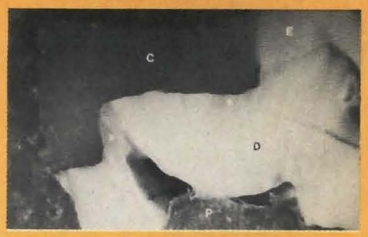

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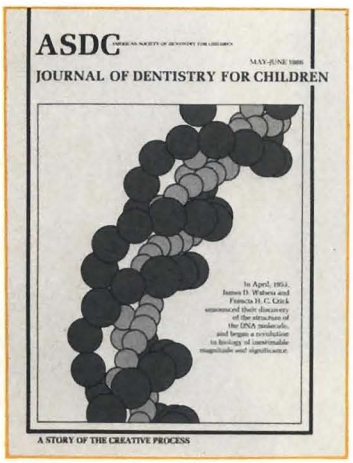
JOURNAL OF DENTISTRY FOR CHILDREN

Volume 53 Number 3 May-June 1986



The cover is symbolic of the molecular structure of deoxyribonucleic acid (DNA). The discovery of the structure by Crick and Watson, with all its biological implications, has been one of the major scientific events of this century. It has literally transformed the science of biochemistry. Cover art by Sharlene Nowak.

CLINIC

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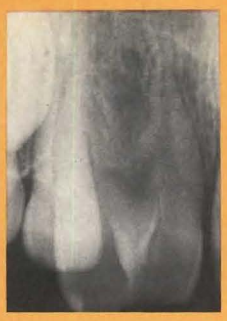

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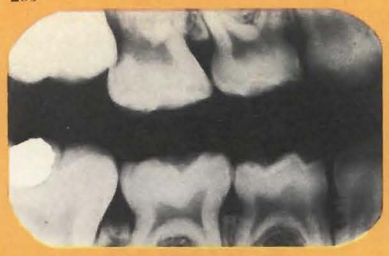

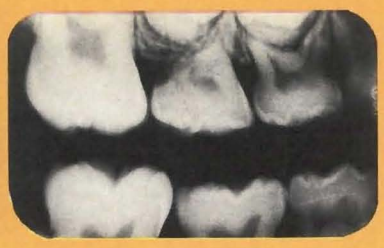

For the busy reader

The cervical margin of amalgam restorations: a radiographic and clinical assessment—page 177

In this study, the cervical margins of class II amalgam restorations in primary molars were assessed both clinically (with mouth mirror and probe) and radiographically; and the two methods were compared, with the type of degradation studied. Using both methods, only 20 percent of the cervical margins seemed to show 'good' adaptation.

Requests for reprints should be addressed to Dr. W. E. van Amerongen, Head, Department of Paedodontics, Free University, de Boelelaan 1115, 1007 MC, Amsterdam, The Netherlands.

An evaluation of pit and fissure caries and caries experience as selection criteria in bitewing examinations for children—page 184

One area of particular concern, on behalf of the pediatric dental patient, is the routine use of low doses of ionizing radiation in the form of bitewing radiographs at semiannual examinations. Patients at high-risk for interproximal caries should receive bitewing examinations at the first clinical evidence of caries. The use of pit and fissure caries as a criterion for determining relative interproximal caries risk may not be reliable in fluoridated communities.

Requests for reprints should be directed to Dr. Jan E. Kronmiller, Assistant Professor, Department of Pediatric Dentistry, School of Dental Medicine, University of Pittsburgh, Pittsburgh, PA 15261.

In vitro assessment of the effect of Scotchbond on the marginal leakage of class II composite restorations in primary molars—page 188

By means of dye penetration, the authors determined the microleakage at the interface of class II composite restorations in thirty-eight extracted or naturally exfoliated primary molars. Scotchbond was used as the bonding agent in half of the prepared cavities' dentin and enamel; the control group (B) used Concise bonding agent in the enamel only. The use of Scotchbond in class II cavities did not prevent marginal leakage under the conditions of this study.

Requests for reprints should be directed to Dr. Gideon Holan, Department of Pedodontics, Hadassah Medical Organization, Hadassah University Hospital, Kiryat Hadassah, P.O.B. 12000, 1-91 120 Jerusalem, Israel.

Localized juvenile periodontitis of the primary dentition—page 193

A case report of an 8-year-old Caucasian boy with localized juvenile periodontitis affecting the primary dentition is described. A method of culturing for *A. actinomycetemcomitans*, frequently assocatied with localized juvenile periodontitis, was used to support the diagnosis.

Requests for reprints should be directed to Dr. Robert L. Mandell, Forsyth Dental Center, 140 The Fenway, Boston, MA 02115.

Localized enamel hypoplasia of the primary canine—page 197

An abnormality of enamel formation, detected only recently in living children, was found in a majority of prehistoric children, some from more than 20,000 years ago. The lesion, roughly a circular area of enamel hypoplasia approximately one millimeter in diameter, occurs on the labial surface of the primary canine tooth. A clinical examination of 2,367 school children in the Vancouver, Canada, area showed the defect occurring in fewer than one percent.

Requests for reprints should be directed to Mark F. Skinner, PhD, Department of Archaeology, Simon Fraser University, Burnaby, B.C. V5A 1S6, Canada

Atypical root resorption of maxillary primary central incisors due to digital sucking: a report of 82 cases—201

To illustrate particular patterns of apical root resorption in primary maxillary central incisors of digital suckers, the radiographs of patients in a private pedodontic practice were evaluated. All cases in this study displayed one of four typical forms of apical root resorption. Radiographic findings appeared in children as young as twenty-five months of age.

Requests for reprints should be directed to Dr. Irving Rubel, 6200 Wilshire Blvd., Suite 1709, Los Angeles, Ca 90048.

Treatment of an unusual case of fusion—page 205

An unusual example of anterior tooth fusion is presented in which the involved tooth had one crown, one talon cusp, two roots, and three root canals. Despite extensive multidisciplinary consultations, treatment failed, resulting in extraction of the tooth.

Requests for reprints should be directed to Dr. Franklin S. Weine, Professor and Director, Postgraduate Endodontics, Loyola University School of Dentistry, 2160 South First Avenue, Maywood, IL 60153.

Ectopic eruption of maxillary first permanent molars: a review—209

This paper presents information on the ectopic eruption of the maxillary first permanent molar, a local eruption disturbance. Two types can be differentiated: reversible and self-correcting, and an irreversible type, in which the permanent molar remains locked. Many treatment methods have been suggested.

Requests for reprints should be directed to Dr. J. Kurol, Senior Consultant Orthodontist, Department of Orthodontics, The Institute for Postgraduate Dental Education, Järnvägsgatan 9, S-552 55 Jönköping, Sweden.

Malocclusion patterns in fourteen children with duchenne's muscular dystrophy—page 215

The purpose of this investigation was to determine whether this myogenic disorder produces a specific pattern of malocclusion. Occlusal relationships, dental arch dimensions, and oral muscular function and morphology were recorded for fourteen boys, aged 8 to 15 years. In all children, a consistently increased arch width indicated oral function patterns that were specific for patients with this type of muscular dystrophy..

Requests for reprints should be directed to Dr. Arild Stenvik, Department of Orthodontics, Dental Faculty, University of Oslo, Geitmyrsveien 71, 0455 Oslo 4, Norway.

Efficacy of fluoride on dental caries reduction by means of a community water supply—page 219

A study of 200 children, patients of the City of New Orleans Health Department Bureau, was made to determine the effects of a fluoridated water supply (the Mississippi River) on their caries experience. Results show that drinking water is a very cost-effective vehicle of fluoride administration, and that a fluoridated community water supply does indeed reduce the numbers of caries lesions and extractions.

Requests for reprints should be directed to Dr. Peter J. Fos, 4400 Trenton Street, Suite G, Metairie, LA 70002.

Hereditary hypophosphatemia rickets: an important awareness for dentists—page 223

Characteristic dental findings of X-linked hypophosphatemia vitamin D-resistant rickets are often the first clinically noticeable signs of the disease. Confirmation with dental findings will permit early diagnosis and prevent deformities that would otherwise occur. Prophylactic full-coverage restorations are indicated.

Rquests for reprints should be directed to Dr. Frank L. Herbert, Associate Professor, Department of General Dentistry, LSU School of Dentistry, LSUSD Box 127, 1100 Florida Avenue, New Orleans, LA 70119-2799.

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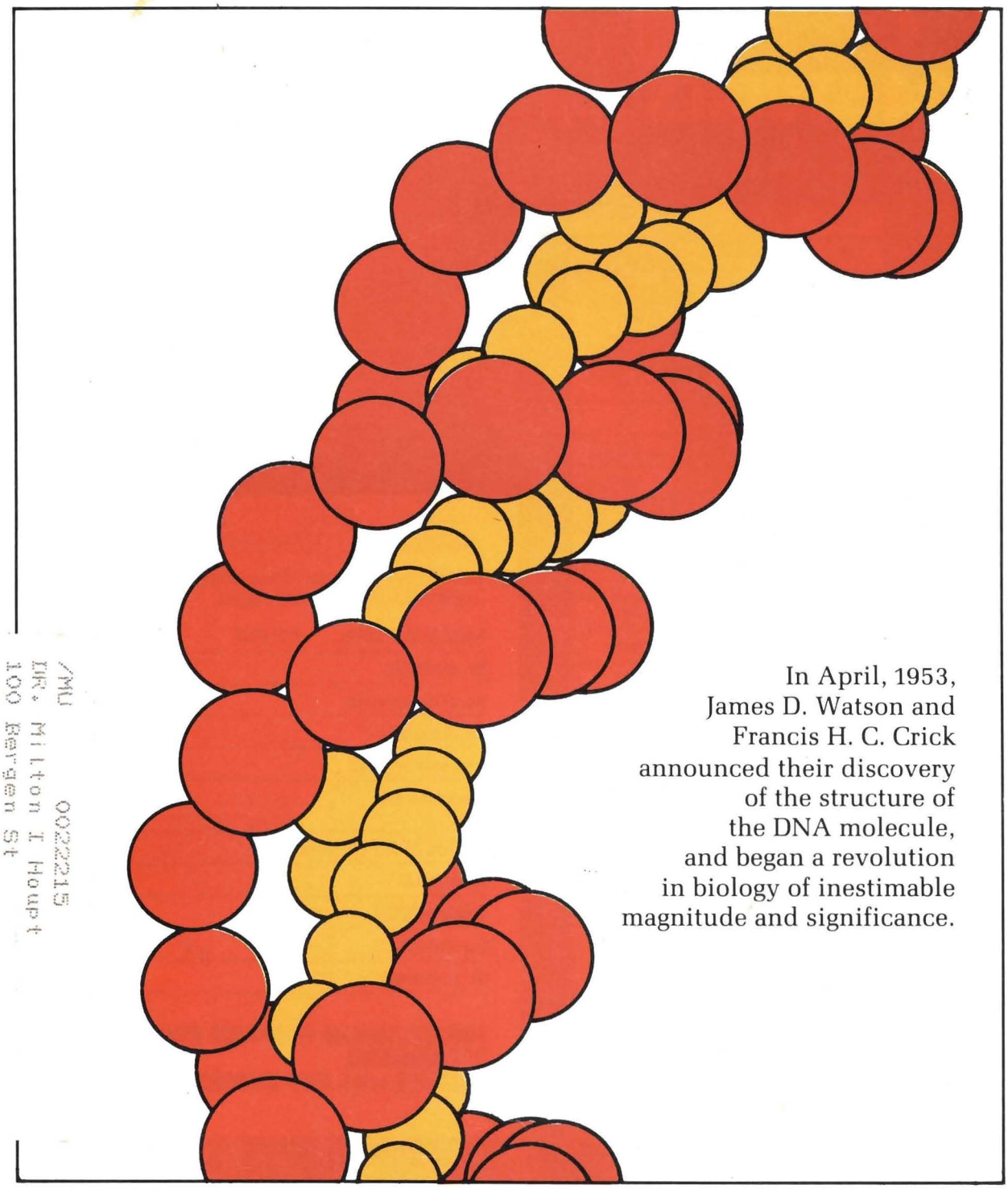


In April, 1953, James D. Watson and Francis H. C. Crick announced their discovery of the structure of the DNA molecule, and began a revolution in biology of inestimable magnitude and significance.

A STORY OF THE CREATIVE PROCESS

The cervical margin of amalgam restorations: a radiographic and clinical assessment

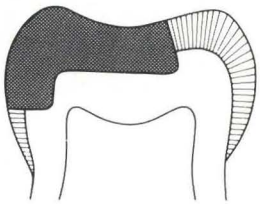
Clinic

Willem E. van Amerongen
Christiaan O. Eggink

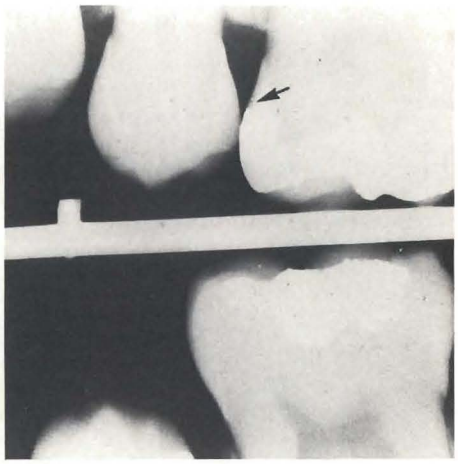
In the past few years, much clinical research has been devoted to the study of the quality of amalgam restorations and to the determination of criteria that would make it possible to establish a standard of durability. Marginal adaptation has been a feature of all this research. That most of these studies, however, focused on the occlusal surface of the amalgam restoration is understandable for reasons of visibility or accessibility, but is cause for casting doubt on the value of the results.[1,2] Admittedly, the quality of the marginal adaptation supplies no information on the condition of the tooth structure in contact with the restoration. The adaptation of the cervical margin of an amalgam restoration is another matter. Although for this part of the restoration, the focus has been less on deterioration of quality over a period of years than on the quality immediately after the placing of the restoration, its importance has long been understood. Many investigators have demonstrated that overhanging or open margins can cause periodontal lesions, because of increased plaque retention and mechanical irritation.[3-7] Nevertheless, many developments have affected the quality of the material itself. Little attention has been paid, however, to improvements of technique that might ensure that good adaptation to the cervical margin is achieved and then

Dr. van Amerongen is Head of the Department of Paedodontics, Free University, de Boelelaan 1115, P.O. Box 7161, 1007 MC Amsterdam, The Netherlands. Professor Eggink is Head of the Department of Conservative Dentistry at Free University.

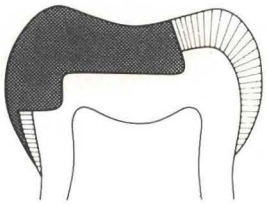
Figure. A through F are different categories of cervical marginal adaptation. Observations were made in one vertical and two horizontal directions (d.n.a. = does not apply).



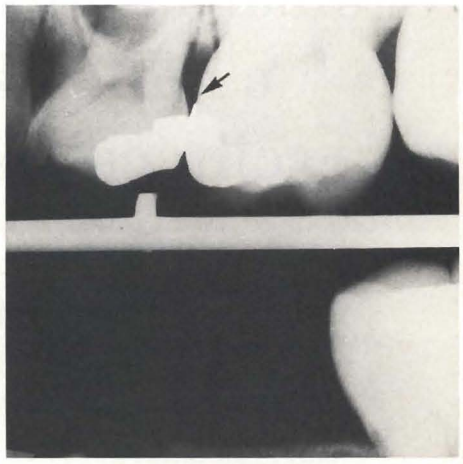
A.
horizontal 1 = d.n.a.
horizontal 2 = ,, ,,
vertical = ,, ,,



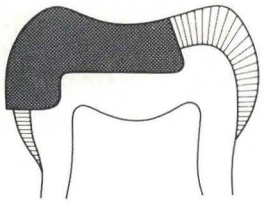
A. Marginal adaptation is good. Neither in vertical nor in horizontal direction is a projecting or receding restoration observed.



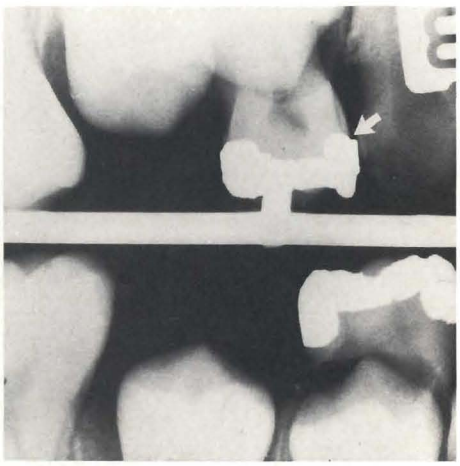
C.
horizontal 1 = pos.
horizontal 2 = O
vertical = pos.



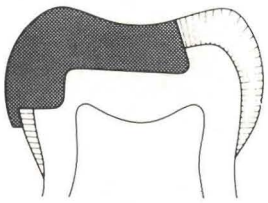
C. Marginal adaptation is not good. A restoration projects in vertical as well as horizontal direction, with smooth transition to the tooth in apical direction.



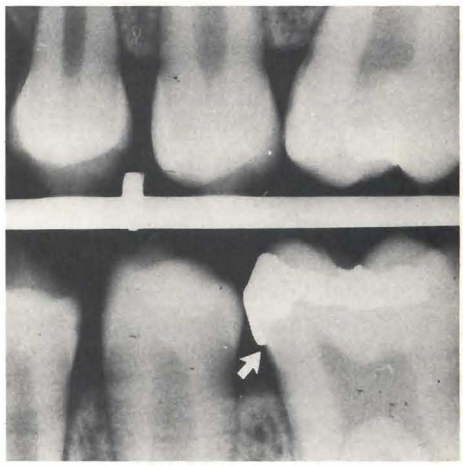
B.
horizontal 1 = pos.
horizontal 2 = d.n.a.
vertical = ,, ,,



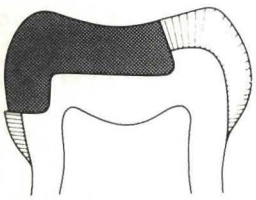
B. Marginal adaptation is not good. A restoration projects in horizontal direction at the level of the cervical margin of the specimen (horizontal 1).



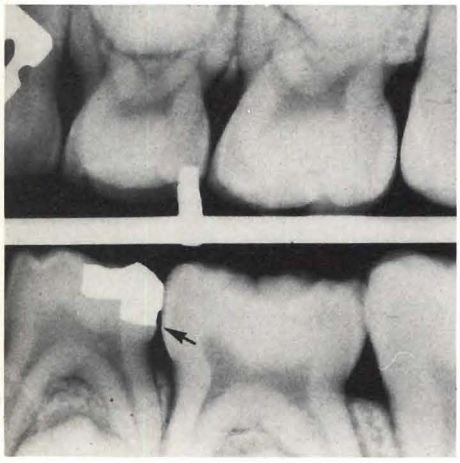
D.
horizontal 1 = pos.
horizontal 2 = ,, ,,
vertical = ,, ,,



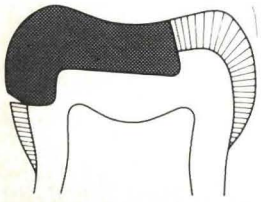
D. Marginal adaptation is not good. A restoration projects horizontal direction at the level of the cervical margin (horizontal 1) and at the level of the more apical margin of the restoration (horizontal 2) as well as in vertical direction.



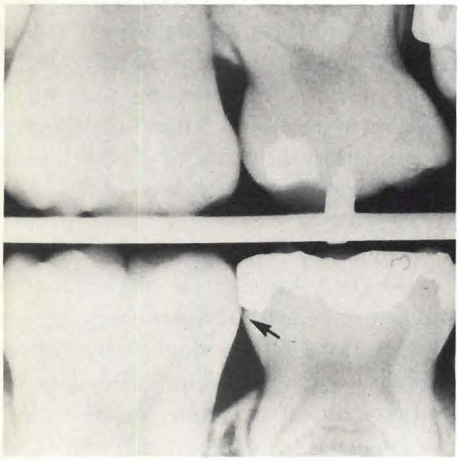
E.
horizontal 1 = neg.
horizontal 2 = d.n.a.
vertical = ,, ,,



E. Marginal adaptation is not good. A restoration recedes in horizontal direction (horizontal 1).



F.
horizontal 1 = 0
horizontal 2 = neg.
vertical = ,, ,,



F. Marginal adaptation is not good. The restoration shows a wedge-shaped groove at cervical margin of the specimen. Proceeding from the assumption that, as in categories C and D, horizontal 1 is localized more occlusally than horizontal 2, the latter is in this case localized at the level of the cervical margin of the specimen and has a negative value at vertical measurement.

maintained. Although it is generally recognized that it is difficult to achieve good adaptation to the cervical margin, there are no reliable data on the percentage of "failures".[8] This is due to the methods used to study this criterion. Some investigators used a mouth mirror and probe, while others preferred radiographic assessment.[9] It was never established whether these methods provide proper insight into the actual situation, or whether one method is superior to the other. Insight into the various types of cervical margin degradation, is, furthermore, likewise limited.

In this study, the cervical margins of class II amalgam restorations in primary molars were assessed both clinically (with mouth mirror and probe) and radiographically. The two methods were compared, and the type of degradation was studied. To determine the true value of the two methods, the restorations in several primary molars were reassessed after shedding (*in vitro* study).

