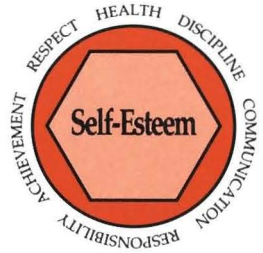
ASDC
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN
JOURNAL OF DENTISTRY FOR CHILDREN
MARCH–APRIL 1992

Self-esteem is central to what we make of our lives—the loyalty we have to developing ourselves and to caring about others, and it is at the heart of what we will achieve in the course of our lifetime. Perhaps nothing affects health and energy, peace of mind, the goals we set and achieve, our inner happiness, the quality of our relationships, our competence, performance, and productivity, quite as much as the health of our self-esteem.

The best chance a child has of securing a level of high self-esteem is to have parents who possess it, model it, and want to instill it in their children. If parents rear their children with love and respect, allow them to experience consistent and benevolent acceptance, give them the supporting structure of appropriate rules and reasonable expectations, if they don't assail them with contradictions, don't resort to ridicule, humiliation, or physical abuse as a means of controlling them, and help them believe in their abilities and goodness—then children have a chance of internalizing those attitudes and thereby acquiring the foundation for healthy self-esteem.

–Bettie B. Youngs,Ph.D.



EVERY NEW ADJUSTMENT IS A CRISIS IN SELF-ESTEEM.
—*Eric Hoffer*: The Ordeal of Change

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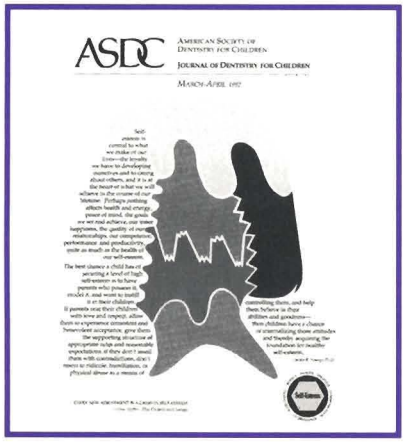

Self-esteem is at the heart of what one will achieve in the course of a lifetime.

Cover art and design by Sharlene Nowak-Stellmach.

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BEHAVIOR

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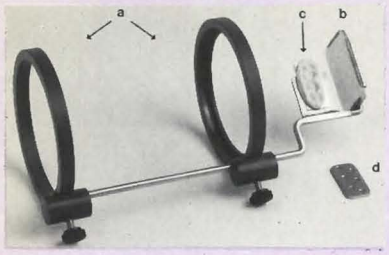

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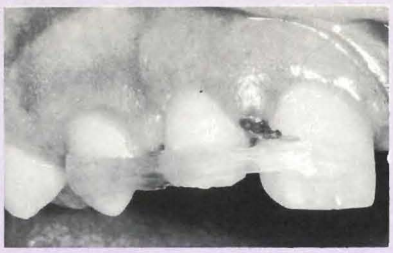

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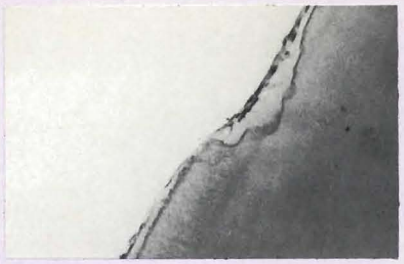

DEVELOPMENT

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EPIDEMIOLOGY

REVIEW

CASE REPORTS

For the busy reader

Radiographic assessments of class II resin composite restorations in a clinical study: Baseline results—page 97

In this article, the baseline results of radiographic evaluation of both Class II resin composite and Class II amalgam restorations are discussed. Attention is paid to the quality of the cervical margin adaptation, the occurrence of radiolucencies adjacent to the restoration, and the homogeneity of the resin composite.

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

Diffusion of buffered glutaraldehyde and formocresol from pulpotomized primary teeth—page 108

The medicaments used in this study were buffered glutaraldehyde and formocresol. Schiff's reagent was used in the quantification of aldehyde released into the collecting medium. The results of this study clearly show that formocresol diffused throughout the dentine and cementum within fifteen minutes following a pulpotomy procedure, whereas no diffusion of buffered glutaraldehyde was observed.

Requests for reprints should be directed to Dr. Mean Rusmah, Lecturer, Department of Children's Dentistry, Faculty of Dentistry, University of Malaya, 59100 Kuala Lumpur, Malaysia.

Mandibular fractures in Sri Lankan children: A study of clinical aspects, treatment needs and complications—page 111

Thirty-seven children younger than age eleven who had incurred mandibular fractures were studied. Age, sex, type of fracture, incidence, etiology, treatment methods, and complications were examined.

Requests for reprints should be directed to Dr. N.A. de S. Asoka Amaratunga, Division of Oral Surgery, Dental School, University of Peradeniya, Peradeniya, Sri Lanka.

Dental treatment of fearful children using nitrous oxide. Part II: The parents' point of view—page 115

This survey was conducted among the parents of fifty-two children with severe dental anxiety, who were between the ages of six and eleven years of age. Results showed that parents consider the dentist to be the sole source of their child's dental anxiety. Personality and/or environment are unconnected to their child's fear, in their opinion.

Requests for reprints should be directed to Dr. J.S.J. Veerkamp, ACTA, Louwesweg 1, 1066 EA Amsterdam, The Netherlands.

Eruption pattern in the primary dentition of premature low-birth-weight children—page 120

This study evaluated the eruption pattern of primary teeth in thirty-one prematurely born children with low birth-weight. Compared with the normal development pattern, the prematurely-born children younger than age twenty-four months had 28 percent fewer erupted teeth, on average.

Requests for reprints should be directed to Dr. Shahrbanoo Fadavi, Department of Pediatric Dentistry (MC 850), University of Illinois at Chicago, 801 South Paulina Street, Chicago, IL 60612.

There really are a lot of poor children in the United States—page 123

More than 20 percent of children in this country live in poverty; in cities, the figure is 30 percent. One fourth of all children younger than age six—and the same proportion of rural children—are living in poverty. Malnutrition affects nearly 500,000 children.

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.

Do the parent(s) of your pediatric patients smoke?—page 126

Parental smoking (and smoking by young teenagers) is the concern of pediatric dentists. A review is provided of the incidence of smoking and its consequences.

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.

Children without health-care coverage—page 129

A review is provided of various federal reports on the health-care coverage of children. The role of pediatric dentists in helping to advance improvements and access to care is underscored.

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.

Distribution of missing teeth and tooth morphology in patients with oligodontia—page 133

In a population of 196 patients with isolated oligodontia (/I) and oligodontia as part of a syndrome (/S), the distribution of missing teeth was recorded. Patients with oligodontia/S were missing significantly more teeth than patients with oligodontia/I.

Requests for reprints should be directed to Dr. Yvonne van der Weide, Department of Oral-Maxillofacial Surgery, Prosthodontics and Special Dental Care, University of Utrecht, Padualaan 14, 3584 CH Utrecht, The Netherlands.

Considerations for the direct pulp capping procedure in primary teeth: A review of the literature—page 141

Direct pulp capping of pulp exposures in primary teeth remains controversial. This review of the literature presents findings and recommendations to encourage a more favorable success rate of direct pulp capping, comparable to the more invasive techniques such as pulpotomies and pulpectomies in primary teeth.

Requests for reprints should be directed to Dr. Hugh M. Kopel, University of Southern California, School of Dentistry, Children's Hospital of Los Angeles, Los Angeles, CA 90054-0700.

Milk as storage medium for exarticulated teeth: Report of case—page 150

In Scandinavia, traumatic dental injuries can usually be treated professionally within a few hours. A fourteen-year-old boy suffered an avulsed tooth while playing basketball. Despite an extraoral period of more than twelve hours, a functional PDL survived.

Requests for reprints should be directed to Dr. Karl-Johann Nordenvall, Department of Pedodontics, Folktandvarden, Fjärilen, Turingegatan 26, 151 34 Södertälje, Sweden.

An unusual case of dental anomaly: A "facial" talon cusp—page 156

Requests for reprints should be directed to Dr. Najwa M. Jowharji, 2160 S. First Avenue, Maywood, IL 60153.

CLINIC

Radiographic assessments of class II resin composite restorations in a clinical study: Baseline results

C.M. Kreulen, DDS
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The evaluation of marginal adaptation plays a prominent role in clinical studies on the behavior of resin composites in Class II cavities.[1] When assessing this characteristic, a mirror and dental probe are usually used. This evaluation faces few problems on the occlusal surface, but it is often difficult proximally, due to limited accessibility. Alternative methods are thus needed to obtain supplementary information about the box margins, particularly because of the poor access and limited visibility that contribute to the imperfections of the restorative treatment.

Such an alternative evaluation is bitewing radiography. The radiographic image of the restoration often reveals that part of the box that is difficult to evaluate directly. The collection of subsequent radiographs enables, in contrast to clinical data, comparison of the situation at any time.[2]

It is generally accepted that posterior resin composites should be radiopaque. There is, however, no consensus as to what the right degree of radiopacity should be.[3] Radiopaque composite materials assist in the detection of restorations with cervical marginal defects, and in distinguishing between the restoration and carious tooth tissue.[4,5] Moreover, the radiopacity helps in determining the homogeneity of the material used in resin composite restorations.[6]

In this article, the baseline results of radiographic evaluation of both Class II resin composite and Class II amalgam restorations will be discussed. In this discussion, attention will be paid to the quality of the cervical marginal adaptation, the occurrence of radiolucencies adjacent to the restoration, and the homogeneity of the resin composite. These characteristics will be related to the following factors: choice of material, type of restoration, type of tooth and (level of experience of the) dentist.

MATERIALS AND METHODS

This study forms part of a longitudinal clinical study in which 244 Class II restorations fitted to fifty-six patients are evaluated.[7] Each patient received a series of three resin composite restorations[a] and one amalgam restoration[b]; the amalgam restoration served as a con-

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The authors would like to thank Kerr and Kuraray/Cavex Holland for their support and to express their thanks to Professor C.L. Davidson for his advice.

[a]Herculite XR (Kerr, Santa Ana, CA, U.S.A.).
Clearfil Ray Posterior (Cavex/Kuraray, Haarlem, The Netherlands)
Visionmolar (ESPE, Oberbay, Germany)

[b]Tytin (Kerr, Santa Ana, CA, U.S.A.).

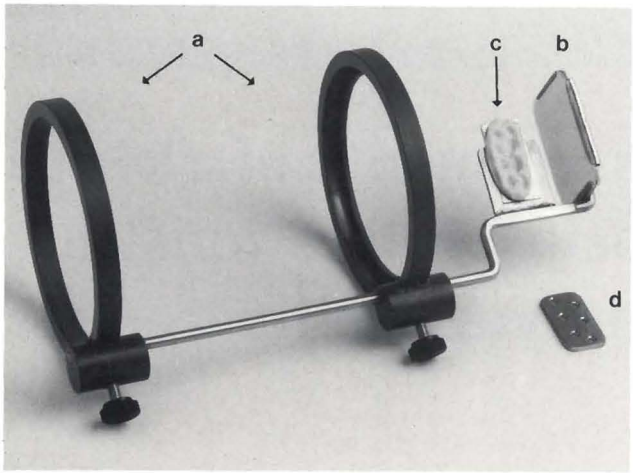

Figure 1. Picture of the aiming device used: a = rings of the tube mount; b = filmholder; c = occlusion mount; d = exchangeable perforated metal plate (here without resin).

trol. A number of patients received two series. The materials were allocated randomly to the teeth to be restored. Two dentists each produced twenty series of four restorations, and a third dentist made twenty-one series. The difference in levels of experience between these three dentists is described in an article by Kreulen *et al.*[8] The restorations of standard size were made in accordance with a detailed protocol, using a direct filling technique.[7]

Generally within three months after treatment, two bitewing radiographs (one right and one left) were taken of each patient, using a standardized procedure. A beam aiming device as described by van der Linden was used for this purpose (Figure 1).[9] A long- cone X-ray tube can be passed through the rings of the tube mount. The film holder, the tube mount, and the occlusion mount are attached rigidly to one another by means of a metal rod, and this construction ensures that the beam meets the film perpendicular. Occlusal mounting is achieved by applying self-polymerizing resin[c] to the perforated metal plate, and this exchangeable relocation jig can be fitted to the equipment for every patient individually. Using the imprints of the lower and upper teeth in the resin, it is possible to determine whether the projection of the proximal surfaces will show overlap, before the radiograph is taken.

The bitewings were produced using a long-cone X-ray unit[d] (70 kV, 15 mA) with 2 mm aluminum added filtration. The focus-film distance of the aiming device was 38 cm. Each film[e] was exposed for 0.4 seconds, using an electronic timer, after which the radiograph was developed directly in an automatic developing unit (AC 245 L, Dürr Dental), using a developing time of 8 minutes at 28° C.

The bitewings were evaluated by two trained dentists who were not involved in making the restorations. They used a viewbox and an X-viewer[f] for this purpose. The viewbox provides steady illumination under an opal glass object holder (light intensity approximately 2900 lx and color temperature 2800-2900° Kelvin, both measured at the surface of the glass). The X-viewer enlarges by a factor of 2 and shields against excess light. The evaluation took place in a semidarkened room.[10]

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[c]Formatray (Kerr Europe, Basel, Switzerland).

[d]GE 1000 (General Electric, Milwaukee, WI, U.S.A.).

[e]Kodak Ektaspeed E, size 2, double pack (Kodak-Pathé, France).

[f]X-viewer (X-Produkter, Malmö, Sweden).

The cervical margins of resin composite restorations (30 percent) appear to show fewer defects than the cervical margins of amalgam restorations (40 percent).

Evaluation of the radiographs was conducted on the basis of the items and criteria described in Figure 2. A distinction was made between the box(es) and the step in the evaluation of each restoration. Where there was overlap between the restorations to be evaluated, or where part of the restoration was not shown on the radiograph, a score of 8 was given to that part.

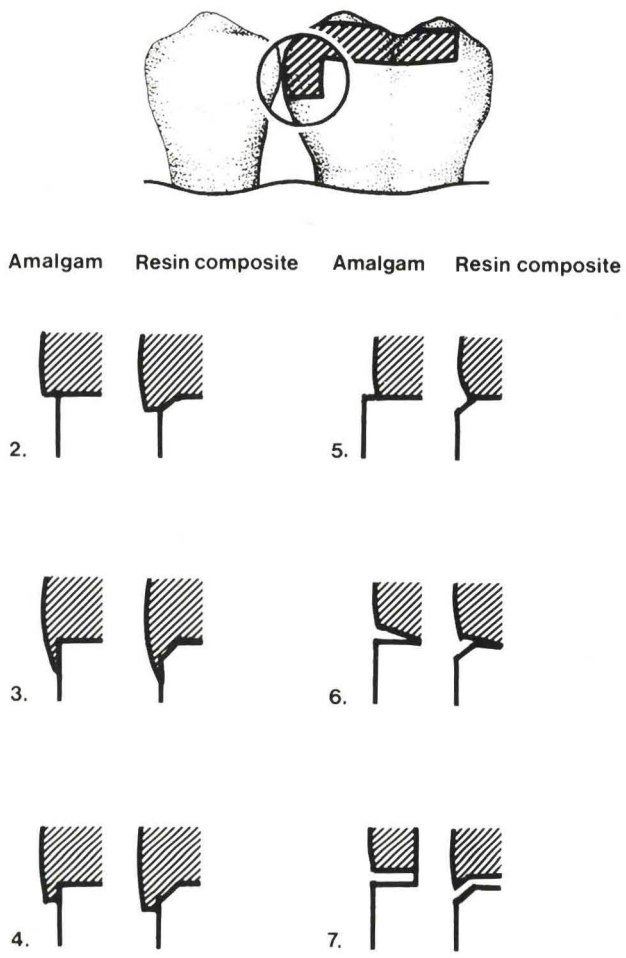

Figure 2. Evaluation items and criteria. 1) Cervical marginal adaptation: 1 = The cervical marginal adaptation is good; 2 - 7 = The cervical marginal adaptation shows defects, as shown in the diagrams above, divided into resin composite and amalgam restorations; 8 = The cervical marginal adaptation cannot be evaluated. 2) Voids (as an indication of the homogeneity of the restorative material): 1 = No detectable voids; 2 = Uncertain whether voids are present; 3 = Detectable voids; 8 = Evaluation not possible. 3) Adaptation to the vertical wall of the step: 1 = The adaptation is good; 2 = The adaptation is poor; 8 = Evaluation not possible. 4) Radiolucencies adjacent to the restoration: 1 = No detectable radiolucencies; 2 = Uncertain whether radiolucencies are present; 3 = Detectable radiolucencies; 8 = Evaluation not possible.

The evaluations of cervical marginal adaptation were based upon the criteria of cervical marginal defects of amalgam restorations as described by van Amerongen *et al.*[11] Unlike the amalgam restorations, the resin composite restorations made have a bevel, and this bevel can be distinguished as such on the radiographic image of the restoration (Figure 2). The homogeneity of the resin composite is determined on the basis of the presence or absence of voids.

Radiolucent areas adjacent to the restorations are evaluated in the vicinity of the gingival and pulpal walls for the box, while for the step, the pulpal floor is examined. The step of a two-surface restoration radiographically also has a vertical wall. The adaptation to this wall is evaluated in the same way as for cervical marginal adaptation, except that a distinction is only made between good and poor marginal adaptation. The presence or absence of radiolucencies in this vertical wall is also evaluated.

The two researchers evaluated the radiographs separately. Where there were differences between their evaluations, joint decision-making was used. In cases where agreement could not be reached, the most negative assessment was used. Interexaminer and intraexaminer agreement was determined, using Cohen's Kappa statistics. Frequency analyses were carried out on the evaluation data, and the influence of the indicated variables upon the results was determined using Chi-square tests (SPSS/PC).

RESULTS

Calculation of interexaminer agreement yields a mean Kappa value of 0.87, with a range from 1.00 (for evaluation of radiolucencies in the vertical wall of the step) to 0.77 (for evaluation of the distal cervical marginal adaptation). Intraexaminer agreement has a mean Kappa value of 0.79, with a range from 1.00 (for evaluation of radiolucencies in the vertical wall of the step) to 0.31 (for evaluation of radiolucencies adjacent to the distal box).

Frequency of deficiencies

The cervical margins of resin composite restorations appear to show fewer defects than the cervical margins of amalgam restorations (Figures 3A and 3B, good cervical margins: 75.3 percent (mesial) and 60.3 percent (distal) compared with 68.8 percent and 48.9 percent,

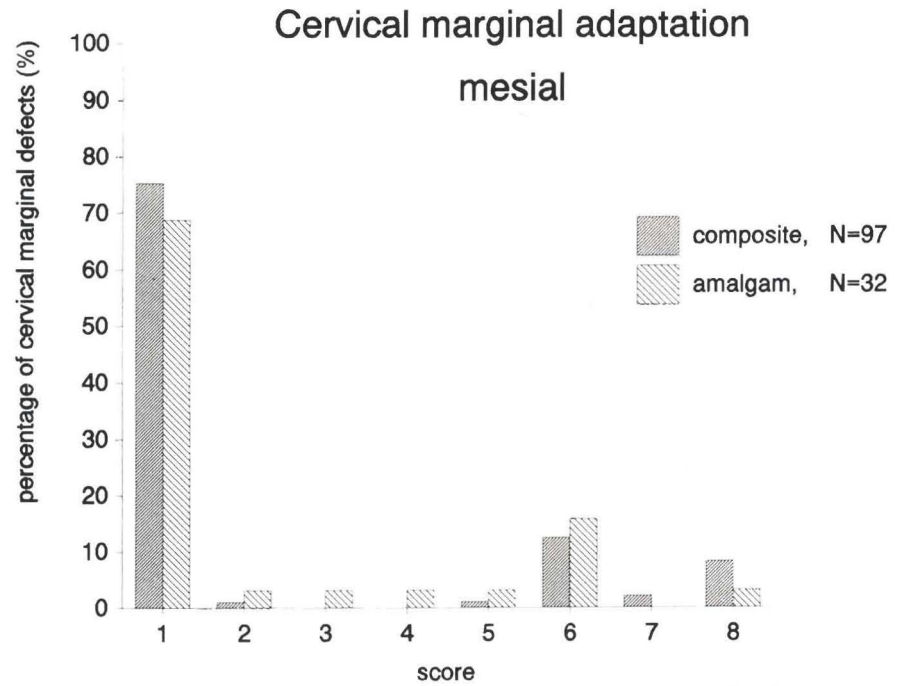

Figure 3A. Frequency distributions of the mesial cervical marginal adaptation, in percentages.

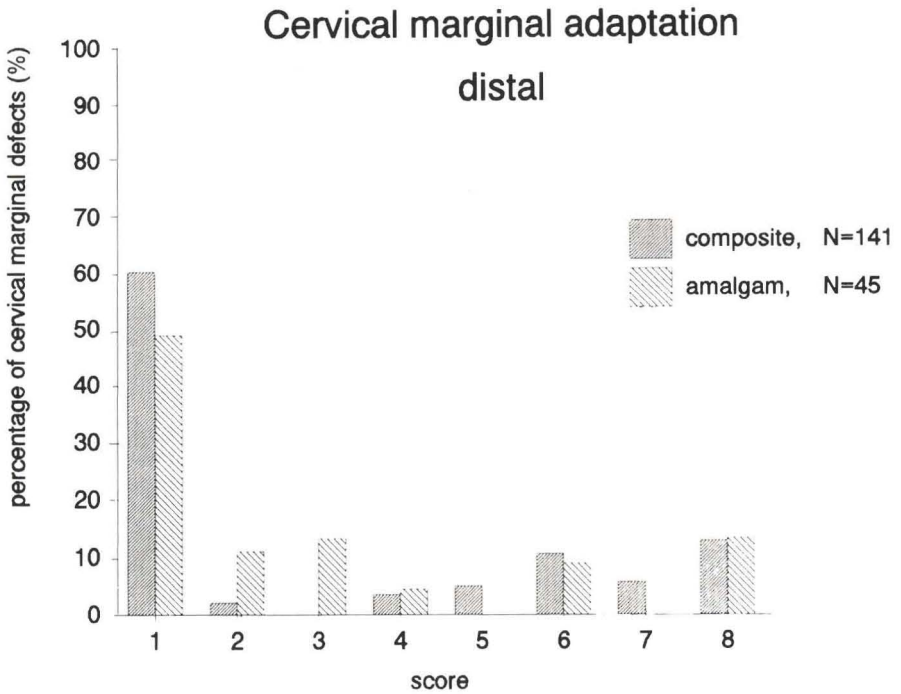

Figure 3B. Frequency distributions of the distal cervical marginal adaptation, in percentages.

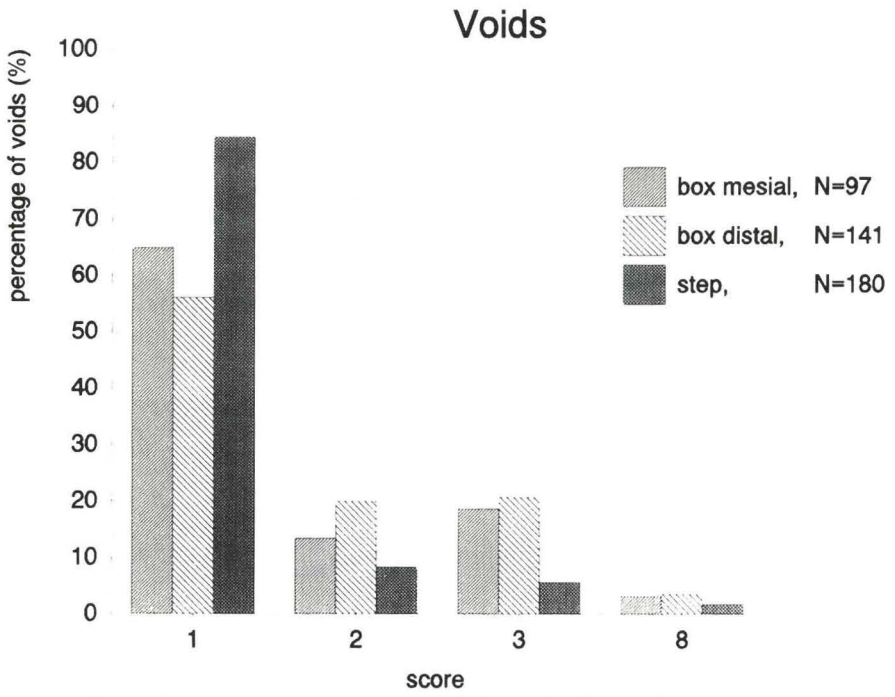

Figure 4. Frequency distributions of the voids observed, in percentages.

respectively). Cervical marginal defects receiving a score of 6 are found for both types of restorative material, while the scores 2 and 3 are mainly awarded to distal boxes of amalgam restorations.

