their entire length (Figure 2). The reason for this technique is illustrated in Figures 3 and 4.

The root canal filling material used was the KRI 1 paste.† The filling material was transported to the canals with a lentulo, the active portion of which has been

*Pharmachemie AG, Drusberstr. 125, CH-8053 Zürich, Switzerland (25% p-chlorophenol, 60% camphor, 15% menthol).

†2.025% p-chlorophenol; 4.86% camphor; 1.215% menthol; 80.8% iodoform.

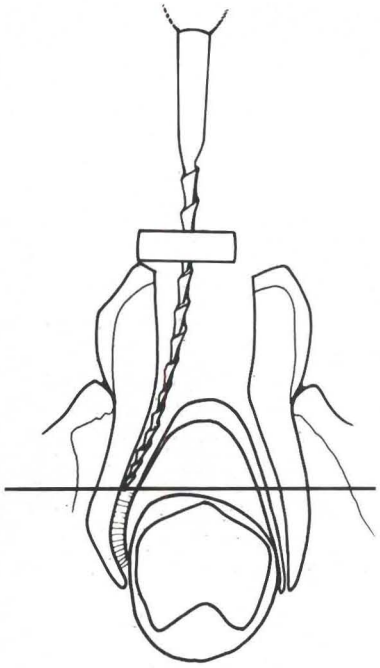

Figure 3. If the permanent tooth bud is within the furcation area, use of instruments should be limited to a plane just occlusally to occlusal plane of the permanent tooth.

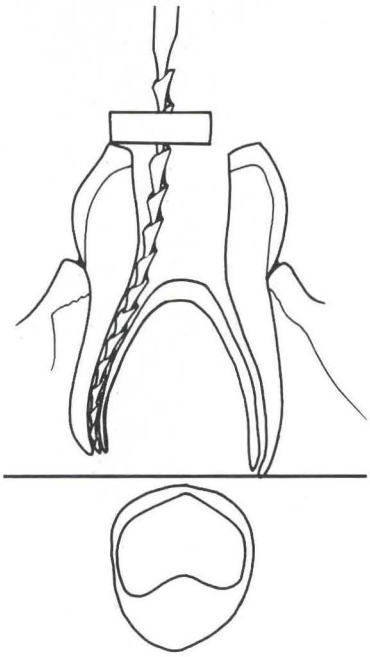

Figure 4. If the permanent tooth bud is below the apices of the primary tooth, instruments can be used for the entire length of the canals.

trimmed to 10 mm and placed in a low-speed handpiece. The lentulo was withdrawn gently from the canals while still rotating. This was done three to five times. Immediately, a small amount of the paste was placed in the pulp chamber and pressure was exerted, using several cotton pellets in the chamber and one held with a cotton plier. For the narrower root canals, one drop of KRI 3 liquid was added to the KRI 1 paste to facilitate its flow.

After the canals were filled, a thick paste of reinforced zinc oxide eugenol[‡] was placed. Amalgam restorations were placed on anterior teeth and stainless steel crowns on posterior teeth.

At six-month intervals, following the root canal treatment, clinical and radiographic examinations were made. The treatment was considered successful, if clinically, the tooth was painless, without pathologic mobility, and the gingiva was healthy and without a fistula. Radiographically, any preexisting radiolucencies had to be resolving within six months.

Clinical and radiographic follow-up varied from six to twenty-four months (Table).

RESULTS

Of the fifty-five treated teeth, ten were not available for the six to twenty-four-month follow-up, because the patients had moved or failed to appear for the recall examination.

The Table presents the rate of clinical and radiographic success, at the time the tooth was last examined. Two teeth were considered failures, one because no reduction in size of a preexisting radiolucency had occurred, and the other because persistence of the fistulous tract after fifteen days, postoperatively.

No clinical or radiographic signs or symptoms of failure were observed in 95.6 percent of the treated teeth (forty-three out of forty-five teeth).

Figures 5 to 9 show cases treated with the iodoform paste.

DISCUSSION

The present study shows that from a clinical and radiographic standpoint, the iodoform paste evaluated is an effective root canal filling material for infected primary teeth.

The iodoform paste is bactericidal to microorganisms in the root canal and loses only 20 percent of its potency over a 10 year period.[14] On the other hand, zinc oxide eugenol pastes are not bactericidal, unless mixed with drugs, such as formocresol.[15] Because formaldehyde has

Table ☐ Clinical success rate of root canal treatment with an iodoform paste.

Time in months after treatment	Clinical status at the last examination		
	Success	Failure	Total
1-6	1	1	2
7-12	2	1	3
13-18	8	0	8
19-24	32	0	32
Total	43	2	45

[‡]IRM, L.D. Caulk Co., Milford, DE 19963.

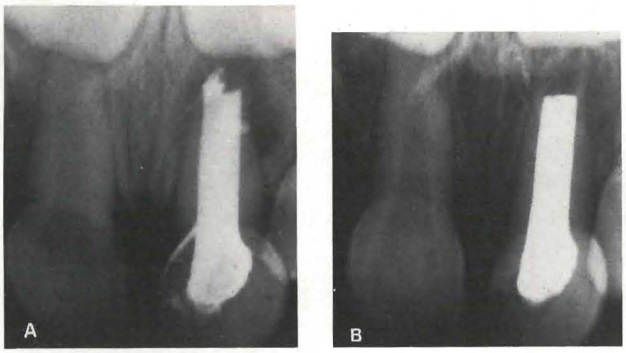

Figure 5. A, Preoperative and B, twelve-month postoperative radiographs of a primary central incisor treated with KRI paste.

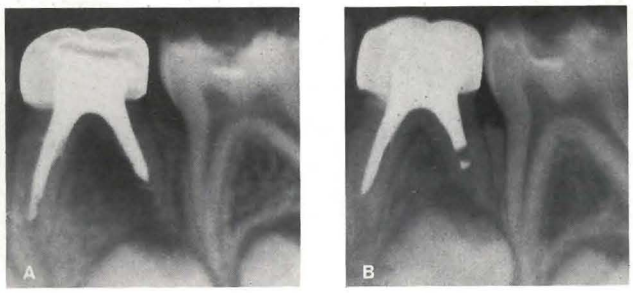

Figure 6. A, Preoperative and B, twelve-month postoperative radiographs of a primary first molar treated with KRI paste. Note resorption of paste within the distal root.

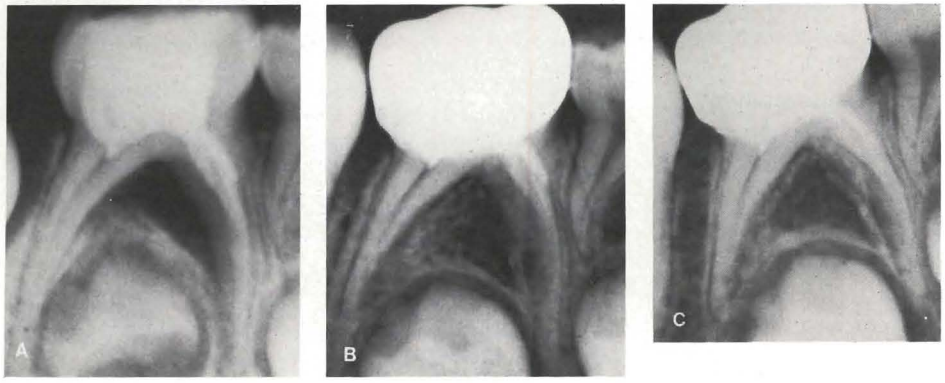

Figure 7. Partial pulpectomy with KRI paste. A, immediate postope rative, B, 18-month, and C, 24-month postoperatively.

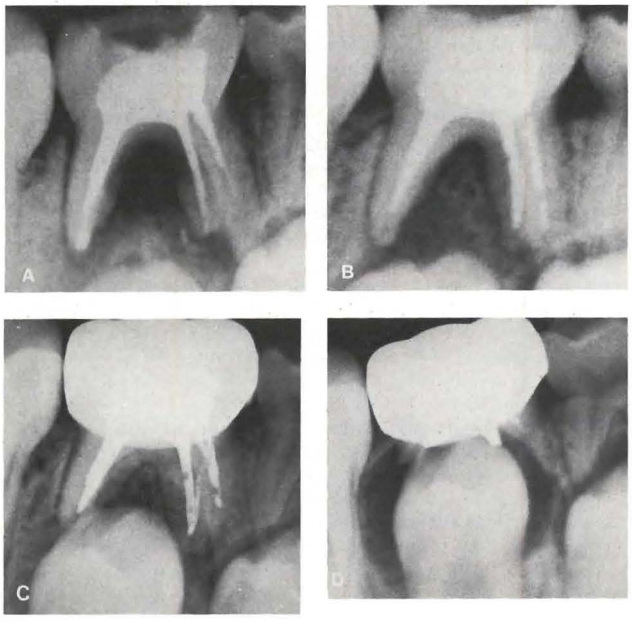

Figure 8. A, immediate postoperative, B, six-month, C, 14-month, and D, 24-month postoperative radiographs of a second primary molar treated with KRI paste. Note the adequate resorption of the paste not interfering with the premolar eruption, advanced resolution of furcation morbidity at six months, and complete resolution at 14 months.

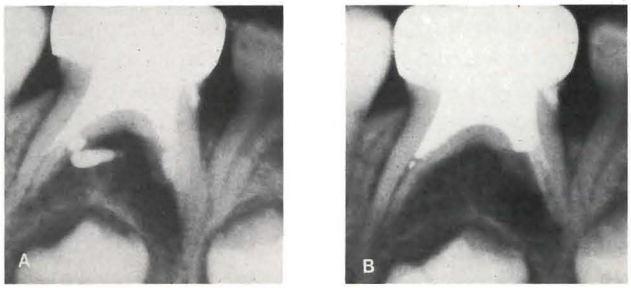

Figure 9. A, immediate postoperative, and B, six-month radiographs of a treated second primary molar. Note that furcation morbidity has been arrested and excess paste in the furcation and mesial root has been resorbed.

been shown to diffuse through the organism, however, its use in pulp therapy has been questioned.[16-20]

Erausquin and Muruzabal have shown that zinc oxide eugenol is irritating to the periapical tissues and may produce necrosis of bone and cementum.[12] Also, excess material forced through the apex during filling procedures could remain in the apical tissue during the process of physiological root resorption and could take months or even years to resorb.[11,21] KRI paste, the iodoform filling material used in the present study, resorbs from the periapical and furcation areas by the macrophages in one to two weeks. This resorption was observed in the present study and by others.[7,11] Another advantage of the KRI paste is that it remains in paste form and never sets to a hard mass; removal of the

material for retreatment, therefore, is very easy.

In a recent study, it was shown that none of the succedaneous teeth in cases treated with KRI paste had enamel disturbances or other morphological defects.[8] Color changes in the form of small white spots of approximately 1 mm in size were present in three cases. In the present study, it was not possible to evaluate this aspect of the KRI pulpectomy.

The principal advantages of the KRI paste are that it is bactericidal in the root canals, resorbs from the apical tissues in one to two weeks, is apparently harmless to permanent tooth germs, is radiopaque, does not set to a hard mass, and is easily inserted and removed.[7]

In the present study, a yellowish-brown discoloration was seen in the crowns of many of the maxillary anterior teeth treated with the KRI paste. Similar discolorations have been reported by other authors using iodoform pastes.[23] We recommend that in anterior teeth, the KRI paste be confined to the root canal and care taken to avoid placing it in the pulp chamber. The pulp chamber should be carefully cleaned and filled with any other cement.

Tagger and Sarnat have stated that root canal instrumentation in primary teeth becomes similar to "surgical debridement" in permanent teeth (removal of necrotic tissue as completely as possible) without harming adjacent healthy or vital structures.[10] To compensate for the incomplete debridement due to the complexity of the root canal system of primary teeth, it becomes necessary to destroy the microorganisms in tissue remnants and to render them unsuitable for supporting microbial life. The KRI paste could achieve this.[14,22]

Root canal instrumentation in infected primary molars or in molars with their permanent successors within the furcation area should be performed with caution as the resorptive process could have thinned the roots in the furcation area. These thinner roots could easily be perforated, if standard endodontic procedures are used to enlarge the canals. The technique used in this study should avoid possible damages to the unerupted permanent tooth, because instrumentation of the root canal is limited to a level above the occlusal plane of the unerupted permanent successor and the Hedstrom file with a pull-back direction is the only active instrument used.

The two-session technique presented in this clinical evaluation is supported in part by a recent study showing that more than one appointment and the supporting action of an antibacterial medicament between appointments would be necessary to achieve bacteria-free root canals in infected teeth in a predictable manner.[24]

Further studies should determine whether a two-step pulpectomy technique is necessary, considering the strong bactericidal properties of the iodoform paste.