MATERIAL AND METHOD

Clinical assessment

Mouth mirror and probe (Maillefer number 6) were used in the clinical assessment of 400 cervical margins of two- and three-surface amalgam restorations. The ratings assigned at each assessment were:

- ☐ + = the margin is hardly detectable, if at all.
- ☐ ± = the margin is detectable, but the probe does not catch.
- ☐ − = the margin is readily detectable and the probe catches somewhere on the margin.

Each of the three ratings could apply to the restoration with a bulging or protruding proximal surface or defective margins. The study was performed by two investigators. A comparable study revealed sufficient agreement between the two raters (75 percent) to lump their ratings subsequently in a single value judgment.[10] Whenever the ratings differed, the most negative rating was used.

Radiographic assessment

In addition to the clinical assessment, a radiographic assessment was made, using bitewing radiographs obtained with the aid of focusing equipment according to Backer Dirks.[11] Although this technique was introduced for detection of caries on proximal surfaces, it is suitable also for assessment of proximal restorations.

With the aid of these radiographs, cervical-margin

defects were divided into six categories, depending on size and locality (Figure).

Because this division differentiates between horizontal and vertical defects, measurement of these components is possible. For this purpose, we used a stereomicroscope in which one ocular element was graduated, making it possible to measure each defect within 0.05 mm. This was done at the beginning and at the end of each restoration (horizontal Î and horizontal II). The height of the defect was measured as well (vertical). A pilot study of thirty-five restored proximal surfaces revealed 87 percent agreement between the measurements made by the two investigators. It was decided, therefore, that these time-consuming measurements would subsequently be made by one of the two.

The 400 cervical margins of two- and three-surface amalgam restorations considered in the clinical assessment were also used in the radiographic assessment, using the method described above. Of these margins, eighteen could not be assessed due to overlapping of the film images. Categorization makes it possible to establish whether there is agreement between the radiographic assessment of this part of the marginal adaptation, divided into classes A through F, and the clinical assessment expressed in the ratings +, ± or −.

The *in-vitro* study

Since it is conceivable that neither radiographic nor clinical assessment can provide an adequate impression of the situation, forty-six teeth were submitted to an *in vitro* study, using the criteria of assessment and ratings described for clinical assessment.

Statistical analysis

Since presentation and interpretation of the results were the sole objectives, statistical methods were not used in analyzing the data obtained in this study. Only in the comparison of related results was the X^2 test occasionally applied, proceeding from the postulate that the fail probability should not exceed 5 percent.

RESULTS

As indicated in the preceding section (Figure), radiographic assessment was not only based on a division into categories, but also included measurements. Of the defects observed, both the vertical and the horizontal dimensions were measured.

Table 1 ☐ Distribution of 382 cervical margins over categories A through F. The degree of degradation in horizontal and/or vertical direction per category is expressed in millimeters.

	protruding			shrinkage			
	positive deviation			negative deviation			
categories	Hor 1	Hor 2	Vert	Hor 1	Hor 2	Vert	number
A	----	----	----	----	----	----	169
B 0 – 0.25	50						
0.25 – 0.50	18						
0.50 – 0.75	2						71
0.15 – 1.00	1						
1.00 – 1.50	0						
1.50 –	0						
C 0 – 0.25	10		9				
0.25 – 0.50	7		8				
0.50 – 0.75	1		1				18
0.15 – 1.00	0		0				
1.00 – 1.50	0		0				
1.50 –	0		0				
D 0 – 0.25	16	19	16				
0.25 – 0.50	28	30	27				
0.50 – 0.75	15	11	9				96
0.15 – 1.00	2	2	10				
1.00 – 1.50	3	2	3				
1.50 –	32	32	31				
E 0 – 0.25				7			
0.25 – 0.50				2			
0.50 – 0.75				0			9
0.15 – 1.00				0			
1.00 – 1.50				0			
1.50 –				0			
F 0 – 0.25					10	12	
0.25 – 0.50					5	5	
0.50 – 0.75					2	1	19
0.15 – 1.00					0	0	
1.00 – 1.50					1	0	
1.50 –					1	1	
Total							382

Table 1 shows that 44 percent of the cervical margins on the bitewing radiographs were assessed as good (category A), 49 percent as protruding (categories B, C and D) and 7 percent as open (categories E and F). Of the seventy-one assessments in category B, sixty-eight proved to protrude no more than 0.5 mm in horizontal direction. The same (but in the horizontal as well as in the vertical direction) applied to seventeen of the eighteen assessments in category C.

In category D (which encompasses 25 percent of all assessments), however, the degree of protrusion showed a deviant pattern. In thirty-two cases, i.e. in one-third of all observations in this category, the restoration was protruded 1.5 mm or more. The data on shrunken restorations reveal situations more or less identical to those reported for categories B and C; in most cases the amount of shrinkage did not exceed 0.5 mm.

Comparison of the clinical assessments with the radiographic categories yields the results presented in Table 2.

In can be deduced from Table 2 that there was little

Table 2 ☐ Radiographic assessment of the cervical margin, with A indicating good marginal adaptation and the other letters indicating various categories of cervical margin degradation. How this criterion was clinically assessed is also indicated per category.

Radiographic assessment Category	Number of facets	Clinical assessment % +	% ±	% −
A	169	46.0	32.8	21.1
B	71	39.8	22.8	37.4
C	18	59.6	25.5	14.9
D	96	34.3	29.5	36.2
E	9	6.4	45.2	48.4
F	19	31.5	15.8	52.6

agreement between the radiographic assessments in categories and the clinical assessments in classes. When the data are lumped in "good" and "not good" assessments (Table 3), however, no significant differences are found between clinical and radiographic assessments ($X_1^2 = 0.77$; $p = 0.4$). In other words, one method of assessment is not superior to the other.

A second look at the data in Table 3 also reveals, however, that of the 382 cervical margins assessed, 41 percent were clinically assessed as "good". These results are halved when the two methods are taken together: of the 382 cervical margins, only 78 (20.4 percent) were qualified as "good", versus 35 percent as "not good". This is interpreted to mean that about 50 percent of the clinical assessments yielded results that differed from those of the radiographic assessments.

In summary, the data seem to warrant the conclusion that it makes no difference whether one assesses clinically or radiographically. In order to gain the most realistic impression of the true situation in terms of cervical marginal adaptation, however, it is preferable to use both methods. Even so, the degree of protrusion or degradation of the cervical margin can influence the results of both methods of assessment. To gain some insight into this influence, the data were rearranged on the basis of the unit of measurement (Table 4 versus Table 1).

All assessments of radiographs encompassed one vertical and two horizontal observations. A comparison with clinical assessments should be confined to observations that can also be made with the dental probe. For the various categories this implies that the following measurements in this study are of value:

- ☐ Category A: no measurements were made.
- ☐ Category B: the measurements of Horizontal I.
- ☐ Category C: measurements (smooth transition between restoration and tooth).
- ☐ Category D: the measurements of Horizontal II.
- ☐ Category E: the mesaurements of Horizontal I.
- ☐ Category F: the measurements of Horizontal II.

Because no measurements were made in category A and the measurements of category C are not clinically verifiable, the data on these two categories are not included in Table 4. The remaining categories have been lumped in one group in which the cervical margin was radiographically assessed as "not good". Only the

Table 3 ☐ Comparison of cervical margins clinically and radiographically assessed as "good" and as "not good".

Radiographically \ Clinically	good	not good	
good	78	91	169
not good	79	134	213
	157	225	382

Table 4 ☐ Degree of cervical margin degradation expressed in millimeters, comparing radiographic (bitewing radiographs) with clinical assessment.

Radiographic assessment Hor.I/Hor. II	Number of facets	Clinical assessment % +	% ±	% −
0.00 – 0.25	97	40.8	26.2	33.0
0.25 – 0.50	62	34.6	24.2	41.2
0.50 – 0.75	15	26.8	19.5	53.7
0.75 – 1.00	3	10.0	10.0	80.0
1.00 – 1.50	3	0.0	8.3	91.7
1.50 –	33	41.2	41.2	17.6

degradation measured in horizontal direction, which is relevant in a comparison with the clinical assessment, is shown in order of magnitude.

Table 4 shows that, with the exception of defects in excess of 1.5 mm, the percentage of negative clinical assessments increases with an increase in the degree of degradation in horizontal direction. This warrants two conclusions: the more the restoration protrudes, the more likely is clinical assessment to lead to a negative qualification; a clinical assessment of "not good" can be confirmed by radiographic assessment, and vice versa. In this way the postulate that a radiograph does not permit adequate assessment of a class II restoration due to overprotrusion, is largely refuted.

The comparative study of clinical and radiographic assessments has shown that the use of one method reveals twice as many "good" marginal adaptations as does the use of both methods, in the attempt to assess the quality of the cervical margin. In order to establish whether in this way the true situation is approximated, the following *in vitro* study was performed: a number of patients were asked to save the restored primary molars after shedding. Of the fifty primary molars thus obtained, forty-six had a multiple-surface restoration (thirty-six with a two-surface and ten with a three-surface amalgam restoration). The fifty-six cervical margins of these teeth were submitted to the criteria of assessment and ratings used in clinical assessment. The results thus obtained revealed a "good" cervical marginal adaptation in only eleven instances (20 percent), as shown in Table 5. This percentage is in agreement with

Table 5 ☐ Clinical assessment of shed primary molars (*in vitro* study).

+	±	−	total
11	18	27	56

the results of combined clinical and radiographic assessments.

Not all the shed teeth in the *in-vitro* study had been clinically assessed before shedding, but bitewing radiographs that were obtained for caries diagnosis were available. Because the restorations in the shed teeth differed from those considered in the clinical and radiographic assessment, it had to be demonstrated for purposes of comparison that the restorations in the forty-six shed teeth were of the same quality as those studied before shedding. After radiographic assessment, it was found that the group of 382 cervical margins (Tables 2 and 4) did not differ significantly from the group of shed teeth, either in terms of a division into categories ($X^2_5 = 4.28$; $p = 0.5$) or in terms of a division according to degree of degradation ($X^2_6 = 5.29$; $p = 0.5$). These data are presented in Table 6.

The marginal adaptation was assessed as "good" in virtually the same percentage of cases: 44 percent in the former group, and 43 percent in the group of the shed teeth. These results warrant the conclusion that the result obtained by means of one of the methods of assessment used in this study of quality must be divided by two in order to gain an accurate impression of the percentage of good cervical marginal adaptations.

Table 6 ☐ Results of radiographic assessment of fifty-six cervical margins of shed primary molars compared with the results presented in tables 2 and 4. Percentages in parentheses.

Radiographic assessment		Number of facets
Category	Shed teeth	Result in table 2
A	24 (43)	169 (44)
B	12 (21)	71 (19)
C	5 (9)	18 (5)
D	9 (16)	96 (25)
E	2 (4)	9 (2)
F	4 (7)	19 (5)
Total	56	382

Radiographic assessment		Number of facets
Hor.I/Hor.II	Shed teeth	Result in table 4
0.00	24 (43)	169 (44)
0.00 – 0.25	19 (34)	97 (25)
0.25 – 0.50	10 (18)	62 (16)
0.50 – 0.75	2 (4)	15 (4)
0.75 – 1.00	0 (0)	3 (1)
1.00 – 1.50	0 (0)	3 (1)
1.50 –	1 (2)	33 (9)
Total	56	382

DISCUSSION

Despite the use of the Backer Dirks focusing equipment, successive radiographs of the same teeth showed minor differences due to slight differences in positioning and beam direction. This means that possible dimensional changes were not always discernible. We did not differentiate, therefore, between the radiographs obtained every six months (at least not in the assessment of the proximal surfaces). The bitewing radiogaphs selected for this purpose yielded, therefore, no absolute values when measured stereomicroscopically, but gave a relative impression of the degree of degradation in horizontal and in vertical directions.

Regardless of the minor deviations from the standard procedure in obtaining bitewing radiographs, there is another factor that made it impossible to use the measurements performed as absolute values. Because the radiograph shows a projection of a three-dimensional object on a flat plane, the observation can only be regarded as the resultant of the criterion to be assessed (in this case the cervical margin). In the end, only the most prominent part becomes visible.

In the classification of the cervical marginal adaptation of class II amalgam restorations, the following rule of thumb was applied: The horizontal part of the protruded or shrunken restoration should be as small as possible. A deviation in the direction of the beam in the horizontal plane would make the restoration seem too wide in this area. The value measured in a vertical direction should be as great as possible. A deviation of the beam in this direction would make the restoration seem too small in this area. Applying this rule, only eighteen of the 400 cervical margins to be assessed had to be discarded because of overlap.

One of the questions considered in comparing the results of clinical and radiographic assessments of the quality of the adaptation of the cervical margin was whether a correlation could be found between the degree of degradation observed and the chance of verification with the aid of the dental probe. The fact that only measurements in a horizontal direction are of direct importance in this comparison, does not imply that the values measured in the vertical sense would exert no influence on the result. After all, a high degree of vertical projection may make the horizontal deviation clinically invisible, because it is localized at a level not readily accessible to the probe, *i.e.* deep beneath the gingival margin.

This may explain why the number of clinically assessed deviations rated as – diminishes significantly when the radiographically assessed deviations exceed 1.5 mm (Table 4). In these cases the value measured in horizontal direction is nearly always associated with the same degree of projection in a vertical direction (Table 1).

One of the conclusions to be drawn from Table 3 is that neither clinical nor radiographic assessment of the cervical margin can adequately detect and qualify all defects. An amalgam restoration that inadequately fills the angle between the vertical wall and the cervical margin of the specimen can be clinically identified as such, but not radiographically. On the other hand, class II amalgam restorations with a protruding cervical margin, but with a smooth transition between restoration and tooth (category C) can be radiographically identified as such, but not clinically.

The number of multiple-surface restorations in primary molars that seemed to show good cervical marginal adaptation by both clinical and radiographic assessments, did not exceed 20 percent (Table 3). In view of the large number of defects, therefore, both methods of assessment will have to be used in order to obtain optimal information. This conclusion is supported by the results of the *in vitro* study.

The conclusions drawn from a comparative study of clinical and radiographic assessments and an associated *in-vitro* study are based on different data. The results of clinical assessment reflected the conditions of the multiple-surface amalgam restorations at a particular time, while the radiographic assessment covered still other times. Because the method of clinical assessment used, however, is very accurate, we proceeded from the assumption that dimensional changes that may have occurred would not be discernible. This postulate is confirmed in a study by Philips, who found that no dimensional changes were clinically observed in restorations of six different types of amalgam with known physical characteristics.[12] For assessment of the cervical margin it seems unnecessary, therefore, to differentiate the ages of the restorations. In another publication, this postulate is confirmed on the basis of a comparison of restorations aged 0, 6, 12, and 18 months.[10]

Although the data for the present study were collected from class II amalgam restorations, the results show that any sufficiently radiopaque restoration can be assessed by the methods described.

SUMMARY AND CONCLUSIONS

In a study of the condition of the cervical margin of class II amalgam restorations, a radiographic method of assessment was compared with clinical assessment using mouth mirror and probe. The radiographic method assesses the type of defect of the cervical margin on the one hand, and the degree of defect on the other. In addition, an *in-vitro* study was performed of forty-six primary molars with multiple-surface amalgam restorations, in order to evaluate the results of the comparative study.

Comparison of the results of clinical assessment with those of radiographic assessment revealed that:

- ☐ There is no correlation between the type of defect of the cervical margins of the amalgam restorations and their clinical assessment.
- ☐ There is a correlation between the degree of cervical margin defect and clinical assessment, as long as the defect does not exceed 1.5 mm.
- ☐ There is no significant difference between the clinical and the radiographic methods of assessment.
- ☐ Using both methods, only 20 percent of the cervical margins seemed to show "good" adaptation.

The results of the *in-vitro* study warrant the conclusion that:

- ☐ In this study, too, only 20 percent of the cervical margins showed good adaptation.
- ☐ The restorations of the forty-six shed primary molars were of the same quality as those involved in the comparative study.
- ☐ These two conclusions warrant the postulate that, when only one of the two methods of assessment is used, the results must be divided by two in order to gain an accurate impression of the condition of the cervical margins.

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An evaluation of pit and fissure caries and caries experience as selection criteria in bitewing examinations for children

Jan. E. Kronmiller, DDS
Ronald F. Nirschl, DDS, MSD
Thomas G. Zullo, PhD

There has recently been considerable attention focused on minimizing exposure of pediatric dental patients to low doses of ionizing radiation.[1,2] The use of routine radiographic examinations is contraindicated according to recent guidelines.[1,2] These guidelines emphasize maximizing the benefit-to-risk ratio of diagnostic radiographic examinations. One particular area of concern is the routine use of bitewing radiographs at semiannual examinations.[3,4]

The use of posterior bitewing radiographs for detection of interproximal caries has been well substantiated.[5-7] Studies have shown a range of 28 to 75 percent of all posterior interproximal lesions in preschool children are detectable only by bitewing radiographs.[8,9] Stephens *et al* evaluated the use of bitewing radiographs in thirteen- to thirty-year-old patients, and found that 89 percent had interproximal caries that could only be detected by bitewing radiographs.[10]

While bitewing radiographs are indicated at the initial oral examination, when posterior teeth are in contact, their routine use at semiannual examinations is not.[3,4] Selection criteria have been proposed for determining the relative caries risk of each patient and, consequently, the frequency of bitewing examinations. Patients at high risk for interproximal caries should receive bitewing examinations more frequently than those at low risk. Criteria which indicate a high risk of caries include: poor oral hygiene, fluoride deficiency, high carbohydrate diet, poor family dental health, developmental enamel defects, developmental disability and acute or chronic medical history, and genetic abnormality. It has also been stated that bitewing examinations are recommended at the first clinical evidence of caries.[4]

The multifactorial etiology of dental caries makes it difficult to determine the relative importance of each of the criteria that place a patient at high risk for interproximal caries.[11,12] The purpose of this study was to examine the relationship of pit and fissure caries and caries experience to interproximal caries.

METHODS AND MATERIALS

Data for this study were collected by examination of the records of patients in the pediatric dental clinic of the University of Pittsburgh. The 100 records examined were selected using the following guidelines:

- ☐ Either of two investigators completed the oral examination at the patient's initial appointment.
- ☐ Patients were between the ages of three and twelve years.
- ☐ Bitewing radiographs were diagnostic for the detection of interproximal caries by the following cri-

Dr. Kronmiller is Assistant Professor, Department of Pediatric Dentistry; Dr. Nirschl is Associate Professor, Department of Pediatric Dentistry; and Dr. Zullo is Professor and Director, Division of Learning Resources, School of Dental Medicine, University of Pittsburgh, Pittsburgh, PA 152681.

Table 1 ☐ Prevalence of pit and fissure and interproximal caries.

Dentition	Number of patients	Percent of patients with pit and fissure caries	Percent of patients with proximal caries
Primary	14	46	50
Mixed	72	60	62
Permanent	14	71	14
All	100	58	54

Table 2 ☐ Prevalence of pit and fissure and proximal surface restorations.

Dentition	Number of patients	Percent of patients with pit and fissure surface restorations	Percent of patients with proximal surface restorations
Primary	14	0	7
Mixed	72	46	26
Permanent	14	21	14
All	100	36	22

Table 3 ☐ Prevalence of caries-free patients by age.

Age	Number of patients examined	Number of patients who were caries-free	Percent of patients who were caries-free
3	2	1	50.0
4	5	1	20.0
5	5	0	0.0
6	10	4	40.0
7	15	2	13.3
8	18	1	5.5
9	8	0	0.0
10	6	0	0.0
11	13	1	7.6
12	18	3	16.6
All	100	13	13.0

Table 4 ☐ Relationship of occlusal, buccal and lingual caries to interproximal caries.

a. All dentitions

		Interproximal caries −	+	Total
Occlusal, buccal and lingual caries	−	18	24	42
	+	28	30	58
	Total	46	54	100

$X^2 = .11$

b. Primary dentition

		Interproximal caries −	+	Total
Occlusal, buccal and lingual caries	−	4	5	9
	+	3	2	5
	Total	7	7	14

Fisher's Exact Probability Test shows no statistically significant difference.

c. Mixed dentition

		Interproximal caries −	+	Total
Occlusal, buccal and lingual caries	−	10	19	29
	+	17	26	43
	Total	27	45	72

$X^2 = .03$

d. Permanent dentition

		Interproximal caries −	+	Total
Occlusal, buccal and lingual caries	−	4	0	4
	+	8	2	10
	Total	12	2	14

Fisher's Exact Probability Test shows no statistically significant difference.

teria: Radiographs were considered nondiagnostic for interproximal caries detection, if the distal contacts of the canines or the distal-most contact areas of the quadrants were not clearly visible. A pair of radiographs of a primary dentition were considered nondiagnostic for interproximal caries detection, if two or more contact areas overlapped. A pair of radiographs of a mixed or permanent dentition was considered nondiagnostic for interproximal caries detection, if four or more contact areas were overlapped.

A positive finding for interproximal caries was recorded at the first evidence of a defect in the enamel. Radiographs were examined on a viewbox in a dimly lighted room. All radiographs were examined once.

Prior to the initiation of the study, a pilot study was conducted. Twenty-two cases were evaluated using the previously stated criteria. The primary purpose of the pilot study was to evaluate the percent agreement between the two investigators. The percent agreement for detection of interproximal caries was 79.1 percent.