Voids are frequently reported in resin composite restorations (Figure 4, score 3). This is more often the case in the boxes (approximately 20 percent) than in the steps of these restorations (5.6 percent). In many cases, there is uncertainty about whether voids are present (for example 19.9 percent in case of distal boxes).

Radiolucent areas are sometimes found adjacent to resin composite restorations, especially adjacent to the distal box (Figures 5A and 5B). When determining the presence or absence of radiolucencies, however, the evaluators are more uncertain for the resin composite restorations than for the amalgam restorations. Deficient adaptation of the restorative material to the vertical wall of the step as well as radiolucencies in this wall appear to be found seldomly or not at all.

In the interests of data reduction, in further calculations the only distinction made is between the presence or absence of deficiencies. Where there is uncertainty about whether or not a defect is present, these cases are included in the group with deficiencies. The division of each restoration into a step and a box (or boxes) is also dropped, and instead the restoration is examined as a whole. In summary, the calculations are based on the following definition:

Score 1: Restoration shows no deficiencies.

Score 2: a) Two-surface restoration, with a deficient box, step, or both. b) Three-surface restoration with a deficiency in one or both of the boxes, or in the step, or a combination of these.

Table 1 gives the results of this reclassification. Almost 30 percent of the resin composite restorations and over 40 percent of the amalgam restorations show cervical marginal defects. The difference found, however, is not significant ($p > .05$). A significant difference ($p < .05$) can be observed between resin composite and

Table 1 ☐ Frequency distribution in which no distinction is made between box(es) and step of the restorations, in percentages.
score 1 = no deficiency
score 2 = one or more deficiencies.

	Restorative material	N	score 1	score 2	p-value
Cervical marginal adaptation	Composite	156	71.2%	28.8%	N.S.*
	Amalgam	52	57.7%	42.3%	
Voids	Composite	172	47.1%	52.9%	
Radiolucencies	Composite	173	78.6%	21.4%	<0.05
	Amalgam	56	92.2%	7.8%	
Adaptation vertical wall step	Composite	116	92.2%	7.8%	
	Amalgam	41	100%	0%	

*N.S. = not significant

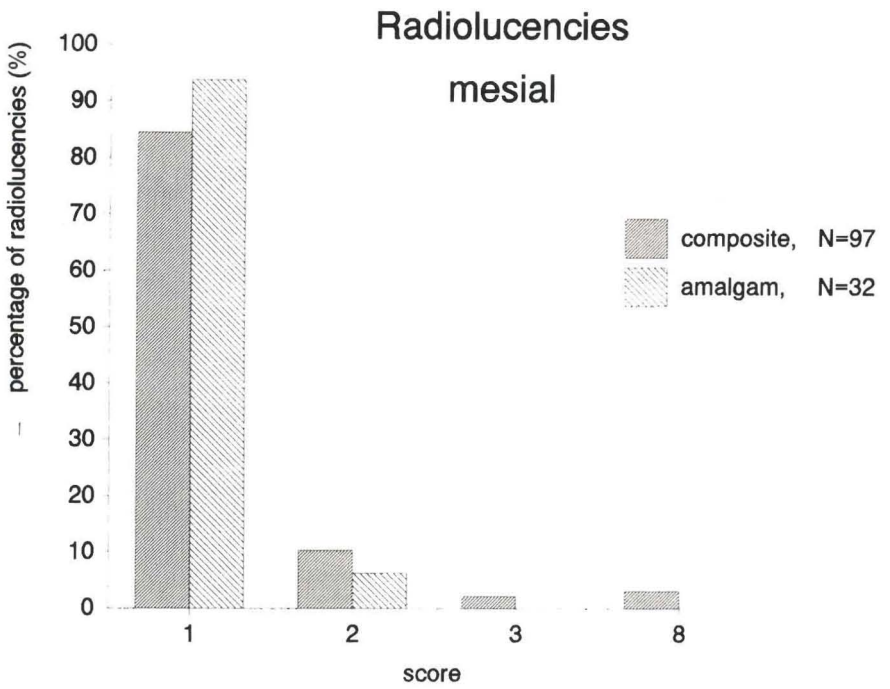

Figure 5A. Frequency distributions of the radiolucencies observed adjacent to mesial boxes, in percentages.

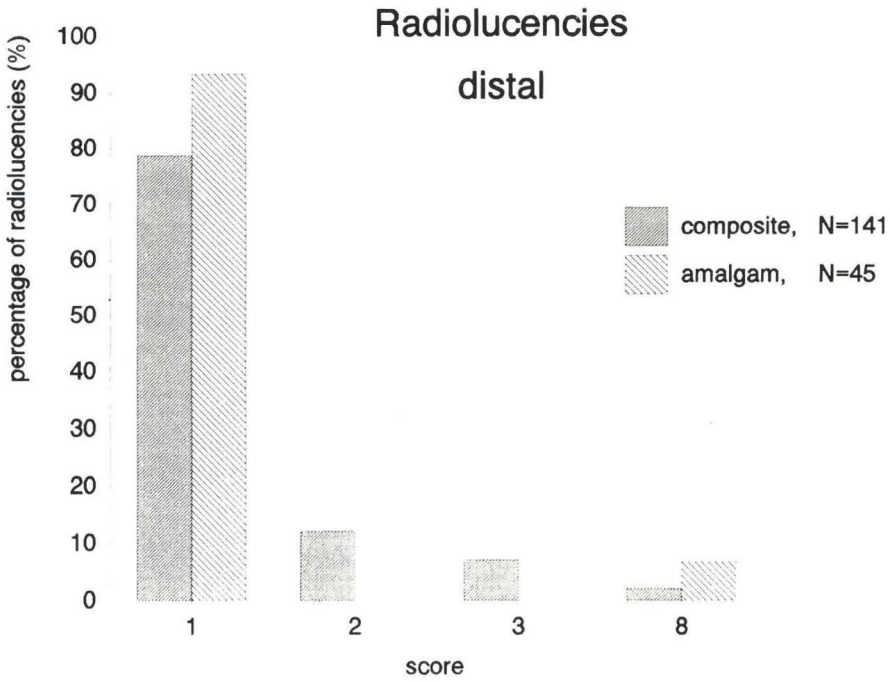

Figure 5B. Frequency distributions of the radiolucencies observed adjacent to distal boxes, in percentages.

Table 2A ☐ Influence of the factors examined on the deficiencies found, resin composite restorations.

Resin composite restorations		Dentist			Type of restoration		Type of composite resin			Type of tooth	
		1	2	3	Two-surface	Three-surface	Hercu-lite XR	Clear-fil Ray	Visio-molar	Pre-molar	Molar
Cervical marginal adaptation	N	47	51	58	109	47	52	53	51	103	53
	score 1	72.3%	47.1%	91.4%	75.2%	61.7%	73.1%	81.1%	58.8%	70.9%	71.7%
	score 2	27.7%	52.9%	8.6%	24.8%	38.3%	26.9%	18.9%	41.2%	29.1%	28.3%
	p-value		<0.001		N.S.*			<0.05		N.S.*	
Voids	N	52	58	62	116	56	57	58	57	113	59
	score 1	48.1%	36.2%	56.5%	50.9%	39.3%	40.4%	39.7%	61.4%	46.0%	49.2%
	score 2	51.9%	63.8%	43.5%	49.1%	60.7%	59.6%	60.3%	38.6%	54.0%	50.8%
	p-value		N.S.*		N.S.*			<0.05		N.S.*	
Radiolucencies	N	53	58	62	117	56	58	58	57	114	59
	score 1	67.9%	81.0%	85.5%	82.1%	71.4%	74.1%	81.0%	80.7%	73.7%	88.1%
	score 2	32.1%	19.0%	14.5%	17.9%	28.6%	25.9%	19.0%	19.3%	26.3%	11.9%
	p-value		N.S.*		N.S.*			N.S.*		<0.05	
Adaptation vertical wall step	N	39	36	41	116		32	39	45%	80	36
	score 1	89.7%	88.9%	97.6%	92.2%		93.7%	92.3%	91.1%	91.2%	94.4%
	score 2	10.3%	11.1%	2.4%	7.8%		6.3%	7.7%	8.9%	8.8%	5.6%
	p-value		N.S.*					N.S.*		N.S.*	

*N.S. = not significant.

Table 2B ☐ Influence of the factors examined on the deficiencies found, amalgam restorations.

Amalgam restorations		Dentist			Type of restoration		Type of tooth	
		1	2	3	Two-surface	Three-surface	Pre-molar	Molar
Cervical marginal adaptation	N	16	16	20	39	13	25	27
	score 1	62.5%	62.5%	50.0%	61.5%	46.2%	52.0%	63.0%
	score 2	37.5%	37.5%	50.0%	38.5%	53.8%	48.0%	37.0%
	p-value		N.S.*		N.S.*		N.S.*	
Radiolucencies	N	18	17	21	39	13	25	27
	score 1	83.3%	94.1%	100%	92.5%	93.7%	96.4%	89.3%
	score 2	16.7%	5.9%	0%	7.5%	6.3%	3.6%	10.7%
	p-value		N.S.*		N.S.*		N.S.*	
Adaptation vertical wall step	N	14	15	12	41		18	23
	score 1	100%	100%	100%	100%		100%	100%
	score 2	0%	0%	0%	0%		0%	0%
	p-value		N.S.*				N.S.*	

*N.S. = not significant

amalgam in terms of the occurrence of radiolucencies adjacent to the restorations. Deficient adaptations to the vertical wall are not found in the amalgam restorations, and no testing of these, therefore, has been carried out. It further appears that the percentage of resin composite restorations with voids amounts to 52.9 percent.

Influence of the factors examined

The results of the study on the influence of different factors on the deficiencies found are summarized in Tables 2A and 2B. The independent variables are set out horizontally: dentist, type of restoration (two- or three-surface), type of resin composite, and type of tooth (premolar/molar). The dependent variables are given vertically: cervical marginal adaptation, voids in the material, radiolucencies adjacent to the restoration, and adaptation to the vertical wall of the step. A distinction has been made between resin composite (Table 2A) and amalgam restorations (Table 2B).

The dentist appears to have a significant influence on the quality of the cervical margin in the case of resin composite restorations ($p < .001$). The least experienced dentist (dentist 3) produced restorations with the fewest marginal defects (8.6 percent). The dentist with the most experience (dentist 1) approached the mean value for the deficient cervical margins (mean 28.8 percent, Table 1), and the restorations produced by dentist 2 showed the largest number of defects of the cervical margin (52.9 percent). Radiolucencies adjacent to, and voids in, the restoration also appear to show a dentist-effect in the case of resin composite restorations (for example voids: the highest frequency was 63.8 percent, the lowest 43.5 percent). The differences found, however, are not significant ($p > .05$).

In addition to the differences between amalgam and resin composite restorations, the effects of the three different composite materials can be studied. There is a significant difference in cervical marginal adaptation between the three resin composites, such that Visiomolar shows the largest number of deficient cervical margins (41.2 percent), and Clearfil the fewest (18.9 percent, $p < .05$). Conversely, as regards the presence of voids in the material, Visiomolar shows the fewest defects (38.6 percent), while there is very little difference between Herculite and Clearfil (59.6 percent and 60.3 percent, $p < .05$).

An effect of the type of tooth is only evident as regards radiolucencies adjacent to resin composite restorations. Here, premolars show more defects than molars (73.7 percent and 88.1 percent, $p < .05$), whereas this difference is not found in the case of amalgam restorations.

DISCUSSION

Eidelman *et al* stated that radiographs are necessary to evaluate the cervical marginal adaptation of Class II resin composite restorations.[12] Evaluation of bitewing radiographs is complicated, however, by the occurrence of overlaps.[13] Moreover, the extent of the cervical marginal defects observed is strongly influenced by the angulation of the X-ray beam.[2,14] Thus the results of radiographic studies will be dependent upon the technique used.

The present study is based on the use of focusing equipment that permits one to assume that bitewings of different patients are comparable, and also that successive radiographs made of one patient at given intervals will correspond in their composition. The latter is important for future longitudinal comparative research. By using an individual jig, the position of the film relative to the X-ray beam and to the teeth is fixed. Advokaat reports, however, that even using this method, deviations can occur after a number of years.[15]

Density problems affect the interpretation of radiographs. The incorporation of a step wedge in the aiming device offers a possibility for making corrections, and in the future it will probably be possible to use image processing techniques.[16] The dimensions of a step wedge leave no room for it, however, in the adapted equipment.[17]

As well as standardizing the direction of the beam, it has also been attempted to achieve standardization of the focus-film distance, the exposure time, the tube voltage, and the developing procedure. A number of authors report that the interobserver variation has a greater influence on the evaluation of radiographs than differences in evaluation conditions.[10,18] Nevertheless, these differences have been avoided as far as possible. In addition, the Kappa values calculated give some idea of the degree of interobserver and intraobserver agreement.

According to Espelid and Tveit, detection of caries near resin composite restorations is easier than it is with amalgam restorations.[4,19] The evaluator's subjective perception will cause an apparent increase in the existing contrast between two zones with different densities (Mach effect). The observer will thus unconsciously overestimate the contrast between the dentin and amalgam on the interface, and will interpret the radiolucency of the dentin as caries (false positive).[4] The relatively low radiopacity of resin composites results in a low level of contrast with the dentin, and hence Mach effects will seldom occur. In the present study, a glassionomer cement is used as a replacement for dentin. Its radiopacity is comparable to those of the

There are significant differences in cervical marginal adaptation among the three resin composites.

resin composites used and greater than that of enamel.[20] Thus, few false positive evaluations of caries should be made.

Differences between restorative materials in the detection of radiolucencies can also be influenced by the superimposition of radiopaque restorative materials on tooth tissue.[4,21] Radiolucencies bordering the restoration can be masked in this way, thus increasing the probability of false negative judgments.

Given the above factors, the difference observed between resin composite and amalgam restorations as regards the occurrence of radiolucencies adjacent to the restorations cannot be interpreted precisely. Radiolucent areas (halos) as a result of an excess of bonding agent is not probable in the light of the restorative technique used.[22] Moreover, these defects are likely to be circumscribed, in contrast to the deficiencies observed. Radiolucencies can easily be related to remaining dental caries, but this disregards the fact that observations were done on restorations that were placed with great care.

In removing a dental caries lesion, no difference can be expected between composite and amalgam restorations. An effect of the material (composite versus amalgam), however, was observed in this study. The distribution of the type of tooth for resin composite restorations is skewed with more premolars restored than molars (data not shown). Premolars show more radiolucencies than molars with respect to resin composite restorations. These findings may be an indication that treatment differences between premolars and molars have an influence on the results rather than the factor material. When preparing a premolar, the dentist may tend to be cautious in the removal of caries in a deep lesion, due to the dimensions of the tooth. The distance to the pulp is relatively small, and exposure of the pulp will thus be avoided. In addition, the box preparation will generally be narrow and shallow. Hence the visibility is less than in the treatment of a molar, and remaining caries will more easily remain undetected.

Voids in the restorative material are attributed to the properties of the material or to technique-specific properties.[23] The possible negative consequences of these may include discoloration, plaque accumulation and the propagation of internal cracks.[24,25] According to Feilzer *et al*, however, porosity may be able to help reduce shrinkage stress in the resin composite.[26] In vitro research, where the material was polymerized under pressure, yielded porosity percentages in the material, varying from 0.01 percent to 6.8 percent, and this can be regarded as material-specific porosity.[23,24] Using extracted teeth, a radiographic investigation of inhomogeneity resulting from the filling technique yielded a figure of 15 percent voids for the bulk technique and 30 percent for layered fillings.[6] The question arises, however, as to which types of porosity are detected by radiographic evaluation in clinical research, and which cannot be detected.

In the present study, 38 percent (Figure 4, minus score 8) of the boxes show voids, and 14 percent of the steps, and this seems to agree reasonably well with the findings described above. It should be borne in mind, however, that where there is uncertainty about a defect, it is recorded as being present. The fact that a difference was found between the three resin composites could be accounted for by differences in stickiness and condensability. Voids can more easily occur during the application and condensation of a "sticky" material.

Clinical research on the cervical marginal adaptation of Class II resin composite restorations yields a figure of 33 percent for defects of boxes filled with a layered

Differences in stickiness and condensability may contribute to the differences in numbers of defects found among the three resin composites.

technique, and 40 percent for a bulk technique.[12] The percentage of cervical marginal defects observed with resin composite restorations in the present study amounts to 28.8 percent. There appears to be a difference between the three resin composites. No plausible explanation can be given for this at the moment.

As regards the presence of poor cervical marginal adaptation of amalgam restorations, the literature reveals a wide variety of figures, varying from 33 percent to 80 percent.[11,27,28] The percentage of 42.3 percent found for amalgam restorations in the present study thus appears to be entirely reasonable, in this respect. In contrast to the findings of Advokaat, the "dentist" factor has no significant effect upon the occurrence of cervical marginal defects with amalgam restorations.[15] This factor does, however, have an influence upon resin composite restorations.

Despite striving to achieve high quality, a fairly high percentage of restorations with cervical marginal defects was found in this study. In this respect, the restorative technique can interfere, because it is not capable of closely following anatomical shapes, and in particular irregular curvature of the proximal surface, so that it is not possible to achieve an ideal cervical profile.

The anatomical shape can also influence the interpretation of the radiographic image.[29] Radiographs represent a three-dimensional object on a flat surface and superimposition of tooth structures can lead to failure to recognize an overhang of a restoration. In this study, some allowance has been made for this problem by evaluating the doubtful cases as deficient. As van Amerongen indicates, the clinical and radiographic data should be placed side by side, therefore, in order to make the picture of the box more complete.[11] This material will be explored in greater depth, in a subsequent article.

CONCLUSION

- ☐ Defects of the cervical margin of newly placed Class II restorations occur fairly frequently, in both amalgam and resin composite restorations.
- ☐ Voids are very frequently found in Class II resin composite restorations.
- ☐ For resin composite restorations, the above deficiencies seem to be affected by the dentist providing the treatment.

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ASTHMA IN CHILDREN OF SMOKING MOTHERS

Our data suggest that children of lower socioeconomic status are at increased risk of developing asthma if their mothers smoke 10 or more cigarettes per day. This finding may have important implications for our understanding of recently described changes in the epidemiology of asthma. Several reports have indicated that prevalence of asthma, hospitalization rates for acute asthma, and asthma mortality in children and young adults increased during the period between 1975 and 1985. Data from the National Center for Health Statistics indicate that increases in morbidity and mortality rates for asthma are particularly important among minorities. A recent study suggested that these differences between ethnic groups are in large part attributable to socioeconomic factors. Studies on the prevalence of smoking in the United States also show that cigarette consumption increased until the mid-1970s and declined steadily since then, but until 1985, smoking initiation rates significantly increased among women with 12 or fewer years of education. Our results are therefore compatible with the hypothesis that the recent increases in prevalence and severity of childhood asthma may be at least in part attributable to an increase in the prevalence of smoking among less educated mothers.

In conclusion, our results suggest that many cases of childhood asthma may be prevented through a sustained effort to discourage smoking initiation and to encourage smoking cessation, particularly among less educated women of childbearing age.

Martinez, F.D. *et al*: Increased incidence
of asthma in children of smoking mothers.
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Diffusion of buffered glutaraldehyde and formocresol from pulpotomized primary teeth

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The formocresol pulpotomy technique has been the procedure of choice for the treatment of cariously exposed vital primary molars.[1-3] The clinical success rate of formocresol pulpotomies has been above the 90.0 percent level.[4-7]

In recent years, scientific evaluation of the medicament has indicated that it is strongly toxic and is distributed systemically.[8-11]

Due to the deficiencies of formocresol, glutaraldehyde, a saturated dialdehyde, has been suggested as a potential pulpotomy medicament. In vitro studies by Nelson and co-workers and Ranly and Lazzari demonstrated its excellent tissue fixative properties.[12,13] Animal studies by Fuks *et al* and Davis *et al*, and human studies by Seow *et al* have all demonstrated its low toxicity and poor potential as a causative agent for inflammatory destruction of pulpal tissue.[14-16]

The pH of the environment has a complex effect on the binding of aldehydes to proteins.[17] In the aqueous form, glutaraldehyde is slightly acidic and weakly antimicrobial. Increasing the pH to 7.5 or more would significantly increase, however, its antimicrobial and fixative activity.[18,19] A recent study showed that buffered glutaraldehyde produced well-circumscribed, relatively inflammation-free lesions in connective tissue of rats.[20] This suggests its high initial molecular protein cross-linking, with minimal or no diffusion of buffered glutaraldehyde into adjacent tissues.

The efficacy of 2 percent w/v buffered glutaraldehyde as a medicament in pulpotomies of carious primary molars has been investigated by Fuks *et al*.[21] They observed a 90.0-94.0 percent success rate.

This study was to investigate the diffusion of buffered glutaraldehyde and formocresol through dentine and cementum of primary teeth treated by pulpotomy.

MATERIALS AND METHOD

The medicaments used in this study were buffered glutaraldehyde and formocresol. The 2 percent w/v buffered glutaraldehyde was prepared from a 25 percent stock solution* by diluting it with a sodium phosphate buffer (pH 9.2). The formocresol used was Buckley's formocresol, diluted with glycerol and water, in a 1:5 ratio.

Thirty freshly extracted primary molars from children between the ages of four to seven years were selected for this study. The teeth were caries-free or with initial caries. More than two-thirds of the roots of all teeth were present. All the teeth were thoroughly cleaned by brushing in running tap water, immediately after extraction.

Access to the pulp chamber was obtained through the occlusal surface using a 330 carbide bur in a high-speed handpiece with water spray. Coronal pulpal tis-

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*Glutaraldialdehydiosung, 25 percent, B. Merck, Darmstardt.

Formocresol diffused out through dentine and cementum within fifteen minutes.

sue was amputated using a sterile sharp spoon excavator. The pulp chamber was then irrigated with normal saline and dried with sterile cotton pellet.

A sterile cotton pellet was then moistened with 2 percent w/v buffered glutaraldehyde or formocresol, placed over the pulp stumps for a period of three minutes and then removed. The access cavity was then sealed with quick setting zinc-oxide eugenol cement. For control, distilled water instead of buffered glutaraldehyde/formocresol was used to moisten the cotton pellet.

Determination of diffusible aldehydes

Each tooth was suspended with the roots immersed in 15 mls of collecting medium (phosphate buffer, pH 9.2) in a plastic bottle at room temperature. At every fifteen-minute interval up to an hour, at every hour interval for the next four hours, and then at every twenty-four hours up to ninety-six hours, 1 ml of the collecting medium was pipetted out into respective test-tubes and analyzed for glutaraldehyde content, using a modification of the method described by 's-Gravenmade *et al.*

To 1 ml of the collecting medium, 1 ml of distilled water was added, followed by 0.5 ml of 0.01 M hydrochloric acid and 0.5 ml of Schiff's reagent. The mixture was mixed thoroughly and left overnight at room temperature before measuring the absorbance spectrophotometrically at 560 nm against a reagent blank. The amount of aldehyde diffusing into the collecting medium was calculated from a standard glutaraldehyde formocresol curve.

RESULTS

There was no diffusion of 2 percent w/v buffered glutaraldehyde through the dentine and cementum into the collecting medium from eight our of eleven pulpotomy-treated primary molars. The collecting medium from three other teeth, however, showed traces of glutaraldehyde, after three hours of incubation. The traces were not within detectable limits.

All the formocresol treated teeth had traceable amounts of formocresol (0.119 ± 0.0138 μgm/ml) in the collecting medium as early as fifteen minutes after the pulpotomy procedure. The concentration of formocresol was observed to gradually increase and reach 4.4780 ± 0.2999 μgm/ml after four hours (Table).

DISCUSSION

Schiff's reagent was used in the quantification of aldehyde released into the collecting medium, because it has been shown to be an effective reagent by other workers, in the quantitative measurements of the diffusion *in vitro* of some aldehydes from root canals of human teeth. The method employed in this study was a modification of the method described by 's-Gravenmade *et al.*[22] The reaction mixture was left overnight at room temperature and this was necessary for the maximum development of color for Schiff's reagent, especially for very low concentration of aldehyde.

The results of this study clearly demonstrate that formocresol diffused out through dentine and cementum within fifteen minutes, following a pulpotomy procedure, while no diffusion of buffered glutaraldehyde was observed.

Absence of diffusion of glutaraldehyde is in accordance with that reported by Dankert *et al* and Kopel *et*

Table ☐ Diffusion of formocresol and buffered gluteraldehyde (mean S.D) as a function of time.