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Consequences of endodontic treatment of primary teeth Part II. A clinical investigation into the influence of formocresol pulpotomy on the permanent successor

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W. E. van Amerongen, DDS, PhD
P. A. Vingerling, PhD

Part I of this report described a clinical and radiological study of the influence of formocresol pulpotomy on the life-span of primary molars.[1] The study demonstrated that the exfoliation of teeth so treated is not affected, thus supporting the conclusion that formocresol pulpotomy is a successful method of treating primary teeth with an exposed pulp. The study focused, however, on the influence on the primary teeth and provided no information on the possible influence on their permanent successors. Only a few studies, to date, made an attempt to assess the relationship between formocresol pulpotomies on primary teeth and enamel lesions of their permanent successors. The results of studies on this relationship are varied. Pruhls *et al*, for example, concluded from their study of twenty-five pairs of teeth that premolars succeeding primary teeth treated by formocresol pulpotomy stood a greater chance of affliction with an enamel lesion than the contralateral premolars.[2] Only successful pulpotomies were considered in this study.

On the basis of their study of fifty-two pairs of teeth, including both successful and unsuccessful pulpotomies, Rölling and Poulsen contradicted the conclusion stated above.[3] Their results seem to indicate that formocresol has no effect on the enamel of the succedaneous teeth. From their study of forty-seven pairs of "selected" premolars, Jeurissen and Schols concluded that the risk of enamel hypoplasia is significantly increased for the succedaneous teeth, after formocresol pulpotomy on the primary teeth.[4]

Since the results are fairly diverse, a more extensive study has been prepared, to establish whether formocresol pulpotomy on primary teeth does or does not have detrimental consequences for the permanent successors.

MATERIALS AND METHODS

The 141 patients involved in the first part of the study were all summoned for a follow-up after complete eruption of the premolars. Of the 141 patients, thirteen failed to report, and in the remaining 128 patients 139 pairs of premolars were clinically evaluated. Because very few patients had more than one pulpotomy, the few who did were not studied as a separate group. During the period of the clinical study, the ages of the patients ranged from thirteen to nineteen years. Table 1 shows the distribution of the premolars that succeeded the teeth (test teeth) on which a pulpotomy had been performed. The distribution of the corresponding teeth assessed on the contralateral side (control teeth) can be readily determined from this table.

All teeth were submitted to careful examination by

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two independent observers. During assessment, it was not known whether the premolars examined belonged to the test group or to the control group. The observers scored opacities and hypoplasias, which were defined as follows.

- ☐ An opacity is a qualitative enamel lesion visible to the naked eye as abnormal transparency of the enamel, characterized by a white, cream colored, brownish, or yellowish area. The enamel surface is smooth and enamel thickness is normal. Despite this definition it is possible, particularly on rather smooth surfaces, that opacities are assessed as initial caries lesions and vice versa. To overcome this problem, the postulate that, unlike opacities, caries lesions always follow the contours of the gingiva was accepted.
- ☐ Hypoplasia is a quantitative enamel lesion which is visible to the naked eye and morphologically characterized by involvement of the enamel surface and a reduced thickness of the enamel.

The opacities and hypoplasias were classified by size, as follows:

— none;
— less than 1.5 mm;
— more than 1.5 mm but less than one-half of the mesiodistal width of the tooth;
— more than one-half of the mesiodistal width of the tooth.

Both test teeth and control teeth were examined for the presence of opacities and hypoplasias at five different sites:

— buccal surface;
— palatine/lingual surface;
— occlusal surface;
— buccal cusp tip;
— palatine/lingual cusp tip.

STATISTICAL ANALYSIS

Since presentation and interpretation of the results are the sole objectives, statistical methods were not used to analyze the data. Only in the comparison of related results is the X^2-test applied, proceeding from the postulate that the tail probability should not exceed 5 percent.

The purpose of the study

On the basis of the plan of the study, its aim can be further defined with the following questions:

- ☐ Can a difference be observed between the incidence of enamel lesions in the test group and that in the control group?
- ☐ Does the patient's age at the time of the pulpotomy influence the development of enamel lesions in the permanent successors?
- ☐ Can a difference be observed between the incidence of opacities in the test group and that in the control group?
- ☐ Does the patient's age at the time of the pulpotomy influence the development of opacities in the permanent successors?
- ☐ Can a difference be observed between the incidence of hypoplasias in the test group and that in the control group?
- ☐ Does the patient's age at the time of the pulpotomy influence the development of hypoplasias in the permanent successors?

RESULTS

Clinical assessment

Before discussing the results of this study we should establish the reliability of the method of clinical assessment used. In order to gain an impression of this, the degree of interrater agreement was determined.

As already mentioned, the two observers independently assessed 139 pairs of teeth (always a test tooth and a control tooth). Five observations per tooth were scored (ten per pair of teeth). The degree of interrater agreement can thus range from ten identical to ten different

Table 1 ☐ Distribution of clinically examined test teeth, successors of the primary teeth submitted to formocresol pulpotomy.

	14/24	34/44	15/25	35/45	total
boys	24	19	23	26	92
girls	15	10	6	16	47
total	39	29	29	42	139

Table 2 ☐ Degree of agreement between the two observers, determined per pair of teeth assessed.

Number of identical observations	Number of pairs of teeth assessed
10	99
9	27
8	10
7	2
6	1
	Total 139

Table 3 ☐ Number of teeth with and without enamel lesions on the test side and on the control side.

	number of test teeth			number of control teeth		
	♂	♀	total	♂	♀	total
with enamel lesions	37	18	55	32	22	54
without enamel lesions	55	29	84	60	25	85
total	92	44	139	92	47	139

Table 4 ☐ Enamel lesions in test and control teeth related to the patient's age (in years) at pulpotomy.

Age at time of pulpotomy	Total number of teeth with enamel lesions	
	Test side	Control side
4	7	6
5	7	5
6	11	8
7	12	15
8	11	13
9	6	4
10	1	3
Total	55	54

Table 5 ☐ Number of teeth with one or more opacities and total number of opacities.

Opacities	Test side	Control side
Number of teeth	34	35
Total number	77	79

Table 6 ☐ Number of teeth with opacities on test side and control side, related to patient's age (in years) at pulpotomy.

Age at time of pulpotomy	Total number of teeth with opacities	
	Test side	Control side
4	6	4
5	5	5
6	6	6
7	4	9
8	8	9
9	4	1
10	1	1
Total	34	35

observations. The degree of interrater agreement is presented in Table 2, which shows that less than six identical observations per pair of assessed premolars never occurred, and that 1333 of the total of 1390 observations were identical. This indicates a degree of interrater agreement of 96 percent. In view of this finding, it was decided not to make any further distinction between the two observers. In the case of a difference between the two, the largest enamel lesion observed was accepted in collating the following results.

Total number of enamel lesions

Because it was demonstrated in Part I of the study that first and second primary molars and mandibular and maxillary premolars did not differ in life-span from the time of pulpotomy on, this distinction was not made in Part II, to ensure reader convenience. After all, this life-span determines the time during which the formocresol used might influence the formation of the permanent successor.

In order to gain a preliminary impression of possible differences in observed enamel lesions between test side and control side, a survey of all premolars with and without enamel lesions is shown in Table 3, without differentiation by specific surface or type of enamel lesion.

The Table shows no difference in total number of teeth with enamel lesions between the test side ($n = 55$) and the control side ($n = 54$) ($X_1^2 = 0.01$; $p = 0.09$). Nor was a significant difference observed in this respect between boys and girls (Girls: $X_1^2 = 0.69$; $p = 0.59$. Boys: $X_1^2 = 0.57$; $p = 0.55$). In the girls, more enamel lesions were in fact observed on the control side than on the test side. Comparing the incidence of enamel lesions in boys with that in girls, no significant difference was observed either ($X_1^2 = 0.75$; $p = 0.61$). In view of this finding, which is analogous to that in Part I of the study, no further distinction will be made in this respect either.

Total number of enamel lesions related to age at time of pulpotomy

The patient's age at the time of the pulpotomy might play a role in the development or absence of enamel lesions in the permanent successors. The mineralized crowns of the premolars are present by about the sixth year of life.[5] A pulpotomy performed on a primary molar before the sixth year of life might, therefore, be more likely to have consequences for the permanent successor than one performed at a later age. Table 4 presents the results obtained by relating the presence of enamel lesions in test teeth and control teeth to the patient's age at the time of the pulpotomy on the primary teeth.

The data in this table warrant the conclusion that the observed number of teeth with enamel lesions is unrelated to the age at the time of the pulpotomy. Comparison of test side with control side reveals no significant difference ($X^2_6 = 2.77$; $p = 0.16$). Proceeding from the postulate that enamel lesions in premolars are more likely to develop in the developmental phase before the sixth year of life, those in the group aged four and five years were summated, as were those in the group aged six to ten years. Even so, the difference proved to be too small ($X_1^2 = 0.39$; $p = 0.47$) to corroborate that postulate.

The results presented so far demonstrate that there are no demonstrable differences between test side and control side upon evaluation of all enamel lesions observed. This does not mean, however, that the results for each type of enamel lesion separately should show a similar tendency. This is why a second comparison will be presented, in the same way as in Tables 3 and 4; but separately for opacities and hypoplasias.

Opacities

Table 5 shows the number of teeth in which opacities were observed. Like Table 3, this table fails to reveal any significant difference between test side and control side (thirty-four and thirty-five teeth, respectively, with one or more opacities). In addition to the number of teeth with opacities, this table also shows the total number of opacities observed on the test side and on the control side; after all, each tooth was assessed at five different sites. In this respect too, the difference is small (seventy-seven opacities on the test side and seventy-nine on the control side).

Finally the possible influence of the age at which pulpotomy was performed on the development of opacities was studied. Table 6 relates this age to the number of teeth with opacities. The data show that the observed number of teeth with opacities is unrelated to the age at which pulpotomy was performed. Comparison between test side and control side reveals no significant difference ($X^2_6 = 4.168$; $p = 0.34$). Proceeding from the postulate that opacities are more likely to develop after a pulpotomy performed before the sixth year of life, the pulpotomies performed at ages four and five were again summated and compared with those performed in the age- group, six to ten years. Again the difference is too small ($X_1^2 = 0.36$; $p = 0.45$) to corroborate the postulate.

Hypoplasias

The results with regard to hypoplasias are no more spectacular than those with regard to opacities. Table 7 lists the numbers of teeth showing hypoplasia and the total number of hypoplasias registered after assessment at five sites per tooth. Test side and control side again do not differ significantly either in number of teeth with hypoplasia or in total number of hypoplasias found.

Comparison between the data in Tables 5 and 7 and those in Table 3 reveals that the sum of the teeth with opacities and those with hypoplasias exceeds the total number of teeth with enamel lesions. The difference amounts to two teeth on the test side and three on the control side, and is due to the fact that in these teeth both opacities and hypoplasias were observed; thus they were included in both categories.

A possible relationship between hypoplasia and age at which pulpotomy was performed was also investigated. Table 8 presents the results, which in fact do not differ from earlier findings: the observed number of teeth with hypoplasias is unrelated to age at pulpotomy, and there is not significant difference between test side and control side ($X^2_6 = 4.55$; $p = 0.39$).

Table 7 ☐ Number of teeth with one or more hypoplasias and total number of hypoplasias.

Hypoplasias	Test side	Control side
Number of teeth	23	22
Total number	28	26

Distinguishing between the four-year-old and five-year-old patients on the one hand and those aged six to ten years on the other, it is still impossible to demonstrate that age at which pulpotomy was performed may have exerted an influence on the formation of the permanent successors of the pulpotomized teeth ($X_1^2 = 0.07$; $p = 0.21$; NS).

DISCUSSION

The results do not corroborate the postulate that formocresol used in a pulpotomy of a primary tooth could have a detrimental effect on the formation of its permanent successor. The question remains, however, whether this demonstrates with certainty that formocresol is harmless to the tissues surrounding the teeth containing it. In this respect, it should be pointed out that for example the data in Table 4 may be a distorted reflection of reality, due to the relatively small number of premolars with enamel lesions observed in the original group, aged four and five years (fourteen on the test side and eleven on the control side). The number of pulpotomies performed in this patient category, however, totals 32 (almost 25 percent of the total). This figure should be considered sufficient to ensure a fairly accurate interpretation of the data.

It is striking, nevertheless, that so many enamel lesions were observed both on the test side and on the control side. Many factors have probably played a role in the pathogenesis of these lesions, illness, medication, fluoride application, and local factors such as pulp lesions, dental caries, and restorations in the primary teeth.[6-12] These factors will not be discussed in detail, if only because illness and medication are ill-defined concepts. Studies of the influence of fluoride on the formation of permanent teeth are extremely complicated. Because in the context of this study it would be impossible to give a detailed account of the influence of fluorides, we confined ourselves to an attempt to establish how many enamel lesions were observed in patients given no fluoride at all. Only six patients proved to be in this category: too small a number to warrant any conclusion.