Data collected from these records included age of patient, sex, stage of dentition; number of carious occlusal, buccal and lingual surfaces; number of carious proximal surfaces; number of filled proximal surfaces; number of filled occlusal, buccal and lingual (pit and fissure) surfaces. Data were collected for posterior teeth only. Patients in this study lived in a fluoridated community.

RESULTS

For all patients, 58 percent had carious pit and fissure surfaces, 54 percent had carious proximal surfaces and 39 percent had both pit and fissure and interproximal caries lesions. Caries experience was determined by the number of filled pit and fissure and proximal surfaces. Thirty-six percent had restorations in pit and fissure surfaces and 22 percent had filled proximal surfaces. A breakdown of carious and filled surfaces by stage of dentition is given in Tables 1 and 2.

Overall, 13 percent of the patients examined were caries-free. The prevalence of caries-free patients as a function of age is given in Table 3. Thirty (51 percent) of the fifty-eight patients with pit and fissure caries had untreated interproximal caries (Table 4a). Forty percent of the patients in the primary dentition stage who had pit and fissure caries also had interproximal caries (Table 4b); whereas 60 percent of the patients in the mixed dentition stage who had pit and fissure caries had untreated interproximal caries (Table 4c). Only 20 percent of the patients in the permanent dentition stage who had pit and fissure caries had interproximal caries (Table 4d).

Twenty-three (59 percent) of the thirty-nine patients who had a history of caries also had interproximal caries (Table 5a). The only patient, however, in the primary dentition stage who had restorations had no interproximal caries (Table 5b). Of the patients with a caries experience in the mixed and permanent dentitions, 65 percent and 25 percent, respectively, had interproximal caries (Tables 5c and 5d).

The relationship of caries experience to new interproximal caries was further examined by looking at those patients who had a history of interproximal caries. Of the twenty-two patients who had proximal restorations, sixteen had new interproximal lesions (Table 6a). All but one of these sixteen patients were in the mixed dentition stage.

The relationship between interproximal caries and interproximal restorations in the primary dentition is given in Table 6B. Of the nineteen patients in the mixed dentition stage who had a history of interproximal restorations, fifteen were found to have new interproximal lesions. In the mixed dentition, the relationship of interproximal caries to interproximal restorations was statistically significant (Table 6c). The relationship between interproximal caries and interproximal restorations in the permanent dentition is given in Table 6d.

DISCUSSION

The prevalence of caries-free patients as a function of age closely resembles data previously reported.[13] In Bohannan's data for permanent teeth, comparison of data for twelve-year-old patients shows general agreement.[13] Interproximal caries accounted for 34 percent of the total caries in this study. This is dissimilar to the distribution of caries by tooth surface reported for fluoridated communities, but the current study included caries in primary teeth.[13] Relatively few patients examined in the current study were in the primary and early permanent dentition stages, whereas a large proportion of patients were in the mixed dentition stage. The patients in the mixed dentition stage represent individuals whose proximal surfaces were at risk for caries development for a longer period of time than the patients in the primary and early permanent dentition stages. Results of a recent survey showed peaks in incremental rates of caries experience in permanent teeth at ages eight to nine years, eleven to thirteen years, and fifteen to sixteen years.[14] These peaks would correspond to first permanent molars, premolars, and second permanent molars, respectively, at risk for proximal caries for periods of two to three years. Studies of lesion progression in proximal surfaces show results comparable to this two-to-three-year period.[15-17] A recent longitudinal caries

Table 5 ☐ Relationship of interproximal caries to caries experience.

a. All dentitions

		Interproximal caries −	+	Total
Caries experience	−	30	31	61
	+	16	23	39
	Total	46	54	100

$X^2 = .35$

b. Primary dentition

		Interproximal caries −	+	Total
Caries experience	−	6	7	13
	+	1	0	1
	Total	7	7	14

Fisher's Exact Probability Test shows no statistically significant difference.

c. Mixed dentition

		Interproximal caries −	+	Total
Caries experience	−	15	23	38
	+	12	22	34
	Total	27	45	72

$X^2 = .01$

d. Permanent dentition

		Interproximal caries −	+	Total
Caries experience	−	9	1	10
	+	3	1	4
	Total	12	2	14

Fisher's Exact Probability Test shows no statistically significant difference.

Table 6 ☐ Relationship of interproximal caries to interproximal fillings.

a. All dentitions

		Filled interproximal surfaces −	+	Total
Carious interproximal surfaces	−	40	6	46
	+	38	16	54
	Total	78	22	100

$X^2 = 3.07$

b. Primary dentition

		Filled interproximal surfaces −	+	Total
Carious interproximal surfaces	−	6	1	7
	+	7	0	7
	Total	13	1	14

Fisher's Exact Probability Test shows no statistically significant difference.

c. Mixed dentition

		Filled interproximal surfaces −	+	Total
Carious interproximal surfaces	−	23	4	27
	+	30	15	45
	Total	53	19	72

$X^2 = 22.10$ ($p = .01$)

d. Permanent dentition

		Filled interproximal surfaces −	+	Total
Carious interproximal surfaces	−	11	1	12
	+	1	1	2
	Total	12	2	14

Fisher's Exact Probability Test shows no statistically significant difference.

study of permanent tooth surfaces at risk also emphasized the importance of consideration of each interdental area as a unit in determining the time of carious attack on the proximal surfaces of teeth.[18] It would appear from these results that recommendations for frequency of radiographs for interproximal caries detection should be different for children in the mixed dentition stage than for children in the primary and early permanent dentition stages. This should account for the period during which proximal surfaces were at risk for caries.

It has been demonstrated that in fluoridated communities 94 percent of the caries lesions involved noninterproximal surfaces.[13] The present study showed a higher relative proportion of interproximal caries. Nonetheless, a poor correlation between the radiographic diagnosis of interproximal caries and pit and fissure caries exists in this study. This suggests that the use of pit and fissure caries as a criterion for determining interproximal caries risk may not be reliable in fluoridated communities.

In this study, the relationship between interproximal caries and caries experience was found not to be statistically significant. The relationship between previous interproximal caries and new interproximal caries was found only to be statistically significant in the mixed dentition stage.

CONCLUSIONS

- ☐ The use of pit and fissure caries as a criterion for determining relative interproximal caries risk may not be reliable in fluoridated communities.
- ☐ The use of caries experience as a criterion for determining relative interproximal caries risk may not be reliable in fluoridated communities.
- ☐ The use of interproximal caries experience as a criterion in determining relative interproximal caries risk may be reliable in the mixed dentition stage.

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In vitro assessment of the effect of Scotchbond on the marginal leakage of class II composite restorations in primary molars

Gideon Holan, DMD
Anna B. Fuks, CD
Rafael Grajower, PhD
Aubrey Chosack, BDS, MSD

Development of composite resins in the last decade brought hope that they could be used as posterior restorative materials. Several clinical studies were undertaken, and some are still in progress.[1-8] Clinical researchers were encouraged by the report that, in addition to improved esthetics, the marginal integrity of composite material used with the acid-etch technique is comparable to or better than amalgam.[9] Excessive wear, however, is the most serious limitation of these materials, and it is the major factor restricting their use as a replacement for amalgam restorations in permanent teeth.[1-3] Composite restorations have been utilized successfully in primary molars, and this technique has been recommended by some authors when the "life expectancy" of the tooth is limited to three years.[8]

Even if the problem of wear was solved and composite resins were used as substitutes for class I amalgam restorations, leakage at the gingival margin would still preclude their use in class II cavities. It would be ideal to have a restorative material with adhesive properties that could eliminate the problem of leakage at the gingival margin. Commercial dental adhesive resins have appeared only recently on the market. Several authors demonstrated that the application of one of these resins (Scotchbond†) helped in preventing microleakage.[10-13]

Dr. Holan is Instructor, Department of Pedodontics; Dr. Fuks is Senior Lecturer, Department of Pedodontics; Dr. Grajower is Associate Professor and Head, Unit for Dental Materials; Dr. Chosack is Associate Professor, Department of Pedodontics, The Hebrew University, Hadassah Faculty of Dental Medicine, Jerusalem, Israel.

The objective of the present investigation was to determine, by means of dye penetration, the microleakage at the interface of class II composite restorations placed in extracted or exfoliated primary molars, employing Scotchbond as a bonding agent.

MATERIALS AND METHODS

The experimental material consisted of thirty-eight extracted or naturally exfoliated primary molars. Some of these teeth were intact, some had minimal to moderate caries lesions, and others had amalgam restorations that were removed.

Forty-eight conventional class II cavities were prepared in either the mesial or distal surface or in both surfaces of these teeth. The enamel adjoining the cavities was beveled at an angle of 45 degrees to a width of 1 to 1.5 mm. The total sample of teeth was randomly divided into two groups of twenty-four class II cavity preparations. The beveled enamel in both groups was etched, and different bonding agents were used as follows:

- ☐ Group A (Experimental): Scotchbond was used as a bonding material to dentin and enamel.
- ☐ Group B (Control): The dentin was covered with Dycal* and the conventional bonding agent, supplied with Concise†, was used on the enamel only.

*L.D. Caulk Company, Milford, Delaware 19963
†Dental Products, 3M St. Paul, MN 55144

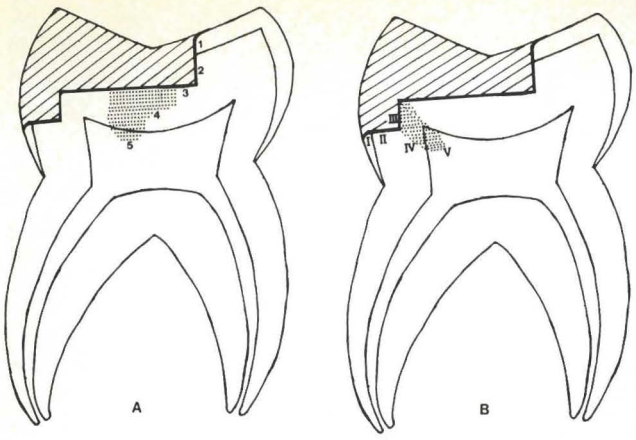

Figure 1. Diagram showing the method of evaluating the various depths of dye penetration at the occlusal (A) and proximal (B) surfaces.

After placing the bonding material, metal matrix bands were adapted to the teeth by means of a Tofflemire matrix holder. The composite resin (P10†) was used as the restorative material in both groups. This resin was placed initially at the proximal box and condensed with an amalgam plugger. Carving and smoothing of the occlusal surface was accomplished with a ball burnisher.

After the material had set, the occlusal surfaces and margins were finished with Alpine stones, followed by Sof-lex† finishing discs. The proximal surfaces were left untouched, since smoothness was achieved by close adaptation of the matrix band, and in cases where excess was inadvertently left at the margins, it was removed with Sof-lex discs. The teeth were kept at room temperature, but in a moist environment, to prevent dehydration.

Ten days later the restored teeth were thermocycled 60 times between 4°C ± 2 and 60°C ± 2 with dwell times of one minute in each bath. Following thermocycling, the teeth were triple-coated with a layer of nail polish, melted utility wax and a second layer of nail polish, excluding the restoration and approximately 0.5 to 1 mm of the surrounding enamel margin.

The coated teeth were then immersed in a 2 percent solution of basic fuchsin for twenty-four hours. After removal from the dye, the coatings were stripped from the teeth by peeling and, where necessary, by scraping. The teeth were then thoroughly washed in water, dried and embedded in acrylic resin. Mesiodistal sections were obtained by grinding off the embedded teeth parallel to their axes. The sections were polished under running water and examined under a dissecting microscope. The procedure of grinding and polishing was repeated to allow evaluation of five to seven sections of each tooth, depending on the bucco-lingual width of the proximal part of the restoration.

The depth of dye penetration was evaluated by using a modified standardized system as suggested by Going *et al* and modified by Fuks and Shey (Figures 1 a, b).[14,15]

- ☐ Degree 0: No penetration of dye.
- ☐ Degree 1 or I: Penetration of dye along the occlusal or gingival wall limited to the enamel.
- ☐ Degree 2 or II: Penetration of dye along the complete length of the occlusal or gingival wall but not along the pulpal wall.
- ☐ Degree 3 or III: Penetration of dye along the pulpal wall.
- ☐ Degree 4 or IV: Diffusion of dye into the dentin under the pulpal wall.

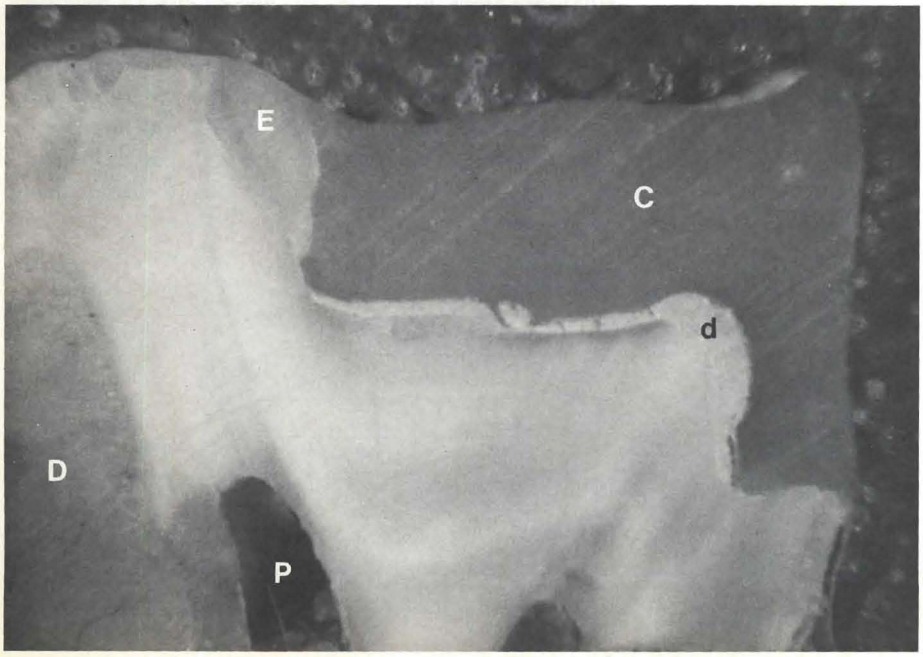

Figure 2. Photograph showing a longitudinal section of a class II composite restoration without Scotchbond. Notice the absence of dye penetration (degree 0) at the occlusal and cervical margins. C = composite; E = enamel; D = dentin; P = pulp; d = dycal.

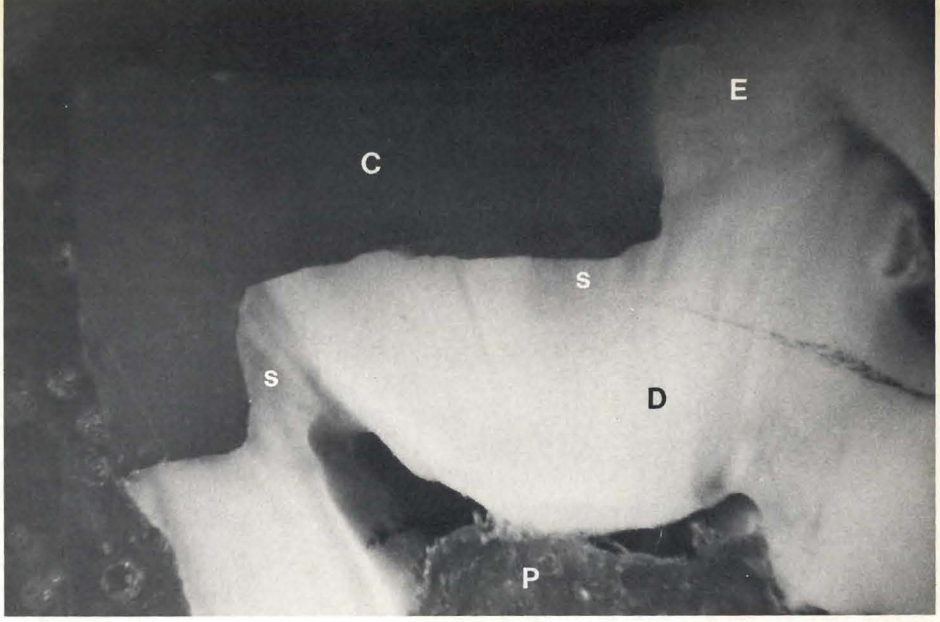

Figure 3. Photograph of a longitudinal section of a class II composite restoration of the experimental group (with Scotchbond). Notice the dye penetration into the dentin at the occlusal surface (degree 4) and into the pulp at the proximal surface (degree V). C = composite; E = enamel; D = dentin; P = pulp; s = stain.

□ Degree 5 or V: Penetration of dye through the dentin to the pulp chamber.

Every section was rated separately for the occlusal and gingival margins. The highest score for each surface was taken as the representative of the evaluated tooth.

RESULTS

The results are summarized in the Table.

Occlusal surface

Only two (8.3 percent) restorations of group A had no leakage, as compared to nineteen (79.2 percent) of group B (Figure 2). In addition, fifteen (62.5 percent) restorations of group A showed dye penetration deep into the dentin (Figure 3) and in some cases reached the pulp (degree 4 + 5), as opposed to none in group B.

Proximal surface

Two (8.3 percent) restorations of group A had no leakage as compared to six (25 percent) in group B. The dye penetration was scored 'V' in thirteen (54.2 percent) restorations of group A compared to only three (12.5 percent) restorations of group B (Figure 4).

Both differences were statistically significant ($P < 0.05$) when submitted to a chi-square test (Figure 5a, b).

There were only four teeth with no dye penetration through both occlusal and proximal surfaces, all of them from the control group.

DISCUSSION

The importance of bonding filling materials to hard tooth tissue for the prevention of secondary caries formation has been recognized for many decades.[16-18] Furthermore, this bond could also provide additional resistance and retention, prevent marginal microleakage and eliminate postoperative pain.

Many attempts have been made to develop methods for the adhesion of restorative materials to dentin.[19-24] Glass ionomer cements have the property of adhering to enamel and dentin without conditioning.[25-27] A clinical study utilizing one of these materials in class II restorations in primary molars, however, resulted in a high failure rate.[28]

Several authors showed that the application of Scotchbond could reduce marginal leakage, when used to restore class II cavities *in vitro*.[10-13] More recently, good retention rates were reported when cervical erosions were successfully restored without cavity preparations, utilizing Scotchbond and composite resins.[29,30] Other investigators, however, showed that leakage occurred in class II and class V composite fillings using Scotchbond.[31,32]

Table □ Assessment of marginal leakage by depth of dye penetration.

Depth of dye penetration	Total number of restorations			
	Experimental (A)		Control (B)	
	Occlusal	Proximal	Occlusal	Proximal
0	2 (8.3)*	2 (8.3)	19 (79.2)	6 (25)
1 - I	0 (0)	0 (0)	1 (4.2)	8 (33.3)
2 - II	1 (4.2)	1 (4.2)	0 (0)	4 (16.6)
3 - III	6 (25)	6 (25)	4 (16.6)	2 (8.3)
4 - IV	6 (25)	2 (8.3)	0 (0)	1 (4.2)
5 - V	9 (37.5)	13 (54.2)	0 (0)	3 (12.5)
Total	24 (100)	24 (100)	24 (100)	24 (99.9)

*Numbers in brackets correspond to percentage values.

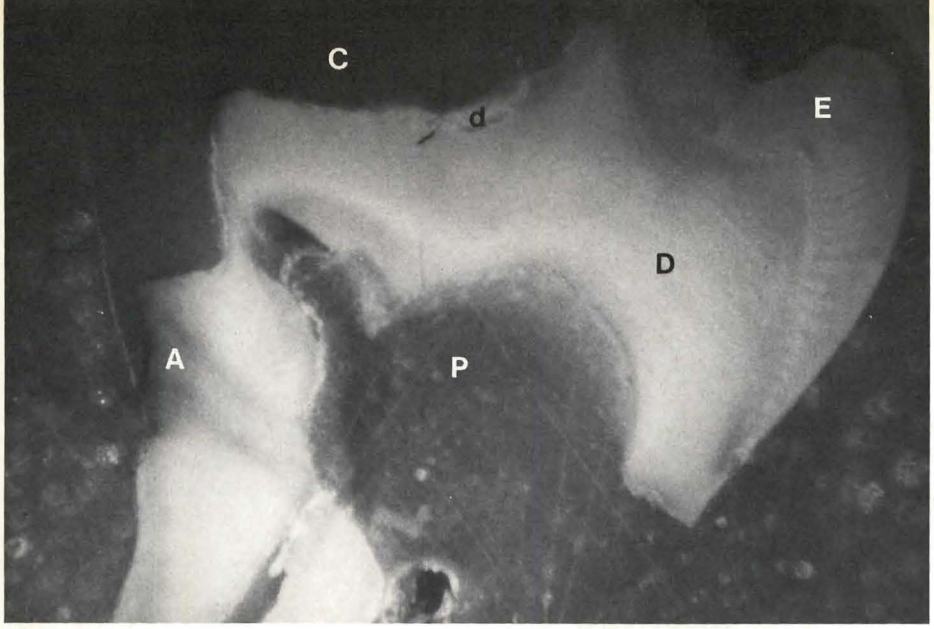

Figure 4. Photograph of a longitudinal section of a class II composite restoration of the control group (without Scotchbond). Notice the dye penetration into the pulp through the proximal surface (degree V). C = composite; E = enamel; D = dentin; P = pulp; d = dycal; s = stain.