Time (mins)	Formocresol μmg/ml (9)	Glutaraldehyde μmg/ml (11)	Control (10)
15 mins	0.1190 ± 0.0138	N.D	N.D
30 mins	0.2245 ± 0.0119	N.D	N.D
45 mins	0.3259 ± 0.032	N.D	N.D
60 mins	0.4727 ± 0.0820	N.D	N.D
2 hrs	0.8480 ± 0.0820	N.D	N.D
3 hrs	1.4616 ± 0.2654	Traces*	N.D
4 hrs	4.4780 ± 0.2999	Traces*	N.D
5 hrs	4.5876 ± 2543	Traces*	N.D
24 hrs	5.1910 ± 0.4325	Traces*	N.D
48 hrs	5.6473 ± 0.4211	Traces*	N.D
72 hrs	5.8345 ± 0.5321	Traces*	N.D
96 hrs	5.9231 ± 0.2124	Traces*	N.D

Number in parenthesis represented the number of teeth used in the study.
N.D = not detected.
*Observed in three teeth only.

al.[23,24] The exact mechanism underlying this phenomenon is unknown. The possible explanation for the lack of diffusion of 2 percent w/v buffered glutaraldehyde from pulpotomy-treated primary teeth is the pulpal tissue-glutaraldehyde interaction. Glutaraldehyde, being a saturated dialdehyde has a strong ability to combine with amino acids of proteins, to form inter- and intramolecular methylene bridges of macromolecular sizes, thus reducing its diffusion and solubility. It has also been reported that the fixation of bovine pulp by 2 percent and 5 percent glutaraldehyde is greatly enhanced by the alkaline pH.[15]

On the contrary, formocresol's reaction with proteins is slow and its smaller molecular size compared to glutaraldehyde further facilitates its diffusion. s'Gravenmade believes that satisfactory fixation by formocresol requires an excess of the medicament and a long period of interaction.[17] Unfortunately, these two conditions will only enhance the undesirable side effects. Application of excess formocresol will saturate the remaining pulpal tissue, providing a larger amount to diffuse out into surrounding tissues to cause tissue damage. Another disadvantage of formocresol is that most of its reactions are reversible, thus the possibility of recurrence of inflammation in the remaining pulpal tissue.

Slight diffusion of glutaraldehyde was observed by 's-Gravenmade *et al* and Lekka *et al*.[22,25] 's-Gravenmade *et al* measured the diffusion of unbuffered glutaraldehyde through endodontically treated teeth. As such, no pulpal tissue-glutaraldehyde interaction was possible. Leaving the glutaraldehyde soaked cotton pellet on the amputated pulp stumps throughout the study could be the contributing factor for the slight diffusion observed by Lekka *et al* in their study.

CONCLUSION

Results of this study showed that unlike formocresol, 2 percent w/v buffered glutaraldehyde did not diffuse out of the cementum and dentine of pulpotomized primary teeth. This tends to support the use of buffered glutaraldehyde as a medicament in pulpotomy for primary teeth.

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Mandibular fractures in Sri Lankan children: A study of clinical aspects, treatment needs and complications

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Mandibular fractures in children are rare compared with those in adults.[1] Yet they are not less important, for several factors make them somewhat different from those in adults.

With regard to treatment there are disadvantages associated with the treatment of children as well as advantages. It is difficult to make use of the teeth for fixation of fractures. Primary teeth may either be insufficient in number or their roots may be resorbed. Permanent teeth may be incompletely formed. The shape of the primary tooth crown is also not favorable for retention of wires, splints, etc.[2]

On the other hand, mandibular fractures in children heal more rapidly. Furthermore, discrepancies in alignment and occlusion would be corrected by the natural remodelling of bone.[2]

Ankylosis of the temporomandibular joint, causing impairment of function, and damage to the condylar growth center that could result in facial deformity are potentially serious complications.

There is also the question regarding the duration of maxillomandibular fixation. Is it possible to shorten this period without jeopardizing union? Fractures in children heal quickly, and it may be possible, therefore, to reduce the period of maxillomandibular fixation. It may be useful to investigate this aspect.

Sri Lanka has gone through a period of heightened economic activity. Facial injuries seem to have increased during this time. Mandibular fractures in children too may have risen. It may be opportune to inquire into the incidence, the clinical, and treatment aspects and the sequelae of these injuries in Sri Lanka.

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MATERIALS AND METHODS

Thirty-seven children less than eleven years of age, admitted to the Provincial Hospital in Kandy, Sri Lanka, for treatment of mandibular fractures, during the period from January 1973 until December 1984, formed the material for this study. Children over eleven years were not included as they may have passed the mixed dentition stage.[2]

Fractures were diagnosed by the clinical and radiological methods given in the standard texts.[3] Classification of fracture sites was as follows: symphysial, body, angle, ramus, condylar, alveolar, and combinations of these in bilateral fractures.[1]

Management of these patients was conducted in the manner described in the literature, making use of simple and inexpensive methods available in Sri Lanka.[2] All patients were given an antibiotic.

Union of the fracture was ascertained by testing for mobility at the fracture site, and also by getting the patient to open the mouth against moderate pressure, applied at the point of the chin via the operator's palm, and if this caused pain, union was considered to be insufficient.

Patients were reviewed at monthly intervals for a period of six months, and those who had condylar fractures for a further period of twelve to eighteen months, to determine whether facial growth is affected. Func-

tional mobility of the lower jaw was assessed by measuring the interincisal distance with the mouth open to the maximum extent, to which was added the incisal overbite. Maximal mouth opening was determined in an age and sex matched control group of thirty-seven children who had no history of mandibular fractures.

"Student's t Test" was used to compare averages.

RESULTS

The thirty-seven children with mandibular fractures formed 4.5 percent of a total number of 818 patients who had received facial fractures and were admitted to the same hospital during the same period as the subjects.

Twenty-six of the patients were males, and eleven were females; nine (1 percent) were below five years and twenty-eight (3.4 percent) were in the group, six to eleven years of age (Table 1).

Twenty-four of the fractures were unilateral and thirteen were bilateral. Their distribution according to the site is given in Table 2. In seventeen patients (45.9 percent), the condyle was involved. In the tooth bearing area, there were fourteen fractures in the body region, compared to ten at the angle and four in the symphysis. Only three patients (8.1 percent) had alveolar fractures.

Falls from heights had caused eighteen (48.6 percent) of the fractures and seem to be the most common cause; followed by traffic accidents, which accounted for eleven (29.7 percent); and accidents connected with sports, which were responsible for six (16.2 percent) of the fractures (Table 3).

Although condylar fractures are common, complications appear to occur only rarely.

Maxillomandibular fixation using eyelet wiring and arch bars, and acrylic splint on the lower arch fixed with two circumferential wires were the most frequently employed methods of treatment (Table 4). Open reduction with interosseous wiring was necessary in three patients. Nine children could be managed with no fixation; four of them had condylar fractures and the other five had undisplaced hairline fractures in the body.

Table 5 reveals the complications that developed in these children. Infections occurred in two patients.

Table 1 ☐ Age and sex distribution of patients and control group.

	Patients			Control Group		
Age	Male	Female	Totals	Male	Female	Totals
– 5	6	3	9	6	3	9
6 – 11	20	8	28	20	8	28
Totals	26	11	37	26	11	37

Table 2 ☐ Distribution according to fracture site.

Symp.	Body	Angle	Ramus	Condyle	Alveolar	Totals
4	8	5	0	4	3	24
Body + Condyle		Angle + Condyle		Bilateral Condyle		
6		5		2		13

Table 3 ☐ Distribution by cause.

Cause	Number	%
Fall from height	18	48.6
Road traffic acc.	11	29.8
Sports acc.	6	16.2
Miscellaneous	2	5.4
Totals	37	100

Table 4 ☐ Methods of treatment.

Treatment method	Number	Percent
Immobilization with eyelets	13	35.1
Acrylic splint on mandible	12	32.4
Interosseous wiring	3	8.1
No treatment	9	24.4
Totals	37	100

Table 5 ☐ Complications of mandibular fractures in children.

	Number	Percent
Infection	2	5.4
Nonunion	0	0
Malunion	0	0
Ankylosis	1	2.7
Retarded growth		
No complications	34	91.9
Totals	37	100

Table 6 ☐ Average maximal mouth opening three months after treatment.

Method of treatment	Patients Number	Average opening ± SD (in MM)	Control group: Number	Average opening ± SD (in MM)	P
Immobilization	12	38.1 ± 2.96	12	41.8 ± 3.44	>0.05
No immobilization	24	40.3 ± 3.14	24	42.3 ± 3.47	>0.05

Malunion and nonunion were not observed. Ankylosis and retarded facial growth were detected in one patient.

Maximal mouth opening was not significantly reduced in the patients, compared with the control group (Table 6). Of course in this regard the patient with ankylosis was not taken into account.

DISCUSSION

The findings of this study support the view that the frequency of mandibular fractures in children is not high. The incidence reported in the U.K. is 4.81 percent, which is very close to the figure recorded in the present series, 4.5 percent.[4] A somewhat higher incidence of 10 percent has been recorded in the U.S.A.[3]

Mandibular fractures seem to be less common in children less than five years of age than in those over six years. This is in agreement with the findings of previous studies.[4,5] This could be attributed to the fact that the younger children are less active and are lighter in weight and fall, therefore, less frequently and less heavily.

Unilateral fractures recorded a higher proportion in the present series. Combinations of fractures seem to be more common, however, in the western countries.[1] These patterns may be a reflection on the etiological factors that are operative in a particular society.[6]

Involvement of the condyle was observed in 45.9 percent of patients, either alone or in combination with other fractures. Yet only one patient (2.7 percent) had ankylosis of the temporomandibular joint and damage to the growth center. Though condylar fractures are common, complications resulting from them seem to be extremely rare. Walker reported that these complications are usually associated with crush injury of the head of the condyle, which fortunately are rare.

The etiological pattern of mandibular fractures in children in Sri Lanka seems to be different from that experienced in the western countries. In the present series, falls from heights was by far the most common cause and second in order of frequency were traffic accidents. Khosla and Boren were of the opinion that traffic accidents are the number one etiological factor in the U.S.A.[1]

Contrary to general belief that teeth in children cannot be used for fixation, adequate immobilization was achieved with eyelet wiring and arch-bars in a majority of patients (35.1 percent) who needed treatment.

Where immobilization was thought to be undesirable, a prefabricated acrylic splint was used on the mandible. MacLennan had used this method with substantial success.[8] This method is cheap as the appliance is reusable and suitable, therefore, for countries with limited resources.

Interosseous wiring at the lower border was performed only in cases where the fracture was behind the tooth-bearing area at the angle. No difficulties were encountered as the bur holes were placed in a region where there were no teeth. Rowe and Killey and also Khosla and Boren were of the opinion that interosseous wiring could be done in the tooth-bearing areas too.[1,2,]

A shortened period of two weeks of maxillomandibular fixation was attempted and was successful in every

In order to avoid ankylosis, it is important to avoid immobilization of the mandible as far as possible.

instance. Rowe and Killey recommended a three to four-week period of immobilization.[2] Later workers found that a shorter duration could be effective.[9] Present study lends support to this concept. Loss of weight, which may result from treatment, could be something that Third World children can ill afford. Functional impairment following prolonged immobilization has also been reported.[10] Furthermore, evidence of degenerative changes in the joint cartilage has been discovered in experimental animals subjected to jaw immobilization.[11]

The fact that complications of mandibular fractures in children are rare is supported by the findings of this study.

Non-union and malunion were not observed. Infection was seen only in two patients. The excellent blood supply enjoyed by the facial regions and the healing powers of young bone seem to ensure rapid and uncomplicated healing.

Ankylosis of the temporomandibular joint, which is a possible complication in condylar fractures, could be prevented to a great degree by avoiding immobilization of the mandible as far as possible. This was the method adopted in the present series. Open reduction of condylar fractures are seldom indicated. Even displaced condylar heads regain a functional position by a process of bone remodelling.[12]

It is possible that the reduction in maximal mouth opening could be minimized by shortening the duration of immobilization. In the present series immobilization was shortened to two weeks and there was no significant reduction in mouth opening and jaw mobility in these patients. Further controlled research into this aspect would be necessary, however, before conclusions could be drawn.

Undisplaced fractures of the body of the mandible in children could be successfully managed with no fixations. Most of the authors advocate such an approach toward the management of this type of fracture.[2]

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BURNING OF A NEONATE, USING PULSE OXIMETER

Injuries secondary to pulse oximetry have been recently reported due to the incompatibility of wiring between the sensor of one manufacturer and the pulse oximeter of another, pressure necrosis, and overheating due to shattering of the light-emitting diode portion. Caution must be exercised even when using safe, noninvasive monitoring devices such as oxygen saturation monitors. There continues to be a need for true electrical safety to be established and enforced for the manufacturers of pulse oximeters as well as all medical equipment. Pediatricians and neonatologists may be unaware of this "burning" complication of pulse oximeter monitoring as it has been previously published in pediatric literature.

Sobel, D.B. *et al*: Burning of a neonate due to a pulse oximeter: Arterial saturation monitoring.
Pediatrics, 89:154-155, January 1992.

BEHAVIOR

Dental treatment of fearful children using nitrous oxide. Part 2: The parents' point of view

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Extensive research has been conducted on dental anxiety, among children and adults. With adults, some form of self-reporting has generally been used, while for children the various techniques used have included observation of behavior, self-reporting, or reporting done by the parents.[1-9] The studies that involve reports by the parents (generally the mother) indicate that the opinion of the mother about the level of her child's fear during dental treatment shows a positive correlation with the behavior of the child during treatment, and that the child behaves more anxiously if the mother herself is afraid of dental treatment.[7,9,10] This observation is supported by other studies: surveys conducted among adults with dental anxiety reveal that these people can often recall that their own mothers were afraid of the dentist.[10,11] Although a causal link does not necessarily follow from these relationships, it can nevertheless be assumed that parents, or at any rate mothers, are (among the) determinants of their child's dental anxiety.[12]

The studies in which parents are asked to give their opinion about their child's dental anxiety involve children of all age-groups and levels of anxiety. There are no known data concerning the opinions of the parents of children with a defined (high) level of anxiety.[12]

Studies of extreme dental anxiety among children still leave a number of questions unanswered: what ideas do the parents have about the causes of their child's fear? Can they cope with this themselves? Who or what do they regard as being the main cause of this fear, and do they also distinguish other contributing factors? It is assumed that the parents know their child the best, and should be able to give a full picture of the nature, the origins and the way to deal with their child's fear of the dentist.

This study, based on a survey conducted among the parents of a group of highly anxious children, aims to give a preliminary impression of this issue.

MATERIALS AND METHODS

The survey was conducted among the parents of fifty-two children, between six and eleven years of age, who were referred to the department of pediatric dentistry of the Academic Centre for Dentistry in Amsterdam (ACTA), because regular treatment was not possible.[13] The majority were referred by the family dentist (90.2 percent, N = 47), while the rest (9.8 percent, N = 5) found their own way to this department. The survey was given out during the first (introductory) session. The questionnaire was anonymous and consisted of

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thirty-eight statements. Besides some basic information, questions were asked about the child's disposition to anxiety, the causes of dental anxiety, tensions elsewhere, the nature and character of the child, the parents' expectations and how they prepared the child for the treatment. Questions on subjects that were closely related to each other were presented in succession. For each, the respondent could choose a number between 1 and 5, according to the extent of his/her agreement with the statement.

The results were analyzed using SPSS.[14]

RESULTS

Some of the parents did not respond to all the statements when completing the questionnaire. Since this occurred only occasionally, no further investigation of this was made.

The great majority of the respondents were female (78.8 percent, N = 41), with an average age of 33.2 years (s.d. = 5.2) and most had completed a secondary school education (72 percent, N = 36). A minority had completed elementary education only (22 percent, N = 11), while only a few had been enrolled in higher education (6 percent, N = 3).

As can be seen from Table 1, the parent generally reports that the child is very frightened of the dentist, and that the child is also much more frightened of the dentist than of the physician. The scores for the question in which the child's fear of the dentist is compared with the fear felt by other children of the same age, are less distinct, but still clear. Few parents report that they themselves are afraid of the dentist.

Most parents (Table 2) report that their child, although referred because of resistance to treatment, is not nervous by nature, is no more anxious than other children of the same age, and certainly does not suffer from night terrors. Only the question asking whether the child is easily frightened receives a positive answer more often.

In answer to the questions about the cause of the child's fear of the dentist (Table 3), parents most frequently associate this with pain and with (the visit to) the dentist him/herself. The influence of doctors and fear-inducing persons in the environment (brothers and sisters) upon this fear was seen as being small. Furthermore, the child's fear is hardly attributed at all to the fear felt by the parents (the respondents themselves), although some doubt is expressed by many parents.

As can be seen in Table 4, the parents report that the causes of their child's anxious behavior are also not to be found among factors at school or within the family. There are no tensions at home, nor at school, and there are no problems with the upbringing of the child.

In answer to the questions about the character and nature of the child (Table 5), the parents tend to give a fairly standard picture of their children. They report that their children are absolutely average as regards shyness. The children are reasonably independent on average, and possibly somewhat bossy. They are reasonably affectionate, but are certainly not spoiled, nei-

Table 1 ☐ Fear of the dentist. The figures give the number of respondents to each score (from 1 to 5).

	1 Not afraid	2	3	4	5 Greatly afraid
Child afraid of the dentist	2	3	6	10	31
Child more afraid of the dentist than of the doctor?	6	4	8	5	29
Child more afraid of the dentist than other children of same age?	3	5	15	12	16
Parent afraid of the dentist?	23	14	9	2	4

Table 2 ☐ Anxious disposition.

	1 No never	2	3	4	5 Yes always
Child is nervous	24	16	5	2	4
Child is easily frightened	16	15	8	1	10
Child is more frightened than other children of same age	16	12	15	4	4
Child often has nightmares	40	7	1	1	2

Table 3 ☐ Causes of dental anxiety.

	1 Never	2	3	4	5 Always
Afraid of pain	1	9	10	6	24
Bad experiences with dentists	7	8	8	12	16
Bad experiences with doctors	18	8	14	5	6
Parents afraid themselves	9	19	18	2	3
Brothers/sisters make child afraid	24	12	13	2	1

Table 4 ☐ Other causes: does your child have any problems with tensions at home or at school?

	1 Disagree completely	2	3	4	5 Agree completely
Tensions at school	28	13	6	2	2
Tensions at home	31	9	5	2	5
Problems with upbringing	38	9	1	2	1

ther at home nor by other people. They tend to be open rather than closed in their contacts with others, and make friends easily.

As Table 6 shows, the parents have a definite opinion about the success to be expected from the treatment. They do make a gradual distinction here: a small number of the parents wonder whether, after treatment is completed, the child will also be less frightened of the dentist.

When preparing their child for dental treatment (Table 7), the large majority of the parents say that they choose one or a few strategies. The most frequent choice is to explain that the treatment is necessary. Ignoring the fear is by far the least frequently chosen strategy.

None of the solutions chosen appears to them to be clearly better than the others. In their own opinion, the parents are not very successful with their strategies for reducing fear.

In this survey, children appeared more fearful of the dentist than of the physician.

Table 5 ☐ Other causes: description of the nature and character of the child.

My child . . .	1 Disagree completely	2	3	4	5 Agree completely
Gets along well at school	1	3	10	5	32
Is socially skilled	3	3	10	6	30
Is affectionate	4	6	9	9	23
Is independent	2	2	12	18	18
Wants to be the boss	13	10	14	5	10
Is shy by nature	4	8	21	11	8
Is closed by nature	19	6	15	4	7
Is spoiled:					
- at home	25	4	11	4	8
- by others	26	7	7	2	9
Is submissive	35	6	9	1	1

Table 6 ☐ Parents' expectations of treatment at the clinic.

	1 Certainly not	2	3	4	5 Certainly will
Will treatment be successful?	-	1	5	5	40
Will child be less afraid afterwards?	-	4	7	10	28

Table 7 ☐ Parents preparing their child for a visit to the dentist, and the reported effectiveness of these strategies. (In parentheses: the number of parents who chose each strategy. More than one option may have been chosen.)

	1 No effect	2	3	4	5 Strong effect
1. Try to distract the child (37)	11	9	12	2	3
2. Try to calm the child (47)	11	9	17	6	4
3. Explain what is going to happen (44)	11	6	15	9	3
4. Promise something (reward) (38)	12	7	10	4	5
5. Promise to stay with the child (34)	13	3	5	5	8
6. Tell the child that it is necessary (48)	8	14	13	8	5
7. Ignore the fear (8)	3	-	4		1

DISCUSSION

With respect to the composition of the group of respondents, no systematic differences were found between the variables. The composition of the group is not different from those found in other studies.[12]

The parents were referred to the department of pediatric dentistry because their child distressed by dental anxiety could not be treated by the family dentist.[13] It is highly probable then, that they will describe their child as highly anxious (Table 1). The parents do not connect the dental anxiety with their child's general disposition toward anxiety (Table 2), although research shows that this relationship is to be found, both for children and for adults.[9,12,15] The parents consider the pain experienced or anticipated during dental treatment as the source of the child's fear (Table 3). The parents' agreement with the (passively formulated) statement, "My child is easily frightened", possibly indicates that they tend to explain their child's behavior in terms of the circumstances in which it occurs, rather than in terms of other factors centered in the child. Other responses to questions about the nature of the child confirm this first impression. The parents picture a group of normal children, who are not exposed to or have no trouble with other anxiety-provoking factors (Tables 3, 4, and 5).

It becomes highly important that the mother is taught how to prepare her child for a visit to the dentist.

were given here, the possibility remains that a certain degree of bias does play a part: the starting point (the referral) clearly suggests that the cause of the untreatability can be attributed to the dentist; if there is a treatment problem with which the dentist cannot cope, why look for other causes?

The results in Table 7 are predictable. The parents do try to calm their child, but do not succeed sufficiently in doing so. If the family dentist cannot then cope with the problems, referral follows. These results agree with the findings of Bailey.[9] His study reveals that the child's anticipatory fear becomes even greater, if the mother attempts to make him/her less afraid. It is worth mentioning here that the children in his study report that they do appreciate being given information beforehand. Bailey concludes that the element of surprise needs to be taken away from the visit to the dentist. In the case of highly anxious children, it becomes very important that the mother be taught how to prepare her child for the visit to the dentist.

It is surprising that the parents report that they themselves have little or no fear of the dentist. This does not agree with the results of other studies, which reveal that around 25 percent of the population is frightened of the dentist, and 8 percent of these people are so frightened that they avoid regular check-ups.[1,12,15]

A possible explanation is that parents develop a certain level of coping for themselves, based mainly on acknowledging the discomfort of dental treatment (be brave, it has to be done), rather than on actually dealing with the situation (for example by relaxing). In that case, the parents would not be sufficiently able to understand or share in their child's fear effectively. Research shows that dental anxiety in the mother greatly increases the statistical probability of anxious behavior among her very young children.[7,8] This relationship decreases, however, as the child grows older.[9] This last factor in particular correlates well with the results of this study. In addition, a great deal of research is based on retrospective studies: fearful adults report that their parents were also fearful in the past. It is possible that these adults may attribute more anxiety to their parents than they actually experienced.[10-12]

Further research will need to focus upon better cooperation between the dentist and the parents of highly anxious children, in the areas of preparing and treating these children. It is primarily the dentist's responsibility to support the parents by developing practical applications of existing theoretical models for reducing fear.

CONCLUSION

This survey indicates that:

- ☐ Parents of children with a high level of dental anxiety see the dentist or the pain of the treatment as the most important cause of their child's fear.
- ☐ The parents of these children do not themselves recognize any factors that would promote fear.
- ☐ The parents themselves are not sufficiently able to support their child by preparing them for a visit to the dentist.

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ENSURING INCOME SECURITY

Poverty and economic instability take a dreadful toll on children. Children growing up in poor families, especially those living in troubled neighborhoods, suffer the most health and behavioral problems. They have lower levels of literacy and higher rates of school dropout. They experience more hunger, homelessness, and violence. They are more often removed from their parents' care because of abuse or neglect. Disproportionately, they lack the necessary skills and knowledge to get good jobs, and they have fewer job opportunities. If they live in families headed by a single mother or in families dependent on welfare, they will more likely than not repeat the pattern of their parents' lives and continue the cycle of poverty when they reach adulthood.

The emergence of a permanently poor population is destroying the social fabric of this nation. Too many American children born into abject poverty grow up without hope of a decent and secure future. As young people, they are often dispirited, angry, and hostile. As adults, they may be unable to form strong families and contribute to the life of their communities and the nation. Because children in racial and ethnic minorities are disproportionately poor, disproportionately from single-parent families, and disproportionately living in severely troubled neighborhoods, they are at even greater risk of failing to enter the social and economic mainstream.