A similar investigation was made into the possible influence of local factors. For this purpose patients were

Table 8 ☐ Number of teeth with hypoplasias on test side and control side, related to patient's age (in years) at pulpotomy.

Age at time of pulpotomy	Total number of teeth with hypoplasias	
	Test side	Control side
4	2	4
5	4	1
6	3	6
7	5	4
8	4	4
9	2	2
10	3	1
Total	23	22

Table 9 ☐ Total number of enamel lesions on test side and control side, classified by size.

	Test side	Control side
< 1.5 mm	72	68
$ 1.5 mm, < half the mesiodistal width	22	25
$ half the mesiodistal width	11	12
Total	105	105

selected whose control side primary molars remained intact, until replaced by their permanent successors (excluding an influence of local factors). The total number of teeth in this category was sixteen. Their permanent successors were compared with the contralateral (test side) teeth.

On both the control side and test side, five premolars with enamel lesions were found. Despite the limited size of this group, it seems a plausible conclusion that local factors, too, exert no influence on the formation of permanent successors. Although the cause of the observed enamel lesions remained entirely uncertain in this study, the possible influence of formocresol on the size of the enamel lesions was investigated. In this case, a so-called additional effect should be involved. Given the classification of lesions as less than 1.5 mm; more than 1.5 mm, but less than half the mesiodistal width of the tooth; or more than half the mesiodistal width of the tooth, all the observed opacities and hypoplasias together can be presented as they are shown in Table 9.

Again there is a high degree of similarity between test side and control side: no significant differences in the sizes of the lesions ($X^2_2 = 0.35$; $p = 0.16$).

SUMMARY AND CONCLUSIONS

In the context of a study of the development of enamel lesions in permanent successors of primary molars treated by formocresol pulpotomy, a comparative clinical study was performed of 278 premolars divided equally between test side and control side. These teeth were assessed by two observers who, when finding enamel lesions, always differentiated between opacities and hypoplasias.

The principal conclusions from this study are the following:

- ☐ The degree of agreement between the two observers was 96 percent.
- ☐ There was no significant difference in number of teeth with enamel lesions, between test side and control side.
- ☐ When the observed teeth with enamel lesions were related to the age at which pulpotomy was performed, the difference was still insignificant.
- ☐ Separate comparison of opacities and hypoplasias likewise showed no significant differences, even if related to age at which pulpotomy was performed.
- ☐ A formocresol pulpotomy exerts no influence on the size of enamel lesions found in permanent successors of the teeth on which pulpotomies were performed.

The general conclusion from the results of this study is that formocresol pulpotomy is a successful technique for the treatment of primary teeth, not only as regards the life-span of these primary teeth (see Part I of the study) but also in terms of its effect on their permanent successors.

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Assessment of the systemic distribution and toxicity of formaldehyde following pulpotomy treatment: part two

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The active constituents of formocresol, formaldehyde and cresol, are known toxic agents.[1] Although the local destructiveness of formocresol has long been recognized, it was not until the systemic distribution of formaldehyde from a pulp treated with formocresol was demonstrated that any thought was given to possible somatic injury.[2,3] Follow-up studies were undertaken to evaluate the systemic toxicity of formocresol.[4,5] In one of these, the systemic distribution of formaldehyde from sixteen pulpotomies in a dog was implicated in liver and kidney morbidity.[5] While the concurrent treatment of such a large number of teeth in one patient would be unusual, the oral rehabilitation of children under general anesthesia often involves numerous pulp procedures. Unfortunately, there have been no other studies dealing with the level of formaldehyde or the number of pulpotomies required to endanger the patient. For this reason, a two-part study was initiated to determine the systemic distribution and toxicity of formaldehyde from a pulpotomy site. In the first phase, the systemic distribution of formaldehyde from a pulpotomy in a rat was quantified.[6] In this, the second phase, that quantity was used as a basal dose for toxicity studies. Our objective was to administer increments of formaldehyde until systemic morbidity was demonstrated; in this way, tissue damage could be equated with the number of concurrent pulpotomies required to achieve a toxic body-load.

MATERIALS AND METHODS

Administration of formaldehyde

Male Sprague-Dawley rats weighing 150 gms were anesthetized with Nembutal prior to surgical exposure of the neck region. In order to duplicate as closely as possible the route taken by substances which pass from a pulpotomy into the systemic circulation, doses of formaldehyde were infused slowly into the jugular vein. Several dosages of formaldehyde were administered, each an increment of 0.38 μMoles, which was the amount previously determined as the body-load following pulp treatment. In the hope of avoiding untoward osmotic effects, formaldehyde was diluted in physiologic saline, and no more than 200 μl was ever infused. Following infusion, the wounds were closed with wound clips.

Collection of urine and blood

The rats from which urine was to be harvested were restrained in acrylic cages. Urine was collected by an external catheter (PE 240 tubing) with an expanded tip,

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which was inserted through the opening of the foreskin, around the end of the penis, and secured by ligation.[7] The rats were then loaded intraperitoneally with 2 ml of saline. The urine was collected in ice-cooled graduated centrifuge tubes; the tubes were completely shielded from fecal contamination. After six hours the volumes of urine were recorded, and aliquots were taken for further processing.

Following ether anesthesia, blood was drawn from the renal artery, permitted to initiate a clot, and then centrifuged to separate the serum.

Evaluation of blood factors

BLOOD UREA NITROGEN (BUN)

Urea nitrogen was determined, using a diagnostic kit (Sigma No. 65-UV). This assay is based on the hydrolysis of urea to ammonia, which is then used in a reductive reaction to yield an equivalent amount of nicotinamideadenine dinucleotide (NAD) by oxidation of NADH. The reaction is followed by measuring the decreases in Absorbance at 340 nm. The urea is calculated by the following formula: (Initial Absorbance- Final Absorbance) x 135 = mg urea nitrogen/dL.

SERUM GLUTAMIC-OXALACETIC TRANSAMINASE (SGOT)

Serum glutamic-oxalacetic transaminase was determined using a diagnostic kit (Sigma No. 505). The assay of this enzyme is based on the transfer of amino groups from specific amino acids to α-ketoglutaric acid. The products of this reaction are glutamic acid and oxalacetic acid. These acids are determined colorimetrically after their reaction with 2, 4-dinitrophenylhydrazine at 490 nm. SGOT activity is expressed in Sigma-Frankel (SF) units/ml, derived from a calibration curve.

Evaluation of urinary factors

PROTEINS

For the assay of urinary proteins, the tubes containing the collected urine were subjected to vortical motion and duplicate 50 μl samples were taken for analysis by the method of Bradford. After color development, the samples were read on a spectrophotometer at 595 nm. The total amount of protein excreted was then calculated from the measured volume of urine and is reported as μg/8 hr volume.

ALKALINE PHOSPHATASE

The determination of alkaline phosphatase in the urine was based on standard clinical practice.[8] After measuring the urine volume, an aliquot was adjusted to pH 7.4. The urine was then centrifuged until clear, transferred into dialysis tubing, and weighed. The urine was dialyzed overnight at 4°C against 0.001 Tris Buffer, pH 7.4, to remove inhibitors. After dialysis, the bag was blotted dry, reweighed, and the urine centrifuged. The alkaline phosphatase in the urine was determined by measurement of hydrolyzed phosphate, following incubation of an aliquot with the substrate β-glycerophosphate. The formula for calculating enzyme activity was adjusted for fluid gain or loss, during dialysis; the values for an 8 hr collection were established as the norm. The unit of enzyme activity is defined, therefore, as the amount of enzyme in an 8 hr volume of urine that liberates 1 mg of phosphorous under the conditions of the assay.

LACTATE DEHYDROGENASE (LDH)

The determination of lactate dehydrogenase in urine followed a standard clinical protocol.[8] Urine collected for six hours was placed in dialysis tubing and weighed. The urine was then dialyzed overnight in distilled water at 4°C to remove inhibitors. The bag was next blotted dry, reweighed and assayed for lactate dehydrogenase. The assay used is based on the catalysis of lactic acid to pyruvate and the reduction of NAD to NADH, which is read spectrophotometrically at 340 nm. The formula used to calculate the total activity in the urine adjusted for volume effects of dialysis and normalized the length of collection. The units of enzyme activity (defined as nmol NADH formed/ml/min) are reported, therefore, for 8 hr volumes.

Measurement of respiration by liver slices

The procedure for incubation of liver slices was modified from the protocol of Ramos *et al.*[9] Following 6 hr urine collection, the rats were anesthetized with ether and the ventral lobes of the liver were excised. The lobes were quickly sliced with a Stadie-Riggs tissue slicer. The sections of tissue were then placed in weighed flasks, which contained 3 ml of phosphate buffer and 20 μMoles of succinate. After reweighing, the flasks were sealed with rubber sleeve stoppers with attached plastic wells. One μCi of ^{14}C-succinic acid was added to the incubation media, and the flasks were shaken in a 37°C water bath for 45 min. The ^{14}C-CO_2 liberated by tissue metabolism was captured by methylbenzenethonium hydroxide (Sigma No. M-1756) in the plastic wells. Liver reactions were stopped by the addition of 2 M H_2SO_4, and the wells were removed and added intact to scintillation vials containing a multipurpose scintillation cocktail

(Beckman Ready-Solv MP). After the decay of chemiluminescence, all vials were counted for five minutes, and quenching effects were corrected by internal standards. Radioactivity is reported as dpm/mg of tissue.

Metabolic studies with ^{14}C-formaldehyde

To test the effect of formaldehyde on the later conversion of ^{14}C-formaldehyde to ^{14}C-CO_2, rats were anesthetized with Nembutal and infused with formaldehyde via the jugular vein as described above. After twenty-four hours the rats were injected with 0.5 μCi of ^{14}C-formaldehyde intraperitoneally. The rats were then maintained in a glass metabolic cage, which had outlets for the capture of expired gases. Air was drawn from the cage by a vacuum and was bubbled through two vessels containing 3 N NaOH. After six hours, aliquots of the NaOH were treated with H_2SO_4 in closed vessels, to release the CO_2 from the bicarbonate product. The ^{14}C-CO_2 was then captured in vials of methylbenzethonium hydroxide which had been enclosed in the flasks. Aliquots of the hydroxide were then counted by liquid scintillation spectroscopy as described above. The amount of metabolized ^{14}C-formaldehyde is reported as a percent of the total injected.

Histologic studies

Samples of liver and organ were harvested from rats six and twenty-four hours postinfusion, fixed in 10 percent buffered formalin, and prepared for routine paraffin sectioning. Five μM sections were cut and stained with hematoxylin and eosin.

RESULTS

The results of the biochemical and metabolic studies are listed in the Table. It is apparent that some of the assays were more sensitive indicators of acute toxicity than others. For instance, neither of the blood factors (SGOT or BUN) disclosed a significant difference between the formaldehyde-treated groups and the controls. The dose of formaldehyde evaluated by these factors was 250x that obtained from a single pulpotomy, and the highest used in this study.

In contrast, lactate dehydrogenase and protein values in the urine were significantly elevated by a dose 125x that obtained from a single pulpotomy. The alkaline phosphatase in the urine was not affected at this level. The higher dose (188x) had even a more profound impact on the lactate dehydrogenase, almost doubling the amount present in the urine.

Table ☐ Results of acute toxicity studies (Mean ± S.E.)

Test	Saline	Formaldehyde	Dose	Significance[1]
SGOT* Units/ml	50.3±2.7 n=8	54.7±3.7 n=8	250x	N.S.
BUN† mg/dL	11.3±0.8 n=8	11.3±0.6 n=8	250x	N.S.
LDH ‡ (urine) Units/8 hr vol	38.5±5.0 n=8	45.3±6.6 n=8	125x	$p < .05$
		65.6±9.5 n=8	188x	$p < .001$
Alk Phos** (urine) Units/8 hr vol	.79± .2 n=8	.64± .14 n=8	125x	N.S.
Urine protein ug/6 hr	615±35 n=8	759±75 n=8	125x	$p < .001$
Liver slices Succinate->CO_2 dpm/mg	55.4±9.4 n=8	37.6±2.5 n=8	125x	$p < .001$
		21.5±2.9 n=8	188x	$p < .001$
^{14}C-HCHO Metabolized % of Dose	37.5±2.8 n=4	42.0±3.1 n=4	10x	N.S.
		37.5±3.1 n=4	100x	N.S.

[1]Significance determined by student's T Test.
*Serum glutamic-oxalacetic transaminase
†Blood urea nitrogen
‡Lactate dehydrogenase
**Alkaline phosphatase
The method for reporting values is listed beneath each individual test, and "n" represents sample size.

Liver respiration, as determined by an *in vitro* slice assay, was substantially reduced by the 125x dose, and more than halved by the 188x dose.

A postinfusion effect of formaldehyde on the metabolism of ^{14}C-formaldehyde was not noted. Similarly, histopathologic findings from the observation of kidney and liver sections were consistently negative, at both six and twenty-four hours postinfusion.