The results of the present study confirm some of the previous reports and show that Scotchbond was not effective when used to prevent marginal microleakage.[31,32] Further, the low percentage (8.3) of teeth without or with minimal dye penetration (degree 0-1) in the occlusal margin of the experimental group as opposed to 83.4 percent of the controls suggests that a conventional bonding agent provides a better adaptation to etched enamel than Scotchbond. These findings were surprising, since the manufacturers report bonding values 20 percent higher for Scotchbond than for Enamel Bond, when tested in etched enamel.[33] Since other factors like storage time of the teeth and etching time of beveled primary enamel have been shown not to affect the adhesion, the higher effectiveness of the conventional bond might be the result of its better penetration into etched enamel.[34,35] This assumption, however, is not recorded in the literature.

Scotchbond also compared unfavorably in the cervical margins, since 62.5 percent of these restorations scored the highest degree of dye penetration as compared to 16.7 percent of the controls (Figures 5a,b). Cleansing of the dentin with hydrogen peroxide could have contributed to these results, since a decrease in bond strength has been reported when Scotchbond was applied to dentin pretreated with hydrogen peroxide and a phosphoric acid gel.[36]

It was recently shown that rinsing acid from enamel has a significant effect over the smear layer when the rinse water passes over the dentin.[37] These authors reported that optimum adhesion was obtained by preventing acid contact with the dentin, and suggested that the adhesive film should be cured prior to any acid-etch step.

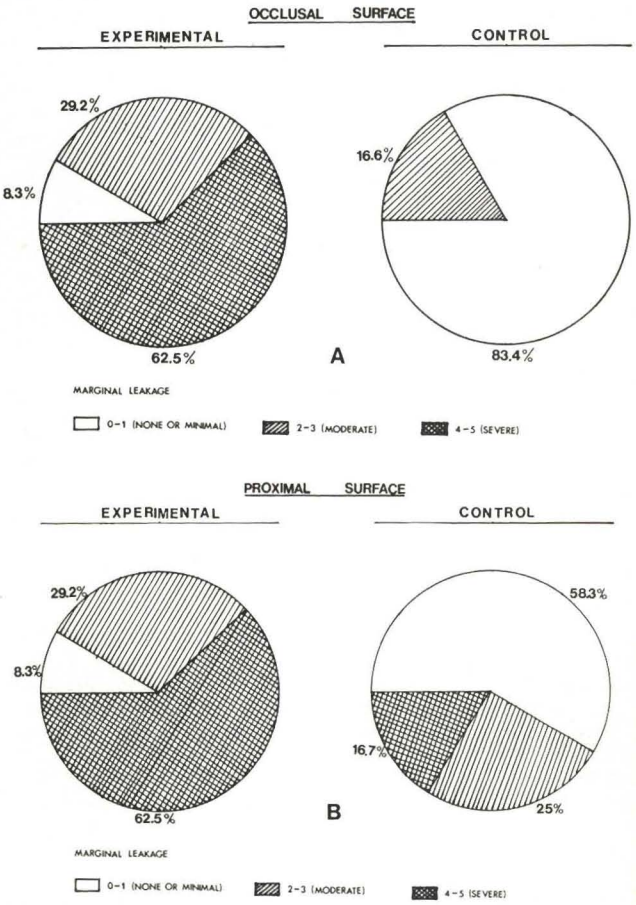

Figure 5. Diagram illustrating the degrees of marginal leakage at the occlusal (A) and proximal (B) margins of P10 restorations with Scotchbond and conventional bonding agent.

CONCLUSION

The use of Scotchbond in class II cavities did not prevent marginal leakage under the conditions of this study. Further investigations, however, should be done, curing the dentin adhesive before etching, before making any definitive conclusions concerning the clinical use of Scotchbond.

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Localized juvenile periodontitis of the primary dentition

Robert L. Mandell, DMD, MMSc
Mark D. Siegal, DDS, MPH
Edith Umland, MD

Juvenile periodontitis, or periodontosis according to Baer, "is a disease of the periodontium occurring in an otherwise healthy adolescent, which is characterized by a rapid loss of the alveolar bone about more than one tooth of the permanent dentition. There are two basic forms in which it occurs. In one form, the teeth affected are the incisors and the first molars; in the other, more generalized form, most of the dentition can be affected. The amount of destruction manifested is not commensurate with the amounts of local irritants present."[1] Baer pointed out seven characteristics that justify the classification of juvenile periodontitis as a distinct periodontal disease:

- ☐ Age of onset (early puberty, between eleven through thirteen years).
- ☐ Sex ratio (female/male ratio 3:1, according to Benjamin and Baer)[2].
- ☐ Familial background.
- ☐ Lack of relationship between local etiological factors and presence of deep periodontal pockets.
- ☐ Distinctive radiographical pattern of alveolar bone loss.
- ☐ Rate of progression (rapid).
- ☐ Lack of involvement of primary teeth.

Estimates of the prevalence of juvenile periodontitis range from 0.1 percent to 17 percent.[3] Barnett *et al* found a 2.4 percent prevalence rate in a population of 2,167 individuals, aged thirteen to thirty years, who presented for treatment in a dental school.[4] It appears that juvenile periodontitis is more prevalent among blacks and females, and may follow an autosomal recessive pattern of inheritance.[2,5,6]

Concepts about localized juvenile periodontitis (LJP) have changed in recent years as research provided more information about this distinct clinical form of periodontal disease. Microbiological studies have revealed a strong association between a gram negative, anaerobic, rod-shaped microorganism, *Actinobacillus actinomycetemcomitans* and localized juvenile periodontitis.[7-10] Ninety-five percent of localized juvenile periodontitis patients examined harbored *A. actinomycetemcomitans* in contrast to 15 percent of other population groups.[11] The microorganism inhabits supragingival plaque, subgingival plaque, and penetrates underlying connective tissue.[7-9,12-15] In addition to the cultural association of *A. actinomycetemcomitans* with localized juvenile periodontitis, a significant increase in gingival crevicular and serum antibodies to the microorganism has been found in patients with the disease.[16,17] Defects in the polymorphonuclear leukocyte functions of chemotaxis and phagocytosis have been reported.[18-20]

There is evidence that therapy for localized juvenile periodontitis patients should be directed at the elimina-

Dr, Mandell is with the Forsyth Dental Center, 140 The Fenway, Boston, MA 02115. Dr. Siegal is Director, Community Dental Programs, Columbus Health Department, Columbus, Ohio.

tion of *A. actinomycetemcomitans.*[21] Scaling and root planing alone and in combination with the antibiotic tetracycline have been insufficient for eliminating the microorganism from a significant proportion of sampled periodontal pockets.[21] It appears that periodontal surgery in combination with systemic antibiotics (tetracycline class antibiotic) has been effective in achieving either clinical improvement or, as recently documented, the elimination of *A. actinomycetemcomitans* from localized juvenile periodontitis lesions.[22-24]

Previously, the overwhelming majority of reports in the literature suggested that localized juvenile periodontitis was limited to the permanent dentition of otherwise healthy adolescents. Two recent reports have described advanced idiopathic alveolar bone destruction in the primary dentitions of healthy prepubescent children.[5,25] These reports challenge the classical definition of localized juvenile periodontitis. Therapies suggested for localized juvenile periodontitis in the primary dention include those mentioned for the permanent dentition plus extraction and space maintenance.[5,25]

The following case report documents the occurrence of localized juvenile periodontitis in several members of a Caucasian family and more specifically an acute infection of *A. actinomycetemcomitans* in the primary dentition of one family member.

CASE REPORT

E. T., an eight-year-old white male with an unremarkable medical history, was evaluated for vertical bone loss and hypermobility of all four first primary molars.

An oral examination of the patient revealed a normal dentition for his age, remarkable for hypermobility of all four first primary molars. Localized gingival recession of 1 mm to 2 mm exposed the cementum of the buccal roots of the mandibular first primary molars and the palatal root of the maxillary first primary molars. Periodontal pocket depths ranging from 4-7 mm were recorded around each affected tooth. Purulent material was expressed from the free gingival margins around the maxillary and mandibular right first primary molars.

Bitewing (Figure 1) and panoramic radiographs furnished by the referring dentist revealed marked vertical bone loss around the first primary molars. The radiographs demonstrate the mandibular teeth more clearly than the maxillary teeth. The distal root of the mandibular left first primary molar was completely resorbed while the distal root of the contralateral tooth was minimally resorbed, consistent with the physiological resorption associated with the eruption of its permanent successor.

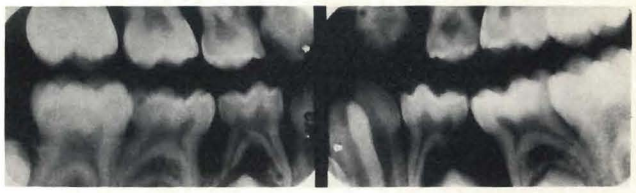

Figure 1. Bitewing radiographs of E.T.

A wedge biopsy of gingival tissue submitted for histological examination was found to exhibit characteristics of chronic gingival inflammation. A pooled plaque sample of gingival microflora from around the first primary molars was collected by introducing fine sterile endodontic paper points (Johnson & Johnson, New Jersey) into the periodontal pockets for ten seconds. The pooled paper points were then placed in Trypticase-soy broth (BBL, Maryland) and transported to a local hospital microbiology laboratory. There the samples were mixed thoroughly on a vortex mixer and replicate portions of the specimen were plated on a modified TSBV media consisting of vancomycin (5 μg/ml) and bacitracin (80 μg/ml) in a Trypticase-soy agar base supplemented with 5 percent sheep's blood defibrinated.[26] Suspected colonies of *A. actinomycetemcomitans* were subcultured and identified by appropriate laboratory tests.[8] Based upon these tests, *A. actinomycetemcomitans* was the predominant cultivable organism recovered from the specimen. Disk diffusion susceptibility testing was performed, and confirmed the isolate to be susceptible to tetracycline class antibiotics, the most commonly used drugs to treat periodontal *A. actinomycetemcomitans* infections. Due to the nature of the bone loss, the histological, and particularly the microbiological findings, the patient's condition was diagnosed as localized juvenile periodontitis. Therapy consisted of the extraction of all first primary molars and a regimen of oral doxycycline, one milligram per pound of body weight taken b.i.d. on the first day, followed by one milligram per pound taken q.d. for the next thirteen days. Bacterial cultures seven weeks postoperatively from the permanent molars and incisors were negative for the presence of *A. actinomycetemcomitans.*

The diagnosis of localized juvenile periodontitis was also supported by the familial pattern of the disease in this instance. A review of the referring dentist's, as well as a previous dentist's, patient records of seven family members (mother, father, and five siblings) revealed the father to be edentulous, but to have suffered severe bone loss previously. Three adolescent siblings showed radiographical evidence of bone loss consistent with localized juvenile periodontitis. The radiographs of the affected siblings are shown in Figures 2-4. The affected

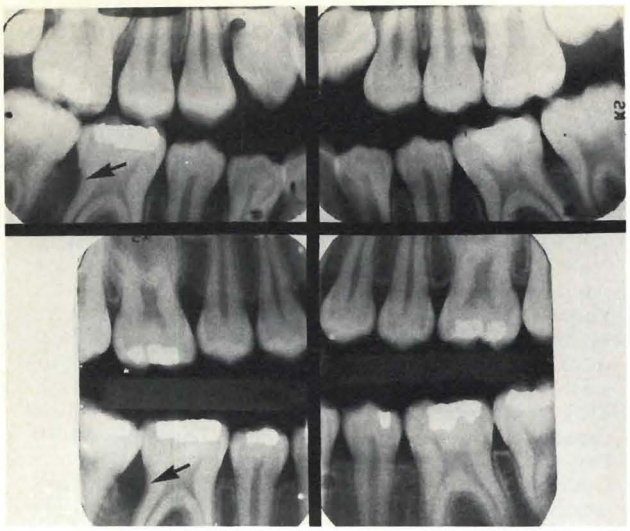

Figure 2. Bitewing radiographs of sister W.T. Top pair: 12-2-82; Bottom pair: 12-10-84. Note the bone loss in conjunction with the lower right permanent first molar (arrow).

family members, all teenagers, were referred to a periodontist for treatment.

DISCUSSION

Destructive periodontal disease in children is a rare occurrence. Previously, localized juvenile periodontitis was throught to be a destructive disease of the periodontium affecting permanent teeth. This case report provides further evidence that *A. actinomycetemcomitans* infection may exist in the mixed dentition, and strongly resembles localized juvenile periodontitis and agrees with other reports.[5,25,27,28]

The tendency toward localized juvenile periodontitis affecting the permanent dentitions of blacks and females is not absolute; E. T. is a white male whose primary teeth were affected. The familial pattern of localized juvenile periodontitis, found in this case is, however, a frequent characteristic of the disease.[3]

The documented association between localized juvenile periodontitis and the microorganism *A. actinomycetemcomitans* may be helpful in diagnosis, along with other appropriate laboratory studies, thorough review of the medical history, and clinical examination. The simple and practical sample collection technique using paper points required no special instrumentation. Local hospital microbiology laboratories should be able to supply appropriate transport fluid and prepare suitable selective media for isolation of *A. actinomycetemcomitans*.[8,25]

The differential diagnosis for advanced periodontal destruction about primary teeth now includes: localized juvenile periodontitis, hypophosphatasia, Papillon-Lefevre syndrome, histiocytosis X, neutropenia, cyclic neutropenia, leukemia, diabetes mellitus, scleroderma, fibrous dysplasia, and acrodynia. Goepford summarized the typical findings for each of these conditions.[25]

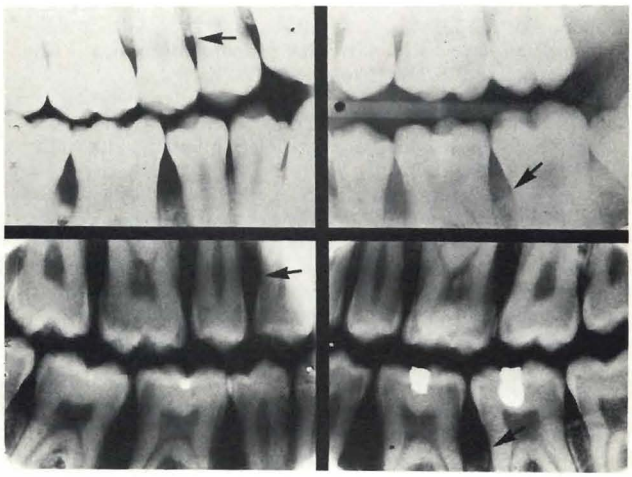

Figure 3. Bitewing radiographs of brother G.T. Top pair: 5-6-81; Bottom pair: 6-20-83. Note the progress of the disease (arrows indicate areas for comparison).

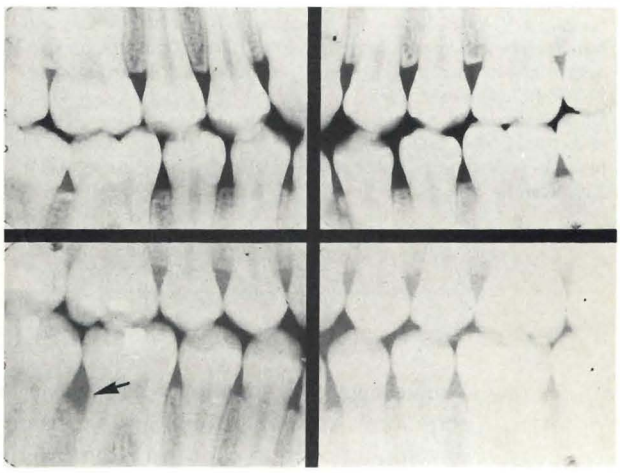

Figure 4. Bitewing radiographs of brother M.T. Top pair: 5-5-81; Bottom pair: 3-21-84. Note the bone loss in conjunction with the lower right first molar (arrow).

The microbial samples were processed in the Microbiology Department, University of New Mexico/BCMC, Albuquerque, NM 87131.

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SMOKELESS TOBACCO

Unlike cigarette smoking which was a long established habit before its health consequences were determined, the consumption of smokeless tobacco products is still a developing phenomenon. In regard to this issue, a variety of health professionals are confronted with the challenge of intervening to prevent further adoption of a behavior which, at best, is esthetically displeasing to many people and which, at worst, has potentially life-threatening consequences. Health gains achieved through the decline in the prevalence of cigarette smoking could be negated partially by increased use of smokeless tobacco.

Marty, P.J. *et al*: Patterns of smokeless tobacco use in a population of high school students. AJPH, 76:190-192, February, 1986.

Localized enamel hypoplasia of the primary canine

Mark F. Skinner, PhD
John Tat Wai Hung, BSc, DMD, MBA

An abnormality of enamel formation, only recently perceived in living children, was found in a majority of prehistoric children, some of them from more than 20,000 years ago.[1-3] The lesion takes the form of a roughly circular area of enamel hypoplasia, a millimeter or so in diameter, on the labial surface of the primary canine tooth (Figure). The condition has not yet been observed on any other primary teeth.

The defect was first reported by Jorgenson (1956) who attributed the condition to a genetic origin.[4] He observed that it occurred in 21 percent to 28 percent of modern and medieval Danish primary canine teeth (n = 688 and 182 teeth, respectively). The defect was shown to occur in similar proportions of a cadaver sample from Calcutta (29 percent, n = 83) but in substantially higher proportions (≥ 50 percent) of canine teeth selected from an Upper Paleolithic sample (n = 20) from Western Europe and a late Neolithic sample (n = 34) from the Middle East.[3] Recently, Badger reported that the defect occurs in 22 percent of a clinical sample of primary canines (n = 220, 45 percent affected individuals).

Immature human remains, particularly of young infants, regularly form more than half the skeletal sample from archaeological sites.[5] This reflects intense selection against particular individuals. For each such site it remains an unresolved but fundamental question of prehistoric human ecology, whether the majority of such individuals were constitutionally predisposed to die or whether much of the death assemblage was selected virtually at random. This bears directly on our attempts to understand the course of human evolution. One solution to this problem is to find hard tissue pathological 'markers', laid down during development, whose presence indicates that the health of particular individuals was significantly compromised prior to death. For example, a higher incidence of striae of Retzius was shown in skeletons from progressively younger children in prehistoric samples from Illinois.[6]

The defect reported here is being studied for its potential as a marker of poor health before death. Since the hypoplastic defect is common in prehistroic samples, and occurs, although more rarely, in recent children from Old and New World contexts, its cause would seem not to be culturally or temporally specific. The location of the defect at about crown one-half indicates that it starts to form at birth or shortly after birth.[7] Enamel deposited prenatally appears normal. This supports an environmental etiology for the condition. Skinner suggested that the cause of the defect may be minor traumata to the developing canine crown of neonates.[3] At birth, cortical bone, which forms the bulging labial wall of the crypt of the primary canine, is normally thin or possibly lacking. In either case the tooth crown may

Dr. Skinner is with the Department of Archaeology, Simon Fraser University, Burnaby, B.C., V5A 1S6 Canada.

Dr. Tat Wai Hung is Director, Burnaby School Dental Program, Clinical Lecturer, University of British Columbia, Faculty of Dentistry, Vancouver.

not be adequately protected from physical trauma. Crowns of adjacent primary teeth are not as subject to trauma through thinned alveolar bone, either because they form in a less vulnerable, less labially projecting position or their crown formation is largely completed at birth.

A metrical study of the size and vertical location of the defect within the primary canine crown showed that larger defects appear to form earlier during ontogeny.[3] This is felt to indicate that infants with larger defects were born relatively early, possibly prematurely. This interpretation assumes that thinning or fenestration through labial cortical bone is more prevalent in progressively younger infants.

The clinical research reported here represents an exploratory study to determine whether the defect is sufficiently common in children living in the Greater Vancouver area to justify further investigation of its etiology. It was anticipated that the effects of variables of sex and socioeconomic status on incidence of the defect could be analyzed.

METHODS AND MATERIALS

The sample consisted of healthy six-to-eight-year-old children from elementary schools served by the Dental Health Program in Burnaby, British Columbia. Observations were made by two dental assistants who had been shown a dry mandible with a primary canine bearing a gross example of the hypoplastic defect (Figure) and a color photograph of the defect in a living child. They were instructed to look closely for the lesion, noting its presence or absence in any of the four dental quadrants. They were specifically asked to score only obvious, not borderline, instances so as not to overestimate its prevalence. The results may be considered conservative, since teeth with restorations in this area were deliberately ignored. Approximately 2,380 mouths were examined for the defect, during normal screening for dental health problems. Racial background was recorded for those children exhibiting the lesion.