National Commission on Children: *Beyond rhetoric.*
Washington, D.C.: Government Printing Office, 1991, pp 79, 80.

DEVELOPMENT

Eruption pattern in the primary dentition of premature low-birth-weight children

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The relationship between the eruption pattern of the primary dentition and a child's general growth and development has been studied by several investigators. Even though the earlier studies did not show any relationship, more recent studies demonstrated a strong correlation between these two factors.[1-5] Most of the previous studies were based on limited numbers of subjects or an inadequate statistical analysis that produced contradictory results. Infante and Owens attempted to determine the relation of primary-tooth emergence to several measures of growth.[5] The results of their study indicated that the timing of primary-tooth emergence is significantly related to child growth and perhaps to nutritional status.

In another research, investigators studied the eruption patterns of primary dentitions in 658 children under three years of age.[6] A correlation between general somatic development and eruption of the primary dentition was demonstrated. On the contrary, another study of timing and sequence of eruption of primary teeth in healthy Australian children showed no significant relationship to growth rate or to psychomotor maturity.[7] The study by Tanguay *et al* confirmed the findings of Infante and Owens.[5,8]

A review of the literature shows very limited knowledge regarding the eruption patterns of the primary dentitions of prematurely born children with low birth-weight. Since prematurely-born children have been found to develop more slowly compared with children born full term, this study was undertaken to investigate the primary-tooth eruption-patterns of these children. The hypotheses tested in this study were:

- ☐ Prematurely-born, orally intubated low-birth-weight children demonstrate delayed tooth eruption, when compared with normative data for full-term children.
- ☐ Eruption of primary teeth in prematurely-born children shows a relationship with their general developmental patterns.

METHODS AND MATERIALS

This study was approved by the Institutional Review Board of the University of Illinois at Chicago. A total of thirty-one children, ages fifteen months to five years, were examined for this investigation. All of the participants were premature children, categorized as very-low-birth-weight (1500 grams or less) and low-birth-weight (2500 grams or less) infants who were intubated orally during their neonatal period and were all being

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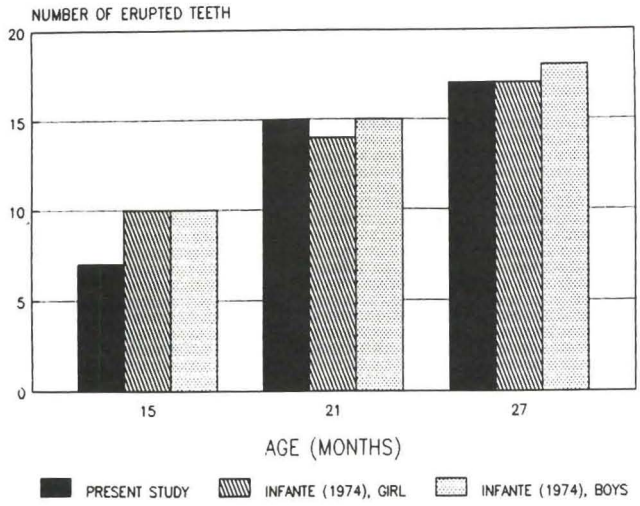

Figure. Eruption pattern of primary teeth (Premature children vs norms).

observed periodically in the Department of Pediatrics of the University of Illinois Hospital in Chicago. Their birth-weight ranged from 680 to 2500 grams and they were intubated for a period of one to seventy-five days. They were evenly distributed by sex (F = 15, M = 16). In order to evaluate the eruption patterns of these children, they were divided among three groups:

Group I consisted of eight children, ages twelve to twenty- four months; *Group II*, fifteen children, ages twenty-five to thirty-six months; and *Group III*, eight others who were more than thirty-six months of age. A clinical oral examination was conducted using mirror and explorer. The number of teeth clinically present was recorded. The methodology and data base from the 1974 Infante study was used as a control in this study.[9] For determining the number of erupted teeth, we used Infante's definition: A tooth was considered "emerged" if any portion of it had pierced the oral mucosa. The total number of teeth present in each group was recorded. The means and standard deviations for the total group by six-month age-intervals were computed separately. Comparison of the means of the erupted teeth and different age-groups was performed, using a t-test. Correlation coefficient was used to analyze the relationship between the period of intubation and the number of teeth present for different age-groups, as well as the relationship between birth weight and the number of erupted teeth. The average numbers of erupted teeth for different age-groups was plotted and compared with those for normal full-term children of respective age-groups, as reported by Infante.

RESULTS

Out of eight children in the first group (twelve to twenty-four months), four boys and two girls showed delayed tooth-eruption (75 percent). In Group II (twenty-five to thirty-six months), out of fifteen children, one girl (6.6 percent) showed delayed tooth eruption. In Group III (older than thirty-six months), all eight children had normal eruption-patterns and no one showed signs of delay (Table).

Table ☐ Distribution of delayed tooth eruption in different age-groups.

Age (m)	Total no. of subjects	Subjects with delayed tooth eruption		Percent of delayed tooth eruption
		Male	Female	
12 - 24	8	4	2	75
25 - 36	15	0	1	6.6
> 36	8	0	0	0

Eruption of the primary dentition was more closely related to the child's growth in height than to her chronological age.

Compared with normative data, the prematurely born children under twenty-four months of age on the average showed 28 percent fewer erupted teeth; whereas those older showed compatible eruption-patterns with those of full-term children (Figure).

When the eruption of primary teeth was evaluated, based on the birth weight or the intubation period, no correlation was observed.

DISCUSSION

The findings of this study indicated a delay of 75 percent in the eruption of the primary dentitions in premature children under twenty-four months of age. This was consistent with another study evaluating the oral development in children with very low birth-weight.[10] In that study the investigators showed delay in dental eruption, which later corrected during the second year of development.

After reviewing their medical charts, it was found that the participants in this study also had delays in their general developmental pattern. When their growth curves were studied, those under twenty-four months of age demonstrated a consistent delay in weight, height, head circumference, and fine motor skills, indicating that eruption of the primary dentition in premature children follows the same pattern as the general developmental pattern. This finding was confirmed by two other studies, which showed that even in healthy full-term children, the eruption of the primary dentition was more closely related to the child's growth in height, rather than chronological age.[5,8]

The fact that we were not able to determine any relationship between the intubation factor and eruption pattern led us to believe that the delayed tooth eruption is more likely a developmental problem, consistent with prematurity, rather than the effects of local trauma such as oral intubation. Parents of prematurely-born children should be informed about the delayed eruption pattern of the primary dentition and advised that a normal eruption pattern usually remains after two years of age.

CONCLUSION

Low-birth-weight, prematurely-born children show a delayed tooth- eruption pattern in their first twenty-four months of life. Following that period, they appear to "catch up" and have comparable norms for the corresponding age-group.

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EFFECT OF VERY LOW BIRTH-WEIGHT AND SUBNORMAL HEAD SIZE ON COGNITIVE ABILITIES AT SCHOOL AGE

The results of the longitudinal follow-up of 249 children with very low birth weights, who were born from 1977 to 1979, confirm our hypothesis that perinatal failure of brain growth, as evidenced by a subnormal head circumference, has serious long-term implications, if catch-up growth does not occur by eight months of corrected age. Furthermore, such catch-up growth only occurs during the first year of life and shifts in head-circumference percentiles after that time are usually minimal.

Hack, M. *et al*: Effect of very low birth weight and subnormal head size on cognitive abilities at school age. N Engl J Med., 325:231-237, July 25, 1991.

DEMOGRAPHY

There really are a lot of poor children in the United States

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

I don't know about you, but I never realized how pervasive poverty is amongst children in our country. I have seen the television documentaries on the horrors of poverty and starvation in various countries throughout the world. Over the years, I have travelled to other countries, taking the obligatory tourist photographs of poverty in Africa, Asia, and South America. (It seems that someone else's poverty tends to be quite photogenic— whether done by professionals or amateurs. Have you ever noted how many critics' awards are granted to black and white pictures of near total deprivation when taken at some particular angle with just the right lighting?) I have even written an article asking whether our concern for the aged has overshadowed the needs of children.[1]

But as our news media increasingly address the extent of an economic recession in the early 1990s, (and fill the evening TV news and the morning newspaper with the consequences of such an event on the already economically devastated segments of our society) I have come to realize the extent of poverty in the ranks of the children in our nation.

Surely, there are few groups with more concern for the well being of our children than pediatric dentists. I thought you would want to know some of the grim facts.

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THERE CONTINUE TO BE MANY POOR PEOPLE IN THIS COUNTRY

During the last twenty years, the number of people below the poverty line has continued to rise from 25 to more than 33 million residents (an increase of from 12.6 percent to 14 percent of the population). But while approximately 10 percent of the white population has been below the poverty line during the past twenty years, during this same period, almost a third of the black and more than a quarter of the Hispanic communities have had incomes that were below the federally defined poverty line.

The poverty line provides a standard financial figure for the minimal subsistence of a family (the figure varies for families of different sizes) in a particular year and residential locale (i.e. urban vs rural). In addition, families living just above this minimum standard are considered "near poor." During the past twenty years, approximately 18 percent of our national population (between 35 and 44 million people) has lived in "poor" or "near poor" economic conditions (Table 1).

THE POOR BY AGE

Many of the aged are experiencing economic difficulties; particularly aged women who do not live in a family setting.[1] But on a numerical and percent basis, far greater are numbers of children living in conditions of poverty than in the case of the elderly. Between 1970 and the 1980s, the number and percent of the elderly poor decreased.[1] During the same period, the number

and percent of poor children increased (Table 2). By 1988, 11.6 million children (20.4 percent of all children) were living in conditions of poverty. The poverty rate for children was double and almost triple the rate for individuals in many of the other age-groups. And even more devastating were the poverty rates for black and Hispanic children. More than 45 percent of black children, and 39 percent of Hispanic children (compared to 15 percent of white children) lived in conditions of poverty (Table 3).

LIVING ARRANGEMENTS

Children who live in poor or near poor economic conditions are not distributed uniformly in various family arrangements. For example, in 1987:

- ☐ Fifteen percent of white children, and 45 percent of black children, living in a male household (includes families with and without female spouses present) lived in poor economic conditions. Fifty-five percent of all children living in a female household (no males present) lived in poor economic conditions.
- ☐ Twenty percent of white children, and 53 percent of black children living in a male household lived in poor or near poor economic conditions. Sixty-two percent of all children living in a female household lived in poor or near poor economic conditions (Table 4).

And economic conditions in female households continue to deteriorate. For example:

- ☐ The median constant dollar incomes (i.e. removing the effects of inflation) of white, black and Hispanic female-led families have decreased throughout the 1980s.
- ☐ In 1987, the median family incomes of black and Hispanic female households were significantly less than that of all black and all Hispanic households

Table 1 ☐ Persons below the poverty line and below 125 percent of poverty level by race, Hispanic origin and year.[2]

	Below poverty line				Below 125% of poverty line
	Number (in millions)				
Year	All races*	White	Black	HispanicII	All races
1970	25.4	17.5	7.5	na	35.6
1975	25.9	17.8	7.5	3.0	37.2
1980	29.3	19.7	8.6	3.5	40.7
1985	33.1	22.9	8.9	5.2	44.2
1988#	31.9	20.8	9.4	5.4	42.6
	Percent				
1970	12.6	9.9	33.5	na	17.6
1975	12.3	9.7	31.5	26.9	17.6
1980	13.0	10.2	32.5	25.7	18.1
1985	14.0	11.4	31.3	29.0	18.7
1988#	13.1	10.1	31.6	26.8	17.5

* Includes other races not shown separately.
** Hispanic persons may be of any race.
# Based upon revised processing procedures; data not directly comparable with earlier years.

Table 2 ☐ Children below poverty level by race and Hispanic Origin: 1970-1987.[2]

	Number below poverty level (in millions)				Percent below poverty level			
Year	All races*	White	Black	Hispanic	All races*	White	Black	Hispanic
1970	10.2	6.1	3.9	na	14.9	10.5	41.5	na
1975	10.8	6.7	3.9	1.6	16.8	12.5	41.4	33.1
1980	11.1	6.8	3.9	1.7	17.9	13.4	42.1	33.0
1985	12.5	7.8	4.1	2.5	20.1	15.6	43.1	39.6
1987	12.4	7.6	4.3	2.6	20.0	15.0	45.1	39.3

*Includes other races not shown separately.

Table 3 ☐ Persons below the poverty level by race, Hispanic origin, and age: 1988.[2]

	Number below poverty level (in millions)				Percent below poverty level			
Age	All races*	White	Black	Hispanic	All races*	White	Black	Hispanic
< 16	11.6	6.9	3.9	2.5	20.4%	15.2%	45.4%	38.9%
16-21	3.3%	2.0	1.0	.6	15.7	11.8	34.0	24.4
22-44	9.5	6.4	2.6	1.6	10.5	8.4	23.6	20.6
45-54	1.9	1.3	.5	.3	7.7	6.0	19.9	17.1
55-59	1.0	.7	.3	.1	9.6	7.7	25.4	18.9
60-64	1.1	.8	.3	.1	10.4	8.7	25.3	19.1
65+	3.5	2.6	.8	.2	12.0	10.0	32.2	22.4
Totals	31.9	20.8	9.4	5.4	13.1%	10.1%	31.6%	26.8%

*Includes other races not shown separately.

Table 4 ☐ Children in families below the poverty line and below 125 percent of poverty level by race of head of the household, family status and year.[2]

	Below poverty level			Below 125% of poverty level		
	Numbers (in millions)					
	1979	1985	1987	1979	1985	1987
Children under 18 in families with:						
Male householder*						
White	5.9	7.8	7.6	8.2	10.3	9.9
Black	3.7	4.1	4.3	4.7	4.8	5.0
Female householder	5.6	6.7	7.1	6.7	7.6	8.0
Other families**	4.4	5.8	5.4	6.6	8.2	7.6
Children under 18 in families with:	Percent					
Male householder*						
White	11.4	15.6	15.0	15.9	20.5	19.6
Black	40.8	43.1	45.1	51.1	51.0	52.8
Female householder	48.6	53.6	54.7	58.0	60.7	61.9
Other families**	8.5	11.7	10.9	13.0	16.5	15.4

*Includes families with both spouses present and families with male householder with no spouse present.
**Includes races and members of unrelated subfamilies not listed separately.

In cities, the percent of children in poverty is 30

(52 percent and 43 percent, of the respective black and Hispanic households) (Table 5).

Unfortunately, the difficulties faced by female households are becoming an increasingly substantial dilemma. By 1988, there were 13.5 million children living only with their mothers (4.3 million children with a mother who had never married). Between 1970 and 1988, there was a 678 percent increase in the number of children living with a mother who had never been married (Table 6).

Not only is the average income of female households with children far less than that of the average income for all families with children, but in 1987, more than three quarters of white, black and Hispanic women, who never married and have children in their households, had incomes below ten thousand dollars (Table 7).

EVEN MORE BAD NEWS

While more than 20 percent of children in this nation live in poverty, in cities, the figure is 30 percent.[4] In addition:

- ☐ Nearly one of every four children under age six is poor; higher than that of any other age-group.[5]
- ☐ One of every four rural children is poor and rural families are less likely than urban ones to receive public assistance.
- ☐ Malnutrition affects nearly a half million children and 100,000 are homeless.[6]

The dire consequences of this continuing horror of poverty surely must pervade the future of each of these children. As practitioners concerned with the well-being of our children, I thought you would want to know some of these grim facts. "Never before has one generation of American teenagers been less healthy, less cared for or less prepared for life than their parents were at the same age."[4]

Table 5 ☐ Median income of families with children by family type, race and Hispanic origin: 1975, 1985, 1987.[3]

	Current dollars			Constant dollars		
	(in thousands)					
	1979	1985	1987	1979	1985	1987
All families						
White	$21.1	$28.9	$32.2	$32.9	$30.6	$32.2
Black	10.7	14.9	15.0	16.7	15.7	15.0
Hispanic	14.0	17.0	17.9	22.0	17.9	17.9
Female-headed families						
White	$ 9.0	$11.3	$11.7	$14.2	$11.9	$11.7
Black	6.6	7.3	7.8	10.3	7.7	7.8
Hispanic	5.9	7.4	7.7	9.2	7.8	7.7

Table 6 ☐ Number of children under eighteen living with only their mothers: 1970, 1980, 1988.[3]

Child lives with mother who is:	1970	1980	1988	Percent change 1970–1988
		(in millions)		
Divorced	2.3	4.8	5.0	119%
Separated	2.3	3.0	2.9	24
Never married	.6	1.8	4.3	678
Widowed	1.4	1.3	.8	−39
Total of children living with only their mother	7.5	11.4	13.5	81

Table 7 ☐ Distribution of income of families with children by family type, race and Hispanic origin: 1987.[3]

	All children	Mother only never married
White		
<$10,000	13.5%	75.6%
$10,000–$19,999	16.0	17.6
$20,000–$29,999	17.6	4.0
$30,000–$49,999	31.1	2.2
$50,000+	21.8	.5
Mean income	$35,953	$7,829
Black		
<$10,000	43.9%	81.2%
$10,000–$19,999	21.7	14.2
$20,000–$29,999	12.6	3.1
$30,000–$49,999	15.3	1.5
$50,000+	6.5	.0
Mean income	$18,500	$6,596
Hispanic		
<$10,000	30.7%	79.7%
$10,000–$19,999	27.1	13.8
$20,000–$29,999	16.7	3.3
$30,000–$49,999	17.6	2.7
$50,000+	8.0	.3
Mean income	$21,921	$7,540

Note: Children in families with "mother only" and "mother only—divorced with spouse absent" have family mean incomes that are a quarter to a third the mean incomes of families with "both parents present."

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Do the parent(s) of your pediatric patients smoke?

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

When a dental practitioner provides services to a youngster, surely the least of the problems to be faced is whether the child's parent(s) smoke cigarettes. Similarly, when providing care to young teenagers, does the dentist really have to be concerned about their smoking habits? At a time when 20 percent of the children live in families with incomes below the poverty line, 5.5 million children under twelve years of age skip meals due to lack of money, and there is a resurgence in the numbers of children who are not properly immunized against common childhood diseases, the consequences of smoking by children and by the parents of our patients seem almost innocuous.[1-3] In addition, have there not been dramatic decreases in the numbers and percent of the population that smoke?

Practitioners do not really need to be concerned, if we believe that the areas of dentistry (particularly pediatric dentistry) limit our concerns to teeth, their decay and alignment, their osseous and periodontal support, and the general condition of the oral cavity.

Yet, if for no other reason than economics, it makes good business sense to create a practice environment that stresses the preventive aspects of total health care. "...pediatric dentists who have been able to create and sustain a perceived need for health maintenance have lucrative recall programs."[4]

But aside from the mercenary aspects, the continuing emphasis on general health status for the pediatric patient and the entire family fulfills the general obligation of all health care professionals. "In performing these activities, these pediatric dentists are fulfilling the true meaning of 'doctor'—a teacher."[4]

SOME REALITIES OF SMOKING

The reality is that in 1990, 32 percent of the population between eighteen and twenty-five years of age continued to smoke (28.7 percent of the total population, eighteen years and over).[5] In addition, cigarette smoking has been identified as the single most avoidable cause of death in the United States. "Some 400,000 lives are lost each year from lung cancer, cardiovascular disease and chronic obstructive lung disease."[5]

"...strong evidence exists that exposed infants and children (to cigarette smoke) are at higher risk for specific health and developmental problems."[6] The effects of environmental exposure to cigarette smoke for preschool children "...are primarily the result of two phenomena: The mother's prenatal smoking practices and the child's subsequent family environment."[7]

Maternal smoking during pregnancy increases the rise of adverse prenatal consequences, including intrauterine growth retardation, low birth-weight, and preterm delivery. "Recent estimates suggest that elimination of smoking during pregnancy could reduce about 5 percent of perinatal deaths."[7] As compared to children

*It seems appropriate that I wrote this material on the first day that smoking, chewing or even possessing tobacco is illegal in the State of Iowa for anyone under 18 years of age, and that the teenager interviewed for the article (reported the day before the law went into force) was smoking in the nonsmoking section at a mall in Iowa.[11]

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whose mothers did not smoke, morbidity in children in the preschool years has been found to be higher for children whose mothers smoked during pregnancy and postnatally, including increases in respiratory tract and middle-ear infections, febrile seizures, and behavioral and cognitive abnormalities affecting mental development.[7]

There are many differences throughout the country in the demographic characteristics of smokers and the quantity of cigarettes smoked each day. For example:

- ☐ Compared to college graduates, about twice as many women between eighteen and forty-four years of age with less than a high school education smoke (15 percent compared to 31 percent).
- ☐ Forty-one percent of those smoking during their childbearing years smoke about a pack a day.
- ☐ Eleven percent of women between eighteen and twenty-four years and 21 percent of women between twenty-five and forty-four years smoke more than a pack a day.[7]

But the overall reality is that the onset of smoking for females is occurring at younger ages.[8]

If you claim that your dental practice is "prevention oriented," the effects of smoking should be an integral part of your office education activity. And this program should not be just for your particular pediatric patient, but for the younger and older children at home (80 percent of families with children have more than one child [Table 1]).

Table 1 ☐ Children in a household living with one or both parents: 1990.[9]

Number of children per household	Number of households	Percent
	(in thousands)	
One	12,007	19.2
Two	25,227	40.4
Three	15,495	24.8
Four	5,837	9.4
Five	2,222	3.6
Six or more	1,581	2.5
Totals	62,370	100.0

I HAVE *NO SMOKING* SIGNS IN THE OFFICE

It is easy to assume that for many many years most everyone has been aware of the dire consequences of smoking. Yet, in a study in the early 1980s:

- ☐ Twenty-four percent of heavy smokers did not know that smoking is dangerous.
- ☐ Forty-one percent did not know that smoking will shorten their life expectancy.
- ☐ Fifty percent did not know that smoking causes heart attacks.
- ☐ Fifty percent of teenagers did not know that smoking is addictive.[10]

Despite your *NO SMOKING* signs, nonsmoking sections in many restaurants and places of business, and the July 1, 1991 antismoking law in the State of Iowa,* based upon the findings of the 1991 National Center for Health Statistics report on smoking, young children, undoubtedly, will continue to be subjected to varying degrees of cigarette smoke. For example, in 1988, among children five years of age and younger:

- ☐ Sixteen percent of them had been exposed prenatally and postnatally to sidestream smoke in the household.
- ☐ Fifty percent of white and 60 percent of black children were exposed to smoking.
- ☐ A greater percent of non-Hispanic than Hispanic children were exposed to smoke; Mexican American children were least likely to be exposed to smoke.

Adverse effects of smoking on all members of a family are real.

Table 2 ☐ Distribution of children five years of age and under by exposure to smoking, and selected characteristics: 1988.[7]

	Percent never exposed to smoking*	Percent exposed to smoking*
All children	49.9	50.1
Race		
White	50.7	49.3
Black	40.3	59.7
Hispanic origin		
Non-Hispanic	49.2	50.8
Hispanic	55.7	44.3
Mexican-American	60.2	39.8
Family income		
Less than $10,000	32.3	67.7
$10,000–$24,999	43.4	56.6
$25,000–$39,999	54.9	45.1
$40,000 or more	64.0	36.0
Poverty status		
In poverty	35.3	64.7
Not in poverty	53.5	46.5
Mother's Education		
Less than 12 years	32.9	67.1
12 years	43.2	56.8
More than 12 years	65.0	35.0
Place of residence		
Metro. stat. area	50.3	49.7
Central city	48.0	52.0
Not central city	51.9	48.1
Not metro. stat. area	48.5	51.5
Assessed health status		
Excellent, very good, good	50.3	49.7
Fair or poor	37.1	62.9

*Includes prenatal and/or postnatal.
Note: Excludes children whose exposure is unknown.