DISCUSSION

This study was undertaken as part of an overall program to evaluate the toxicity of formocresol and possible alternatives. An earlier report dealt with the determination of the systemic load of formaldehyde resulting from a pulpotomy treatment.[6] That basal dose was used as a reference point in the present study, and several increments of the dose were evaluated. This and our former study were limited to an analysis of formaldehyde; LD_{50} data suggests that cresol is equally toxic, and we did not wish to confuse the results by using formocresol.

The findings of this study are provocative, for several reasons. Although small amounts of formaldehyde were shown to be toxic by several biochemical responses, higher levels than those obtained from pulp treatment were necessary. The fact that relatively large doses were required, therefore, to achieve overtly toxic levels is not altogether reassuring, because the visible distress exhibited by many of the infused rats was not in concert with the biochemical data. We observed many physical signs of acute toxicity such as respiratory distress, lacrimation, and nasal congestion; outright respiratory failure and death were not uncommon at higher doses. Yet only a few of the biochemical analyses substantiated that the rats were recipients of an extremely toxic agent.

We are not impressed with the sensitivity of many of the classical methods of toxicity for the assessment of acute toxicity; apparently some histologic and biochemical changes will not develop without chronic insult. For instance, we were never able to observe histopathological findings in either the kidney or liver, even though some of our assays demonstrated clear physiological changes. The negative histological findings were not in keeping with those of Myers *et al*, who reported kidney and liver changes in a dog following sixteen formocresol pulpotomies.[5] The discrepancy might be explained, however, by their use of formocresol rather than formaldehyde alone.

General cytotoxity was determined by the assay for serum glutamatic-oxalacetic transaminase released in the blood by damaged cells. While often associated with the liver, this enzyme is not limited to this organ, but is present in some extrahepatic organs such as heart, skeletal muscle, and kidney. For this reason, it is theoretically a good measure of generalized cellular injury.[8] In this study, at very toxic levels of formaldehyde, the serum levels of this enzyme were not significantly elevated.

The use of liver slices *in vitro* permitted an evaluation of residual hepatic function, isolated from confounding influences of other tissues. This assay proved relatively sensitive to the infusion of formaldehyde, six hours earlier. Because formaldehyde is a normal metabolite, a principal in the so called "One- Carbon" pool, the reduction in succinic acid metabolism cannot be completely ascribed to nonspecific formaldehyde toxicity. The depression may reflect totally, or in part, preoccupation of liver tissue with ridding the system of exogenous formaldehyde. Certainly in the whole-body metabolic study, the conversion of formaldehyde to CO_2 was not inhibited by prior infusion with a 100x dose of the same agent. These results suggest that the depression of succinate metabolism is an indirect consequence of the saturation of common intracellular systems by formaldehyde, rather than direct enzyme poisoning. This would certainly be the preferred explanation, if similar responses to formaldehyde were observed in humans.

Potential nephrotoxicity was evaluated by assays for glomerular and tubular damage. Examination of the former is common in clinical medicine. Proteinuria is a consequence of altered filtration in the glomeruli. In this study we determined the total protein excreted, during six hours. Following a 125x dose of formaldehyde, urine protein was significantly elevated. This effect on glomerular filtration was not paralleled by a change in the blood urinary nitrogen, suggesting that a change in the latter requires more time or more damage to develop.

According to Wright and Plummer, the measurement of several enzymes in the urine are valuable for the detection of acute kidney damage by toxic compounds.[10] Supposedly the enzymes are "markers" for specific regions of the cell, and by measuring their activity in the urine, the site of the primary lesion might be determined. Alkaline phosphatase is considered a marker for the endoplasmic reticulum and plasma membrane, and lactate dehydrogenase is found in the cytoplasm or soluble fraction of the cell. Of the two enzymes, lactate dehydrogenase was a better indicator of nephron damage. Formaldehyde at the 125x dose significantly elevated urinary lactate dehydrogenase, and the effect was amplified by the 188x dose. While Wright and

Plummer reported that alkaline phosphatase was not spilled into the urine to the same extent as lactate dehydrogenase, in response to a variety of nephrotoxic agents, our inability to observe even a minimal increase in alkaline phosphatase suggests that formaldehyde toxicity is limited to the cytoplasm.[10]

The results of this study are interesting from several perspectives. First, it revealed that the histological, physiological, and/or biochemical sequelae of overt, acute toxicity are not uniformly demonstrable by methods used for chronic conditions.

Secondly, the study did demonstrate that small doses of formaldehyde are toxic when administered intravenously, even though comparable levels may not be attained clinically. This supposition is based on a very tenuous extrapolation from rats to humans.

Thirdly, our results raise questions about the conclusions drawn from earlier studies on the toxicity of formocresol.[4,5] In these studies, formocresol was used as the test agent, while most of the morbidity was ascribed to formaldehyde. Compared to the systemic levels attained in doses following multiple pulpotomies (calculated from their data) the doses of formaldehyde required to demonstrate biochemical changes were higher in our study.[2,5] The disparate levels of formaldehyde, however, may not be all that inconsistent. Whereas we limited our study to formaldehyde, formocresol was used in previous studies. Thus, the effects of two drugs simultaneously administered were actually evaluated. Because formaldehyde was the first component of formocresol that was shown to escape from a pulpotomized tooth, it was natural that it became the cynosure for toxicity studies. The preoccupation with formaldehyde, however, may have blinded us to the other toxic constituent. Although we do not know the extent of cresol distribution following a pulpotomy, low levels may prove to be toxic. Further studies could conceivably condemn cresol for much of the systemic toxicity manifested by formocresol.

And finally, our results demonstrated the worth of some assays for acute toxicity studies. We will select the more sensitive ones for future evaluation of cresol and glutaraldehyde, and formaldehyde at lesser doses.

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THE INITIAL LESION

Experimentally induced gingivitis in man has been studied by *Zachrisson* (1968) and by *Payne* et al (1975). The earliest change that can be detected microscopically and clinically is an acute inflammation manifested by vasculitis in the plexus of venules lateral to the junctional epithelium, a greatly increased migration of neutrophilic granulocytes into and through the junctional epithelium and into the gingival sulcus, exudation of fluid from the sulcus, flooding of the connective tissues with serum proteins and fibrin, and loss of the perivascular collagen at the site of inflammation.

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Dimensional stability of alginate impressions immersed in disinfecting solutions

David P. Durr, DMD, MS
Eleanor V. Novak, BDS, LDS

Infectious viral diseases, such as Hepatitis B, Acquired Immune Deficiency Syndrome (AIDS), and Herpes Simplex, may be transmitted via blood and saliva, both of which may be found on the surface of an alginate impression. These impressions present a potential reservoir of virus particles, which may be transferred to the stone models poured from them. Thus, a risk of contamination exists for all those who handle such impressions in the dental operatory and dental laboratory, during the making of stone casts, model trimming, or appliance and prosthetic fabrication. It may be appropriate, therefore, to disinfect alginate impressions immediately after removal from the mouth.[1]

Although procedures that conclusively test the efficacy of disinfecting solutions against the Hepatitis B and AIDS virus have not been developed, it does appear that a ten-minute immersion in a high level germicide, such as a sodium hypochlorite or a potentiated glutaraldehyde solution, will render the surface of a material, virus-free.[2,3] For these agents to be acceptable for disinfection of alginate impressions, they must not affect the dimensional stability of the alginate impressions. Studies by Trevelyan, and Storer and McCabe evaluated the dimensional changes of alginate, following such disinfection procedures; but the objects investigated were of relatively small dimensions, which could not be related to the dimensions of full arch impressions.[4,5]

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The purpose of this investigation was to determine the dimensional stability of full-arch alginate impressions that have been immersed in either 1 percent sodium hypochlorite or 2 percent potentiated glutaraldehyde solutions. The stability was assessed by measuring stone casts which were poured from the alginate impressions. In addition, the surface quality of the stone casts was assessed.

MATERIALS AND METHODS

A plaster-based master cast (Figure 1) was constructed with three embedded measurement posts. The elevated arch form was such that it fit a standard adult disposable[a] impression tray and such that the alginate impression

[a]PCA Dis-A-Tray #1, Silverman's, Plymouth Meeting, PA

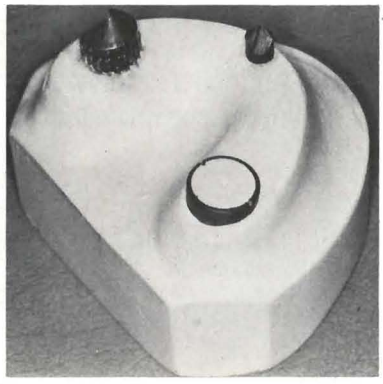

Figure 1. Master cast with three embedded measuring posts.

taken from it would closely resemble in mass distribution, the varying thicknesses of a clinical impression. The measurement posts were made from 1/4"[b] and 1/2"[c] drill bits and a plaster-filled copper ring. The measurement locations (Figure 2) were:

A. Tip of small drill bit
B. Tip of large drill bit
C. Central pit of plaster-filled copper ring
D & E. Notches in copper ring

The surface of the plaster base of the model was smooth, but the surfaces of the drill accessories were detailed, with raised and channeled pattern that could be used for evaluation of any loss of surface detail in the poured stone casts.

Impressions of the master cast were made, using 21 gm of alginate[d] mixed with 60 ml of tap water at 22°C and spatulated for 45 seconds. The alginate was loaded into the impression tray, which was then positioned on the master model. The impression was removed one minute after the point of initial set, by breaking the seal and snapping it from the model.

Control samples were poured directly using a hard dental stone[e] with an extremely low setting expansion of 0.05 percent so as to reduce as far as possible any error caused by expansion of the stone itself. Two hundred gm of this stone were mixed with 46 ml of tap water at 22° and mixed for 60 seconds at 120 rpm on a spatulator[f]. The stone was poured into the alginate impression using a standardized technique with some vibration so as to eliminate as many bubble imperfections as possible. The stone models (Figure 3) were separated from the alginate impressions one hour after pouring.

Experimental samples were poured following sterilization with either 1 percent sodium hypochlorite[g] or 2 percent potentiated glutaraldehyde solution[h]. The potentiated glutaraldehyde solution used in this experiment contained nonionic ethoxylates of isomeric linear alcohols, which are surfactants and act as synergistic agents, to allow better penetration of glutaraldehyde. Plastic containers were filled with freshly prepared solutions and kept covered. Twenty alginate impressions of the master model were completely immersed in the 1 percent sodium hypochlorite solution and a further twenty impressions in the 2 percent potentiated glutaraldehyde solution for ten minutes at room temperature. The impressions were inverted on removal from the solutions and gently shaken to remove excess liquid. The impressions were then rinsed in tap water and stone casts were made immediately, according to the techniques described previously.

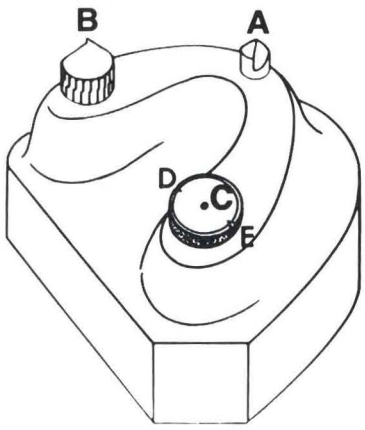

Figure 2. Measurement locations on master cast.

Four linear measurements, coded AB, AC, BC, DE, were made on the master model and on each of the stone casts in the control and experimental groups. The measurements were made using fine point calipers and a Boley gauge, which is a vernier indicator calibrated in tenths of a millimeter. The measurements taken from the control and experimental groups were compared with each other and with those of the master cast, to determine the dimensional changes of the alginate impressions.

The surface quality of the casts was evaluated by direct comparison of the control and experimental casts with the master model. The following characteristics were evaluated: smoothness of the base, reproduction of the points of the measurement posts with attention to blunting, and reproduction of the detail of the measurement posts with their raised and channeled areas. The evaluations were categorized as *excellent quality of reproduction, intermediate quality, or poor quality.*

All linear measurements and surface quality evaluations were performed by the same examiner (E.V.N.). Each cast was evaluated blindly with respect to treatment or control group assignment. Intraexaminer reliability was determined by repeating measurements and surface quality evaluations on fifteen casts. The coefficients of variation for the replicate measures were very low, ranging from 0.1 percent for measurements AB, AC, and BC to 0.3 percent for measurement DE. This indicated that the measurement technique was highly reliable. The surface quality evaluations also were highly reliable as there were no discrepancies between the initial and repeat evaluations.