RESULTS

The results of this investigation are provided in Tables 1 and 2. The defect is apparently quite rare, occurring in less than 1 percent of the sample, in marked contrast to earlier studies, which report that about 45 percent or more of prehistroic or living individuals were affected.[1-3] While males and females appear equally likely to show the condition, there is a tendency in this study

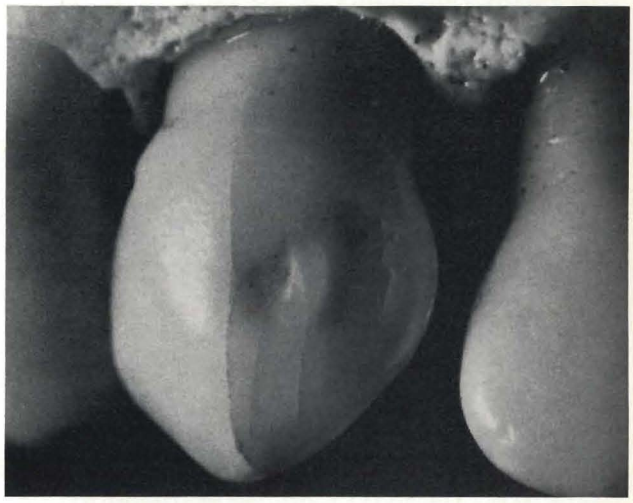

Figure.

for the defect to occur primarily in the female lower jaw but in both jaws of the male. The lesion is slightly more common in children from the school subjectively judged by health care personnel to be in the poorer neighborhood. A larger sample of individuals with the condition will be required to investigate interactions between defect frequency and sex or socioeconomic status.

There is a striking proportion of 'non-whites' who exhibit the condition. Six out of thirteen individuals (58 percent) are Indo-Asian or black. 'Nonwhites" are estimated to constitute only 12 to 15 percent of the Burnaby school children (personal communication, Eliot Grieve, Public Relations Officer). According to mother-tongue data (1981) provided by StatsCanada, Indo-Asians form only 8.1 percent of the Burnaby population.[8] They constitute, however, 38.5 percent of those Burnaby children with the hypoplastic lesion.

DISCUSSION

Because the cause of this hypoplastic flaw on the labial surface of the primary canine is not established, the wide range of variation in its incidence in different populations cannot be explained. Skinner suggested that in certain individuals, alveolar fenestration existing at or shortly after birth over the primary canine crypt, where bone is naturally very thin, exposes the developing tooth crown to physical trauma.[3] If correct, susceptibility then would increase with reduced bone thickness. The observation, here, that Indo-Asians are unusually affected suggests that a link may exist between ethnicity and bone formation. Well-nourished

Table 1 ☐ Proportion of Burnaby schooL children with a hypoplastic area on the labial surface of the primary canine.

	Condition		Sample size	Percent affected
	Present	Absent		
North Burnaby	4	996	1000	0.40
Sample South Burnaby	9	1371	1380	0.65
Totals	13	2367	2380	0.55

Table 2 ☐ Composition of sample of children with a hypoplastic area on the labial surface of the primary canine.

		Quadrant affected				
		Upper		Lower		
Subject	Sex	Left	Right	Left	Right	Racial origin
A	female				X	Indo-Asian
B	female				X	Indo-Asian
C	female			X	X	Indo-Asian
D	female			X		Black
E	female			X		White
F	female			X	X	?
G	female		X			Indo-Asian
H	male			X	X	White
I	male				X	White
J	male			X		'non-White'
K	male	X	X			White
L	male	X				White
M	male	X	X			Indo-Asian
Total	13	3	3	6	6	

Chinese and Japanese Asians are reported to have less compact bone, which is attributed to genetic rather than nutritional factors.[9] East Indians and American students who were equally well-nourished showed no differences, however, in bone density, suggesting that reduced compact bone in East Asian populations is due to dietary factors.[10]

A number of authors have drawn attention to a syndrome known as "Asian rickets". Traditional Asian diet consisting largely of chapatties includes a high content of phytates and fiber which have been shown to interfere with absorption of Vitamin D required for phosphorus and calcium deposition.[11,12] There is debate, however, on the precise cause of Vitamin D deficiency in Asian immigrants living in Britain. Dark skin and the all-enveloping sari of a married female may hinder Vitamin D synthesis under conditions of cultural seclusion from sunlight, particularly in Britain.[13,14] Watney *et al* have shown deficient serum calcium levels in breast-fed Asian infants from Britain, commencing at six days of age. The mothers' diets were particularly high in phytate and low in calcium.[15]

The highest incidence of the enamel defect reported to date is from a Late Neolithic sample of Iranian infants.[3] According to Reinhold *et al*, contemporary methods of milling grain and breadmaking in Iran differ but little from those in Pakistan and India.[16] Reinhold, Ismail-Beigi and Faradji have proposed that the fiber in the diet of contemporary Iranian villagers interferes strongly with calcium absorption.[17] Davies agrees that it is the complexing action of fiber rather than that of phytate that produces mineral deficiency diseaeses in modern Iranian villagers.[18]

A link between size of the hypoplastic defect and apparent prematurity has been found.[3] Defects that commence near the crown tip are significantly larger than those that form near the cervical margin. This indicates that in babies born at a more immature stage of crown formation, the cortical bone is thinned to the point that a larger opening forms over the bulge of the canine crypt. Calcium increments late in gestation normally equal those of an adolescent boy at peak growth velocity.[19] In one study designed to test whether breast milk, which is naturally low in calcium, could meet the calcium requirements of the growing premature infant, it was found that very premature babies fed mothers' milk showed calcium retention levels only 17 percent of normal retention *in utero*.[20] Prolonged feeding of human milk to low birth weight infants results in calcium and phosphorus deficits, manifested as demineralization of growing bones.[21] Also, premature infants fed banked mothers' milk tend to develop phosphorus depletion syndrome.[22] This is characterized by severe bone resorption, elevation in serum calcium, and excretion of up to ten times the normal amount of urinary calcium.[23]

On the basis of current evidence, two factors, prematurity and maternal diet, can be tentatively implicated in producing the canine defect. Both etiologies assume that labial cortical bone thins to the point where fenestration occurs, exposing the developing crown to physical trauma. They are not mutually exclusive. Pregnant females, who for dietary reasons absorb too little Vitamin D, will produce a baby, and the milk to feed it, both of which are Vitamin D deficient.[21] A premature baby born to such a mother is doubly likely to become hypocalcemic and develop net bone loss.

In summary, there is circumstantial evidence to suggest a link between mild hypocalcemia and alveolar fenestration perinatally, which exposes the forming primary canine crown to minor traumata sufficient to produce an area of enamel hypoplasia. The posited effect is likely to be small, not overt as in rickets, but of sufficient intensity to lower an infant below a threshold at which a fenestration appears in naturally thin labial cortical bone. The defect would appear to be most likely to occur

in premature children born to mothers who, for dietary reasons, have breast milk which is hypocalcemic.

CONCLUSION

Chidren from a dental health program in Burnaby, Canada show a very low incidence (< 1 percent) of a circumscribed, circular area of enamel hypoplasia on the labial surface of the primary canine. This lesion is much more common in prehistoric samples. The proximate cause of the lesion is suggested to be by trauma to the developing canine crown, commencing at birth, through a fenestration in the cortical bone overlying the canine crypt. The defect appears, from this study, to be relatively more frequent among Indo-Asian children. Reduced cortical bone thickness in affected individuals is attributed to hypocalcemia stemming from prematurity and/or maternal diets high in phytate and fiber and low in Vitamin D and calcium.

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THREATMENT OF FIBROUS ANKYLOSIS OF THE TMJ WITH A JAW EXERCISER

Limited mouth opening arising from intra- or extra-articular fibrosis appears to be overcome by treatment with jaw exercisers which are more effective than other devices since gradual bilateral opening traction is exerted on the mandible in contradistinction to those devices where the force is applied only to part of the mandible. The scissor type exerciser allows a force to be applied to the whole mandible and this results in a predominantly inferior displacement of the condyles with rotation. This movement may allow a displaced disc, if present, to return into an optimal position in the fossa.

Darveniza, P.J.: A jaw exerciser for fibrous ankylosis of the temporomandibular joint. Australian Dent J, 30:418-422, December, 1985.

Atypical root resorption of maxillary primary central incisors due to digital sucking: a report of 82 cases

Irving Rubel, DDS

Popovich and Thompson, de Rudder, and Gellin in their studies of the effect of thumb- and finger-sucking on occlusion, could not conclude whether such habit patterns alone cause a malocclusion.[1-3] Ripa and Barenie and Andrews stated that digital sucking can cause labial flaring, anterior spacing, protrusion of maxillary anterior teeth, and an anterior open bite.[4,5] One thing appears to be certain: Chronic digital sucking does not produce the same clinical picture in all children nor is the severity of the malocclusion the same.[6] The affecting variables may be grouped into frequency, duration, intensity, and position of influencing digit(s).

Herd stated that apical root resorption may be physiologic in nature or a result of pathologic events.[7] Both processes present similar histologic pictures in that odontoclasts (osteoclasts) are intimately associated with areas of active root resorption. The process is accelerated under morbid conditions such as a pulpal involvement or trauma (occlusal, orthodontic, or accidental).[7] Despite a variety of potential causative factors in root resorption, Diner concluded that a large number of cases cannot be explained and are labeled "idiopathic".[8] Furseth summed up resorptive processes by stating that, under normal conditions, these are not continuous and that they alternate with periods of repair.[9] The time intervals during which resorption takes place are believed, however, to be shorter than periods of repair and rest, and pressure (probably from the erupting tooth) is believed to play a role since resorption of primary teeth is most advanced in areas adjacent to the permanent tooth germ.[9]

Knott and O'Meara studied apical root resorption in maxillary and mandibular primary central and lateral incisors with respect to the eruption of their permanent successors.[10] Variations in resorptive patterns were presented in detail and discussed, with emphasis on initial and advanced stages. In the normal scheme of eruptive patterns of permanent successors, they reported that maxillary primary centrals and maxillary and mandibular primary laterals show lateral resorption more frequently (55 percent equally divided). Also, 50 percent of the maxillary primary central incisors began initial resorption at age 5.8 years, and emergence of the permanent successor took place at age 7 years.

Except for a preliminary report by Taylor and Peterson on the circumferential resorptive patterns in digital sucking based on the dental records of ninety-eight patients, ages two through four years, a search of the literature revealed no mention of the effects of digital habit pressures on apical root resorption.[11]

The purpose of this presentation is to show that digital sucking can produce external apical root resorption as early as two years, one month of age, which varies from the norm referred to by Knott and O'Meara.

MATERIALS AND METHODS

To illustrate particular patterns of apical root resorption in primary maxillary central incisors of digital suckers,

Dr. Rubel is Clinical Professor, Department of Developmental Dentistry, Pediatric Dental Section, University of Southern California, School of Dentistry.

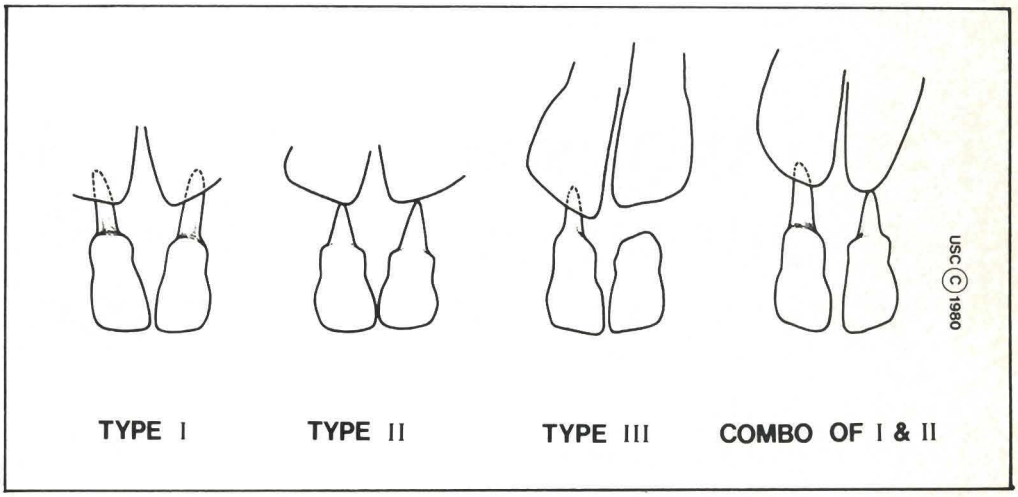


Figure 1. Types of apical root resorption.

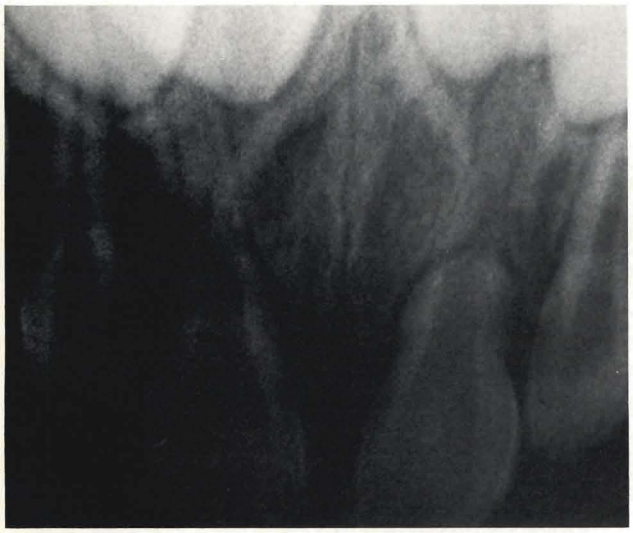


Figure 2. Patient S.F., age 4.1 years, Type I.

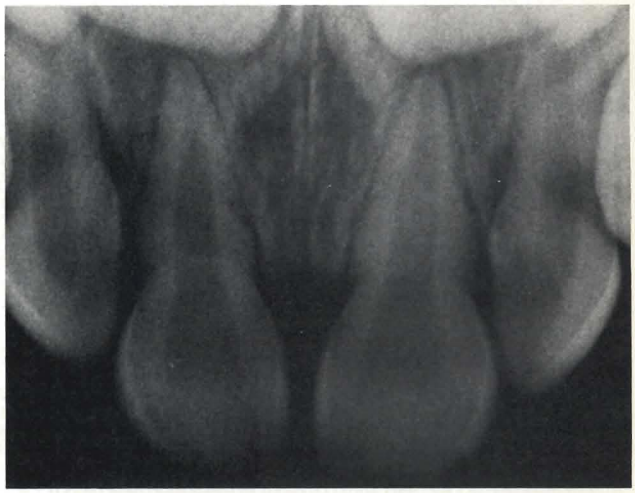


Figure 3. Patient K.A., age 4.2 years, Type II.

the radiographs of patients in a private pedodontic practice were evaluated.

All cases of root resorption in primary maxillary incisors were carefully studied to select only those which had no history of trauma and no radiographic evidence of morbidity.

The maxillary primary central incisors were used in this study because of the size of the roots and the ease of visually assessing the effect of thumb (finger) sucking upon normal apical root resorption.

Cases of digital sucking were included in the study, when the apices of the primary maxillary centrals were radiographically visible. Patients ranged in age from two years, one month to five years, three months (with one case of delayed eruption patterns at age seven years, nine months). The average age of this study group was four years.

For purposes of identifying the pattern of resorption, the author presents the following:

- ☐ Type I: This group appears to show a circumferential process without loss of total root length.
- ☐ Type II: External root resorption results in a conically shaped, apical reduction ("ice cream cone" effect).
- ☐ Type III: There appears to be a rounded or capped reduction of the apical half of the root(s).
- ☐ Combination of Types I and II (see Figure 1).

RESULTS

All cases in this study displayed one of four typical forms of apical root resorption. Following the schematics of

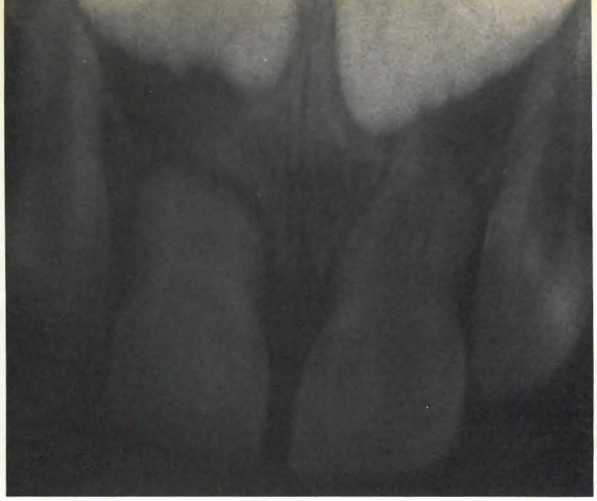
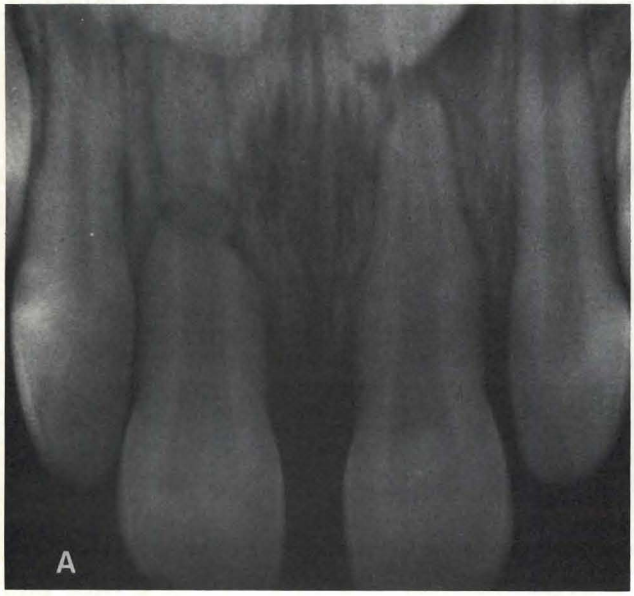


Figure 4. Patient J.A., age 5.3 years, Type III.



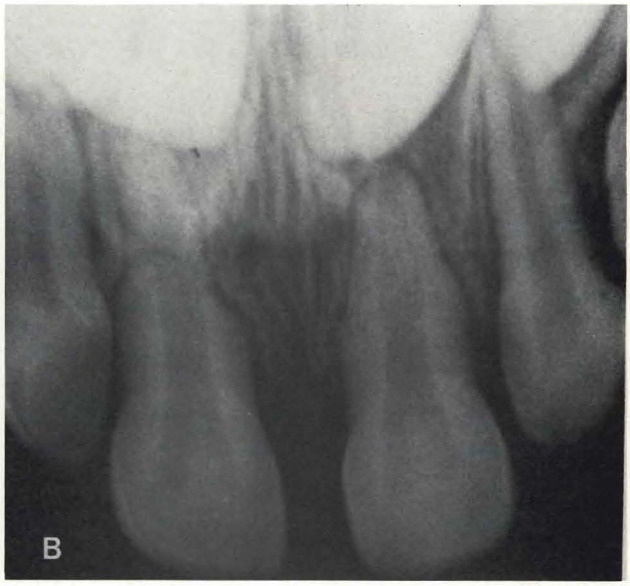
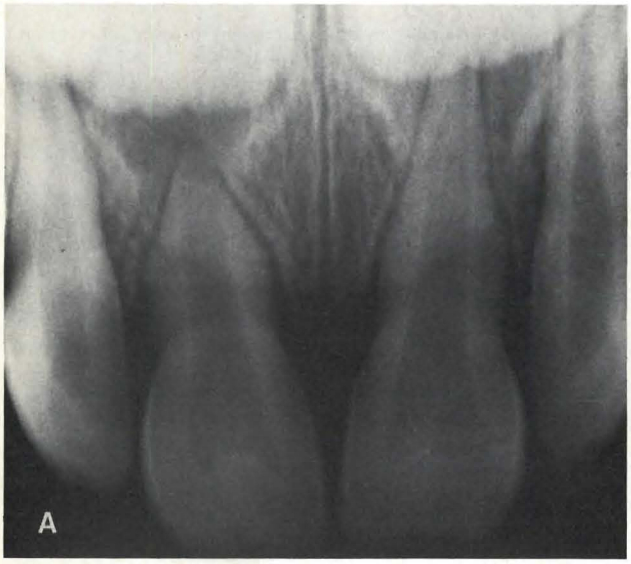


Figure 5. A. Patient K.L., age 4.0 years, Combination of I and II; B. Patient F.B., age 7.9 years, Combination of I and II.



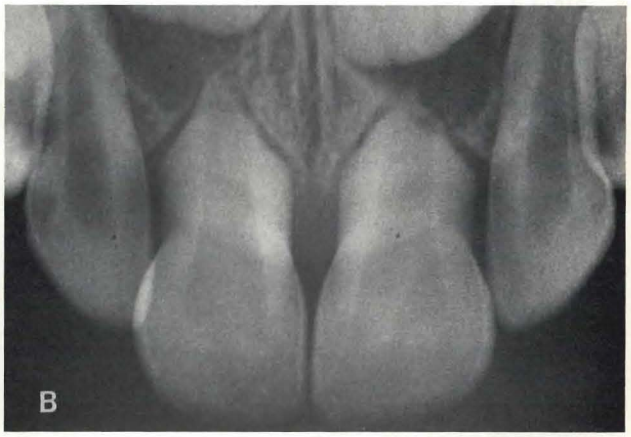


Figure 6. A. Patient F.C., age 3.2 years, Type II; B. Patient J.O., age 5.1 years, Type II.

root resorption (under "Materials and Methods"), radiographs indicative of these types are presented (Figures 2-5). Of particular interest are two representative cases of Type II resorption at different age levels (Figures 6A and 6B) and one case of a Combination of Types I and II in a patient 7.9 years of age with delayed eruption patterns (Figure 5A).

Table ☐ Distribution of habits and types of resorption.

Habit	No. of Cases	Type I	Type II	Type III	Combination I & II
Thumbsucking	80	35	35	6	5
Fingersucking	2		1		
Totals	82	35	36	6	5

The distribution of cases according to types is noted in the chart (Table).