Table 3 ☐ Distribution of children five years of age and under assessed in fair or poor health by exposure to smoking and selected characteristics.[7]

	Percent never exposed to smoking	Percent current smoker in household
All children	2.4	4.1
Race		
White	2.2	4.0
Black	3.1	5.0
Hispanic origin		
Non-Hispanic	2.1	4.0
Hispanic	4.1	5.3
Mexican-American	5.5	5.4
Family income		
Less than, $10,000	4.5	8.0
$10,000–$24,999	2.7	4.6
$25,000–$39,999	2.4	3.3
$40,000 or more	1.1	2.0
Poverty status		
In poverty	3.5	8.0
Not in poverty	2.0	3.2
Mother's Education		
Less than 12 years	3.3	7.3
12 years	2.7	3.6
More than 12 years	1.8	1.9
Place of residence		
Metro. stat. area	1.9	4.1
Central city	1.4	3.9
Not central city	2.2	4.2
Not metro. stat. area	4.0	4.4

- ☐ Almost two thirds of children living below the poverty level were exposed to smoking; compared to 46 percent not in poverty.
- ☐ Two thirds of children with mothers whose education was less than twelve years were exposed to smoking; compared to 35 percent for those with mothers with more than twelve years of schooling.
- ☐ For those children whose health status was reported to be fair or poor, 63 percent were exposed to smoke (Table 2).
- ☐ The relative risk of fair or poor health was almost twice as great for children who live in households with current smokers, as it was for children who were never exposed to smoke (4.1 percent vs. 2.4 percent). This approximate ratio also was observed between the estimates for most of the demographic subgroups (Table 3).

PEDIATRIC DENTIST'S OPTIONS

It would be easy to ignore the subject. Pediatric dentists take care of kids; don't they? But in actuality, dentists (all dentists) take care of families. The adverse effects of smoking on all members of a family are real. We have the ideal opportunity in our programs of preventive health services to include the impact of smoke on children.

What have we got to lose? We might help reduce the estimated one billion packs of cigarettes sold annually in the United States to children under eighteen years of age.[11] We might even save some child and parent from the lament, "if only we had known."

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Children without health-care coverage

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

During 1988, more than half of total gross receipts was paid to independent dentists* through private insurance (48.2 percent) and government programs (4.9 percent).[1] In 1989, 45.1 percent of the total national personal dental expenditures was provided by third parties.[2] An extensive series of reports has linked the increasing use of preventive and general dental services by children and adults to the availability of third-party programs.[3-5] In particular, an earlier report in the Journal of Dentistry for Children noted that:

- ☐ A somewhat higher percentage of male children than female children had dental insurance coverage.
- ☐ Minority children had less insurance coverage than their nonminority counterparts. Black children had the lowest rate of insurance coverage.
- ☐ Children in Metropolitan Statistical Areas (MSAs) had a higher rate of insurance than children in non-MSAs.
- ☐ Children in the West had the highest rate of coverage. Children in the South had the lowest.
- ☐ Children in higher income families had much greater rates of insurance coverage than children in lower income families (Table 1).[3]

The increasing concern over the availability of dental and general health-care coverage has been emphasized in the general press and most recently in the 1991 "Advance Data" report by the National Center for Health Statistics and the report by the National Commission on Children.[6,7] In 1989, 13.9 percent (an estimated 33.9 million persons) in the civilian noninstitutionalized population were reported to lack health-care coverage (including approximately 9.5 million children). Children account for 28.7 percent of all persons without health coverage.[8] (Note: throughout this presen-

Table 1 ☐ Percent of children with dental insurance by various demographic characteristics: 1986.[5]

		Age-group 2-4 yrs.	5-11 yrs.	12-17 yrs.
Gender				
	Male	40.3	43.8	44.0
	Female	40.0	41.7	42.2
Race				
	White	42.6	45.6	45.6
	Black	26.1	28.3	30.2
	Hispanic	33.7	30.7	35.1
Family income				
	Less than $10,000	6.8	7.0	10.6
	$10,000–$19,999	29.5	28.0	28.3
	$20,000–$34,999	52.6	53.7	50.5
	$35,000 or more	61.7	64.0	63.0
Region				
	Northeast	40.6	44.5	45.9
	Midwest	43.2	47.6	49.8
	South	33.0	35.0	34.3
	West	48.2	48.4	48.0
Place of residence				
	Metro. stat. area	44.0	45.4	46.1
	Central city	38.3	39.1	37.8
	Not central city	47.6	49.2	50.9
	Not metro. stat. area	27.7	34.4	34.2

*An independent dentist owns or shares in the ownership of a dental practice. This ownership status includes sole proprietorships, partners, and those who are shareholders in an incorporated practice.[1]

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tation, "health-care coverage" includes private health insurance, Medicare, public assistance [overwhelmingly Medicaid] and military and Veterans' benefits.)

The services provided by pediatric dentists are but one component of the total spectrum of health services required by youngsters. The dental care of children often is carried out in concert with needed general health services. Thus, there is a need for the dental practitioner to maintain a general awareness of the extent of health coverage and the potential for the use of needed services. To this end, the following presentation will review the available data on health-care coverage.

THE GENERAL POPULATION

The results from the National Health Interview Survey indicate that noncoverage was relatively higher for 1) young persons, 2) males, 3) persons who are nonwhite, 4) those with low incomes, 5) persons eighteen years of age and over who are unemployed or had less education, 6) residents of the South and West regions of the country, and 7) residents of central cities in metropolitan statistical areas (Table 2).

Children

Overall health-care coverage. In 1989, 15 percent of all children less than eighteen years of age had no health-care coverage. Almost a third of children in families with incomes between $5,000 and $19,999 had no health-care coverage. Children who are nonwhite, residents of the South and West regions of the nation, and central cities in metropolitan statistical areas have less coverage than their respective counterparts (Table 3). (Note:

Table 2 ☐ Percent of persons less than sixty-five years of age with no health insurance coverage: 1989.[6]

		With no health-care coverage
*All persons**		15.7%
Gender		
	Male	16.7
	Female	14.6
Race		
	White	14.5
	Black	21.9
	Other	20.4
Family income		
	Less than $5,000	31.3
	$5,000–$9,999	36.9
	$10,000–$19,999	30.1
	$20,000–$34,999	11.6
	$35,000–$49,999	6.0
	$50,000 or more	3.7
Poverty status		
	In poverty	36.0
	Not in poverty	11.5
Employment status		
	Currently employed	14.3
	Unemployed	39.2
	Not in labor force	18.5
Education		
	Less than 12 years	30.1
	12 years	16.6
	More than 12 years	9.2
Region		
	Northeast	11.0
	Midwest	10.8
	South	19.7
	West	18.9
Place of residence		
	Metro. stat. area	15.3
	Central city	19.4
	Not central city	12.7
	Not metro. stat. area	17.1

*As a result of the Medicare program, only 1.2 percent of persons 65 years and over did not have health-care coverage.

Note: Percents exclude persons for whom coverage status was not determined.

Private coverage increased directly with increasing family income.

Table 3 ☐ Percent of children less than eighteen years of age with no health insurance coverage: 1989.[6]

		With no health care coverage
All children		14.9%
Gender		
	Male	15.1
	Female	14.7
Race		
	White	14.0
	Black	18.9
	Other	18.9
Family income		
	Less than $5,000	25.5
	$5,000–$9,999	31.6
	$10,000–$19,999	30.2
	$20,000–$34,999	10.9
	$35,000–$49,999	4.0
	$50,000 or more	2.3
Poverty status		
	In poverty	32.5
	Not in poverty	9.6
Region		
	Northeast	9.9
	Midwest	8.8
	South	20.5
	West	16.7
Place of residence		
	Metro. stat. area	14.4
	Central city	18.2
	Not central city	12.1
	Not metro. stat. area	16.5

Note: Percents exclude persons for whom coverage status was not determined

Table 4 ☐ Percent of children less than eighteen years of age with private health insurance, or public assistance coverage by various demographic characteristics: 1989.[6]

		Percent with private health coverage	Percent with public assistance coverage
All children		71.8	11.1
Gender			
	Male	71.9	10.8
	Female	71.7	11.3
Race			
	White	76.3	7.6
	Black	51.6	26.9
	Other	58.2	19.9
Family income			
	Less than $5,000	15.0	60.9
	$5,000–$9,999	19.5	50.3
	$10,000–$19,999	51.3	15.1
	$20,000–$34,999	82.9	3.6
	$35,000–$49,999	92.8	1.7
	$50,000 or more	96.5	0.6
Poverty status			
	In poverty	21.4	46.3
	Not in poverty	85.0	3.3
Region			
	Northeast	78.9	9.2
	Midwest	79.1	12.2
	South	65.9	10.1
	West	67.6	12.9
Place of residence			
	Metro. stat. area	72.1	11.2
	Central city	60.1	18.7
	Not central city	79.7	6.4
	Not metro. stat. area	70.8	10.8

Note: Percents exclude persons for whom coverage status was not determined.

children in the Western region of the nation had the highest rate of dental coverage [Table 1]).

Private health coverage. Almost 72 percent of children had private health care coverage. White children had the highest percentage of private coverage (76 percent); black children the lowest (51 percent). Children residing in the Northeast, Midwest and non-central city areas of metropolitan statistical areas had the highest rate of private coverage. Private coverage increased directly with increasing family income (from 15 percent for families with incomes below $5,000 to 96 percent for families with incomes of $50,000 or more (Table 4).

Public assistance coverage. Eleven percent of children had public assistance coverage (primarily Medicaid). Public assistance coverage was higher for 1) persons who are nonwhite, 2) those with low incomes, and 3) residents of central cities in metropolitan statistical areas (Table 4).

Trends and future directions

Overall, the percent of persons under 65 years of age with no health-care coverage increased from 14.6 percent in 1984 to 15.7 percent in 1989. The largest increases were for individuals in families with incomes in the range of $10,000 to $49,999.[6]

"'Today, children are the poorest Americans.'"[9] That's the finding issued by the National Commission on Children. The Commission estimates that its proposals to combat children's poverty and to guarantee access health care for all children would cost more than $50 billion a year. The majority of the Commission would require employers to provide health insurance for employees and their children or to pay a new tax to finance coverage under a public program. But members of Congress and political consultants are asking, in an era of fiscal difficulties, "are the proposals for children politically realistic?"[7] Unfortunately, the reality is that children don't vote—and election "is the name of the game!" Yet, as we know all too well, the long-term consequences of poverty and neglected health-care for our children can and do have deleterious (and expensive!) consequences throughout the lives of these youngsters.

A major objective of the American Academy of Pediatric Dentistry for the year 2000 is improving access to dental/oral health care for those now deprived of

needed services.[10] The reality is that improving access to dental care is a function of improving the overall access to health care. To some extent, dental services for children may increase as a result of the federal mandate for the state Medicaid programs to include all children below the federal poverty level. (Dental care for children are required services under the Early and Periodic Screening, Diagnosis and Treatment components of state Medicaid programs.)

But there are almost five million children in families, with incomes that are just above the Medicaid eligibility levels, that have no health-care coverage (let alone, dental care coverage)! If efforts by pediatric dentists to extend services to increasing segments of the population are to succeed, they must be coupled with the movement to improve general health-care coverage. As the Congress increasingly turns its attention to the realities that, 1) thirty-four million people in our country do not have health care coverage and 2) "children are the poorest Americans," pediatric practitioners should be positioned to make the necessary case for needed dental care for children. Are you ready for the challenge?

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INCOME SECURITY FOR FAMILIES WITH CHILDREN

A basic step toward ensuring that American children have the opportunity to become healthy, literate, and productive adults is ensuring that they are born into and grow up in families with a decent income. If our society is committed to supporting families as the basic institution for rearing children, and if all children are to have an opportunity to develop to their full potential, then it is necessary for families to be more economically secure. While effective programs to combat many of the devastating effects of poverty and economic instability will continue to be needed for some time, ensuring families an adequate income will significantly reduce economic deprivation. Over time, an adequate income would minimize the need for many specialized subsidies. Moreover, it would directly increase parents' capacity to provide for their children's material needs. It would allow them greater freedom to make basic decisions about how they live their lives and raise their children. And it would ensure that both parents, not just one, maintain financial responsibility for their children's upbringing.

National Commission on Children: *Beyond Rhetoric.*
Washington, D.C.: Government Printing Office, 1991, p 93.

EPIDEMIOLOGY

Distribution of missing teeth and tooth morphology in patients with oligodontia

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Oligodontia is the term used to define the condition in which six or more teeth are congenitally missing, excluding the third molars. Oligodontia can occur alone or as part of a syndrome. These two forms will be referred to as oligodontia/I (isolated oligodontia) and oligodontia/S (oligodontia as part of a syndrome). Apart from the dental symptoms, patients with oligodontia/S have abnormalities of the skin, ears, eyes and skeleton.[1] The dental symptoms occurring in oligodontia are a reduced number of teeth; delayed eruption; abnormalities of the enamel; and abnormalities in tooth morphology, such as reduction in tooth form and tooth size.

The morphology of the teeth is influenced by genetic factors and environmental factors. It is known that reduction in tooth size is associated with oligodontia.[2-5] This reduction is sometimes seen, therefore, as a genetic expression of the same disorder, suggesting that oligodontia is not an isolated phenomenon.[6,7]

Oligodontia is a phenomenon with which every pedodontist can be confronted. It is important for a dentist to be familiar, therefore, with the characteristic symptoms of oligodontia. This enables an early diagnosis. Since the dentist and family doctor should be able to inform the patients about the character and severity of their disorder, detailed information must be available concerning all symptoms of both groups of patients.

The aim of the present study was to examine the distribution of missing teeth, establish the reduction in tooth morphology, and examine eventual differences between the patients with oligodontia/I and oligodontia/S.

PATIENTS AND METHODS

The study included 196 patients with oligodontia. According to the definition, the patients were congenitally missing six or more teeth. The study included 100 males and 96 females. The mean age was twenty-three years, range four to forty-eight years. The patients were enlisted from nine Departments of Oral-Maxillofacial Surgery, Prosthodontics, and Special Dental Care, in various parts of the Netherlands. Of the 196 patients, sixty-two patients had oligodontia/S and ninety-five patients had oligodontia/I. In thirty-nine patients, the diagnosis could not distinguish between oligodontia/I and oligodontia/S. The following criteria were used to distinguish between the different groups; the patient was diagnosed as having oligodontia/I, when the patient showed no symptoms other than the congenitally missing teeth. If the patient showed only one or two other symptoms, but no diagnosis of a syndrome had been made, no discrimination could be made between oligodontia/I and oligodontia/S. Only the patients who showed all or almost all symptoms of a syndrome were diagnosed as having oligodontia/S. The following syn-

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dromes occurred in the patients with oligodontia/S: fifty-six patients had ectodermal dysplasia, in either the hypohidrotic or hyperhidrotic form; one patient with Rieger syndrome; two patients with EEC syndrome (Ectrodactyly-Ectodermal dysplasia- Clefting syndrome); two patients with the Bloch-Sulzberger syndrome, also known as incontinentia pigmenti; and one patient with Cutis marmorata teleangiectatica congenita.

DISTRIBUTION OF MISSING TEETH

The congenitally missing teeth from all 196 patients were recorded. An orthopantomogram was used to score which teeth were missing. The distribution of missing teeth over males and females and over maxilla and mandible were studied. The differences in both distributions for left and right sides were also studied. Discrimination between oligodontia/I and oligodontia/S allowed us to study possible differences in these groups. Chi-square tests and Student's t-tests were used to determine significant differences.[8]

MESIODISTAL DIMENSIONS

The mesiodistal dimensions were measured on casts, using a standard procedure. All impressions were taken with an alginate material, and poured in dental stone. Casts of eighty-two patients who had few or no dental restorations were collected. The maximum mesiodistal dimension of a tooth was obtained only if the tooth was fully erupted and no dental restorations were present. The measurements were made with a digital measuring instrument, accurate within 0.01 mm. Mesiodistal dimensions of left and right teeth were obtained; but in the absence of statistically significant differences between the sides, values averaged from right and left measurements were used in the final analysis. The mesiodistal dimensions of all teeth, except the second and third molars, were made. The measurements were compared with standard mesiodistal dimensions recorded by Moyers *et al.*[9] The statistical procedure was as follows:

- ☐ A Kolmogorov-Smirnov test was performed to confirm the normal distribution of the collected data.[10]
- ☐ Then an F-test was performed to test whether the standard deviations of both populations could be assumed equal.[8]
- ☐ If the standard deviations could be assumed equal a student's-t-test for comparison of two means was used.[8]

In the cases where the standard deviations could not be assumed equal, Welch's test for comparisons involving two variations that must be separately estimated was used.[11]

RESULTS

Distribution of missing teeth

The results of the distribution of missing teeth are given in Tables 1-3. The total population consisted of the two groups oligodontia/I (95) and oligodontia/S 962), and the thirty-nine patients in which the diagnosis could not distinguish between oligodontia/I and oligodontia/S. Males were missing significantly more teeth than females (Table 1).

The difference in mean number of missing teeth between the groups oligodontia/I and oligodontia/S was

The measurements were made with a digital measuring instrument, accurate within 0.01 mm.

Table 1 ☐ Distribution of missing teeth: difference between oligodontia/I and oligodontia/S, males and females.

	Total group N = 196	Oligodontia/I N = 95	t-value	Oligodontia/S N = 62
	Mean ± S.D.	Mean ± S.D.		Mean ± S.D.
Males	14.6 ± 6.0	13.2 ± 5.1	t = 5.1 **	19.4 ± 5.6
Females	12.7 ± 5.2	11.7 ± 4.2	t = 2.4 *	14.7 ± 6.3
	t = 2.4 *	t = 1.4 #		t = 2.4 *
Totals	13.7 ± 5.7	12.6 ± 4.8	t = 4.7 **	16.9 ± 6.4

* = P < 0.01
** = P < 0.001
\# = P > 0.01

Table 2 ☐ Distribution of missing teeth: maxillary and madibular

	Total group N = 196	Oligodontia/I N = 95	Oligodontia/S N = 62
	Mean ± S.D.	Mean ± S.D.	Mean ± S.D.
Maxilla	6.7 ± 2.8	6.2 ± 2.3	8.1 ± 3.2
Mandible	7.0 ± 3.5	6.4 ± 3.1	8.8 ± 3.8
	X^2 = 4.23	5 = 1.1 #	t = 1.1 #
	df = 1#		

\# = P > 0.01

Table 3 ☐ Distribution of missing teeth: left and right sides.

	Left	Right	X^2 df = 1
Oligodontia/I	599	595	0.07 #
Oligodontia/S	527	523	0.03 #
Totals	1345	1335	0.004 #

\# = P > 0.001

significant: males with oligodontia/S were missing significantly more teeth than males with oligodontia/I. For females the difference between oligodontia/S and oligodontia/I was also significant. Males with oligodontia/S were missing significantly more teeth than females with oligodontia/S. The difference between males with oligodontia/I and females with oligodontia/I was not significant (Table 1).

No significant differences were found in the distribution of missing teeth over maxilla and mandible and over left and right sides for males and females (Tables 2,3).

Figure 1 shows the percentages of missing maxillary and mandibular teeth. The second mandibular premolar was the one most frequently missing, followed by the second maxillary premolar, maxillary lateral incisor, and mandibular central incisor.

Mesiodistal dimensions

The mesiodistal dimensions of maxillary and mandibular teeth for males and females are shown in Tables 4-7.

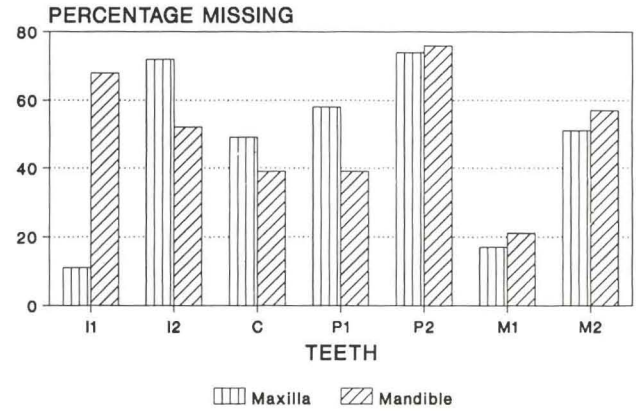

Figure 1. Distribution of the missing teeth of maxilla and mandible. Shown is the percentage for each missing tooth.

The difference in mean number of missing teeth between the groups oligodontia/I and oligodontia/S was significant.

Table 4 ☐ Mesiodistal dimensions of maxillary teeth of females.

	N1	$\overline{X1}$	SD.	N2	$\overline{X2}$	SD.(1)	t-value.	D	D%
I1	35	7.6	0.5	189	8.67	0.57	t = 10.4 **	1.1	13
I2	11	6.0	0.5	172	6.78	0.64	5 = 4.4 **	0.8	12
C	18	7.2	0.6	125	7.49	0.36	d = 1.9 #	0.3	4
P1	13	6.2	0.6	122	6.60	0.46	t = 3.2 *	0.4	6
P2	2	6.0	0.7	99	6.50	0.46	t = 1.6 #	0.5	8
M1	34	9.5	0.8	192	10.18	0.58	t = 5.7 **	0.7	7

SD. = standard deviation.
N1 = sample size
N2 = N from Moyers *et al.*[9]
(1) = from Moyers *et al.*[9]
$\overline{X1}$ = mesiodistal dimension in mm
X2 = mesiodistal dimension in mm[9]
t = t-statistic according to a two-sample t-test[8]
d = d-statistic according to a Welch's test[11]
D = difference of $\overline{X1}$ and $\overline{X2}$
D% = percentage of X2, proportional difference of the mean.

** = $P < 0.001$
* = $P < 0.01$
# = $P < 0.01$

Table 5 ☐ Mesiodistal dimensions of mandibular teeth of females.

	N1	$\overline{X1}$	SD.	N2	$\overline{X2}$	SD.(1)	t-value	D	D%
I1	16	5.1	0.4	196	5.46	0.34	t = 4.1 *	0.4	7
I2	25	5.3	0.4	189	5.92	0.34	t = 8.0 **	0.6	10
C	22	6.2	0.3	148	6.58	0.34	t = 5.4 **	0.4	6
P1	28	6.4	0.4	134	6.78	0.70	d = 3.7 *	0.4	6
P2	5	6.4	0.6	100	7.07	0.46	t = 3.2 *	0.7	10
M1	30	10.3	0.7	191	10.29	0.74	t = 0.7 #	0.01	0.1

SD. = standard deviation.
N1 = sample size
N2 = N from Moyers *et al.*[9]
(1) = from Moyers *et al.*[9]
$\overline{X1}$ = mesiodistal dimension in mm
X2 = mesiodistal dimension in mm[9]
t = t-statistic according to a two-sample t-test[8]
d = d-statistic according to a Welch's test[11]
D = difference of $\overline{X1}$ and $\overline{X2}$
D% = percentage of X2, proportional difference of the mean.

** = $P < 0.001$
* = $P < 0.01$
# = $P > 0.01$

In the absence of significant differences between the mesiodistal dimensions of teeth of patients with oligodontia/I and oligodontia/S for both males and females, values of both groups were combined in the final analysis.

All mesiodistal dimensions of the maxillary teeth of females differed significantly from the mesiodistal dimensions of the teeth of the control group, with the exception of the canine and second premolar (Table 4).[9] The mesiodistal dimensions of central and lateral incisors and the first molar showed the greatest difference (D) of the mean. The greatest proportional differences (D percent) were found with the lateral and central incisors.

The mesiodistal dimensions of the mandibular teeth were also significantly reduced, with the exception of the mesiodistal dimension of the first molar. The greatest difference of the mean, and the greatest proportional difference were seen with the lateral incisor and second premolar (Table 5).

The mesiodistal dimensions of the maxillary teeth of males were significantly reduced (Table 6). The mesiodistal dimensions of incisors and the first molar showed the greatest differences of the mean. The greatest proportional differences were found with the central and lateral incisors and second premolar.

The mesiodistal dimensions of the mandibular teeth were also significantly reduced (Table 7). The second premolar showed the greatest proportional difference, and the greatest difference of the mean.

Figures 2 and 3 show that the mesiodistal dimensions of the upper central incisor (Figure 2) and upper first molar (Figure 3) of females decrease, when the number of missing teeth increases. The correlation

Table 6 □ Mesiodistal dimensions of maxillary teeth of males.

	N1	$\overline{X1}$	SD.	N2	$\overline{X2}$	SD.(1)	t-value	D	D%
I1	37	7.6	0.8	212	8.91	0.59	d = 9.9 *	1.3	15
I2	6	5.6	0.4	201	6.88	0.64	t = 4.9 **	1.3	19
C	11	7.5	0.5	152	7.99	0.42	t = 3.6 **	0.5	6
P1	13	6.1	0.6	157	6.76	0.47	t = 5.0 **	0.7	10
P2	10	5.9	0.5	132	6.67	0.37	t = 6.3 **	0.8	12
M1	32	9.6	0.9	216	10.58	0.56	d = 6.0 *	1.0	9

SD. = standard deviation.
N1 = sample size
N2 = N from Moyers *et al.*[9]
(1) = from Moyers *et al.*[9]
$\overline{X1}$ = mesiodistal dimension in mm
$\overline{X2}$ = mesiodistal dimension in mm[9]
t = t-statistic according to a two-sample t-test[8]
d = d-statistic according to a Welch's test[11]
D = difference of $\overline{X1}$ and $\overline{X2}$
D% = percentage of $\overline{X2}$, proportional difference of the mean.