RESULTS

Table 1 presents the means and standard deviations for the four linear measurements from the master cast and the casts of the control and experimental groups. There

[b]CS-1, Vermont American Tool Company, Loncolnton, NC
[c]U-1584, Black and Decker Manufacturing Company, Townsend, MC
[d]Type II Normal Set (0482581), Healthco, Boston, MA
[e]Type IV Indic-Die Stone, Columbus Dental, St. Louis, MO
[f]Vacuum Spatulator W-107, B.F. Wehmer, Franklin Park, IL
[g]Clorox Bleach (Diluted), Clorox Company, Oakland, CA
[h]Sterall, Hoyt Laboratories, Norwood, MA

Table 1 ☐ Mean linear measurements (mm) of master cast, control group, and experimental groups.

Measurement	Master	Control	Hypochlorite	Glutaraldehyde
AB	31.1	31.1(0.1)	31.1(0.1)	31.1(0.1)
AC	37.8	37.8(0.1)	37.8(0.1)	37.8(0.1)
BC	44.0	44.0(0.1)	43.9(0.1)	43.9(0.1)
DE	14.8	14.8(0.1)	14.7(0.2)	14.8(0.2)

Numbers in parentheses are standard deviations.
n = 20 for each mean

Table 2 ☐ Comparison of linear measurements of master cast with control and experimental groups.

Treatment	Measurement	Mean difference (mm)	p
Control	AB	-0.02(0.09)	0.45
	AC	0.01(0.09)	0.61
	BC	-0.04(0.06)	0.01*
	DE	0.05(0.09)	0.02*
Hypochlorite	AB	-0.04(0.08)	0.05
	AC	0.00(0.14)	1.00
	BC	-0.08(0.07)	0.00*
	DE	-0.08(0.15)	0.03*
Glutaraldehyde	AB	-0.04(0.05)	0.00*
	AC	0.04(0.05)	0.00*
	BC	-0.01(0.08)	0.00*
	DE	0.00(0.15)	0.88

Numbers in parentheses are standard deviations.
*Statistically significant difference from master cast (t-test).

Table 3 ☐ Comparison of linear measurements of control group with experimental groups.

Measurement	F*	p	Interpretation**
AB	0.67	NS	No difference
AC	1.12	NS	No difference
BC	3.80	0.03	Control greater
DE	4.80	0.005	Hypochlorite less

*Analysis of variance, degrees of freedom = 2,57.
**Based on Tukey multiple range comparisons.

Table 4 ☐ Correlations between linear measurements.

Measurements	Control	Hypochlorite	Glutaraldehyde
AB vs. AC	0.02	0.05	-0.08
AB vs. BC	0.24	0.24	-0.26
AB vs. DE	0.01	0.16	0.11
AC vs. BC	-0.20	0.37	0.26
AC vs. DE	-0.07	0.52	0.37
BC vs. DE	0.13	0.41	0.04

Table 5 ☐ Comparison of surface quality of control and experimental groups.

	Control	Hypochlorite	Glutaraldehyde	Total
Excellent	1	5	12	18
Intermediate	19	15	8	42
Poor	0	0	0	0
Total	20	20	20	60

$\chi^2(2) = 14.76$ $p = 0.005$

is remarkably little difference among the means across treatments, and in no case does the difference exceed 0.1 mm. Also the standard deviations of the data are very low, indicating that, for each linear measurement within a particular treatment, all measurements are nearly identical.

Although the means and standard deviations among the treatment groups are very similar, there are subtle differences among the groups which are statistically significant. Table 2 lists the mean differences between the measurements of the master cast and those of the control and experimental groups and presents the results of a t-test, comparing the measurements of the master cast with those of the other groups. Note that the statistically significant differences from the master cast occur in all groups, including the control group.

Table 2 also shows patterns of differences among the groups: AB tends to decrease slightly for all treatments; AC remains the same in the hypochlorite group, but increases slightly for the control and glutaraldehyde groups; BC tends to decrease for all treatments; and DE shows no pattern.

Table 3 presents the results of an analysis of variance for comparing differences among the control and experimental groups for each linear measurement. Only measurements BC and DE exhibit statistically significant differences among the groups. Based on a Tukey multiple range comparison procedure, the control group has greater BC measurements than the two experimental groups, while the hypochlorite group has lower DE measurements than the control or glutaraldehyde groups.

Correlations between measurements on the same cast are listed in Table 4. These indicate no overall pattern of shrinkage or expansion. The largest correlations are artefacts caused by only one or two outlying measurements.

Table 5 presents the results of a Chi-square analysis comparing the surface quality of the control and experi-

Figure 3. Die-stone replicate poured from alginate impression in the control group.

mental casts. In all cases, the casts were judged to be of *excellent* or *intermediate quality*, with glutaraldehyde having more in the *excellent* category than the control and hypochlorite groups combined. These differences are statistically significant.

DISCUSSION AND SUMMARY

The results of this experiment showed that immersion of alginate impressions in either 1 percent sodium hypochlorite or 2 percent potentiated glutaraldehyde solution for ten minutes at room temperature produced statistically significant dimensional changes in the casts poured from these impressions as compared to a master cast and the casts of the control group. Some of the dimensions of the casts in the control group also differed significantly from those of the master cast. In neither of the experimental groups nor in the control group did any of the dimensions differ, however, from the corresponding measurement on the master cast by more than 0.1 mm. Such a discrepancy would be insignificant for many clinical applications, such as the making of study models or working casts for the fabrication of appliances.

The authors gratefully acknowledge the assistance of Mark Espeland, PhD, Biostatistician, Eastman Dental Center for his assistance in the preparation of this manuscript.

The surface quality of the experimental casts was not adversely affected by the immersion of the impressions in the sterilizing solutions. In fact, those immersed in the 2 percent potentiated glutaraldehyde solution seemed to have an improved quality of the smooth areas, definition points, and detailed channeled patterns as compared to the control and hypochlorite groups.

Considering the minimal effect that a ten-minute disinfection with 1 percent sodium hypochlorite or 2 percent potentiated glutaraldehyde appears to have on alginate impressions, it may be advisable to consider such treatment of impressions as routine. Further research into the effectiveness of these and other agents and the time of exposure necessary to assure elimination of any and all bacterial or viral contaminants is warranted.

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DENTAL INJURIES

The results of the present study show that the children from private schools suffered more traumatic dental injuries than the children from public schools ($P<0.05$). The boys from private schools suffered more traumatic dental injuries than the boys from public schools ($P<0.02$). Prevalence of injuries in girls from private schools had no significant difference with that of girls in public schools.

The higher prevalence of traumatic dental injuries in children from private schools is mainly due to one type of injury: the enamel fracture. The higher prevalence of enamel fractures was only observed in boys from private schools as opposed to public schools ($P<0.001$) because girls from private schools showed no siginificant difference from those in public schools, in any type of injury. We cannot explain why boys from private schools suffered more enamel fractures than those from public schools. However, because the Dominican Republic is a third world country, it should be pointed out that children from public schools are largely from a low socioeconomic background, while children from private schools are from middle to high socioeconomic levels.

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Pediatric dentistry in early and mid-1980s: a review of personnel and use of dental services

Demographics

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The Department of Health and Human Services (HHS) recently released the "Fifth Report to the President and Congress on the Status of Health Personnel in the United States" and the report on health expenditures for 1985.[1,2] The two documents describe the changing and generally improving environment for the practice of dentistry in the 1980's and beyond.

Using both an "optimistic" and a "pessimistic" series of forecast indices, HHS predicts a significant increase of from 37.7 percent to 57.4 percent in constant-dollar expenditures between the years 1984 and 2000 (i.e. elimination of the effects of inflation) per active dentist. Should the projections prove accurate, dentists currently earning $65,000 (average income for private practicing dentist in 1984) will have an inflation-adjusted income of between $89,500 and $102,300 by the end of the century.[3]

Reports on 1985 annual dental expenditures confirm the initial stage of this optimism. Between 1984 and 1985, expenditures per active dentist increased from $182,000 to $192,500 (constant-dollar expenditures increased from $58,500 to almost $60,000). Constant-dollar expenditures per active dentist in 1985 were 16.4 percent greater than comparable expenditures in 1978, the last year prior to the recession of the late 1970's and early 1980's.[4,5]

And most important, as a result of the major decreases

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Table 1 ☐ United States population under eighteen years of age and percent of total population: selected years 1970-2000.[11,12]

Year	Population under eighteen (in thousands)	Percent of total population
1970	69,700	34.2
1975	66,300	31.1
1980	64,000	28.2
1983	62,500	26.6
1984	62,700	26.4
1985*	62,838	26.3
1990*	64,337	25.8
1995*	67,134	25.9
2000*	67,389	25.1

*Projections are based upon the middle series of estimates.

Table 2 ☐ The percent of children under seventeen years of age by the time since last dental appointment: selected years 1970 through 1983.[13-15]

Last visit	1970	1975	1980	1981	1983
Less than 1 year	47.0	51.4	50.1	50.0	50.6
1 year	9.5	9.6	10.8	10.8	11.4
Subtotal	56.5	61.0	60.9	60.8	62.0
2 year or more years	8.0	8.1	8.2	8.0	7.6
Never	34.5	30.0	30.1	30.4	30.4
Unknown	1.1	0.9	0.8	0.8	*

*In the report, unknown data were prorated in the specific interval categories.

Table 3 ☐ Number of dental visits per male and female child under age seventeen: selected years 1970 through 1983.[13-15]

Year	Male	Female	Total
1970	1.3	1.5	1.4
1975	1.5	1.7	1.6
1980	1.5	1.8	1.7
1981	1.5	1.7	1.6
1983	na	na	1.9

Table 4 ☐ Number of dental visits for male and female children under age seventeen: selected years 1980 through 1983.[13-15]

	(in thousands)		
Year	Male	Female	Total
1970	45,163	49,708	94,871
1975	47,245	50,252	97,497
1980	44,690	52,568	97,258
1981	46,593	49,608	96,201
1983	na	na	111,585*

*Based upon 1.9 visits per individual and an estimate of the population. Changes in the categories of reported data preclude a direct comparison of data in federal government reports on dental visits.

Table 5 ☐ Number of dental visits per person between two and seventeen years of age by gender, race and family income: 1983.[16]

	2-4 yrs	5-11 yrs	12-17 yrs
Total	0.7	2.1	2.9
Gender			
Male	0.7	1.9	2.5
Female	0.6	2.2	3.3
Race			
White	0.7	2.2	3.2
Black	0.5*	1.4	0.9
All other	0.5*	1.9	2.7
Family Income			
Less than $10,000	0.6	1.4	1.3
$10,000 - $19,999	0.5	1.8	2.0
$20,000- $34,999	1.0	2.5	3.4
$35,000 or more	0.7	2.8	4.4

*Relative standard error of more than 30 percent.

Table 6 ☐ Percent distribution of individuals by race between five and seventeen years by number of dental visits in year: 1983[16]

Number of visits	White	Black
None	29.7	48.3
1	27.3	25.0
2	22.1	13.3
3	6.2	6.2
4	3.7	2.1
5 or more	10.2	3.7

in dental school enrollments, HHS predicts that, for the rest of the century, the number of dentists will increase at the same rate as the population.[1] (These data do not reflect the marked increase in the number of female practitioners. It should be noted that female dentists report less work activity than their male counterparts.[6])

DEVELOPMENTS IN PEDIATRIC DENTISTRY

But are these favorable events reflected in the developments in pediatric dentistry during the early and mid 1980's? Unfortunately, specific economic data are not available from the American Dental Association; since 1981, the Association no longer reports individual specialty income in the Survey of Dental Practice. In addition, reports from other sources, such as proprietary dental publication surveys are suspect.[7] Available personnel data and use of dental services by youngsters do provide, however, information which reflects on some favorable developments in pediatric dentistry. Earlier reports by this author reviewed:

- ☐ The oversupply of pediatric dentists.
- ☐ The need for more improved distribution of pediatric practitioners.
- ☐ The developments in the specialty in the early years of the recovery from the last recession.[8-10]

Table 7 ☐ Number of pediatric dentists: selected years 1970 through 1984.[1,17,18]

Year	Number	
	ADA	Report to President
1970		1,076
1976	1,218	
1979	1,776	
1980		2,063
1982	2,949	
1984		2,398

Table 8 ☐ Pediatric dentists per 100,000 population: selected years 1960 through 1984.[1,19,20]

Year	Pediatric dentists per 100,000 population
1960	0.1
1965	0.3
1970	0.5
1975	0.7
1980	0.9
1984	1.0

Table 9 ☐ Number and percent of senior dental students applying to pediatric dentistry training programs: 1978-79 through 1985-86.[21]

Year	Number	Percent
1978-79	122	2.3
1979-80	130	2.4
1980-81	126	2.4
1981-82	116	2.1
1982-83	91	2.1
1983-84	98	1.7
1984-85	96	1.8
1985-86	102	1.9

The present material will consider developments into the mid 1980's.

Population

Between 1970 and the early 1980's, there was a progressive decrease in the number and percent of the population under eighteen years of age. While the population under eighteen years of age continues to represent a progressively smaller percent of the total population, since 1983 there has been, however, a gradual increase in the absolute number of children in our country. In addition, it is projected by the Department of Commerce that while the population cohort below age eighteen years will represent only one quarter of the total population by the year 2000, the actual number of children below age eighteen will continue to increase and approximate the number of children during the early 1970's (Table 1).