Admittedly, in the collection of cases for this study of digital suckers, there were some which did not show any atypical root resorption regardless of the severity of the habit.

It may be noted that the most prominent types of resorption are Types I and II which appeared to be distributed about equally in 87 percent of the cases. Type III resorption accounted for 7 percent of the cases with a Combination of I and II for 6 percent.

DISCUSSION

Potential manifestations of the digital sucking habit, according to Moyers and Finn are as follows:

- ☐ Maxillary anterior protrusion.
- ☐ Mandibular anterior retrusion due to mentalis muscle contraction compressing the lip inward upon swallowing. The thumb- or finger-sucking adds to the pressure against the lower teeth.
- ☐ Anterior open bite.
- ☐ Tongue thrusting as a result of the open bite and tipping of maxillary incisors.
- ☐ Narrowing of the maxillary arch due to buccal wall contractions.
- ☐ Hypotonicity of the maxillary lip giving a "high lip line", and hyperactivity of the lower lip, which may come to rest behind the labially tilted maxillary incisors.[12,13]
- ☐ To this list of findings, consideration should now be given to the radiographic evidence presented in this paper of the atypical manner in which the apices of the maxillary primary central incisors resorb at any early age in response to "digital sucking trauma".

This last manifestation may be another good reason for the "transferring" or removal of the habit. Further study may show effects of this early digital sucking upon the permanent dentition. Also, radiographic interpretation of apical fracture or other resorptive processes should not be confused with the atypical resorption of digital sucking.

The types of resorption presented in this paper are arbitrary groupings made for convenience of discussion. The chance of a sliding continuation from one group into another is a distinct possibility.

The author proposes that the "syndrome of digital sucking" should include definitive radiographic changes about the apices of the maxillary primary central incisors as well as the already reported changes in bone, soft tissues, teeth, speech and communication, and spacial relationship of the dental arches.

CONCLUSIONS

This report suggests that digital sucking (thumb and/or finger) has an effect upon the normal process of apical root resorption of the maxillary primary central incisors, which can be noted radiographically.

In this investigation, such radiographic findings appeared in children as young as two years, one month of age, and they manifest themselves in four types of apical resorption, of which the most significant and common forms are Types I and II.

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Treatment of an unusual case of fusion

Peter S. Hasiakos, DDS
Franklin S. Weine, DDS, MSD
Daniel G. Ellenz, DDS, MS
Joseph J. Keene, Jr., DDS, MS

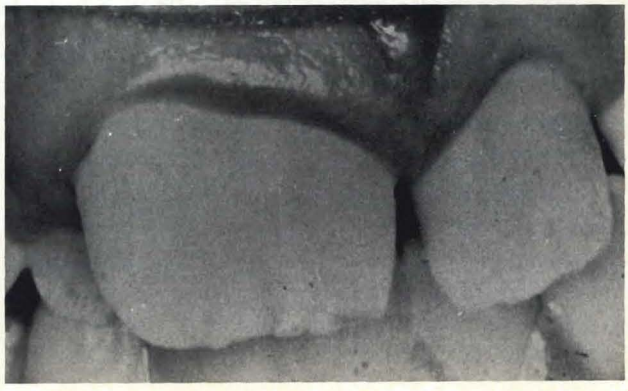
The dental literature contains many reports concerning endodontic treatment of teeth with fusion, gemination and talon cusps.[1-5] This paper reports the treatment of a maxillary right central incisor with a large talon cusp, which was fused to a supernumerary incisor distal to it in arch form. The reasons for failure in this case are different, in part, from those reported by Peikoff and Trout.[6]

As others have stated, it is difficult to differentiate between fusion and gemination specifically, when fusion occurs with a normal tooth and a supernumerary tooth.[7] Fusion is the union of enamel and dentin of two distinct tooth buds. If it occurs early enough in embryonic development, the result may be a tooth of normal or slightly larger than normal size. If it occurs later in development, the result is the more classic example seen clinically, resulting in hypodontia of that dental arch and the presence of a large tooth anomalous in form. Clinically, fusion may be confused with gemination, the latter being an attempt of one tooth bud to divide into two separate dental units. If successful, the result is hyperdontia in that arch with the presence of an extra tooth that is a mirror image of its geminated partner. Meréchaux reported treatment in which the fused teeth had separate and distinct pulp chambers.[8] In this case report, the chambers were confluent and united.

CASE REPORT

The patient was a twelve-year-old white male in good physical health who came to Loyola University School of Dentistry in May, 1983, for routine dental care. Clinical examination revealed a very large right central incisor preventing the maxillary right lateral incisor from at-

Figure 1. Preoperative appearance of anterior teeth of twelve-year-old white male. The maxillary right central incisor measured 12 mm mesiodistally.



Dr. Hasiakos is Assistant Professor of Operative Dentistry; Dr. Weine is Professor and Director Postgraduate Endodontics; Dr. Ellenz was formerly, Graduate Assistant in Endodontics; and Dr. Keene is Associate Professor of Periodontics, Loyola University School of Dentistry, 2160 South First Avenue, Maywood, IL.

taining its normal position in the arch. The lateral incisor was displaced lingually, and, because of the extreme width of the central incisor, adequate space for repositioning the lateral incisor was not available. The patient was very conscious of the large tooth, and was the recipient of some social pressures from his peers, mocking his appearance (Figure 1). The tooth measured 12 mm mesiodistally, 10 mm incisogingivally and 7 mm labiopalatally.

After consultation with the graduate endodontic, periodontic, orthodontic, fixed prosthetic and pedodontic departments, only a guarded prognosis was made, for the following reasons:

- ☐ The pulpal communication between the two discernible roots might extend so far gingivally that amputation of the distal root would yield a subosseous exposed root chamber.
- ☐ This mesial root might then require orthodontic extrusion for proper prosthetic coverage and the resultant crown-to-root ratio would be compromised.
- ☐ The possibility of lateral canals, minor accessory roots that were not radiographically evident, or a distolingual groove (palatogingival groove) persistent at the junction of fusion might cause failure. The radicular lingual groove is sometimes overlooked in clinical diagnosis.[9]

After being told of the guarded prognosis, the parents did not desire extraction and requested that an attempt be made to keep the tooth by any combination of procedures deemed necessary. The alternative treatment was extraction, since the patient would otherwise require orthodontic treatment. It was determined, however, that the central incisor, as presented, could not be treated successfully with orthodontics, due to its size. Retaining the tooth in a *status quo* meant a subsequent discrepancy in arch form with predictably poor periodontal health. Periodontal disease associated with developmental variations of tooth morphology have been documented by Shiloah and Kopczyk.[10]

TREATMENT

Intentional endodontic therapy was initiated on the maxillary right central incisor as part of an attempt to improve the patient's appearance. Procedural difficulties and a guarded prognosis were explained and consented to by his parents. Preoperative radiographs (Figure 2) revealed a small accessory root or fin beneath the talon cusp medially situated between two separate and distinct roots.

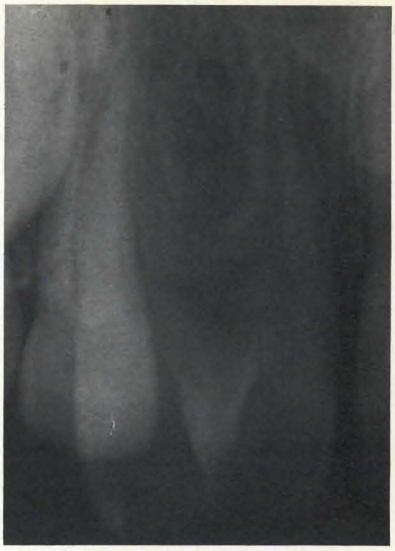


Figure 2. Preoperative radiograph indicating the extreme width of the maxillary right central incisor, which blocks out the right lateral incisor.

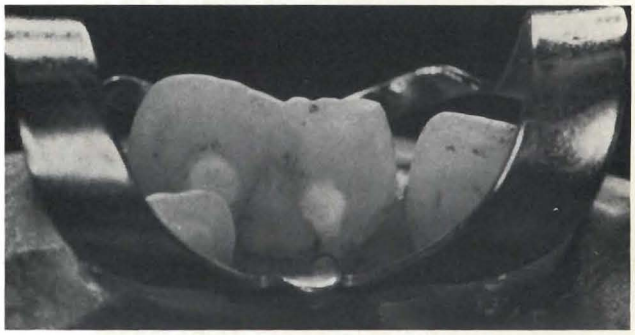


Figure 3. Lingual view, indicating individual entries made into the two canals.

The initial treatment plan was

- ☐ To perform endodontic therapy on all canal systems.
- ☐ Hemisect the tooth by removing the distal root and distal half of the crown.
- ☐ Restore the mesial half.

On June 14, 1983, topical and local anesthetics were administered, the tooth was isolated and two individual lingual entries were made into the major canals (Figure 3). The canals were irrigated with 5.25 percent NaOCl and initial instruments were placed into the canals (Figure 4). Radiographs confirmed the established working lengths. The mesial and distal canals were enlarged to the size of a number 35 file at 23 mm and the size of a number 50 file at 24 mm, respectively (Figure 5). The canals were irrigated, dried, and sealed with zinc oxide eugenol. The small accessory structure was not accessible to instruments.

At the next appointment, June 28, 1983, the patient was asymptomatic. Purulent exudate was observed, however, upon entry to the mesial canal. The access of the tooth was extended to a large, single entry (Figure 6), which revealed granulomatous tissue under the talon cusp. The canals were irrigated with 5.25 percent NaOCl, instruments used again, and the canals flared and closed with zinc oxide eugenol.

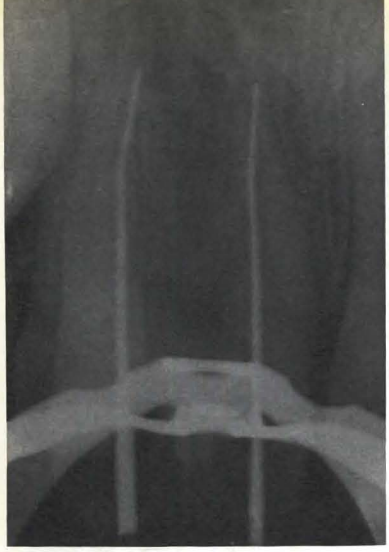


Figure 4. Initial files in place in canals.

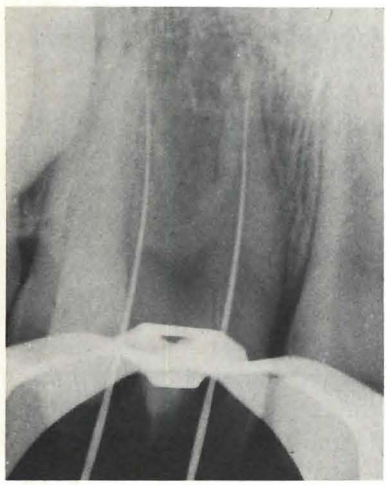


Figure 5. Master apical files in place, the mesial canal enlarged to the size of a number 35 at 23 mm, and the distal canal enlarged to the size of a number 50 at 24 mm.

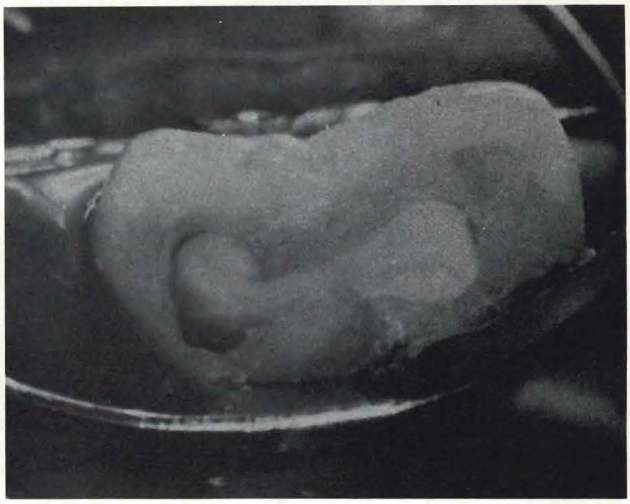


Figure 6. Lingual access widened to make a single confluent entry.

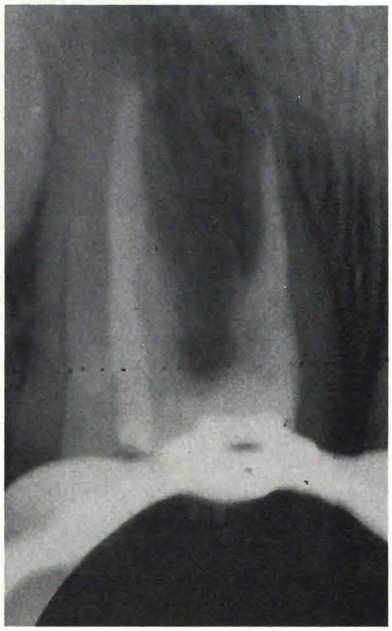


Figure 7. Radiograph after canal fillings with laterally condensed gutta-percha and Wach's Paste as sealer.

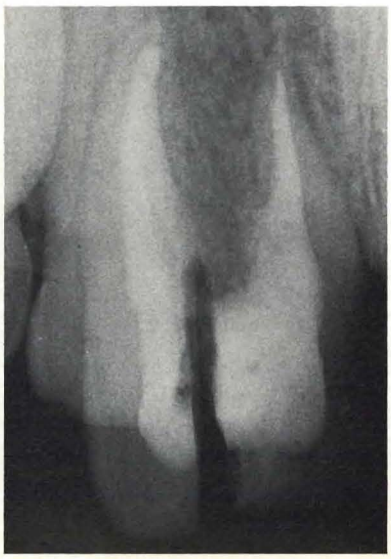


Figure 8. Radiograph following bur cut for hemisection. Cut goes into part of the accessory root.

An additional appointment to cleanse and shape the canals was necessary on July 5, 1983, when the patient presented with moderate swelling of the associated labial mucosa. Purulent exudate again was noted on entering the pulp chamber. The canals were irrigated with warm sterile saline to facilitate drainage. After the exudate diminished, the canals were copiously irrigated with 5.25 percent NaOCl. Additional preparation of the canals was done and a zinc oxide eugenol seal placed. Penicillin V 500 mg, 1 tab q6h x 24 tabs and Tylenol number 3, 1 tab q4h prn pain were prescribed.

On July 13, 1983, the patient returned without complaints or tenderness. The sinus tract had healed and no swelling could be seen. Final canal preparation was performed and the canals were filled with Wach's Paste Sealer* and Kerr gutta-percha†, utilizing lateral condensation as described by Weine.[11] The pulp chamber was packed with warmed gutta-percha under verticle pressure and sealed with zinc oxide eugenol. Post obturation radiographs demonstrated a solid condensation of the canal systems (Figure 7).

A hemisection with removal of the distal segment (Figures 8-10) revealed the furcation was a level closer to

*Sargent's Drugs, Chicago, IL 60602.
†Kerr Manufacturing Co., Romulus, MI 48174

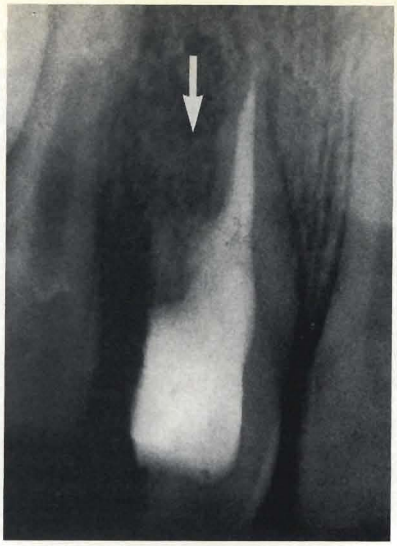


Figure 9. Radiograph following extraction of distal section. There seems to be sufficient room for the lateral incisor now, but notice the remaining accessory root (arrow).

Figure 10. Clinical view, following removal of distal section.

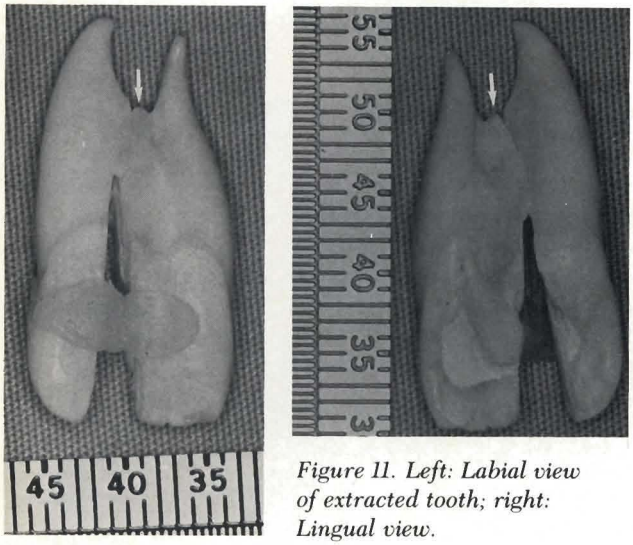


Figure 11. Left: Labial view of extracted tooth; right: Lingual view.

the apex than anticipated preoperatively. In addition, the cut was through part of the inaccessible accessory root that contained some pulp tissue. A widening of the periodontal ligament with periapical changes was noted radiographically. A periodontal and prosthodontic evaluation at this time confirmed the nonrestorability of the retained mesial aspect of the central incisor. Extraction was recommended and the parents were informed (Figure 11). A transitional partial denture was placed until orthodontic treatment could start a few months later. Once the orthodontic appliances were in place, a denture tooth was inserted into the braces for esthetic purposes. The patient presently is under orthodontic treatment.

DISCUSSION

Initially, thought was given to reducing tooth structure mesiodistally and placing a composite veneer, to give the tooth a normal appearance, thus eliminating the need for endodontic therapy and a hemisection. Due to the amount of enamel and dentin that would have been removed, a pulp exposure probably would have resulted. In addition, due to the size and position of the talon cusp, grinding as described by Shey and Eytel was not feasible.[12]

Of specific interest to this case report was the inability to ascertain the depth of the bifurcation, as shown radiographically. Figures 2, 4, 5 and 7 appear to indicate that the bifurcation level was in an acceptable position, so that a successful attempt could be made to retain the mesial root.

In Figure 11A, however, it can be seen that several millimeters of very thin root surface was not visible radiographically, but extended apically from the furcation. It is the opinion of the authors that in the diagnosis and treatment planning of similar cases, prime attention should be given to the level of the furcation. If this distance is greater than 4 mm from the crestal bone, extraction with appropriate prosthetic replacement is advisable. There is very little tolerance for favorable prognosis when furcations occur farther apically.

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Ectopic eruption of maxillary first permanent molars: a review

J. Kurol, DDS, Odont Dr
K. Bjerklin, DDS

Ectopic eruption of the maxillary first permanent molar signifies a disturbance of its path of eruption and is manifested by erupting mesially to its normal path. The permanent molar is initially blocked from complete eruption by the second primary molar. Due to the close contact between these two teeth, resorption occurs on the distal surface of the primary molar. Two types of ectopic eruption are recognized, reversible and irreversible (also called "jump" and "hold" cases).[1,2] If the permanent molar spontaneously frees itself and erupts to occlusion, the reversible type is present (Figure 1). In the irreversible type, the permanent molar remains in the locked position until treatment is provided or premature exfoliation of the primary molar occurs spontaneously (Figure 2).

Irreversible ectopic eruption where the second primary molar is lost often causes mesial tipping and rotation of the permanent molar, unfavorable occlusion and space deficiency for the second premolar (Figure 2). Some sort of treatment is needed, therefore, in these cases.

Ectopic eruption of the maxillary first permanent molar is best assessed from periapical or bitewing radiographs. In a recent study of 126 children with ectopic eruption, it was found that most of the first permanent molars at risk were locked in the atypical areas of resorption in the distal parts of the second primary molars, at six years of age. At seven years, most of the permanent molars in children with reversible ectopic eruption had

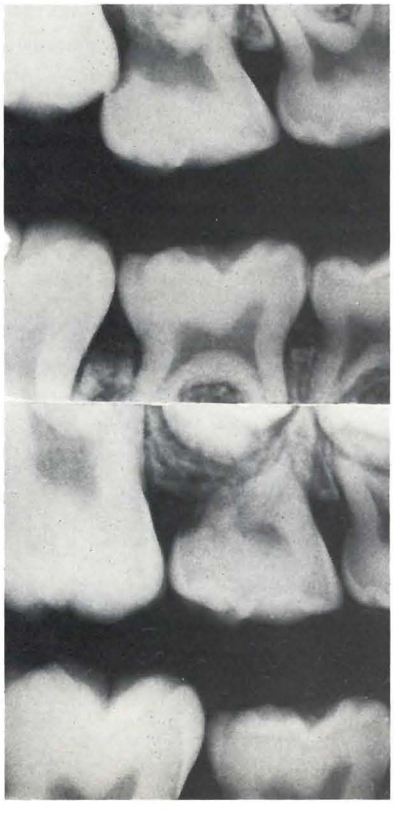

Figure 1. Bitewing radiographs showing reversible ectopic eruption of the maxillary first permanent molar, in a boy age six years, five months (top) and eighteen months later (bottom).