** = $P < 0.001$
* = $P < 0.01$
# = $P > 0.01$

Table 7 □ Mesiodistal dimensions of mandibular teeth of males.

	N1	$\overline{X1}$	SD.	N2	$\overline{X2}$	SD.(1)	t-value	D	D%
I1	10	5.0	0.4	214	5.54	0.32	t = 4.9 **	0.5	9
I2	16	5.5	0.4	208	6.04	0.37	t = 5.6 **	0.5	8
C	12	6.6	0.5	170	6.96	0.40	t = 3.1 *	0.4	6
P1	27	6.5	0.5	159	6.89	0.63	t = 3.3 *	0.4	6
P2	6	5.8	0.6	132	7.22	0.47	t = 7.0 **	1.4	19
M1	31	10.3	0.7	215	10.71	0.60	t = 3.4 *	0.4	4

SD. = standard deviation.
N1 = sample size
N2 = N from Moyers *et al.*[9]
(1) = from Moyers *et al.*[9]
$\overline{X1}$ = mesiodistal dimension in mm
$\overline{X2}$ = mesiodistal dimension in mm[9]
t = t-statistic according to a two-sample t-test[8]
d = d-statistic according to a Welch's test[11]
D = difference of $\overline{X1}$ and $\overline{X2}$
D% = percentage of $\overline{X2}$, proportional difference of the mean.

** = $P < 0.001$
* = $P < 0.01$
# = $P > 0.01$

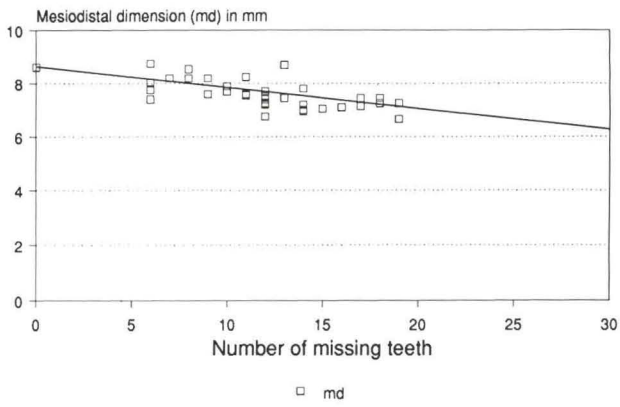

Figure 2. Mesiodistal dimension of the female maxillary central incisor, plotted against the number of missing teeth. Correlation coefficient (R) = -0.63 (P < 0.01); point of intersection vertical axis = 8.7 (N = 35).

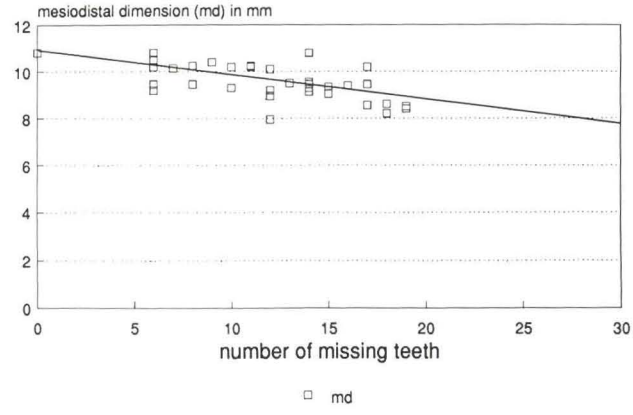

Figure 3. Mesiodistal dimension of the female maxillary first molar, plotted against the number of missing teeth. Correlation coefficient (R) = -0.056 (P = 0.01); point of intersection vertical axis = 10.8 (N = 34).

coefficients were significant ($P < 0.01$) for both teeth. A trend toward a similar correlation was observed for other teeth, although not as clearly significant as for those teeth.

DISCUSSION

The findings presented are of patients with oligodontia, treated in specialized dental centers. The findings are of patients missing six or more teeth, exclusive of the third molars.

Due to the selection procedure, the patients treated in general practice were excluded from the study. Had those patients been included in the study, a reduction in the mean number of missing teeth might be expected, on the premise that the relatively simple cases are treated in general practice.

Discrimination between oligodontia/I and oligodontia/S resulted in a significant difference in the mean number of missing teeth between males and females with oligodontia/S (Table 1). From the literature it is learned that patients with ectodermal dysplasia are afflicted with a severer form of oligodontia.[12] Males with ectodermal dysplasia, often X-linked, show severer signs and suffer more afflictive symptoms than do females. This might explain the larger number of missing teeth in patients with oligodontia/S, especially in males with oligodontia/S, because the group consisted mainly of patients with ectodermal dysplasia.

The difference found between males with oligodontia/I and females with oligodontia/I was not significant. A significant difference in the distribution of missing teeth for males and females was found, however, when no discrimination was made between oligodontia/I and oligodontia/S. The difference found is due to the difference found in males with oligodontia/S and females with oligodontia/S (Table 1). Whisth, Thunold, and Boe report more missing teeth in females than in males; they, however, do not discriminate between oligodontia/I and oligodontia/S.[13] Although Rune and Sarnas do exclude patients with abnormal conditions other than missing teeth from their study, they report no significant difference in the distribution between males and females; which is in agreement with our findings in the group with oligodontia/I.

No significant difference could be observed in both oligodontia/I and oligodontia/S evaluated separately and for the total population, between the distribution of maxillary and mandibular missing teeth. A slightly greater number of teeth were missing, however, in the mandible. A consensus on whether the teeth are more frequently missing in the maxilla or mandible cannot be determined from the literature. Some authors report a greater incidence of missing teeth in the maxilla, whereas others observe a greater incidence of missing teeth in the mandible.[13-15]

No difference could be observed, in the total population and in both groups evaluated separately, between the distribution of missing teeth by left and right sides when both maxillary and mandibular distributions were included. Such a symmetrical distribution is also found by Muller *et al* and by Graber.[14-16] The second maxillary and mandibular premolars, maxillary lateral incisor and mandibular central incisor were most frequently missing (Figure 1). Other investigations report the same result.[13,17] The most stable teeth were the maxillary central incisor and maxillary and mandibular first molars. These are the same as reported by Tso, Crawford and Miller.[18] Apparently both groups, oligodontia/I and oligodontia/S, differed only in the

Oligodontia is not an isolated phenomenon, but is related to a complex of other dental changes.

number of missing teeth. The distributions by left and right sides and by maxilla and mandible were not significantly different. It can be concluded, therefore, that for a correct statement of patients with oligodontia, the two groups should be evaluated separately, if the number of missing teeth is to be considered.

Discrimination between oligodontia/I and oligodontia/S did not result in significant differences in mesiodistal dimensions of the teeth. Patients with oligodontia/S did not have smaller teeth than patients with oligodontia/I. In the total population, almost all mesiodistal dimensions were significantly reduced compared with the mesiodistal dimensions of a normal population. These findings are in concordance with the findings of other authors.[2,3] In concordance with the findings of Brook, the males apparently showed greater differences of the mean than females (Tables 4-7).

Figures 2 and 3 show that the mesiodistal dimensions of the maxillary central incisor and first molar decrease when the number of missing teeth increases; indicating that in patients with multiple missing teeth, the crown-size reduction is severer. For those teeth in females, the correlation coefficients were significant. The other teeth showed more or less the same trend; not all correlation coefficients, however, were significant. It was striking, however, that for all teeth, the points of intersection with the vertical axis had approximately the value of the mesiodistal dimension of the respective teeth of the control group. Garn and Lewis also report an association between reduction in tooth-number and reduction in crown-size.[4]

Crowns of the permanent teeth form throughout infancy and childhood, and their completed size is, in part, a reflection of the individual's morbidity, growth capacity, and general well- being.[19-21] As such, the dentition affords a means of assessing the growth "success" at various ages, stretching from infancy (when the incisors and first molars are undergoing amelogenesis) to late childhood (when the canines and premolars complete crown formation).[22] If oligodontia was a localized, transient disruption in development, only teeth formed in the same period should be affected. From Tables 4-7, it can be seen that almost all teeth, both early and late forming, were affected. Figure 1 also shows that both early and late forming teeth (P2, I2) were frequently missing. In our study, however, not all teeth were equally affected. An interesting phenomenon was the greatest proportional difference of the central and lateral incisors and second premolars (Tables 4-7). Concordance can be seen with the teeth most frequently missing, namely lateral incisor and second premolar, as seen in Figure 1. Bailit reported that not all teeth are equally heritable.[23] The "key" tooth in each morphologic class has the highest heritability, and is the more stable tooth morphologically. The more distal tooth seems to be influenced more by the environment. As noticed in our study, the more distal teeth (P2,I2) seemed to be more severely affected. According to Bailit, the severe reduction in mesiodistal dimensions of lateral incisor and second premolar should, for the most part, be due to environmental factors.[23] The development of the lateral incisor, however, takes place in the 5th/6th month of prenatal life; whereas the development of the second premolar starts in the 9th month postnatal. As such, it is more readily acceptable that those teeth were influenced by genetic factors. Another explanation could be the interaction of genetic and environmental factors, resulting in the more severely reduced distal teeth. The results, taking into account the relatively small number of measurements, support the hypothesis that oligodontia is not an isolated phenomenon, but is related to a complex of other dental changes. Our results support the suggested polygenic mode of inheritance, interaction of many genes with environmental influences as reported by Suarez and Spence and Brook.[5,24]

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PASSIVE TOBACCO SMOKE: HEALTHY CHILDREN 2000

Reduce to no more than 20 percent the proportion of children aged 6 and younger who are regularly exposed to tobacco smoke at home. (Baseline: More than 39 percent in 1986, as 39 percent of households with one or more children aged 6 or younger had a cigarette smoker in the household)

Note: Regular exposure to tobacco smoke at home is defined as the occurrence of tobacco smoking anywhere in the home on more than three days each week.

Baseline data source: Adult Use of Tobacco Survey, CDC.

Environmental tobacco smoke is a cause of disease, including lung cancer, in healthy non-smokers, and is a significant health risk for children. The children of parents who smoke are more likely to develop lower respiratory tract infections, to be hospitalized or see a doctor for these conditions during the first year of life, and to develop middle ear infections than children of parents who do not smoke. Parental smoking may compromise lung function in young children and the developing lungs of the growing child. It may also contribute to the rise of chronic airflow obstruction later in life.

The major source of smoke exposure for young children is their home. Both passive smoking and being nursed by a smoking mother contribute to the amount of tobacco constituents absorbed by infants. In the most recent and comprehensive study of passive smoke exposure of infants, the amount smoked in the same room or vehicle or even the same house as the infant was the major predictor of infants' urinary cotinine levels. Smoking around the infant and putting the infant in a room where smoking occurred recently increased the infant's absorption of environmental tobacco smoke. The authors concluded that "simply blowing smoke away from the infant, going into another room to smoke, or increasing the ventilation in a room will probably not prevent the infant from eventually absorbing tobacco smoke." If people who have contact with children must smoke, they should smoke outdoors or in areas that do not contribute air to places where the child might be.

Although a baseline estimate for this objective is not yet available, 39 percent of households with one or more children aged 6 or younger had a cigarette smoker in the household in 1986. The proportion of children aged 6 and younger exposed to tobacco smoke at home is almost certainly higher.

Healthy Children 2000: U.S. Department of Health and Human Services. Health Resources and Services Administration. September 1991.

REVIEW

Considerations for the direct pulp capping procedure in primary teeth: A review of the literature

Hugh M. Kopel, DDS, MS

Direct pulp capping is defined as the placement of a medicament or a nonmedicated material on a pulp that has been exposed in the course of preparing a cavity in a carious tooth or as the result of trauma. A "mechanical" exposure may also occur during excavation procedures, when the caries has not actually exposed the pulp. DiMaggio *et al* found that the pulps of 75 percent of deeply carious primary and young permanent molars would have been exposed had all carious dentin been removed.[1]

The rationale behind this treatment modality is the encouragement of young healthy pulps to initiate a dentin bridge, thus "walling off" the exposure site. It has been often emphasized that the direct pulp capping in primary teeth should be limited only to small, inadvertent, or "mechanical" exposures that are surrounded by sound dentin.[2-4] The reasons given for limiting this procedure in primary teeth include the potentials for internal resorption, calcifications, chronic pulp inflammation, necrosis, and intraradicular involvement.[2]

Turner *et al* noted, however, that these pathologic reactions were often seen, when calcium hydroxide was used for primary tooth pulpotomies.[5] Nevertheless, the validity of primary teeth as a model for studying the direct pulp capping procedure has been established. Rapp stated that the direct pulp capping treatment is regaining favor as a therapeutic approach following a period of warranted skepticism regarding its effectiveness.[6]

Two arguments have been used for not recommending direct pulp capping in primary teeth. The first relates to primary pulp histology and physiology and its response to irritation, infection, and trauma. The second involves procedural considerations that have been thought to contribute to a poor success rate.

The purpose of this review, therefore, is to present the findings and recommendations in previous investigations of the direct pulp capping procedure in primary teeth and to suggest procedural considerations based on these investigations, in order to encourage a success rate comparable to more invasive techniques, i.e. pulpotomies and pulpectomies.

HISTOLOGIC DIFFERENCES BETWEEN PRIMARY AND PERMANENT TEETH

Fox and Heeley concluded from histologic studies that there are no structural differences between primary pulp tissue and young permanent pulp tissue other than the presence of a cap-like zone of reticular and collagenous fibers in the primary coronal pulp.[7] Many clinicians have noted, however, that the pulps of primary and permanent teeth respond differently to trauma, bacterial invasion, irritation, and medication. Anatomic differences may contribute to these impressions. The roots of the primary teeth have enlarged apical foramina; whereas in time the apices of the permanent teeth become restricted. It is hypothesized, therefore, that a reduced blood supply in permanent teeth favors

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calcific response and healing by "calcific" scarring.[8] This hypothesis is borne out in older pulps, where one finds more calcified nodules and ground substance than in young pulps. Primary teeth, on the other hand, with their abundant blood supply, demonstrate a more typical and faster inflammatory response to irritation than seen in permanent teeth.[3] McDonald also feels that localization of infection and inflammation is poorer in primary pulps than in permanent teeth.[9] It has been shown that the more severe the inflammation in primary teeth the more severe the resorption.[10] The incidence of reparative dentin formation in primary teeth beneath carious lesions has also been found to be more extensive than in permanent teeth.[11-13]

Rayner and Southam stated that the primary pulp responds more rapidly to the effects of dentinal caries than does the permanent pulp.[14] In contrast, Taylor *et al* concluded that in spite of the fact that pulps of primary teeth may be ultimately inflamed and infected by caries, primary molars are capable of marked defensive reactions similar to those observed in permanent teeth.[15]

PULPAL DIAGNOSIS

A correct diagnosis of pulpal conditions in primary teeth is important for an indicated treatment. Obviously the clinical and radiographic findings are paramount in determining this diagnosis and treatment, because histologic findings are not possible in determining a preoperative pulpal status. Pulps can have a reversible or irreversible morbidity, treated or not, depending upon the areas of inflammation and/or pulpitis.

In this regard, Eidelman *et al* and Ishibashi reported poor correlations between clinical and histologic findings in primary teeth, especially with various types of pulpitis.[16,17] Koch and Nyborg, and Schröder were able to find an 80 to 87 percent agreement between the clinical and histologic diagnoses of chronic coronal pulpitis in primary teeth.[18,19] In the Schröder study, the chronic inflammation was found to be closest to an exposure site. Without histologic sections, therefore, the exact pathologic state of an exposed pulp cannot be determined clinically, if radiographs are not conclusive. Thus it is most important for the clinician to be able to decide whether the situation requires conservative or radical treatment. Baratieri *et al* suggested two approaches to the surgical treatment of a reversibly inflamed pulp:

- ☐ Total removal of coronal pulp by pulpotomy.
- ☐ Total or partial conservation of the coronal pulp by direct protection, curettage, or a partial pulpotomy.[20]

It might be remembered that in the carious process, "decalcification precedes bacterial invasion". The dentin in the area of a potential exposure, therefore, might only be minimally inflamed and the pulp also minimally inflamed.

Baume and Holtz felt that the pulp of a primary tooth that was inadvertently (mechanically) exposed and was asymptomatic in the preoperative period without the accepted symptoms of pulpitis is more apt to survive, when capped.[21] The prognosis is less favorable, however, if an attempt is made to cap a pulp that is inflamed and/or infected due to caries or trauma.

Although bacteria were found in the localized area of necrosis following a carious exposure, Lin and Langeland stated that the presence of these live bacteria in the pulp tissue was not a requirement for pulp destruction.[22]

Kennedy and Kapala stated that high cellular content of primary pulp tissue may be responsible for failures of direct pulp capping in primary teeth.[23] Undifferen-

Studies made during the last five years show that pulp healing can take place in the presence of overt inflammation.

tiated mesenchymal cells may differentiate into odontoblastic cells in response to either the caries process or the pulp capping material, which could lead to internal resorption. According to Camp, mechanical exposures have a much better prognosis that do carious exposures, because of the absence of previous inflammation and infection associated with the carious process.[4]

Several investigators have emphasized that the direct pulp capping procedure is contraindicated in the presence of assumed pulpal inflammation.[4,21,24] Tronstad and Major capped experimentally induced pulpal inflammation in monkey teeth with CaOH or zinc oxide eugenol and found no beneficial healing of the exposed pulp, when the CaOH was used.[25] Because of the type of pulpal cellular content in primary teeth, and the possibility of existing pulpal inflammation, infection, and resorption in deeply carious teeth, various pediatric dental clinicians feel, therefore, that the direct capping procedure is contraindicated.[2-4,23,26]

More recently, however, other investigators showed in animal studies that pulp healing can take place in the presence of overt inflammation.[27,28] Cotton observed that when there is minimal pulpal inflammation, a dentin bridge may form against the capping material; but, when the inflammation is more severe, the bridge is apt to form at a distance from the exposure.[29]

Thus, McDonald and Avery and others have felt that a high degree of success with direct pulp capping in primary teeth can be expected in carefully selected cases using specific criteria and treatment methods with proven materials.[5,30-34] (see Table)

The salient features of a successful pulp capping have been described as:

- ☐ Dentin bridging.
- ☐ Maintenance of pulp vitality.
- ☐ Lack of undue sensitivity or pain.
- ☐ Minimum pulpal inflammatory response.
- ☐ The ability of the pulp to maintain itself without progressive degeneration.
- ☐ Lack of internal resorption and/or intraradicular pathosis.[2,23]

The definition of success in direct pulp capping by the presence of a dentin bridge is still controversial, after many years of investigation. Glass and Zander were among the first to use this formation of reparative dentin as a criterion of success.[35] Other investigators who have looked at the clinical and histologic picture in the direct pulp capping procedure also agree with Glass and Zander.[6,36] It has been demonstrated in other studies, however, that a healthy pulp can exist beneath a direct capping, even without a dentin bridge.[37] Seltzer and Bender, Jeffrey, Ulmansky, and Goldberg showed that a dentin bridge is not as complete as it appears and this condition can result in eventual pulp degeneration.[38-42]

TREATMENT CONSIDERATIONS

If one elects to proceed with a direct pulp capping procedure in a primary tooth, the operator should be aware of various treatment considerations which may influence the prognosis for success.

Contraindications

Contraindications to direct pulp capping therapy include a history of (a) severe toothaches at night, (b) spontaneous pain, (c) tooth mobility, (d) thickening of the periodontal membrane, (e) intraradicular radiolucency, (f) excess bleeding at exposure site and, (g) purulent or serous exudate from the exposure.

Table ☐ Direct pulp capping studies in primary teeth.

Author	# of teeth (clinical)	# of teeth (histo)	Age of Patients	Medication	Obs. Period	% Success
Garcia-Godoy	45	–	4-9 yrs	ZOE & FC	18 mo.	96
Jepperson	143	43	2-7	CaOH paste	1-7 yrs.	78
Jerrel et al.	15	15	6-10	Dycal	7-63 d.	95
				Life		
Sawusch	40	2		Dycal	10-63 mo.	80
Sawusch	121	–	–	Dycal	14.8 mo.	85
Schroder et al.	93	–	–	CaOH paste	12 mo.	83
Sveen	120	–	6	ZOE	12 mo.	88
Turner	92	50	6-10	Life		22*
				Dycal	63 d.	33*
				CaOH paste		85*
Weine	40	–	8-10	Dycal	18.6	90

*Dentin bridge percentage

Debridement

Kalins and Frisbee, and Delgado *et al* showed that necrotic and infected dentin chips will invariably be pushed into the exposed pulp during the last stages of caries removal.[43,44] This debris can impede healing in the area by causing further pulpal inflammation and encapsulation of the chips. It is prudent, therefore, to remove peripheral masses of carious dentin before beginning the excavation of the caries where an exposure may occur. Should an exposure occur, nonirritating solutions of normal saline, diluted hydrogen peroxide, or anesthetic solution should be used to cleanse the area and keep the pulp moist.

Bleeding and clotting

A blood clot should not be allowed to form after the cessation of bleeding from the exposure, as it impedes pulpal healing.[27] The clot does not allow the capping material to contact the pulp tissue directly, or subsequently to contact the necrosis-producing, degradation products in the clot, which act as a substrate to attract bacteria, producing inflammation and infection.

Bacterial contamination

Great importance has been given to the observations that bacterial penetration under various restorations is a cause of pulpal damage in deep lesions rather that the properties of the cavity liners and restorative materials.[29,45,46] Kakehashi and colleagues found pulp exposures healed with dentin bridging, even when left uncovered in a germ-free study.[47]

These findings suggest that a more adequate seal of the pulp capping is needed, perhaps by full-crown coverage, rather than by an amalgam alloy or a composite resin, which are prone to bacterial leakage. The stainless steel crown is probably the best coverage for primary teeth to obtain a hermetic seal and to eliminate subsequent bacterial contamination due to microleakage at the enamel or dentin-enamel interface.

Exposure enlargement

Frigoletto noted that small exposures and a good blood supply provide the best healing potential.[48] The current rule of thumb suggests that the size of the exposure be limited to less than 1.0 mm and the exposed tissue exhibits light red bleeding that is easily controlled with a dry cotton pellet applied with minimal pressure.[32]

There have been recommendations that the exposure site be enlarged (sometimes called pulp curettage or partial pulpotomy) before the placement of the capping material.[27,30,49] Enlarging this opening into the pulp itself serves three purposes:

- ☐ It will remove inflamed or infected tissue in the exposed area.
- ☐ It will facilitate washing away carious or noncarious debris.
- ☐ It will allow a closer contact of more capping medicament or material with the actual pulp tissue.

Cvek and Zilberman *et al* have described this partial pulpotomy technique for pulp-exposed, traumatized anterior teeth and carious molars with highly favorable results.[49,50]

Medications and materials used in direct pulp capping

Many medicaments and materials in the years of pub-

Before electing to perform a direct pulp capping in a primary tooth, the contraindications should be carefully considered.

lished dentistry have been suggested to cover over the exposure and initiate pulpal healing and/or hard structure repair. Many of these products have intrigued researchers, because by inference they could produce a new hard tissue barrier.

For over four decades, calcium hydroxide used alone or in combination with a variety of additives has been the most widely investigated and used material in pulp therapy, followed by zinc oxide-eugenol compounds.[51] Antibiotics, calcitonin, collagen, corticoid steroids, cyanoacrylate, formocresol, resorbable tricalcium phosphate ceramic have also been investigated with varying degrees of success. These compounds, with the exception of formocresol, do not seem to have made any impact as a clinical medicament or material for use in the direct pulp capping technique, especially in the pediatric age-groups.

CALCIUM HYDROXIDE

For many years calcium hydroxide in one form or another has been singled out by a myriad of authors as the medicament of choice for pulp exposures.[35,52-54]

CaOH produces coagulation necrosis of the surface tissue of the pulp; and directly under this surface tissue, the underlying tissue differentiates into odontoblasts, which elaborate a matrix in about four weeks.[55] The greatest single benefit of the use of calcium hydroxide is the stimulation it provides for formation of a reparative dentin bridge, perhaps caused by the irritating quality of its high alkalinity, pH 11-12.[54] In this alkaline medium, the enzyme phosphatase is active in releasing inorganic phosphate from the blood, followed by the precipitation of calcium phosphate. The antibacterial action of calcium hydroxide also has been as one of the benefits in capping procedures.[56,57]

There is some question whether Ca++ ions are necessary for the dentinal bridge repair at the exposure site. Sciaky and Pisanti, and Attalla stated that in their radioactive studies, the Ca++ ion in the capping material did not enter into the bridge formation,[58,59] Stark and colleagues, however, believe the Ca++ ion does enter into the bridge formation, as reported in their particular pulp studies.[60] Tagger *et al* recommended further investigations to determine whether a base that releases more CaOH is superior to one that releases less in promoting a biological seal in direct capping.