Time since last dental visit

Through 1975, there had been a progressive increase in the percent of children under seventeen years who had been to the dentist in the past year. Since 1980, however, there has been essentially no change in the percent of the population reporting a visit within the past year (approximately 50 percent). Similarly, there has been minimal increase in the percent of this population group with a visit in the past two years (62.0 percent); and no change in the percent that reported "never" having made a visit to the dentist (30.4 percent) (Table 2).

Number of dental visits

Although in recent years there have been minimal changes in the percent of population below seventeen years of age reporting a visit to the dentist and in the number of visits per person, between 1981 and 1983, there was a 19 percent increase in the number of reported visits per individual (Table 3) and overall increase of 16 percent in the total number of dental visits by children (Table 4).

This increase in the number of visits to the dentist per child did not occur, however, for all population groups. There were marked differences in the number of visits per individual on the basis of gender, race, and family income. Females, whites and children from higher income families reported greater numbers of visits to dentists than their respective counterparts. In particular, black children reported less visits than either white or "all other" groups (Table 5). In addition, almost a half (48.3 percent) of black children between five and seventeen years reported no dental visits in the past year; as compared to 30 percent of white children (Table 6).

Number of pediatric dentists

Between 1970 and 1984, the number of pediatric dentists increased. Although the data from the reports by the American Dental Association and the various reports to the President and the Congress on the status of health personnel differ in absolute number, each series presents a marked increase during the period (ADA: 142 percent; Report to the President: 123 percent) (Table 7).

By 1984, there was one pediatric dentist per 100,000 population, a 100 percent increase since 1970 (Table 8).

Training programs in pediatric dentistry

In the 1985-86 academic year, there was a minor increase over the previous year, in the number of senior dental students applying to pediatric dentistry training programs. The number of applicants was less, however, than the number of seniors who applied to these programs in the late 1970's and early 1980's (Table 9).

In addition, since the 1980-81 academic year, fewer students have been enrolled in the first year of pediatric

Table 10 ☐ Number of students enrolled in first year of pediatric programs: selected years 1972-73 through 1985-86.[19,22]

Year	First year enrollment
1972-73	163
1974-75	177
1976-77	165
1978-79	173
1980-81	190
1981-82	178
1982-83	158
1983-84	149
1984-85	164
1985-86	157

dentistry training programs. This lower level of enrollment was continued in the 1985-86 academic year (Table 10).

OVERVIEW

There have been changes in the general delivery environment for pediatric dental services, since the last recession. While these developments are far from conclusive, they tend to indicate general improvements. For example:

- ☐ Minimal changes in the percent of the population reporting a dental visit in the past year are overshadowed by the increasing use pattern of services, at least for some population groups.
- ☐ There has been a general leveling down in the "production" of dental practitioners and specifically, trained pediatric dentists.

Because children are treated by both the general population of practitioners and by pediatric dentists, any discussion of the evolving use patterns also should consider changes in the overall number of practitioners. Again, there were favorable changes (at least from the practitioner perspective) regarding the changing number of dental visits by children and increasing number of active practitioners. For example, between 1981 and 1983, while there was a 16 percent increase in the total number of dental visits by children under seventeen years (Table 4), there was a 4.6 percent increase in the overall number of active practitioners (from 129,180 to 135,120).[1]

Nevertheless, as the profession in general, and pediatric practitioners in particular, attempt to increase the use of dental services by younger population groups, it is necessary to recognize the reality that there may be limited improvement from efforts that are directed solely to improving dental knowledge and understanding of the need for services. For example, in response to a 1985 national study on health promotion and disease prevention, more than 95 percent of the respondents in all age cohorts over seventeen years reported, it was important to see a dentist on a regular basis; 98 percent recognized the importance of brushing and flossing of teeth; and 96 percent recognized the primary causes of the loss of teeth in children.[23]

This is not meant to imply that programs to maintain the level of dental knowledge and understanding should be downgraded. Rather, in terms of the marginal value of efforts to encourage increased use of services, more return would be obtained from efforts to reach the underserved minority and economically deprived population groups. In particular, improved Medicaid dental programs and general third party dental programs must be encouraged.

The outlook for dentistry, and in particular pediatric dentistry, appears to be more favorable than it has seemed in recent years. Continual review must be maintained, however, to assure that a realistic relationship is maintained between the number of children and pediatric dentists, and evolving dental disease patterns and the use of dental services.

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REDUCTION OF DENTAL FEAR

Why physiological reactions remain when their psychological concomitants are modified can only be a matter of speculation. Bernstein & Kleinknecht who observed no change in an autonomic indicator of stress (Palmer Sweat Index) after reduction of dental fear seem to believe that, while the experience has become "tolerable", post treatment physiological reactions still reflect fear of dentistry. Hirschman *et al* examined the relationship between dental anxiety and an indicator of autonomic arousal (galvanic skin responses). They found no significant difference in the frequency of galvanic skin responses between high and low dentally anxious patients. Hirschman *et al* believe that dental stress may be more a function of cognitive evaluations than increased autonomic arousal. Such data, as well as our observations of ambiguous correlations between clinical and physiologic aspects of dental fear, seem to necessitate a theoretical as well as a methodological reorientation. Our study, like the vast majority of other psychophysiologic investigations of dental fear, relies upon a traditional concept of emotion: an emotion is conceived of as a unitary phenomenon, with different psychologic and physiologic manifestations. The choice of measurement, i.e., whether to study behavioral, subjective or physiologic indicators of emotional change, is often a matter of convenience. However, an alternative view is that emotion is composed of different segments: verbal-cognitive, behavioral and physiologic. Since these three systems are partly independent, emotional change may express itself differently in different cases. In view of this three-system analysis it follows that a reduction of dental fear can be predominantly cognitive, behavioral or physiologic, or any combination. It is thus necessary to observe all three types of change, and to allow for different explanations of dental fear reduction in different patients.

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Case reports

Unusual case of green teeth resulting from neonatal hyperbilirubinemia

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Timothy J. Delcambre, BS, DDS

A bizarre range of tooth colors has been observed in children. Some of these are the result of extrinsic stain deposits from chromogenic bacteria, oral iron preparations or tonics, or the exposure of precarious tooth enamel to stannous fluoride.[1] With the exception of the fluoride staining, such stains can be removed (albeit sometimes with difficulty) by polishing with a rubber cup and appropriate dental polishing materials.

Intrinsic staining or pigmentation associated with an active chemical change in the dentin or enamel poses a special problem for the dentist as well as the patient. The most commonly encountered intrinsic staining is the well-known complication of tetracycline administration during gestation or during the years of tooth development. Other etiological factors associated with tooth pigmentation are:

- ☐ Ingestion of liquid iron preparations reported to be responsible for incorporating black iron stains into developing teeth.[4]
- ☐ Disturbances of porphyrin metabolism.[5]
- ☐ Erythroblastosis fetalis.[6]
- ☐ Hyperbilirubinemia.[7]

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CASE REPORT

A seventeen-month-old black girl was referred to the General Dentistry clinic of Charity Hospital of New Orleans by the Pediatric Department for evaluation of the green coloration of her teeth. Her hospital medical records revealed that she was premature at birth. Upon questioning her legal guardian grandmother, it was found that the child received several months of intensive care at Walter Reed Army Medical Center in Washington, D.C., following her premature birth.

The discharge summary from Walter Reed revealed that the child was the result of a twenty-six week gestation pregnancy, terminated by emergency Caesarian section with a breech presentation. The pregnancy was further complicated by preeclampsia a week before delivery and by multiple drug abuse by the twenty-year-old mother. At birth the child weighed 730 grams, had an APGAR score of 4 at one minute and required intubation at four minutes of life.

After stabilization, the child was brought to the neonatal I.C.U. where she remained for five months. During that five-month period she received treatment for respiratory distress syndrome, including hyaline membrane disease; grade III bronchopulmonary dysplasia; patent ductus arteriosus, requiring ligation; iatrogenic anemia; direct and indirect hyperbilirubinemia; grade III intraventricular hemorrhage; possible pulmonary hemorrhage; early retrolental fibroplasia; hypothyroidism; rickets; hypoglycemia; thrombocytopenia; staphylococcal sepsis.

Pursuant to evaluation of the complaint of "green teeth", initial clinical examination revealed partially erupted maxillary primary central and lateral incisors and mandibular primary central incisors in a more advanced state of eruption. The exposed clinical portion of the four maxillary incisors displayed a distinctly green color (Figure 1), while the mandibular central incisors exhibited a green incisal one-third, in sharp contrast with the normally colored remainder of the tooth crown (Figure 2). The child's mouth was exceptionally clean; the oral soft tissues were healthy, pink and normal. From the shiny, clean character of the tooth enamel, it was obvious that the green stain was intrinsic and not extrinsic in nature.

On a follow-up visit with the child at twenty-six months of age, the maxillary and mandibular primary incisors, canines and first molars were in an advanced state of eruption and displayed a striking demarcation between the green incisal thirds and cusp tips and the normally colored remainder of the clinical crowns (Figure 3). Second primary molars were unerupted at this time, and the oral soft tissues were within normal limits. The child's mouth continued to display a high degree of oral hygiene and testified to the excellent care she received from her grandmother.

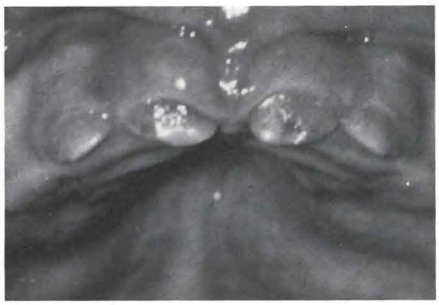

Figure 1. Clinical appearance of maxillary incisors when first examined at seventeen months of age.

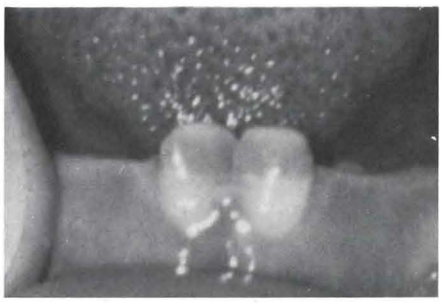

Figure 2. Clinical appearance of mandibular incisors at seventeen months of age.

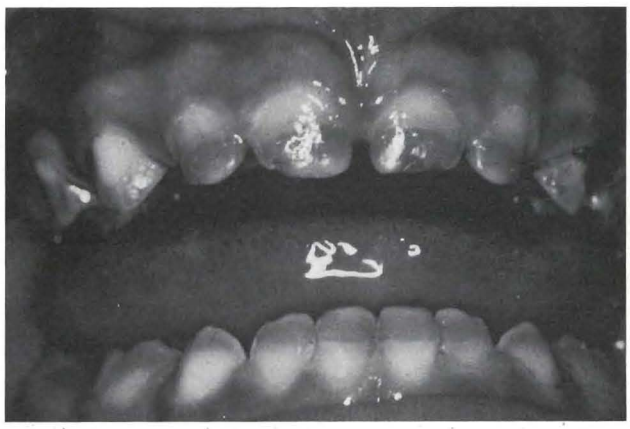

Figure 3. Clinical appearance of the primary teeth at twenty-six months of age, demonstrating the relationship of the pigmentation to the stages of tooth development.

DISCUSSION

Hyperbilirubinemia, especially in neonates, is a fairly common occurrence, usually resulting in a visible jaundice or icterus. It is chemically defined as a serum concentration of bilirubin, greater than 1.5 mg/100 ml. Jaundice, however, is rarely clinically visible until serum concentrations become greater than 7 mg/100 ml.[8] The jaundice results from the catabolism of heme-containing proteins such as hemoglobin and myoglobin into the constituents bilirubin, carbon monoxide, and iron. Excess bilirubin, accumulating in body tissues and interstitial fluid, produces a yellow-green discoloration noted initially in the sclera of the eyes and the skin.[9] Primary teeth in the formative stages may also be affected by the excess bilirubin and become intrinsically stained.[10]

The etiology of neonatal hyperbilirubinemia is varied and can result from several of the problems experienced during the neonatal period of the child reported here.

One cause of neonatal hyperbilirubinemia is premature birth.[8] Because this child was born after a gestation period of only twenty-six weeks, her metabolic and circulatory homeostatic mechanisms were obviously not functioning well enough to permit adequate excretion of bilirubin.

Respiratory distress syndrome has been noted as another cause of neonatal jaundice through the mechanism of keeping the ductus venosus functionally patent and thereby causing poor perfusion of the hepatic sinusoids and decreasing bilirubin excretion.[8]

Extravasation of blood is another possible cause of hyperbilirubinemia.[8] Once outside the circulatory system, the life span of an erythrocyte is reduced dramatically, since macrophages quickly catabolize the red cell hemoglobin to bilirubin. As noted previously, this child had an intraventricular hemorrhage and a possible pulmonary hemorrhage.