Dr. Kurol is Senior Consultant Orthodontist and Dr. Bjerklin is Consultant Orthodontist in the Department of Orthodontics, the Institute for Postgraduate Dental Education, Kärnvägsgatan 9, S-552 55 Jönköping, Sweden.

freed themselves. Only a few of the first permanent molars that were locked at the age of seven freed themselves later.[1] From this study, it seems reasonable to conclude that the type of ectopic eruption can be reliably established after the age of seven.

In 1923, Chapman described this disorder.[3] An apparent increase in prevalence is attributed to evolutionary factors.[4] During the last few years, an increasing number of reports and investigations were presented. The reason for this interest may be related to a pronounced reduction of caries in young children, which earlier may have masked the consequence of ectopic eruption. The purpose of this article is to review current views on the diagnosis, prevalence, etiology and treatment of ectopic eruption of the maxillary first permanent molars.

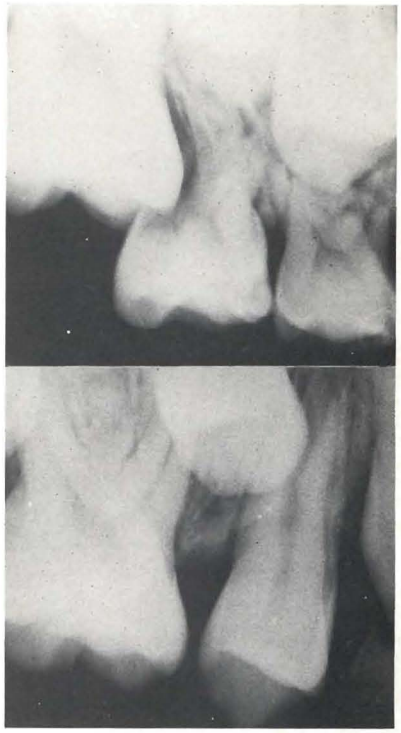

Figure 2. Periapical radiographs showing irreversible ectopic eruption of the maxillary first permanent molar. Note the locked position of the erupting permanent molar and the extensive resorption on the second primary molar, which had to be extracted. From seven years, eight months of age (top) to nine years, eleven months of age (bottom) a loss of space is seen.

PREVALENCE

The prevalence of ectopic eruption is reported to vary between 2 percent and 6 percent (Table).[1,2,5-9] In cleft palate children, a much higher prevalence (25 percent) has been reported.[10] The variation in prevalence from 2 percent to 6 percent may be related to the number of children studied, their ages, and the caries rate. A high caries rate, necessitating early extraction of second primary molars, could affect a diagnosis and could account for failures to detect an ectopic eruption. In an investigation comprising 2,903 children, 126 children (4.3 percent) showed ectopic eruption. Of these 126 children (seventy-five boys and fifty-one girls), 59 percent showed reversible ectopic eruption. Of the 2,903 children investigated, 1.8 percent showed irreversible ectopic eruption and were thus in need of treatment.[1]

ETIOLOGY

Local factors

Many etiological theories have been reported and discussed in the literature, including inadequate arch length, lack of growth in the posterior region of the maxilla, mesially inclined eruption path of the first permanent molar and abnormally large first permanent molars.[3,5-7,11-16] A combination of responsible factors was suggested by Pulver: Abnormally large mean size of the maxillary primary first and second molars, smaller maxillae, posterior position of the maxillae in relation to

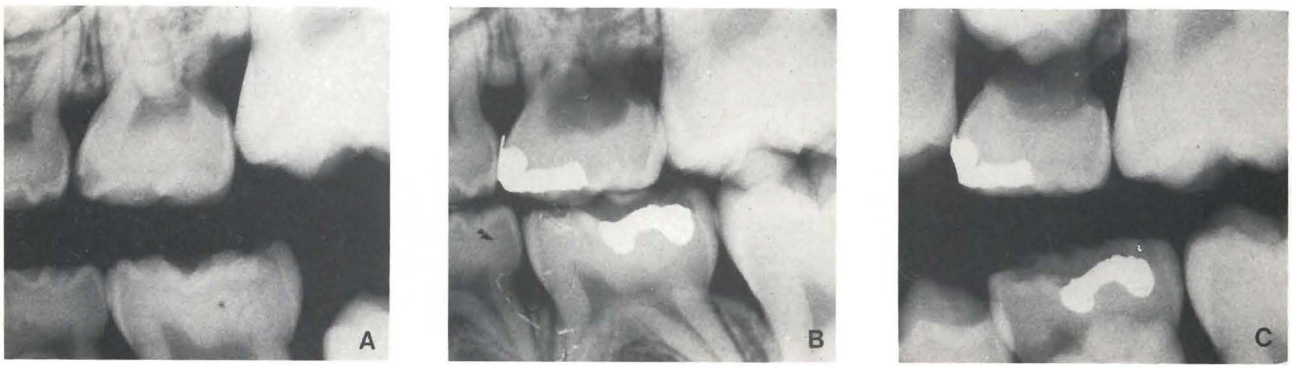

Figure 3. Bitewing radiographs showing the development of the distal resorption in a maxillary second primary molar. A. Reversible ectopic eruption in a boy, age six years, five months. B. Progression of the initial resorption can be seen. C. The second primary molar persisted until the normal exfoliation time. Age ten years, four months.

Table ☐ Reported prevalence of ectopic eruption of the maxillary first permanent molars.

Authors	Year of study	Country	Number of children	Children with ectopic eruption Number	Percent
Cheyne & Wessels	1947	USA	500	9	2
Young	1957	USA	1,619	52	2
O'Meara	1962	USA	315	6	2
Pulver	1968	USA	831	26	3.1
Bjerklin & Kurol	1981	Sweden	2,903	126	4.3
Wäckerle-Heporauta	1981	Switzerland	543	32	6
Kimmel *et al*	1982	USA	5,277	200	3.8

the cranial base, abnormal angulation of the path of eruption of the maxillary first permanent molar, and delayed calcification of some affected first permanent molars.

In order to investigate the importance of different etiologic factors, 129 children (ninety-two with ectopic eruption and thirty-seven matched controls) were studied.[17] Measurements were made on lateral head films, orthopantograms and dental casts. Data were analyzed by discriminate analysis, where factors could be compared separately and in combination. Children with irreversible ectopic eruption had significantly larger permanent molars and a more pronounced mesial angle of eruption. A tendency toward a shorter maxilla was also found. The mesial eruption angle of the first permanent molar was on average increased by fifteen degrees in cases with irreversible ectopic eruption, compared to cases with normal eruption. No significant difference was found between cases with reversible ectopic eruption and cases with normal eruption.[17]

Hereditary factors

Ectopic eruption of the maxillary first permanent molar was found to be more common among siblings (19.8 percent) than in the general population (4.3 percent). This familial tendency is statistically significant.[18] In our study, we found a multifactorial mode of inheritance, where both genetic and local factors can act in combination.[18]

Because early treatment is considered important for ectopic eruption, this familial tendency offers an opportunity to identify and treat some of the children at risk in the early dentition phase.[5,19,20]

TREATMENT

Reversible ectopic eruption

In children with reversible ectopic eruption, no treatment is needed, because the permanent molar spontaneously frees itself and erupts. In order to avoid unnecessary treatment, it is important to differentiate between reversible and irreversible ectopic eruption before treatment is begun.[21] If there is any uncertainty concerning the diagnosis, treatment should be postponed for some months.

The prognosis for the resorbed second primary molar was evaluated in a study where ninety-two such teeth were observed longitudinally.[22] Only two teeth were lost prematurely (within two months) and the remaining ninety affected second primary molars persisted until the normal exfoliation time (Figure 3). During the ob-

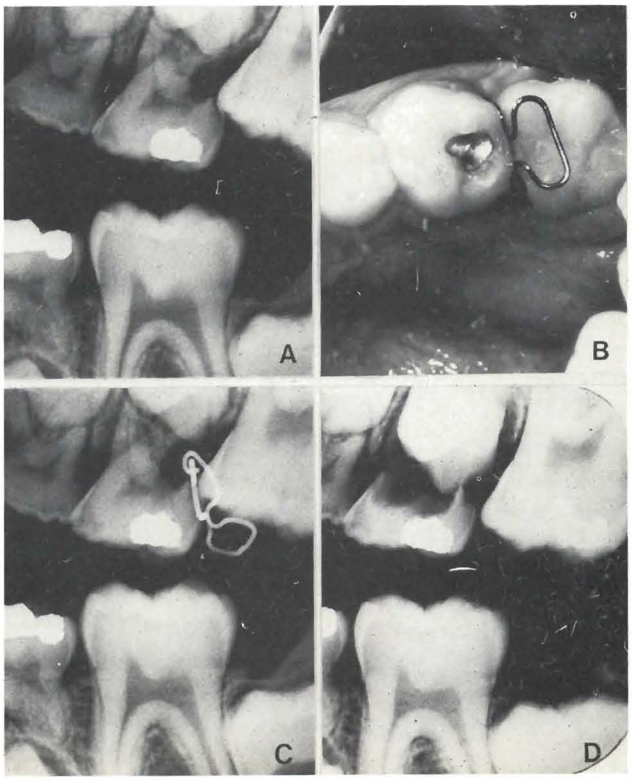

Figure 4. Treatment of ectopic eruption with a separating spring. A. Bitewing radiograph at the start of treatment, at seven years, three months of age. B. Occlusal view with the spring inserted. C. Bitewing radiograph with the spring at the start of treatment. D. Result of successful treatment at eight years, eleven months of age, when the primary molar is near exfoliation.

servation of these teeth, slow progression of the resorption was seen in thirteen teeth, hard tissue repair was noted in fifteen teeth and sixty-two teeth showed no change beyond the initial resorption. Thus, the resorbed second primary molars may serve as excellent maintainers of space and function for a long time with a favorable influence on normal occlusal development. In view of this fact, it is important to avoid damage to second primary molars where the permanent molar may have a chance of freeing itself spontaneously. For these cases, a period of observation is recommended.[22]

Irreversible ectopic eruption

TREATMENT WITHOUT EXTRACTION OF THE PRIMARY SECOND MOLAR

If the permanent molar remains locked in its position in the resorption distal to the primary molar, separation with brass wire is the most commonly recommended treatment.[14,19,23-25] Distal discing or a variety of uses of separating springs have been suggested also.[11,13,16] Treatment with a recently introduced separating spring (De-impactor) is illustrated in Figure 4. In our experience, separating springs are generally effective, but some types may cause occulsal disturbances at the end of the treatment (Figure 4). The efficacy of separation wires is questioned in some cases, and in such instances separating springs may prove useful.[13]

Marked mesial tipping of the maxillary first permanent molar may already be present in the locked position, before extraction of the primary molar. For these cases, more active distal movement of the maxillary first permanent molar is recommended. Humphrey, in 1962, described a type of treatment using a banded second primary molar with a soldered spring acting against the occlusal surface of the permanent molar. A curved tag of composite resin bonded to the occlusal surface of the permanent molar to engage the free end of the spring may improve the efficacy of this type of appliance.[27-29] Biomechanically, because of the reciprocal forces used, this type of appliance may have an unsatisfactory anchorage. If both maxillary second primary molars are banded and united with a soldered transpalatal bar, the stability of the appliance is much improved (Figure 5). An omega loop on the palatal part of the spring can easily be activated with three-beak pliers.

TREATMENT INVOLVING EXTRACTION OF THE PRIMARY SECOND MOLAR

In cases with pronounced resorption or when treatment with separation devices fails, extraction is unavoidable

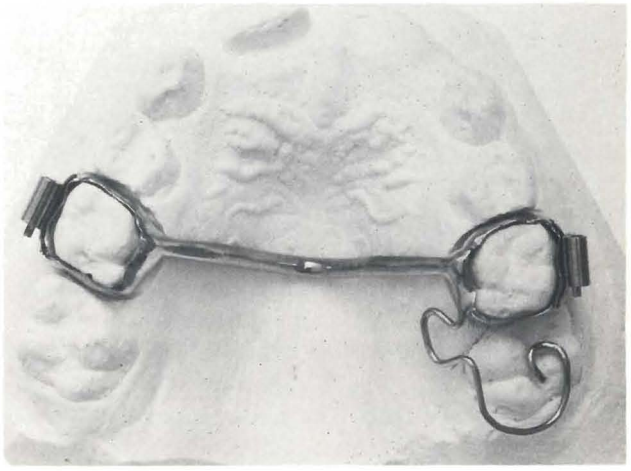

Figure 5. Treatment of irreversible ectopic eruption with a spring acting against the occlusal surface of the left permanent molar. The second primary molars are banded and united with a transpalatal bar for reinforcement of the anchorage. For better retention of the spring, a tag of composite resin may be bonded to the occlusal surface of the permanent molar.

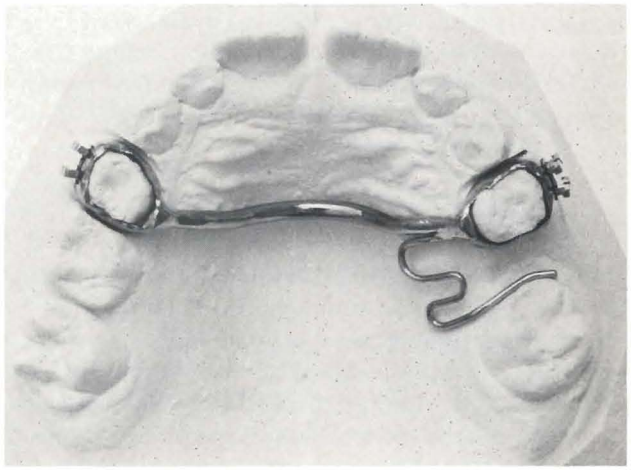

Figure 6. Treatment of irreversible ectopic eruption of the left maxillary first permanent molar, where the second primary molar is lost. The first primary molars are banded and united with a transpalatal bar. The spring acts against the mesial surface of the permanent molar.

and space maintainers should be considered.[5,12,13,16,30] The space maintainer can be designed with occlusal surface support of mesial abutment teeth.[29]

After extraction of the second primary molar, the maxillary first permanent molar erupts with a mesial inclination and space loss results within a short time (Figure 2).

Removable orthodontic plates with springs for the

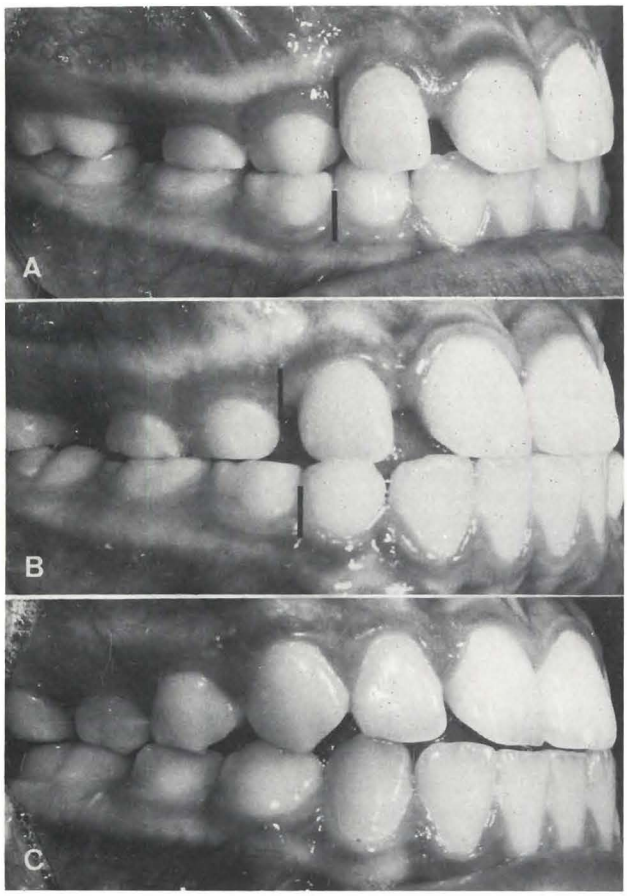

Figure 7. A. Mesially positioned maxillary right first permanent molar due to irreversible ectopic eruption, in a girl, age eight years, three months at the start of treatment. B. After twelve months treatment with cervical traction. Note the change in intermaxillary relationship, which is especially evident in the canine and frontal segment. C. Permanent dentition at twelve years, five months of age.

mesially tipped permanent molar have been described for such cases.[31,32] In our experience, removable plates are effective in unilateral ectopic eruption. In bilateral cases, however, difficulties may arise concerning the anchorage of the plate, as the springs act against the tipped permanent molars, reciprocal forces may displace the plate.

Banded first primary molars with soldered springs acting against the permanent molar were also suggested in cases with extraction of the second primary molars.[9,33-35] The effect of this type of appliance is based on reciprocal forces that act against the primary anchor molar, which may be subject to root resorption. Reinforcement of the anchorage should be considered, therefore, and may be achieved with a transpalatal bar soldered to a banded contralateral molar (Figure 6).

In cases where the second primary molar is lost, distal movement and uprighting of the first permanent molar with cervical traction headgear has been suggested.[11,36]

Several case reports on the treatment methods presented above are available. To our knowledge, however, no investigation of the effect of treatment and follow-up in children with ectopic eruption of the maxillary first permanent molar has been published.

The efect of cervical traction treatment was, therefore, studied longitudinally in children with irreversible ectopic eruption.[37] Forty-six children with irreversible ectopic eruption, for whom the resorbed primary molars were extracted, were treated with the Kloehn cervical traction headgear with straight outer bows. The applicance was used for twelve hours daily. The mean age at the start of the treatment was eight years and three months (6.5 - 9.9 years) and the mean treatment time was eight months (six to twelve months). Orthopantomograms, lateral head films and plaster casts were obtained at the start of treatment, at the end of treatment, and one year posttreatment.

The treatment goals, namely, uprighting to normal inclination and occlusion as well as regaining of space for the second premolar, were attained in thirty-one of the forty-six children treated. Uprighting of the first permanent molar to good occlusion was seen in all cases. In fifteen children, sufficient space was not regained for a second premolar and extraction of permanent teeth became necessary. These children showed the greatest space deficiency initially, but cooperation problems also contributed to the unsatisfactory treatment results.

The main effects of cervical traction treatment were uprighting of the permanent molar and a change in intermaxillary relationship, which may be attributed to a restriction of maxillary anterior growth caused by the cervical traction.[37] Treatment effects, both positive and negative, are illustrated in Figure 7. The registered effects of treatment, such as the rapid and comparatively great changes in intermaxillary sagittal and vertical relationships, necessitate a thorough orthodontic analysis before cervical traction treatment is started, to avoid unnecessary side effects and complications (Figure 7).

The most favorable treatment results with cervical traction were found in the older children whose second premolars were nearly erupted or were erupting at the end of treatment. Cervical traction treatment can be started, therefore, after the extraction of the second primary molar, thereby reducing the need for space maintainers.[37]

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Malocclusion patterns in fourteen children with Duchenne's muscular dystrophy

Arild Stenvik, Lic Odont
Kari Storhaug, Cand Odont

Duchenne's muscular dystrophy is a severe x-linked hereditary condition with an incidence of 1 in 3,000 to 4,000 male births and a population prevalence of 3 per 100,000. First symptoms are usually detectable between three and five years of age, and the majority of those afflicted lose the ability to walk, when between seven and thirteen years of age, due to muscle degeneration. Muscle tissue is replaced by fat and connective tissue, and in late stages, respiratory and cardiac muscles may be affected.

A linear decline in strength is a fundamental characteristic of this disorder.[1] Primary involvement is of the voluntary muscles, and muscles selectively involved by weakness and atrophy contrast with "pseudo-hypertrophic" muscles.[2] Affection of the central nervous system with cerebral atrophy has also been reported.[3] For the masticatory muscles, damage to the reflex arches and alteration in information from dystrophic muscle spindles have been suggested.[4,5]

As the head and neck contain muscles of varying derivation, affection of certain muscle groups may result in imbalances that will adversely influence the dental arches and occlusal relations. Although no accurate documentation of occlusal relationships in randomly selected groups of Duchenne patients is available, frequent occurrences of open bite and expanded dental

Drs. Stenvik and Storhaug are with the Department of Orthodontics, Dental Faculty, University of Oslo, Geitmyrsveien 71, 0455 Oslo 4, Norway.

Table ☐ Age (year), molar and incisor (mm) relationships and dental arch width and length (mm) in fourteen children with Duchenne's muscular dystrophy. Incisor relationship and arch width and length are measured according to specifications established by Moyers *et al.*[9]

		Molar relation (Angle class)				Crossbite		Maxillary molar		Mandibular molar	
No.	Age	Left	Right	Overjet	Overbite	Left	Right	Width	Length	Width	Length
1	8	II	II	5.0	1.8	+	+	47.2	40.2	47.7	35.4
2	9	I	I	5.2	0.0	−	−	49.7	38.3	43.3	33.3
3	9	II	II	12.7	−1.0	−	+	49.0	42.2	49.7	30.4
4	10	II	I	6.0	−8.0	+	+	50.3	32.0	53.2	27.2
5	10	I	I	2.0	3.6	−	−	53.0	32.6	49.2	28.4
6	10	I	I	8.3	−3.3	−	−	52.2	37.3	48.0	30.2
7	11	I	I	2.5	2.1	−	−	55.2	34.6	49.8	32.2
8	12	III	III	8.2	2.2	+	+	46.3	44.5	46.4	31.8
9	13	II	II	8.6	−2.0	+	−	48.3	38.6	48.5	32.3
10	13	II	II	1.5	0.0	+	+	54.0	33.7	57.7	34.0
11	13	I	III	2.0	0.0	+	+	52.0	33.6	51.8	29.0
12	14	I	I	3.5	4.2	+	+	57.3	34.2	58.4	29.4
13	15	III	III	1.0	0.0	+	+	54.4	32.6	57.2	25.0
14	15	I	I	3.0	3.1	−	−	57.4	38.0	51.6	32.8

arches have been reported.[6,7] The purpose of the present investigation was to determine whether this myogenic disorder, which may be expected to involve selectively specific muscle groups of the head and neck, will produce a consistent pattern of malocclusion.