Seltzer and Bender mentioned the osteogenic potential of CaOH, which is capable of completely obliterating the pulp chamber and root canals—a concern to clinicians.[38] Lim and Kirk in their extensive review of direct pulp capping studies find very little support for this suggestion of pulp obliteration as well as internal resorption as a major complication of pulp capping.[36]

Internal resorption and chronic pulpal inflammation have been cited as frequent sequelae, when CaOH is used as a capping material in primary teeth.[62-64] Ranly states that internal resorption may be seen, when CaOH is applied to the inflamed radicular pulp in a primary tooth that has undergone a pulpotomy procedure.[64] These findings by many clinicians suggest that the response of the radicular pulp tissue following a complete coronal pulp amputation (pulpotomy) cannot be compared to the response of minimally compromised coronal pulp tissue, when a direct pulp capping procedure is undertaken.

A review of the literature failed to provide significant evidence of internal resorption as a cause of failure, when CaOH is used as direct pulp capping material.

Hard-set CaOH liners and CaOH paste can be used without causing internal resorption or obliteration of the pulp.

Jepperson reported 97.6 percent clinical success and 88.4 percent histologic success in a long-term study on the use of a creamy mix of CaOH placed on exposed pulps of primary teeth.[33] Although CaOH pastes have been shown to be efficacious in promoting dentin bridges, their high pH, water solubility, and lack of physical strength led manufacturers to introduce modified lower pH CaOH cements, which set quickly and hard for lining cavities and capping pulps.

After clinical investigation of two formulas of a hard, self-setting CaOH compound (Dycal), Sawusch stated that the materials were effective agents for direct and indirect pulp capping in both primary and young permanent teeth.[65] It is of interest that almost twenty years earlier, Sawusch reported that the percentage of success of direct pulp capping of exposed pulps of primary teeth using a calcium hydroxide paste or a hard set material was relatively "high".[66]

Various studies in recent years have shown successful results (up to 80 percent) with CaOH pulp capping of primary teeth with or without coronal inflammation.[5,27,34,65] These investigations demonstrate that the hard-set, CaOH liners can be used as well as the CaOH pastes without causing any pathologic sequelae, such as internal resorption or pulpal obliteration. For example, the so-called "necrobiotic" and inflammatory zones are minimal or absent and dentin bridges are found to form directly under these commercial compounds, instead of at a distance from the paste types.[67,68] Also, adequate sealing against microleakage, antibacterial properties, and physical strength to support dental restorations have been shown for the hard-set, CaOH cements.[69,70]

With the advent of visible-light-curing, restorative resins, it was inevitable that in the interest of saving time and providing a harder, cavity-lining material, light-cured, CaOH, pulp capping products have been introduced. Stanley and Pameijer, and Seale and Stanley, in histologic studies, found that a CaOH product (Prisma VLC Dycal), cured by visible light, maintained all the characteristics of healing and bridge formation, equivalent to the original, self-curing Dycal.[71,72] Lado and Stanley, in an *in vitro* study, compared the bacterial inhibition of these new light-cured products to the self-setting, CaOH cements and found no differences.

Meeker *et al*, however, did not find the antibacterial properties of the light-cured Dycal to persist after seventy-two hours.[74]

Pitt Ford and Roberts showed that the success rate of pulp capping delayed for twenty-four hours was as high as that for immediate capping, when a light-cured, calcium hydroxide preparation was used and sealed with a zinc oxide-eugenol cement.[75] This again shows how important it is to have an adequate seal over the actual pulp-capping material.

OTHER AGENTS SUGGESTED FOR DIRECT PULP CAPPING IN PRIMARY AND PERMANENT TEETH

☐ *Zinc oxide-eugenol cement* Glass and Zander found that zinc oxide and eugenol in direct contact with the pulp tissue produced chronic inflammation, prevented formation of a calcific barrier, and caused pulpal necrosis.[35]

Hembree and Andrews in a literature review of ZOE used as a direct pulp-capping material could find no positive recommendations.[76] Watts also found mild to moderate inflammation and no calcific bridges in the specimens under his study and confirmed by Holland.[77,78] Weiss and Bjorvatn, on the other hand, noted negligible necrosis of the pulp in direct contact with ZOE and stated that any calcific bridging of an exposure site was probably a layer of dentinal chips.[37] They also found no differences in the pulp reactions of primary and permanent teeth.

In spite of the reported lack of success with ZOE cement, Sveen reported that he had 87 percent success with the capping of primary teeth with zinc oxide-eugenol in ideal situations of pulp exposure.[79] He offered no histologic evidence, but Tronstad and Mjor, comparing ZOE with CaOH, found ZOE more beneficial for inflamed, exposed pulps, and felt that the production of a calcific bridge is not necessary, if the pulp is free of inflammation following treatment.[25]

☐ *Corticosteroids and antibiotics.* Corticosteroids and/or antibiotics were suggested for direct pulp testing in the pretreatment phase; and also to be mixed with CaOH with the thought of reducing or preventing pulp inflammation. These agents included neomycin and hydrocortisone, Cleocin, cortisone, Ledermix (CaOH plus prednisolone), penicillin and Keflin.[80-85] While many of these combinations reduced pain for the most part, they were found only to preserve chronic inflammation and/or reduce sensitive dentin. Also, Watts and Paterson cautioned that anti-inflammatory compounds should not be used in patients at risk from bacteremia.[86] Gardner, Mitchell, and McDonald found that vancomycin in combination with CaOH was somewhat more effective than when CaOH was used alone, and stimulated a more regular reparative dentin bridge.[87]

- ☐ *The polycarboxylate cements.* These cements also have been suggested as a direct capping material. The material was shown to lack an antibacterial effect and did not stimulate calcific bridging in the pulps of primary and permanent monkey-teeth.[85] In contrast, Negm, Grant, and Comb placed CaOH and zinc oxide into a 42 percent aqueous polyacrylic acid and used the combination to treat direct pulp exposures in patients from ten to forty-five years of age.[88] This mixture showed faster dentin bridging in 88-91 percent of the patients, compared to Dycal as the control.
- ☐ *Inert materials.* Isobutyl cyanoacrylate and tricalcium phosphate ceramic for example have also been investigated as direct pulp capping materials.[89,90] While pulpal responses in the form of reduced inflammation and unpredictable dentin bridging were found, to date none of these materials has been promoted as a viable medication.

Recently, Heys *et al* compared the healing of exposed primate dental pulps capped with an inert material such as Teflon with two calcium hydroxide materials.[91] Teflon proved to have a similar soft tissue healing pattern at a slower rate; hard tissue formation at the exposure site, however, was infrequent. The investigators agreed with previous studies that an active material was necessary to stimulate the deposition of minerals.

- ☐ *Collagen fibers.* Because collagen fibers are known to influence mineralization, Dick and Carmichael placed modified wet collagen sponges with reduced antigenicity in pulp exposed teeth of young dogs.[92] While the material was found to be relatively less irritating than CaOH and with minimal dentin bridging in eight weeks, it was concluded that collagen was not as effective in promoting a dentin bridge as was CaOH. Fuks *et al* found dentin bridges after two months in 73 percent of pulpotomies in which enriched collagen solution was used.[93] They felt that a different mechanism exists for the production of a truer dentin when a collagen solution is used than with CaOH as no coagulation necrosis was seen.
- ☐ *Formocresol.* Because of the clinical success of formocresol when used in primary pulp therapy, such as pulpotomies and pulpectomies, several investigators have been intrigued by the possibility of its use as a medicament in direct pulp-capping therapy. Arnold applied full strength formocresol for two minutes over pulp exposures in primary teeth that had been enlarged and found a 97 percent clinical success after six months.[94] Ibrahim, Mitchell, and Healy reported, in an animal study, the absence of inflammation along with dentin bridging in fifteen experimental teeth, when the exposure was medicated with formocresol for five minutes and capped with a mixture of formocresol and zinc oxide eugenol cement.[95] More recently, Garcia-Godoy obtained a 96 percent clinical and radiographic success rate in human exposed primary molars, when capped with a paste of one fifth diluted formocresol mixed with a zinc oxide-eugenol paste and covered with a reinforced ZOE cement.[96] Stainless steel crowns were then adapted to the experimental teeth. The author noted that in some cases the exposure had been enlarged to obtain better contact with the zinc oxide-eugenol paste.

SUMMARY

In reviewing the various studies concerning the direct pulp capping procedure in primary teeth, using rigid criteria for case selection and procedure appears to insure a significant amount of success. It is acknowledged that vital primary pulp tissue is capable of healing without resorting to complete pulpectomy, although statistically direct pulp capping has been found to be less successful in primary teeth than indirect pulp therapy or coronal amputation (pulpotomy).

To achieve success for direct pulp capping in primary teeth, the considerations involve: selecting teeth with minimal or no clinical signs of pulpal inflammation; or pretreating the carious tooth with a sedative restoration before excavating the caries; disinfecting the cavity floor; enlarging the actual exposure, and flushing out dentinal debris with mild solutions; controlling bleeding by not allowing a clot to form; placing a hard-set, CaOH material over the exposure, followed by a fast-setting, zinc oxide-eugenol cement to achieve a hermetic seal; and lastly, placing a stainless steel crown to minimize microleakage and prevent a fractured or defective restoration.

These procedural steps can hardly ensure complete success in direct pulp capping of a primary tooth; but, based on the many cited investigations in this review, a significant amount of success can be expected without resorting more frequently to invasive techniques.

Surely the evidence presented leading to the feasibility of direct pulp capping in primary teeth merits further investigations, before dogmatically rejecting this procedure of pulp therapy.

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REPORTS

Milk as storage medium for exarticulated teeth: Report of case

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A vital periodontal ligament (PDL) is important for proper physiologic healing of replanted teeth.[1,2] In vitro studies on human and monkey teeth and in vivo investigations in monkeys indicated that milk is a suitable storage medium for traumatically exarticulated teeth that cannot be replanted immediately.[3-7] Critical factors affecting the outcome are:

- ☐ The interval between exarticulation and replantation.
- ☐ The way the tooth is handled during the extraoral period.

If the tooth is stored in water, the PDL cells will swell until they burst. If the tooth is kept in air on the other hand, the cells will shrink due to dehydration. Both situations involve a disturbance of cell metabolism, enhancing the risk of replacement resorption.[1-10] A suitable storage medium should have a physiologic osmolality in order to keep the PDL cells in as healthy a condition as possible. Furthermore, the storage medium must be available when needed. Milk has an osmolality that ranges within the physiologic limit for cells and accordingly may be the storage medium of choice in situations where immediate replantation cannot be performed.[5] Another approach to preventing the PDL cells from dehydration is to wrap the tooth in thin plastic foil. Monkey teeth stored in this way for an hour before replanting had the same prognosis as teeth replanted immediately after extraction.[9]

Some years ago a campaign was conducted in Sweden on how to handle exarticulated teeth. Milk packages carried a printed message: *Try to put the avulsed tooth back in position immediately after the accident, without any cleaning. If that is not possible, put the tooth in your mouth or in milk. Seek professional help as soon as possible.*

As a result of this campaign, a large portion of "the milk-drinking population" in Sweden knows how to act in an emergency. At least people associate avulsed teeth with milk.

In Scandinavia, traumatic dental injuries can usually be treated professionally within a few hours. Only occasionally does dental treatment have to be postponed. The case presented here concerns a boy with an avulsed tooth, for which dental care was delayed for twelve hours.

CASE REPORT

The patient was a healthy Swedish boy age fourteen years, three months. On Sunday evening, while practicing basketball in the backyard at home, he jumped to put the ball into the goal. The net got twisted around his right lateral maxillary incisor, which was exarticulated and fell to the gravel below the goal. This unusual accident can be explained by the boy's exceptional height and the spacing of his anterior maxillary teeth. Immediately after the accident, the boy grabbed the tooth and ran into the house. His mother, recalling parts of the campaign text, promptly put the tooth into a glass filled with standard milk, which she then stored in the refrigerator. According to the patient, only five to ten

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minutes had passed since the accident.

The mother failed to get in touch by telephone with the oral surgeon on duty and treatment was delayed, therefore, until Monday morning. She contacted the school clinic, which referred the case to the pedodontic clinic in Södertälje. The patient and his tooth arrived at the clinic, twelve hours after the accident.

STATUS

Extraorally there was no sign of trauma. Intraorally the right lateral maxillary incisor was missing. The soft tissue mesial to the alveolus was torn. A blood clot was present in the alveolus. Clinically and radiographically there was no evidence of fracture of the supporting bone (Figure 1). The adjacent teeth were judged to be free of traumatic injuries. The avulsed tooth, which on arrival at the clinic was transferred to sterile physiologic saline at room temperature, appeared undamaged. The root was slender and fully developed.

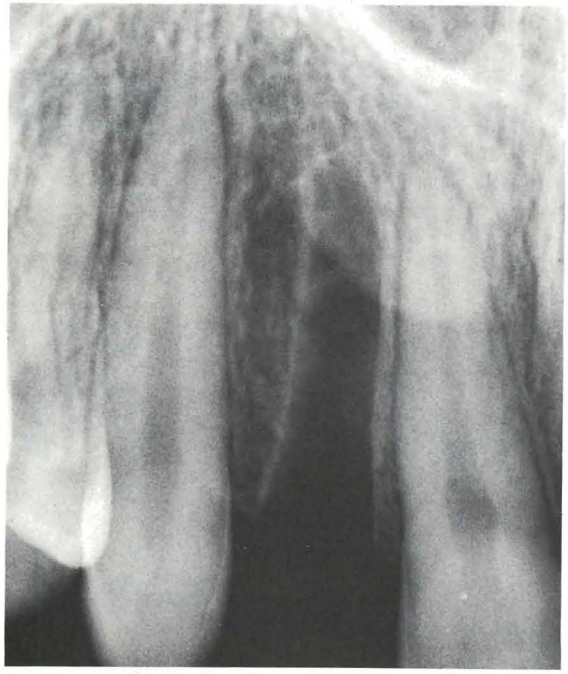

Figure 1. A radiograph taken twelve hours after exarticulation of the right lateral maxillary incisor. The alveolus seems to be intact.

DIAGNOSIS

The injury was classified as exarticulation (N 873.68) of the right lateral maxillary incisor, combined with laceration of the gingiva (N 873.69).

TREATMENT

Although the operator (K-J N.) judged the long-term prognosis for replantation to be poor, due to the long extraoral period, he decided to replant the tooth. He considered that the patient had nothing to lose, especially since the spacing of the anterior teeth would interfere with esthetic prosthetic therapy. Furthermore, no teeth were available for transplantation. Once replantation had been decided upon, a local infiltration anesthetic was applied.* Citanest Octapressin 3 percent was used to minimize local circulatory disturbance, which might aggravate existing tissue damage. Removal of necrotic pulp tissue is usually postponed for one to two weeks in connection with replantation procedures to avoid further damage of the PDL cells. In view of the long extraoral period and the potential risk of introducing milk protein into the circulatory system, however, the operator chose to start the endodontic treatment before replantation. During trephination of the tooth and removal of the pulp tissue, the operator was careful not to compromise the root surface. The root was copiously irrigated with physiological saline throughout the endodontic procedure and again immersed in physiologic saline. The alveolus was also irrigated with saline to remove the rather firm blood clot. Using a blunt instrument, the alveolus was judged to be intact and the tooth was gently positioned without difficulty. To ensure proper adaptation at the gingival margin and of the soft tissue tear, a single suture was applied mesial of the tooth. A loosely packed calcium hydroxide dressing, Calasept**, was applied in the root canal, which then was sealed off with IRM*** (Figure 2). The dressing was applied after the replantation, because the author perceived a risk of contaminating the root surface with the medicaments, if applied before replantation.

Fixation was done with a fiberglass plait, soaked in composite resin, Silux†, diluted with Enamel Bond resin. The splint, which was flexible, was bonded to the buc-

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**Scania Dental AB, Box 5, S-741 00 Knivsta, Sweden.

***L.D. Caulk, Co., P.O. Box 359, Milford, DE, U.S.A.

†Minnesota Mining and Manufacturing Co., St. Paul, MN, U.S.A.

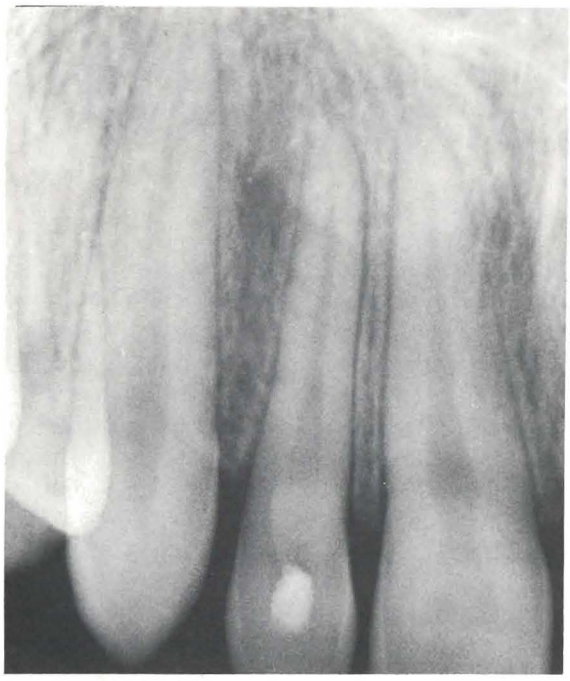

Figure 2. A radiograph taken immediately after replantation and application of a loosely packed, calcium hydroxide pulpal dressing. The tooth is in its proper position.

cal surfaces of the neighboring teeth (Figures 3,4). The patient was given instructions about diet and oral hygiene. Antibiotics were prescribed (Kåvepenin‡ tablets, 1 g twice a day for 10 days) and the patient was referred to hospital for tetanus prophylaxis.

FOLLOW UP

The patient was seen again after one week. Healing of the soft tissue was uncomplicated (Figures 3,4). The patient felt no discomfort. The fixation was removed after another three days. At the one-month control, the tooth mobility was within the normal range and the gingival condition was almost normal.

At the three-month control, the tooth mobility was slightly reduced compared with the contralateral tooth. Percussion suggested development of replacement resorption. Radiographs revealed a rough periodontal contour together with periapical rarefication (Figure 5).

The pulpal dressing was changed. Calasept was densely packed in the root canal.

‡Astra Läkemedel AB, S-151 85 Södertälje, Sweden.

At the six-month control, tooth mobility was nonexistent, indicating ankylosis. Radiographs, however, failed to reveal any replacement resorption. The periodontal contour was still rough, but apical rarefaction was no longer evident. Once again the pulpal dressing was changed.

At the one-year control, there was still no sign of infraposition. The tooth exhibited some mobility, but less than contralaterally. Percussion in the axial direction did not suggest the presence of replacement resorption. A comparison of the radiographs clearly showed that the root had "narrowed" since replantation, but as a PDL appeared to circle the root completely, the narrowing was judged to be a result of extensive surface resorption rather than replacement resorption. Apical obstruction, due to hard tissue formation was clinically evident. After preparation, the root canal was obtur-

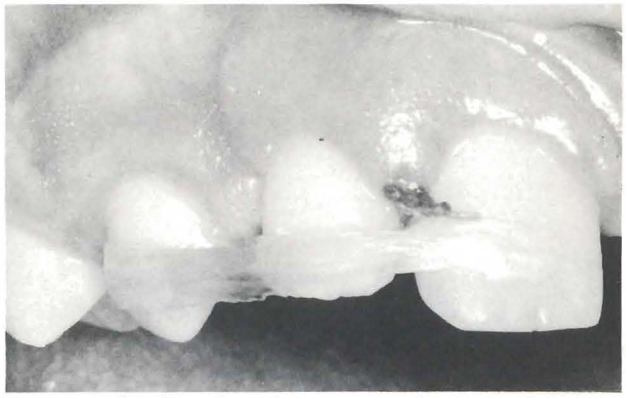

Figure 3. The clinical condition one week after replantation. A flexible fiberglass plait is used for fixation of the replanted tooth. A suture is still in place.

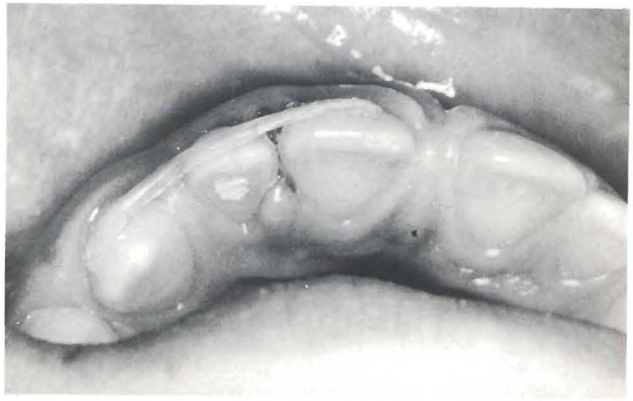

Figure 4. The same condition as in Figure 3.

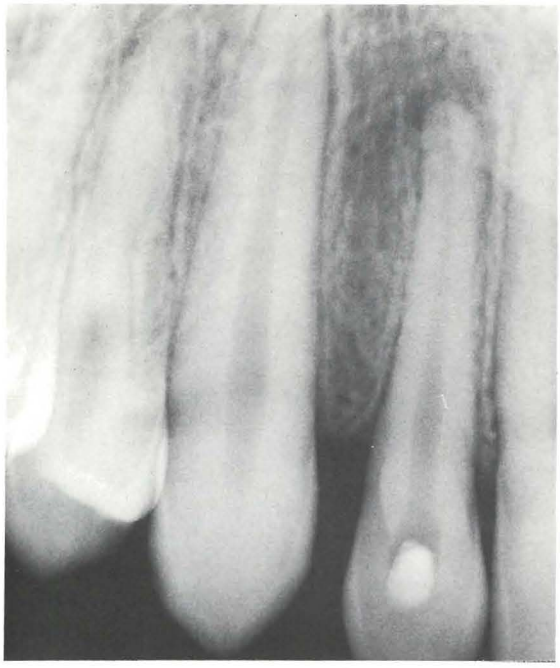

Figure 5. A radiograph taken at the three-month control. The periodontal contour is rough and apical rarefaction is present.

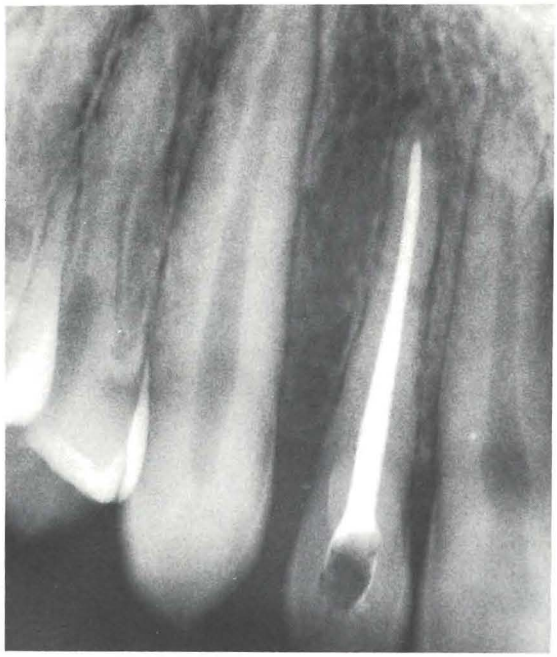

Figure 6. A radiograph taken one year after replantation. Narrowing of the root is evident, compared with Figures 2 and 5. There is no evidence, however, of replacement resorption. The root canal has been obturated with gutta-percha.

ated, therefore, with gutta-percha. The entrance to the pulp chamber was sealed off with composite resin, using the acid-etch technique (Figure 6). At the 1.5,2,3 and 4-year controls, the clinical situation was unchanged, compared with that of one year after replantation. The radiographs indicated successful endodontic treatment. Some surface resorption occurred between one and two years after replantation, but not later on, suggesting the presence of a functional PDL (Figure 7). A five-year control was planned, but a month before the appointment the root of the tooth in question was fractured horizontally and the coronal fragment was lost during a game of field hockey (Figure 8). Since the remaining fragment was judged to be too thin and fragile for prosthetic purposes, surgical removal followed by insertion of an implant was chosen instead of intraalveolar transplantation or orthodontic extrusion. The root was removed without difficulty in one piece, eleven weeks posttrauma, and showing absence of replacement resorption. The root was prepared for examina-

Care must be taken not to contaminate the root surface with medicaments.