Congenital hypothyroidism has also been noted as a cause of jaundice.[8] The exact reason for this is not completely understood, but several animal studies have shown the need for thyroxine in hepatic clearance of bilirubin. There are many other causes of jaundice, but none pertain to this report.

As noted clinically, all erupted primary teeth were affected to some degree by the hyperbilirubinemia and displayed the green pigmentation (chlorodontia).[11,12] Approximately one-half of the incisor crowns, one-third of the canine crowns, and the cusp tips of the first primary molars were affected by the intrinsic staining. This relates very accurately to the time frame during which the child was jaundiced and enamel and dentin formation was occurring. It is consistent, furthermore, with information obtained from human dentition chronology charts.[13]

SUMMARY

Despite its rarity, the unusual green pigmentation of a child's teeth causes great anxiety within the family and peer problems of significance for the child. Timely initiation of dental treatment to prevent disintegration of hypoplastic teeth and to offer cosmetic improvement of unusually discolored teeth must be made available to these children, to assure their normal physical, psychological, and social development. The methods, techniques, and materials available today make this possible.

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Familial dysautonomia with Riga-Fede's disease: report of case

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Mira Frand, MD
Natan Brand, MD

Familial dysautonomia was originally described by Riley *et al* in 1949.[1] This syndrome is characterized by its multiorgan involvement, predominantly a dysfunction of the autonomic and sensory central nervous systems.

One of the well-known features of this disease is insensitivity to pain, which eventually leads to diffuse scarring and repeated ulcerations caused by trauma.[2-4]

Riga-Fede's disease is characterized chiefly by ulcerations on the lingual frenum of infants.[5-8] The histories of these patients associate the ulcerations with trauma inflicted by the primary teeth.

Riga, in 1881, presented a clinical description; and Fede, in 1890, described a histologic examination of this oral phenomenon. This lesion has also been termed *sublingual growth in infants, sublingual ulcer, sublingual granuloma* and *reparative lesion of the tongue.*

In this report, we describe the occurrence of Riga-Fede's disease in a child with familial dysautonomia, following the eruption of mandibular and maxillary primary incisors, in all probability related to insensitivity to pain.

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CASE REPORT

M.S. was born to healthy East European parents. He had a stormy perinatal course with feeding problems, choking inspirations, pneumonia, and failure to thrive. At the age of three months, a diagnosis of familial dysautonomia was made. At that time he suffered from corneal ulcerations, and characteristic reactions to denervation after intradermal injections of histamine.

At the age of ten months, he was seen in the pediatric clinic with extensive ulcerative lesions on the base and dorsum of the tongue. At the time of examination he had erupted the maxillary and mandibular primary central incisors. Biopsy and excision of the lesions was performed in the oral surgery service, with no subsequent healing of the lesions. The report of the pathological examination of the specimen stated *nonspecific inflammatory process.* At that stage, the infant was referred to the pediatric dental clinic for consultation. Upon examination of the tongue, a raised, yellow exophytic lesion (1.5 cm x 2.0 cm) with ulcerative borders occupied most of the dorsum of the tongue (Figure 1). The ventrum of the tongue presented a similar exophytic ulcerative lesion (1.0 cm x 1.0 cm) (Figures 2,3). A diagnosis of Riga-Fede's disease was established and the decision made to cover the incisal edges of the maxillary and mandibular primary incisors with composite material, using an acid-etch technique.

The patient was sedated with chloral hydrate p.r. 1cc/1kg (100 mg/kg) supplemented with 40 percent N_2O/O_2 and the composite coverage was performed (Figure 3). Examinations in one and two weeks showed an impressive healing of the ulcerative lesions on both sides of the tongue (Figure 4).

Nine weeks later, the patient returned to the pediatric dental clinic with the complaint of a new lesion on the right sublingual area of the tongue. On examination, a new ulcerative lesion (1cm x 0.5 cm), opposite the primary mandibular right lateral incisor, which had just reached the occlusal incisal level of the neighboring central incisors, was seen (Figure 5).

The patient was sedated in the same manner described above and the incisal surfaces of the mandibular incisors were covered with composite, to create an edgeless smooth surface, intended to prevent future trauma to the tongue.

Oral examination of the patient, two weeks later, showed a complete healing of the lesion had occurred.

DISCUSSION

Familial dysautonomia is an autosomal recessive disease, characterized by extensive disturbances of the nervous system, in which, however, autonomic and sensory disorder predominates. Most of the clinical features are due to disturbed function of the autonomic nervous system, absence of tears, excessive perspiration, muscular incoordination, and indifference to pain.

An anatomically reduced number of unmyelinated fibers in the sural nerve and reduced numbers in the autonomic ganglia, with marked diminution in sensory axons in the tongue, have been found.[9,10]

Gadoth suggested that the absence of fungiform papillae in classic dysautonomia is not conclusive, unless a proper examination of the tongue is made.[9,11] Careful examination of the patient's tongue showed diminished fungiform papillae, but not a total absence (Figures 2,4). Examination of the patient and questioning of the mother revealed a forceful tongue thrust between the edges of the erupting primary maxillary and mandibular incisors. Despite the enormous size of the lesions, the infant showed no signs of discomfort or pain, during the three months the lesions existed.

Healing of the lesions did not follow their surgical excision as described previously.[12] Reports in the past mentioned various methods of treatment, starting from discing the traumatizing tooth and ending in its extraction.[12] Composite coverage of the traumatic incisal edges of the primary incisors proved, however, to help in

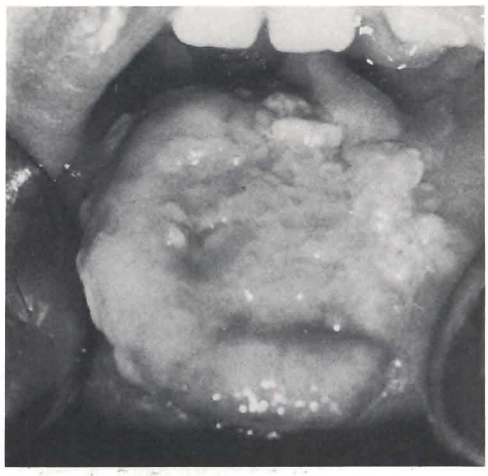

Figure 1. Exophytic ulcerative lesion on dorsum of tongue (1.5 cm x 2.0 cm). Age, twelve months.

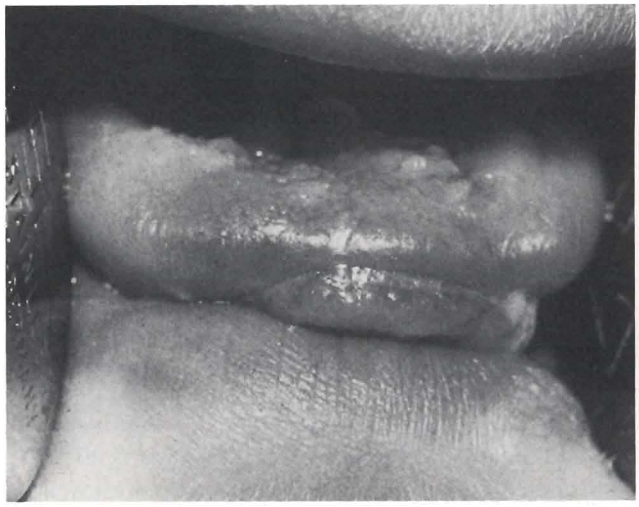

Figure 2. Exophytic ulcerative lesion on ventrum of tongue (1.0 x 1.0 cm).

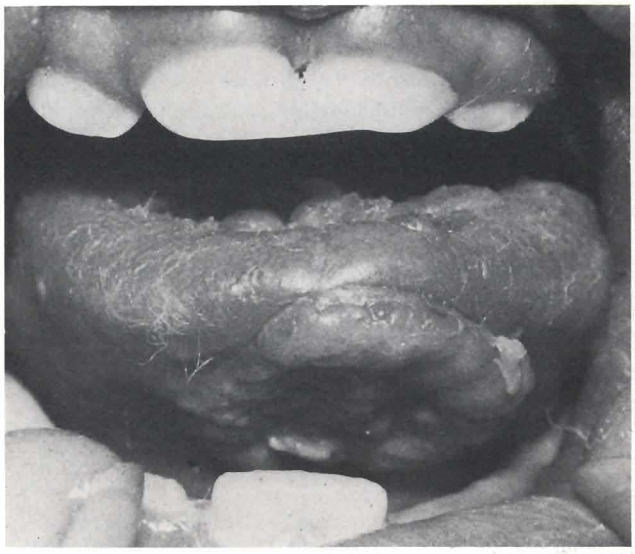

Figure 3. Composite coverage of the primary central incisors.

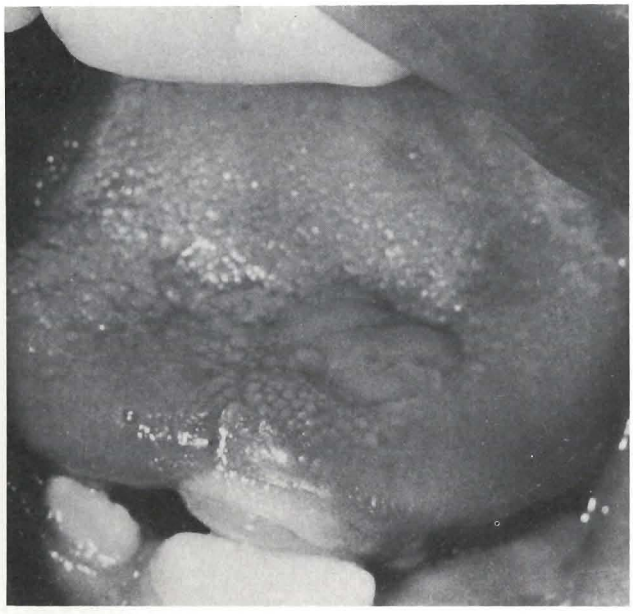

Figure 4. Healing of the dorsal and ventral lesions occurred in two weeks. Small lesions (0.3 cm x 0.5 cm) receded before total disappearance.

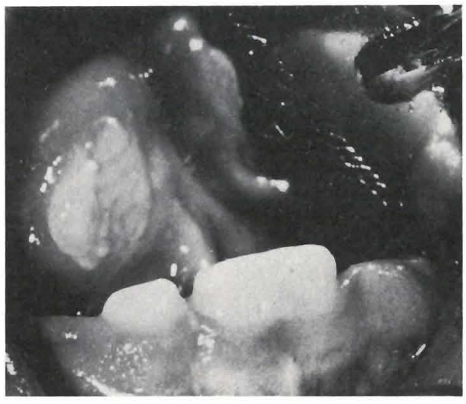

Figure 5. An ulcerative lesion facing the primary mandibular right lateral incisor, nine weeks later (1.0 cm x 0.5 cm).

the rapid healing of the ulcerations.

The extensive disturbances of the nervous system, with indifference to pain a major finding, emphasize the traumatic etiology of the lesions on the tongue. The forceful tongue thrust, with the incisal surfaces of the erupting incisors as the insulting factors, led us to the plan for eliminating those surfaces.

SUMMARY

Due to insensitivity to pain, various complications in familial dysautonomia were reported. To the best of our knowledge, occurrence of Riga-Fede disease was not described. A simple restorative procedure helped to bring about the rapid healing of the ulcerative lesions of Riga- Fede disease.

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Hyperdontia in the primary dentition: report of case

Edna L. Pashley, DMD, MEd

Several excellent reviews have recently been published on the incidence of hyperdontia and hypodontia.[1-3] The reviews established several important generalizations about supernumerary teeth, which warrant review. They point out the very low frequency of supernumerary teeth at about 16 per 1000, being higher in males than females, and showing up more frequently in the maxilla than the mandible (8:1). Bodin *et al* stated that "hyperdontia in the primary dentition was very rare."[2] Only seven cases were found in 21,609 patients or 0.3 per 1000.

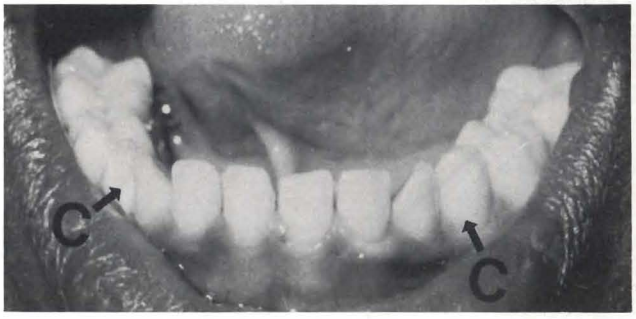
This report describes a case of hyperdontia in a primary, mandibular dentition, in which the supernumerary teeth were fully erupted and in functional occlusion. The combination of these factors makes this a very rare and interesting case.

CASE REPORT

A well-developed, six-year-old, black male, was brought to my office for routine dental care. The mother was only aware that her child needed several restorations. During routine evaluation it was noticed that the child had six apparently normal mandibular incisors between his canines (Figure 1). Occlusal radiographs revealed six mandibular primary incisors between the canines, with a normal number of succedaneous teeth developing in the anterior segment of the mandible (Figure 2) and maxillae (not shown). A panographic radiograph revealed no posterior hyperdontia although it showed the absence of a mandibular second premolar on the right side (not shown).

Medical and dental histories were unremarkable. The dental examination showed the presence of numerous caries lesions in the posterior teeth. The mandibular incisors were all of normal size, shape and color and they were fully erupted in reasonable alignment, and undergoing normal root resorption for a child of that age. The family was not aware of the condition until it was brought to their attention.

Figure 1. Photograph of patient's six primary mandibular incisors, located between the primary canines (designated by letter C).



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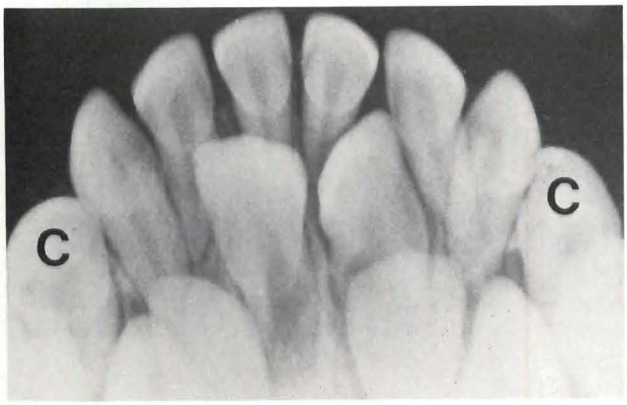

Figure 2. Occlusal radiograph of the same teeth as shown in the previous illustration. Canines are designated by letter C. Permanent incisors are developing normally.

DISCUSSION

Humerfelt *et al* recently reviewed hyperdontia in the primary dentition. They reported the incidence to be between 0.03 and 1.9 percent. They point out that hyperdontia in the primary dentition is often overlooked, because the teeth often erupt normally, are often of normal shape and frequently appear to be in proper alignment, as was the case in this child. They suggest that the prevalence of hyperdontia in the primary dentition has been greatly underestimated.

Hyperdontia seems to show a predilection for premaxillary sites.[4-7] Humerfelt's group reported no differences between sexes in the incidence of supernumerary primary teeth, in agreement with Luten, but in contrast to reports of a marked predilection for hyperdontia in males, in the permanent dentition.[1,5] According to previous reports, a low percentage of permanent supernumerary teeth erupt, while Humerfelt *et al* reported that 73 percent of the primary supernumerary teeth erupted.

The high incidence of eruption of supernumerary primary teeth is probably due to the space created in the primary arch by normal growth. Removal of these supernumerary teeth should await the normal eruption of the underlying permanent teeth, and is seldom required as the primary teeth exfoliate normally. When observed, hyperdontia in the primary dentition should alert the clinician to the possibility of hyperdontia in the permanent dentition. A careful radiographic survey of both dental arches will provide the clinician and the parents with a preview of any further problems that might develop during the course of the child's growth and development.

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COMPARISON OF TREATMENT DATA

Comparison of treatment data performed by successive classes (1980 to 1984) in a predoctoral pedodontic clinic suggests that the restorative needs of pedodontic patients are decreasing in quantity and complexity. The data support empirical observations of educators that the clinical experiences of predoctoral dental students are being affected by the changing patterns in caries prevalance. While the issues are complex and do not lend themselves to universal solutions, it is clear that pedodontic educators must evaluate and coordiante predoctoral and graduate pedodontic programs to reflect both the changing needs of children and the realities of the professional marketplace.

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Goldenhar's syndrome and hypodontia: report of case

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Goldenhar's syndrome (oculo-auriculo-vertebral dysplasia) is an otocraniofacial malformation syndrome in which defects of the eyes, ears, and vertebrae are associated with orofacial, cardiac, respiratory, renal, gastrointestinal and nervous system abnormalities.[1-4] Hypodontia has been reported in association with a number of other syndromes, but there are no previous reports linking hypodontia with Goldenhar's syndrome.[5] We report a case of Goldenhar's syndrome with hypodontia.

CASE REPORT

A nine-year-old boy with Goldenhar's syndrome was referred by his consultant pediatrician to the Department of Child Dental Health, after falling and traumatizing an anterior tooth.

Goldenhar's syndrome was diagnosed at birth after a normal confinement and delivery. Mother had remained well during pregnancy and there was no relevant family history. Abnormalities present at birth were: an epibulbar dermoid at the left lateral corneoscleral junction, a left ear deformity with associated auricular appendages, left malar hypoplasia, widening of the left angle of the mandible, and left sixth and seventh cranial nerve palsies.

During infancy, surgery to remove the epibulbar dermoid, correct strabismus and remove the auricular appendages was completed, and an intravenous pyelogram showed no renal abnormalities. Full face and lateral profile views taken at examination are shown in Figures 1 and 2. Dental examination revealed normal intraoral soft tissues and the following teeth were present:

6	E	D	C	B	1		1	B	C	D	E	6
6	E	D	C	2	1		1	2	C	D	E	6

A superficial enamel fracture involving the incisal edge of the maxillary left central incisor was noted, but this tooth was asymptomatic and vital. The midline of the mandibular arch was deviated 3 mm to the left and the mandible deviated to the left on opening. The lower right second primary molar was submerging.

Radiographic examination revealed that the maxillary left and mandibular right second premolars, and the maxillary left lateral incisor were not developing. The maxillary right lateral incisor was diminutive and there was no periapical morbidity associated with the maxillary central incisors. A flat gonial angle on the left was associated with a 20 percent reduction in the length of the left mandibular ramus (Figure 3).

DENTAL TREATMENT

In view of the lack of symptoms from the vital maxillary left central incisor, which was smooth and both func-

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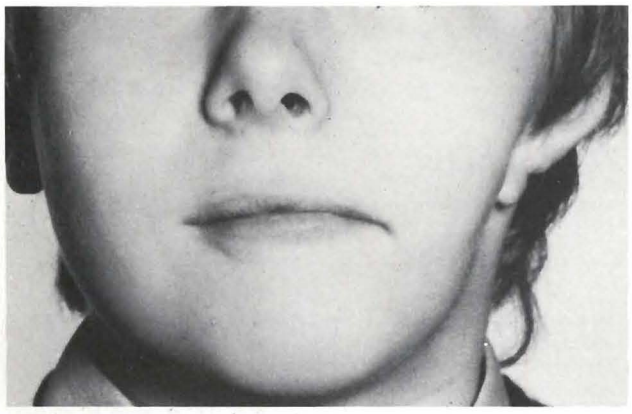

Figure 1. The patient on examination at age nine with left seventh cranial nerve palsy and flattening of the left gonial angle.

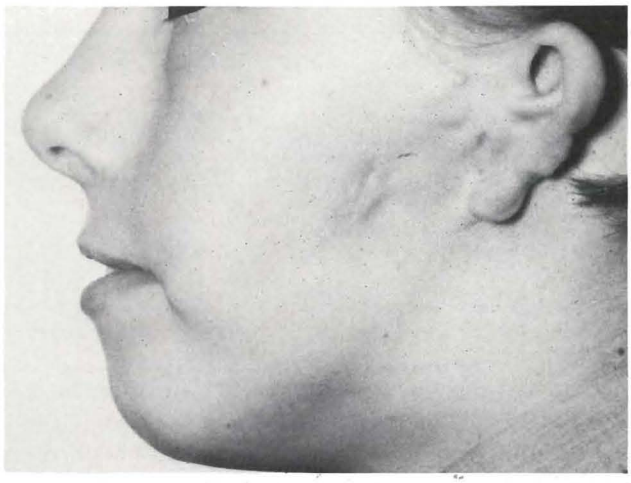

Figure 2. Lateral profile of the patient of examination at age nine, showing abnormal left ear and surgical scar from removal of auricular appendages, left malar hypoplasia and flat left gonial angle.

tionally and aesthetically sound, it was decided that a six-month review would be suitable, when further orthodontic diagnosis would be possible, and a decision made regarding the prognosis for the maxillary right lateral incisor and the mandibular right second primary molar.

DISCUSSION

The syndrome was probably first described by Von Arlt (1845), although it was not until 1952 that Goldenhar clearly defined the syndrome that now bears his name.[6] By 1978, more than 114 cases had been described in the world literature.[7] Incidences of between 1 in 5600 births and 1 in 3500 births have been claimed, although these seem unusually high in view of the number of reported cases.[8,9] The reported orofacial manifestations of this syndrome are presented in the Table.

The etiology and genetics of Goldenhar's syndrome are unresolved. Chromosome analysis has revealed a normal karyotype in all but one case where the Goldenhar variant was seen in association with a 5p karyotype.[10] Dermatoglyphic analysis similarly has not revealed characteristic patterns. The majority of cases occur sporadically, but incidences have been observed in successive generations.[8,11] Embryologically, the syndrome is thought to derive from an abnormality of mesoblastic development affecting the formation of branchial and vertebral systems. The abnormality is unknown,

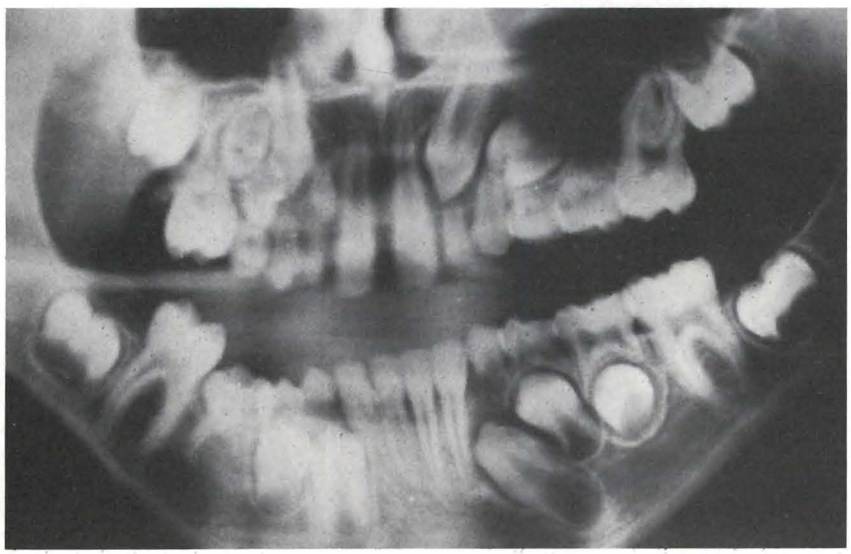

Figure 3. An OPG of the patient at age nine showing a 20 percent reduction in the length of the left mandibular ramus and absence of developing maxillary left lateral incisor and second premolar, and mandibular right second premolar.

Table ☐ Reported orofacial manifestations of Goldenhar's syndrome.

Frontal bossing	Decreased palatal width from midline to lingual surface of teeth on affected side
Unilateral facial hypoplasia	Bifid tongue
Zygomatic, temporal, and maxillary hypoplasia	Bifid uvula
Aplasia or hypoplasia of mandibular ramus and/or condyle with absence of the glenoid fossa	Double lingual frenum
Flattening of the gonial angle	Enlarged philtrum
Macrostomia in one third of patients with agenesis of the mandibular ramus	Malocclusion
High palatal vault	Agenesis of ipsilateral parotid gland and surrounding muscles
Cleft lip and palate	Aberrant salivary gland tissue
Palate and tongue muscles unilaterally hypoplastic and/or paralysed	Salivary fistulas

however, and this does not explain the involvement of other body systems in this syndrome.

Although hypodontia has not previously been reported in association with Goldenhar's syndrome, it is suggested that an increased awareness of possible dental abnormalities by medical staff, together with dental radiological review of these patients into adolescence may reveal further reports of hypodontia.

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PEDODONTIC PATIENT SHORTAGE

Accentuating the problem of insufficient patient numbers are concerns of pedodontic chairmen that shortages of basic procedural techniques such as stainless steel crowns and pulpotomies could have a negative effect on students' ability to provide quality care for children after graduation. The observed shortages in restorative procedures are related to documented changes in the caries experience of children during the decade of the 1970s. As demonstrated by Brunelle and Carlos, comparison of national DMFS data between 1971 and 1980 indicates a greater than 30 percent decline in caries prevalence among U.S. schoolchildren and a dramatic increase in the number of caries-free children, from 28 percent to 37 percent of all children examined. The decline in caries prevalence is also reflected in reports of a "busyness" problem and a decreasing requirement for restorative services in children by practicing pedodontists.

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