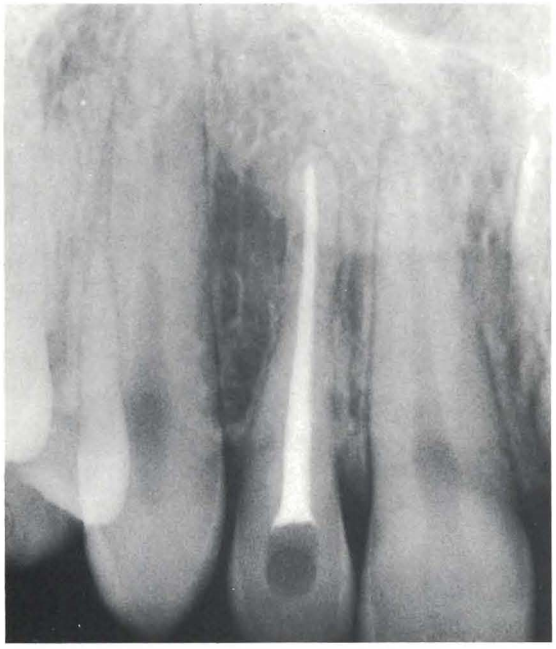

Figure 7. A radiograph taken four years after replantation. A PDL could be traced around the entire root. The result of the endodontic treatment is excellent. Compared with the two-year control, no further root resorption is seen.

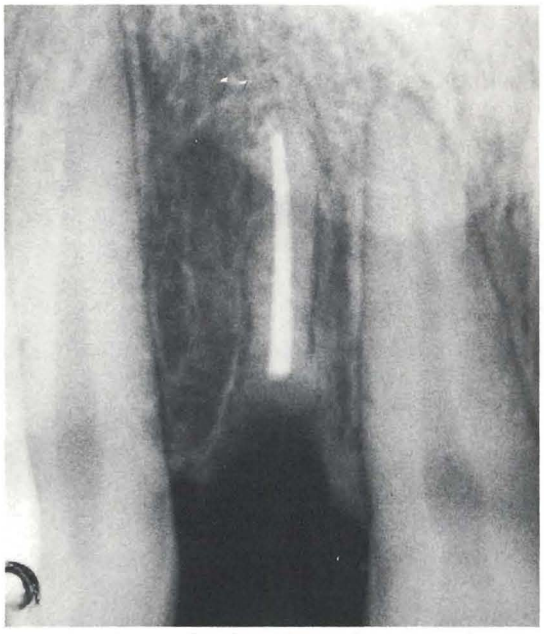

Figure 8. A radiograph taken almost five years after replantation. The tooth has been exposed to a new trauma, causing a root fracture and loss of the coronal fragment.

tion under a light microscope, using routine histologic technique. Sagittal sectioning and Htx-eosin staining were chosen.

The histologic examination revealed fragments of the periodontal membrane. These were free from inflammation. In some areas the root cementum was judged to be intact, while in other places it had undergone resorption and the dentin was covered with cells resembling fibroblasts. Resorption lacunae frequently exhibited healing. No replacement resorption was seen (Figure 9).

DISCUSSION

The treatment of this case followed modern concepts of traumatology.[1] Despite an extraoral period of more than twelve hours, a sufficient number of PDL cells survived in the milk environment to establish a functional PDL. This is in agreement with the results of Blomlöf and co-workers, obtained in vivo on monkeys. Thus it seems that storage of avulsed teeth in milk can be recommended in an emergency. Other factors that affect the prognosis are type and length of fixation,

At the six-month control, lack of mobility indicated ankylosis.

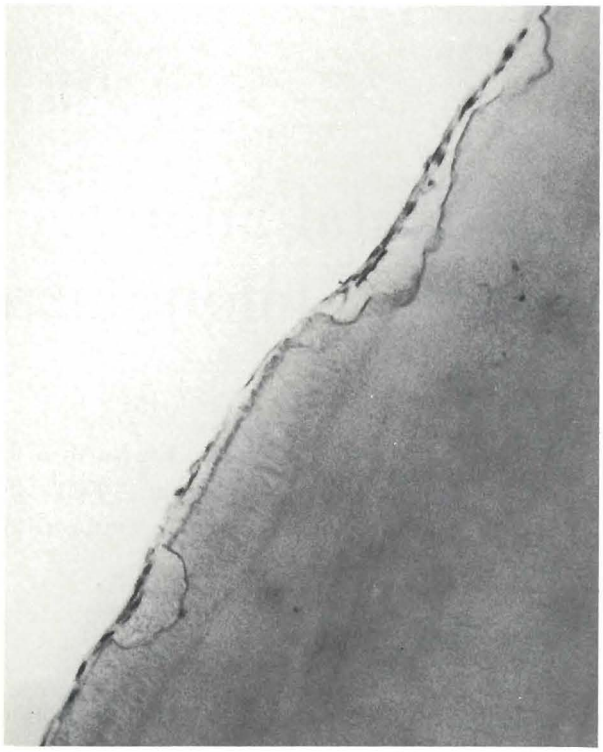

Figure 9. Representative appearance of the root surface. The root has undergone surface resorption. Healing is evident.

presence of necrotic pulp tissue, and microorganisms. Fixation was undertaken with a flexible fiberglass plait, permitting minor tooth movements. Furthermore, the fixation period was limited to ten days. Rigid fixation for a long period of time increases the risk of replacement root-resorption.[1,2,11,12]

The necrotic pulp tissue was removed in connection with the replantation procedure and the patient was treated with antibiotics. Infected necrotic pulp tissue is known to cause inflammatory root resorption.[1,2,13,14]

The endodontic treatment followed current principles for handling of traumatized young permanent teeth.[15] The histologic examination showed that root resorption had taken place, but resorption lacunae frequently exhibited healing. Resorption was detected at the one-year control. The 1.5 and 2-year controls indicated continued resorption, while later controls suggested healing. At the earlier controls, replacement resorption was suspected, because tooth mobility was reduced or absent. Moreover, a high-pitched sound was noted on percussion. According to Andersson *et al*, at least 20 percent of the root surface must be ankylotic for this to be revealed by percussion in an axial direction.[16] Since tooth mobility returned, although it was less than the mobility of the contralateral tooth, the ankylosis was no doubt transient. This type of ankylosis was described earlier by Andreasen.[17] Because of the second trauma, one can only speculate about the long-term prognosis. Although resorption lacunae frequently exhibited healing, active surface resorption sites were also present. It is possible that this resorption was a result of the new trauma and would have healed with time. If so, the author believes that the long-term prognosis would have been good.

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An unusual case of dental anomaly: A "facial" talon cusp

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Roger G. Noonan, DMD, MS
Joseph A. Tylka, DDS

The talon cusp is a supernumerary crown structure composed of enamel, dentin, and a varying degree of pulp tissue. Typically it resembles an eagle's talon and projects incisally from the cingulum area of incisors.[1] This lingual location, which frequently affects occlusion, is pathognomonic of the talon cusp. This report describes a supernumerary cusp. talon-shaped, and labially located: a facial talon cusp.

The talon cusp (so described in 1892) arises during the morphodifferentiation phase of tooth development and has been reported to be associated with Mohr and Rubinstein-Taybi syndromes, and with anomalies such as the complex odontome and impaction.[2-6] This supernumerary structure is rare in occurrence and is most commonly seen on the lingual surface of permanent maxillary incisors.[1,7,8] Permanent mandibular incisors and primary incisors manifest this anomaly.[3,7,9,10] Chen suggests that talon cusps may be seen in primary incisors of Chinese children as frequently as in their permanent incisors.[11]

In addition to shape and location, size has entered into defining the talon cusp, with Mader and others stating that the length be at least half the distance from the cementoenamel junction to the incisal edge.[9,12]

The extension of pulp tissue into the cusp may be present or absent.[1,6,13,14] Radiographic examination has not been decisive in determining the presence or configuration of pulp tissue.[10]

Early diagnosis of a talon cusp has been reported to be difficult, clinically and radiographically. It can mimic a supernumerary tooth, a compound odontoma, or a dens in dente.[8,12,14]

Problems attributed to talon cusps are: compromised aesthetics; occlusal interference; displacement of teeth; caries; periodontal problems; and irritation of the tongue during speech and mastication.[8,9,12,15]

Treatment modalities have included gradual, periodic reduction of the cusp with fluoride as a desensitizing agent; single appointment reduction with and without pulp therapy; sealant for developmental grooves; and partial reduction with composite camouflage.[7,13-16] In addition, orthodontic correction has been necessary.[6,16]

The talon cusp has been reported always to originate from the lingual surface of an incisor tooth. This report describes a cusp on the facial surface of a permanent maxillary central incisor.

CASE REPORT

An eight-year-old, black female was brought to the Pediatric Dentistry Clinic at Loyola University School of

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Dentistry for routine dental care. The patient's medical history was unremarkable. Examination of the oral cavity revealed normal soft tissues, a slight CL III tendency of occlusal development, and normal development of existing primary and permanent dentitions. The right maxillary central incisor exhibited a well defined cusp on the facial surface, pyramidal in shape and extending from the cementoenamel junction to within 1 mm of the incisal edge. The tip of the anomalous cusp was rounded and not irritating. The patient was not concerned with appearance (Figure 1).

A talon cusp can cause serious dental problems.

Noncarious developmental grooves were present at the junction of the cusp with the facial surface of the tooth (Figure 2). The cusp measured 2 mm in width and 7 mm in length from the base to the tip. A pulp chamber was radiographically visible extending into the cusp (Figure 3). The diagnosis of a talon cusp of the maxillary right central incisor was made.

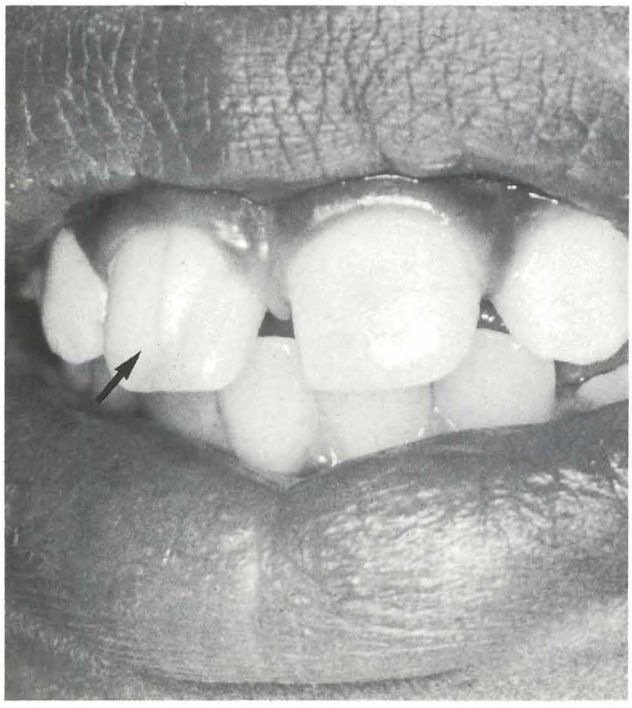

Figure 1. Frontal view of maxillary right central incisor shows facially positioned cusp (arrow).

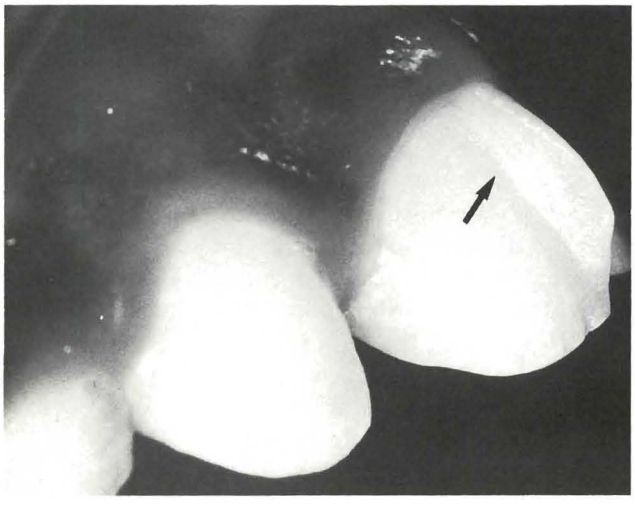

Figure 2. Lateral view of cusp (arrow) connected to the facial surface.

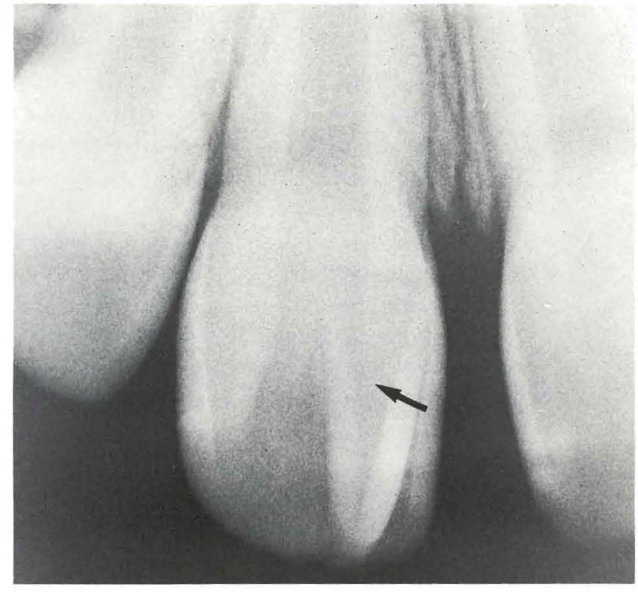

Figure 3. Periapical radiograph shows cusp with pulpal extension.

DISCUSSION

The literature concurs that talon cusps are accessory, cusp-like structures that project from the cingulum area of the incisors.

Controversy exists, however, regarding size of the cusp, presence of pulp, early diagnosis, treatment method, and whether this extra cusp can occur on primary incisors or mandibular permanent incisors.

This case adds to the controversy in that it presents a talon-shaped cusp with a pulp chamber that projects from the facial surface of a maxillary permanent incisor.

Aesthetics was questionable by our standards; the parent refused treatment, however, stating that "the appearance was not causing any trouble".

CONCLUSION

In conclusion, this case shows that a talon cusp may occur on the facial surface of an incisor; hence the following definition is suggested: A talon cusp is a supernumerary accessory talon-shaped cusp projecting from the lingual or the facial surface of the crown of an incisor and extending at least half the distance from the cementoenamel junction to the incisal edge.

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HIV INSTRUCTION, KNOWLEDGE, AND DRUG INJECTION

In our survey and in others, adolescents were quite knowledgeable about AIDS and HIV infection—at least about the major modes of HIV transmission. But few previously reported studies focused on behavior, and only one that we found, which was limited to a single high school, also examined determinants of HIV-related behaviors in youth.

While our analysis showed a relationship between knowledge and HIV-related drug behavior, we do not want to imply that knowledge is the only factor that may be related to behavioral change. Other factors, not measured in the current analysis, may be important predictors of behavioral change in addition to knowledge, for example, certain attitudes, social skills and support, perceived norms, and perceived efficacy in avoiding risks. Thus, it should not be inferred from the results that educational programs designed to increase HIV knowledge alone will have an effect on behavior. But, while other factors that may affect drug-injection behavior need to be investigated, our results suggest that knowledge about HIV may be one factor in the complex process of behavioral change. Comprehensive school health education programs that increase knowledge, as well as specific skills that may influence HIV-related behavior, could be an important means to help prevent the further spread of HIV infection among our nation's youth.

Holtzman, D. *et al:* HIV instruction, HIV knowledge, and drug injection among high school students in the United States. Am J Public Health, 81:1596-1601, December 1991.

ABSTRACTS

Kreulen, C.M.; van Amerongen, W.E.; Akerboom, H.B.M.; Borgmeijer, P.J.; Gruythuysen, R.J.M.: Radiographic assessments of class II resin composite restorations in a clinical study: Baseline results. J Dent Child, 59:97-107, March-April 1992.

Baseline results of a radiographic evaluation of 244 Class II resin composite and amalgam restorations in a clinical study are reported. Bitewing radiographs were taken of each patient using a beam-aiming device. The most important characteristics examined are: cervical marginal adaptation, voids in the material, and radiolucencies in the dentin adjacent to the restoration. These were related to the restorative material, the type of restoration, the type of tooth, and the dentists involved in the study. Cervical marginal defects were observed with almost 30 percent of the composite and with more than 40 percent of the amalgam restorations. More than half of the composite restorations show voids. Radiolucencies adjacent to the restorations were seen for both types of restorative material but these cannot be interpreted unambiguously. For composite restorations there seems to be an effect according to the dentist's experience level, with respect to the above characteristics.

Radiography, bitewing; Restorations, Class II, resin composite [and] amalgam; Radiolucencies; Margins, cervical

Rusmah, Mean and Rahim, Zubaidah, H.A.: Diffusion of buffered glutaraldehyde and formocresol from pulpotomized primary teeth. J Dent Child, 59:108-110, March-April 1992.

The diffusion of 2 percent w/v buffered glutaraldehyde and formocresol (Buckley's formula) from pulpotomized primary teeth was studied. The measurement was based on the reaction of aldehyde with Schiff's reagent. No diffusion of buffered glutaraldehyde was observed from all the glutaraldehyde-treated teeth. Traceable amounts of formocresol were, however, detected in the collecting medium of all formocresol-treated teeth as soon as 15 minutes after treatment. The amount gradually increases and peaked after 4 hours. This suggests that 2 percent w/v buffered glutaraldehyde is a better fixative with low diffusion and is biologically acceptable as a medicament in the pulpotomy of primary teeth.

Glutaraldehyde, buffered; Formocresol; Medicaments; Buckley's formula; Schiff's reagent

Amaratunga, N.A. de S. Asoka: Mandibular fractures in Sri Lankan children: A study of clinical aspects, treatment needs and complications. J Dent Child, 59:111-114, March-April 1992.

A total of 37 children younger than 11 years of age who had mandibular fractures were studied. Age, sex, type of fracture, incidence, etiology, methods of treatment and complications were compared in this study. Frequency of these injuries in 818 hospitalized children was 4.5 percent of all facial injuries. Male to female ratio was 2.4:1. Unilateral fractures were more common than combinations. Condyle was involved in 45.9 percent of fractures. Maxillomandibular fixation for two weeks was the method of treatment employed in 35.1 percent. Complications were rare; infection was observed in two cases, malunion and non-union were not seen. Infection was seen in only two patients. Undisplaced fractures can be successfully managed with no fixations.

Fractures, mandibular; Children; Injuries, facial

Veerkamp, J.S.J.; Gruythuysen, R.J.M.; van Amerongen, W.E.; Hoogstraten, J.: Dental treatment of fearful children using nitrous oxide. Part II: The parents' point of view. J Dent Child, 59:115-119, March-April 1992.

A survey carried out among the parents of children with severe dental anxiety reveals that the parents see the dentist as being the sole source of their child's fear. They do not make any connection with any generally anxious disposition which the child may have, or with circumstances which tend to provoke anxiety. The parents themselves have difficulty in coping with their child's fear of dentistry; strategy now often consists of stressing "bravery" over "relaxation". Further study is recommended into developing practical strategies to be used by the parents for reducing anxiety.

Anxiety, dental; Pediatric dentistry; Practitioners; Attitudes, parental

Fadavi, Shahrbanoo; Punwani, Indru C.; Adeni, Sikander; Vidyasagar, Dharampuri. Eruption pattern in the primary dentition of premature low-birth-weight children. J Dent Child, 59:120-122, March-April 1992.

This study evaluated the eruption pattern of primary teeth in prematurely-born children with low birth weight who were intubated orally during their neonatal period. A total of thirty-one premature children, with ages between fifteen months to five years, were examined clinically. The birth weights ranged from 1000 grams (n = 9); 1000-1500 grams (n = 15); and 1500 grams or more (n = 7). The age-groups ranged from 12-24 months (n = 8); 25-36 months (n = 15); and 37 months or more (n = 8). The period of intubation ranged from one to seventy-five days, with 55 percent of children intubated for two weeks or less. No correlation was observed between the number of erupted teeth and either birth weight or intubation period. When subjects in different age-groups were evaluated, however, it was found that 75 percent under 24 months of age had delayed

tooth eruption. The average number of erupted teeth for different age-groups were plotted and compared with those for normal, full-term children reported by Infante. Compared with the normal pattern, the prematurely born children under 24 months of age on the average showed 28 percent fewer erupted teeth.
Tooth eruption; Dentition, primary; Low birth-weight; Intubation, neonatal

Waldman, H. Barry: There really are a lot of poor children in the United States. J Dent Child, 59:123-125, March-April 1992.
A review is provided of the extent of poverty in the United States; particularly among children. Nearly one child in four younger than age six is poor. Malnutrition and homelessness are widespread and have reached tragic proportions in children. Pediatric dentists can advocate for these children.
Well-being, children's; Poverty; Pediatric dentistry

Waldman, H. Barry: Do the parent(s) of your pediatric patients smoke? J Dent Child, 59:126-128, March-April 1992.
Parental smoking (and smoking by young teenagers) is the concern of pediatric dentists. A review is provided of the incidence of smoking (400,000-plus deaths every year) and consequences (including intrauterine, and those to infants and children). This review is an effort to stimulate the education efforts of dental practitioners.
Smoking and health; Families and smoking

Waldman, H. Barry: Children without health-care coverage. J Dent Child, 59:129-132, March-April 1992.
A review is provided of various federal reports on the health-care coverage of children. The role of pediatric dentists in helping to advance improvements is emphasized.
Insurance, health [and] dental; Children; Pediatric dentistry

van der Weide, Yvonne; Steen, William H.A.; Bosman, Frederick. Distribution of missing teeth and tooth morphology in patients with oligodontia. J Dent Child, 59:133-140, March-April 1992.
In a population of 196 patients with isolated oligodontia (oligodontia/I) and oligodontia as part of a syndrome (oligodontia/S) the distribution of missing teeth was recorded. The mesiodistal dimensions of the teeth were measured on 82 casts. The aim of the study was to evaluate the distribution of missing teeth and the reduction in tooth morphology. Patients with oligodontia/S were missing significantly ($P < 0.01$) more teeth than patients with oligodontia/I. The difference between males and females with oligodontia/S was significant ($P < 0.01$). The difference between males and females with oligodontia/I, and the differences in distribution of missing teeth over maxilla/mandible and left/right sides were not significant. Almost all differences of the mesiodistal dimensions of the teeth, compared to the data of the control-group, were significant ($P < 0.01$ and $P < 0.001$). The results support the hypothesis that oligodontia shows a polygenic mode of inheritance.
Oligodontia; Genetics

Kopel, Hugh M.: Considerations for the direct pulp capping procedure in primary teeth: A review of the literature. J Dent Child, 59:141-149, March-April 1992.
Direct capping of pulp exposures in primary teeth has remained a controversial procedure for many years. It has been emphasized that this procedure should be limited only to small inadvertent or "mechanical" exposures. Current literature is demonstrating an understanding of previous skepticism, however, and now offers certain procedural considerations—based on scientific investigations—to employ this type of therapeutic approach for "mechanical" or small carious exposures in the primary dentition. This review of the literature presents findings and recommendations to encourage a more favorable success rate of direct pulp capping, comparable to the more invasive techniques such as pulpotomies and pulpectomies in primary teeth.
Direct pulp capping; Pulpotomy; Pulpectomy; Primary teeth

Nordenvall, Karl-Johan: Milk as storage medium for exarticulated teeth: Report of case. J Dent Child, 59:150-155, March-April 1992.
A fourteen-year-old boy had his right lateral maxillary incisor exarticulated accidently during a basketball practice at home on a Sunday evening. The tooth was promptly put in a glass of milk by his quick-thinking mother, who had instantly associated the avulsed tooth with a message she had seen on a milk carton. The periodontal ligament remained vital despite a twelve-hour extraoral period until replantation by the pediatric dentist. Treatment was successful and was followed-up for fifty-nine months, when the same tooth suffered a horizontal root-fracture during a game of field hockey and a coronal fragment was lost. An implant was chosen for replacement therapy.
Milk; Periodontal ligament; Trauma; Extraoral period; Replantation; Treatment, endodontic

Jowharji, Najwa; Noonan, Roger G.; Tylka, Joseph A.: An unusual case of dental anomaly: A "facial" talon cusp. J Dent Child, 59:156-158, March-April 1992.
The talon cusp has been described to be a cusp-like structure originating from the cingulum area of incisor teeth. Previous reports have always reported the talon cusp to originate from the lingual surface of an incisor tooth. This report of an eight-year-old black girl's maxillary incisor suggests, however, that a talon cusp may be labially located: a "facial" talon cusp.
Talon cusp; Incisors, primary [and] permanent; Lingual [or] facial crown surface; Anomalies, dental