SURVEY POPULATION AND METHODS

Fourteen male children (mean age, 11.6 years; range, 8 to 15 years), confined to wheel chairs with Duchenne's muscular dystrophy, and who were participating in a treatment and information course at Frambu Health Centre, formed the basis of a study of oral function and occlusal relationships.[8] The total population of these patients in the region of Norway covered by the Centre is thirty-three, and the fourteen boys included in the study were randomly selected with regard to malocclusion. Impressions for plaster casts and intraoral and extraoral photographs were taken. Muscular function and morphology were determined by visual observation, by palpation, and by listening for sounds produced during function of the masticatory system.

Measurements of dental arch width and length were made with calipers to the nearest 0.10 mm according to conditions established by Moyers *et al.*[9] The mean values and standard deviations from their investigation were used for comparison. Sagittal, transversal and vertical occlusal relationships were also recorded.

RESULTS

Occlusal and dental arch characteristics of the fourteen boys are presented in the Table. An increased mandibular dental arch width at the first molars and decreased arch length, measured from the incisors to the first molars, were seen in all patients. Figures 1 and 2 represent the values for mandibular and maxillary arch width and length for each patient. Deviation from the mean for the corresponding age-group is expressed in standard deviations.

Figures 1-3. Deviation in mandibular (figure 1) and maxillary (Figure 2) dental arch width and length and incisor relationship (Figure 3), in fourteen children with Duchenne's muscular dystrophy arranged from left to right according to chronologic age. Deviation from mean value for the age-group is for each patient expressed in standard deviations according to values established by Moyers et al.[9]

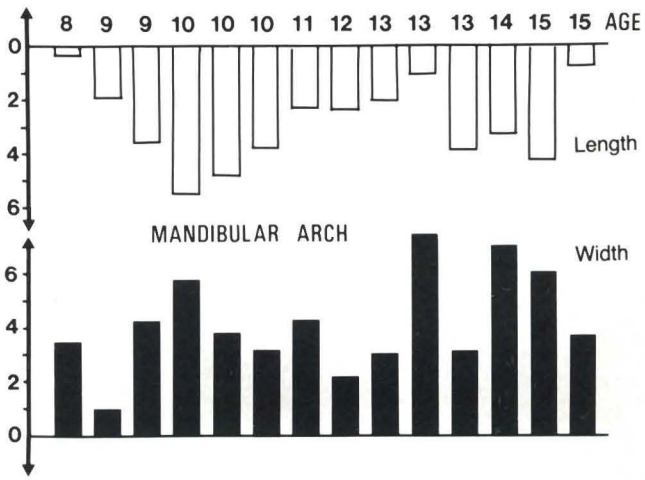

Figure 1.

Open bite did not occur consistently. The deviation in vertical and horizontal incisor relationship for each patient is presented in Figure 3. A marked tendency toward reduced overbite is apparent for the total group.

Lisping or other speech problems could be observed to a varying extent, and either frontal and/or lateral

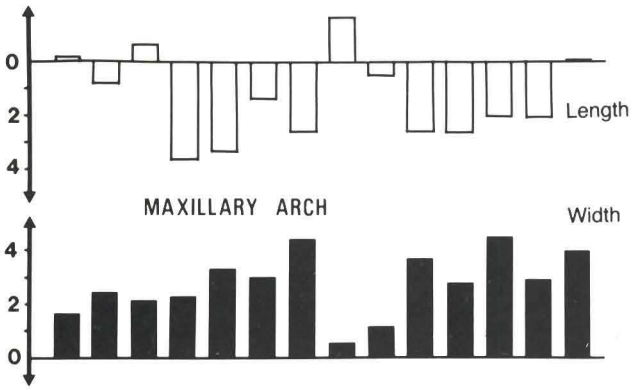

Figure 2.

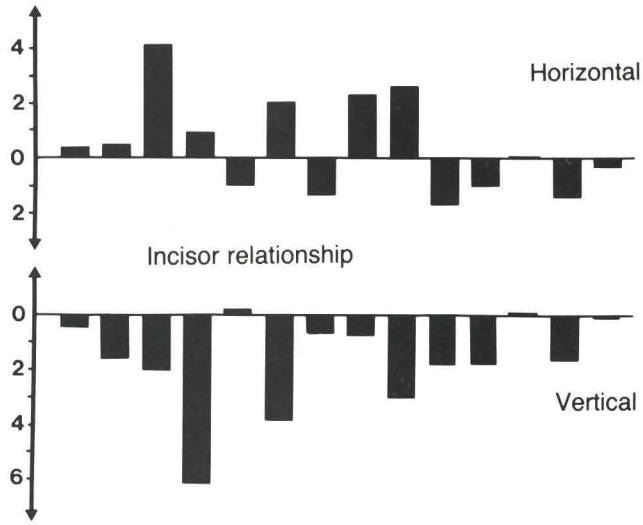

Figure 3.

tongue pressure during swallowing was seen in all patients. The soft tissue of the mental and submandibular areas appeared greater in volume (Figure 4) and was firm upon palpation. Pronounced muscular activity in this region during function contrasted with the hypotonic and inactive condition of the other facial muscles in most patients.

DISCUSSION

A tendency toward open bite and overexpanded dental arches was previously presented in case reports and in more extensive studies.[7,10-12] In the present survey

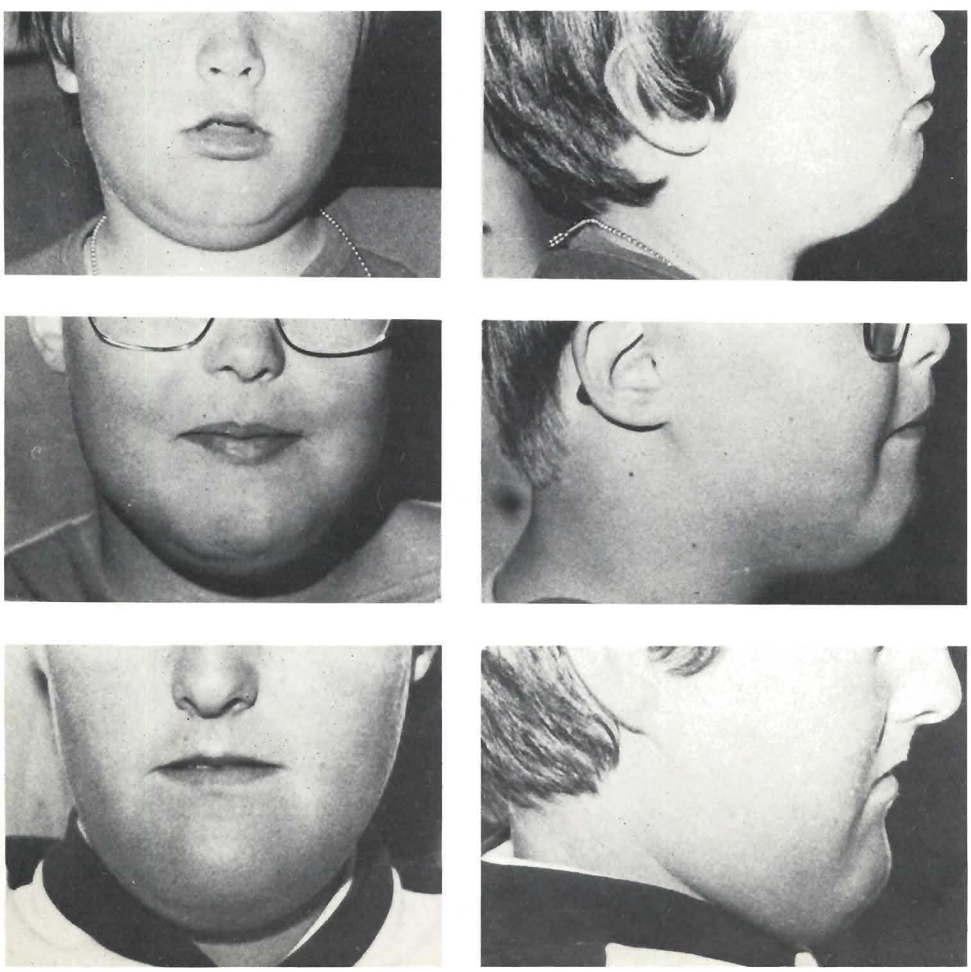

Figure 4. Characteristic morphology of lower face in three children with Duchenne's muscular dystrophy: Soft tissue of the mental and submandibular area appears to be increased in volume.

group, the largest diviations were an increase in the width of both arches and a decrease in the length of the mandibular arch. For these conditions, the same pattern was evident in all patients; for six patients, mandibular arch width exceeded normal values by four to seven standard deviations.

No typical skeletal pattern for Duchenne patients with open bite has been established; nor has any difference in masticatory muscle performance been observed in patients with or without open bite.[13,14] Although a reduced maximum biting force was demonstrated in Duchenne patients, muscle weakness *per se* is considered secondary to the functional neuromuscular damage of muscles in the development of malocclusion.[15]

The effect of the tongue upon the dental arches is difficult to establish. Some reports refer to macroglossia in these patients, but a radiographic examination of tongue size demonstrated a large tongue in only three of twenty patients with muscular dystrophy.[6,10,16] Active voluntary maximum muscular pressure from the tongue has been reported to exceed pressure from muscles external to the dental arch in Duchenne patients; but, as suggested for masticatory muscles, the role of muscle strength may be of less importance than the neuromuscular disturbances.[12] The abnormal movements of the facial muscles and of the swallowing pattern, observed in the present study, are consistent with a previous report.[13] It seems possible that certain muscle groups that participate in oropharyngeal functions may become selectively involved in this disorder. A consequent change in functional patterns may be specific to these patients and could thus give rise to an increased palatal width in the molar region, as established in the survey group.

The indication for orthodontic treatment to establish better masticatory function is uncertain. A progressive development of both the malocclusion and the disease makes the prognosis for such treatment dubious. No longitudinal studies of occlusal relationships in Duchenne patients, however, have been reported. A beneficial effect of masticatory muscle training was presented, and therapeutic exercises should, therefore, be considered for patients with Duchenne's muscular dystrophy.[14]

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Efficacy of fluoride on dental caries reduction by means of a community water supply

Prevention

Peter J. Fos, DDS, MPH
James M. Pittman, DDS, MPH

Fluoride's effectiveness in caries reduction has been studied for many years. Since the realization of fluoride's efficacy in the reduction of dental caries, local water supplies in many communities in this country have been fluoridated to optimal levels. Recent investigations indicated that there is an inverse relationship between the amount of fluoride in the water supply and the number of caries lesions.[1-4] Along with the fact that fluoride is a natural constituent of many water supplies, the fluoridation of water supplies has been found to be the most practical and effective method of fluoride administration.

The community water supply of Orleans Parish, Louisiana (which includes the city of New Orleans) has been fluoridated to optimal levels since June of 1974. This action was mandated after twenty years of discussion and controversy. The official resolution mandates that the fluoride level of the drinking water ". . . is to be maintained no lower than 0.7 parts per million (ppm) fluoride and not more than 1.0 parts per million (ppm)."[5,6] The water source for the drinking water in Orleans Parish, the Mississippi River, is naturally fluoridated. For the twenty-year period from 1960 to 1980, the fluoride level of the Mississippi River averaged 0.42 ppm. To comply with the mandated resolution, the Sewerage and Water Board (the public body that is

Dr. Fos is a part time clinician with the New Orleans Department of Health, Dental Division; and conducts a private practice in general dentistry.
Dr. Pittman is chief of the Dental Division.

responsible for the water needs of Orleans Parish) adds the appropriate amount of fluoride to reach optimal levels.

The optimal fluoride level in drinking water is that level that produces the greatest protection against dental caries, with the least risk of fluorosis.[7] Studies showed that the optimal level of fluoride in drinking water is 1.0 parts per million (ppm).[8] The drinking habits of the population of a specific community will have an influence on the amount of fluoride ingested and the optimal level. Because of the changing climate, water consumption will vary during the year. So, the fluoride content of the water supply must be adjusted periodically throughout the year, if compliance with optimal levels is to occur. When the water consumption is at its lowest (during the winter months), the fluoride concentration of the drinking water is at its highest level. Conversely, when the water consumption is at its greatest the fluoride concentration is decreased. A formula dependent on temperature has been derived for determining the optimal fluoride level:

$$\text{ppm fluoride} = 0.34/E$$

$$E = 0.038 + 0.0062 \times \text{avg. max. temp. (°F)}$$

For Orleans Parish the average yearly optimal fluoride concentration is equal to 0.75 ppm.[9]

Fluoride is added to the Orleans Parish water supply in the form of 25 percent hydrofluosilicic acid. To ensure safety, daily monitoring occurs with daily adjustment of the fluoride concentration. By adjusting the rate of the feeder pump (stroke of the piston) the fluoride concentration is increased or decreased. The end product of the pump setting is the introduction into the drinking water of a specific amount of pound equivalents of hydrofluosilicic acid per gallon that will yield the optimum level of fluoride. Table 1 shows the fluoride levels in the drinking water in Orleans Parish since 1974.[6]

Table 1 ☐ Fluoride levels in the drinking water in Orleans Parish since 1974.

Year	FLUORIDE(ppm)
1974	.61
1975	.74
1976	.74
	.75
1978	.71
1979	.71
1980	.80
1981	.74
1982	.81
1983	.75
1984	.78

JUNE TO DECEMBER 1974

SOURCE: Sewerage and Water Board, East Bank Treatment Plant, New Orleans, Louisiana.

METHODOLOGY

Subjects

In an attempt to study and analyze the caries reduction efficacy of fluoride, as well as cost-effectiveness of the fluoridation system, 200 children were selected. These children were welfare-assistance recipients (Title XIX) and patients of the City of New Orleans Health Department Dental Bureau. The subjects were divided into two groups. In Group I, 100 children born after June of 1974 were studied. Group II consisted of 100 children born before January of 1971. The primary molars were studied. Group I experienced optimal systemic fluoride exposure during molar development, while Group II had no systemic fluoride exposure during molar development.

Table 2 ☐ Results of analysis of the data.

GROUP	PATIENTS EXAMINED	MEAN def SURFACES	MEAN NUMBER EXTRACTIONS
I	100	4.16	0.32
II	100	6.14	0.92

Data collection

To obtain the necessary data, the def indices were collected for the eight primary molars per child. Data were collected using visual oral examinations and dental records. The teeth in question were studied, when the subjects in both groups were seven to eight years of age.

RESULTS

Table 2 shows the results of an analysis of the data. Subjects in Group I (the fluoridated group) exhibited 35 percent with no caries experience (35 out of 100); the remaining sixty-five subjects exhibited various degrees of decay. Also in Group I, 19 percent of the subjects (19

Figure 1. Calculations based on the data regarding decayed and filled surfaces of the teeth.

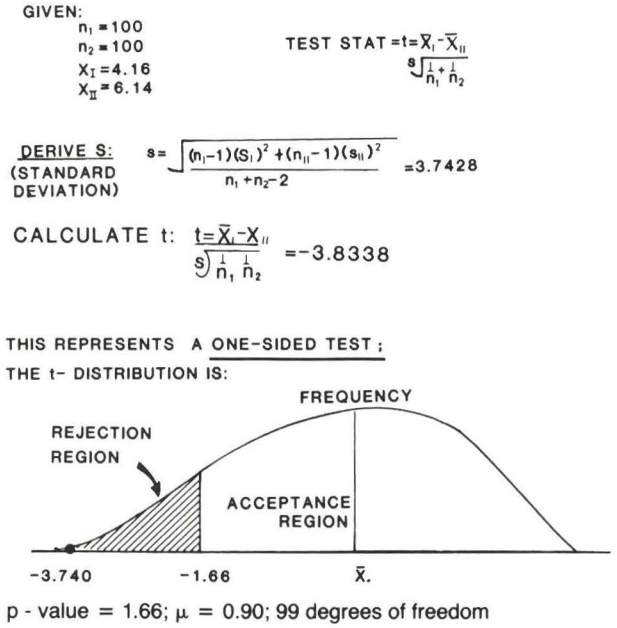
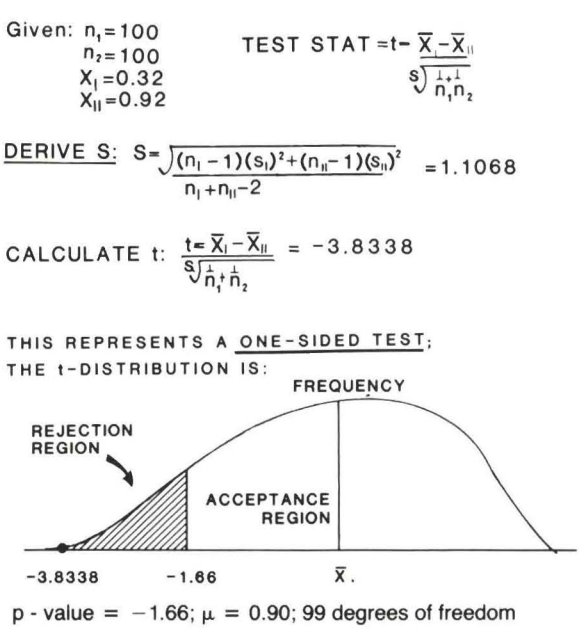


Figure 2. Calculations based on the data regarding extracted teeth.



out of 100) had experienced extractions (one or more). Subjects in Group II (the control group) exhibited 37 percent with no dental caries experience (37 out of 100); the remaining sixty-three subjects exhibited varying degrees of caries experience. But in Group II, 37 percent of the subjects (37 out of 100) had experienced extractions (one or more).

The mean number of decayed or filled surfaces per subject in Group I equaled 4.16; the mean number of decayed or filled surfaces per subject in Group II was 6.14. With respect to extraction, Group I exhibited a mean number per subject of 0.32; Group II subjects exhibited a mean number per subject of 0.92.

Calculations

Hypothesis testing was utilized to determine the validity of the collected data. Hypothesis testing provides a framework for making decisions on an objective basis by weighing the relative probabilities of different hypotheses. By hypothesis testing, it was attempted to determine whether the fluoridated group benefitted with reduced caries experience. To determine the significance of the data and to develop a statistical model for the hypothesis testing, the two-sample t-test was utilized. This statistical test was used, because only the variance of the sample groups could be determined; the population variance was unknown.

The Null hypothesis (Ho) states that the two groups should exhibit similar caries experience. The Alternative hypothesis (Hi) states that the fluoridated group (Group I) should show a greater reduction in caries. The Null and Alternative Hypotheses are:

- ☐ 1a. *Null Hypothesis:* the mean number of decayed/filled surfaces of Group I equals the mean number of decayed/filled surfaces of Group II.
- ☐ 1b. *Alternative Hypothesis:* The mean number of decayed/filled surfaces per subject in Group I is less than the mean number of decayed/filled surfaces per subject in Group II.
- ☐ 2a. *Null Hypothesis:* The mean number of extractions per subject in Group I equals the mean number of extractions per subject in Group II.
- ☐ 2b. *Alternative Hypothesis:* The mean number of extractions per subject in Group I is less than the mean number of extractions per subject in Group II.

The two-sample t-test analyzes independent samples with equal variances. The calculations are performed in several steps (See Figures 1 and 2). To calculate the test stat (t), derivation of the standard deviation(s) must occur first. Then the test stat can be computated and the t-distribution can be plotted. The t-test in this study was performed at a significance level (sigma) of 0.90, with 99 degrees of freedom and a p-value of −1.66. The p-value (critical value) is the probability of obtaining a result as extreme or more extreme than the actual sample value obtained, given that the Null hypothesis is true. The p-

value establishes the acceptance and rejection regions of the t-distribution. The acceptance region is the group of values of t that will accept the Null hypothesis. The rejection region is the group of values of t that will reject the Null hypothesis.[10] A one-sided test distribution was used to indicate an increase or decrease in caries experience.

The Null hypothesis is accepted for any t-value that is greater than the p-value (to the right of the p-value). In both t-tests, decayed/filled surfaces and extractions, the Null hypotheses were rejected. The test stat for the decayed/filled surfaces was calculated as −3.740, which is to the left of the p-value. The test stat for number of extractions was calculated as −3.8338, which is also to the left of the p-value. So, the Alternative hypotheses are true: the mean number of decayed/filled surfaces per subject in Group I is less than the number of decayed/filled surfaces per subject in Group II; the mean number of extractions per subject in Group I is less than the mean number of extractions per subject in Group II.

DISCUSSION AND CONCLUSION

The analysis of the data collected shows that fluoride has had an effect in reducing the caries experience in the subjects in this study. It is also apparent that the severity of decay was reduced by the optimal levels of fluoride. There is a large increase in the number of extractions in Group II, the control group. This indicates that the affected teeth in Group II were decayed to the extent that extraction was a frequent treatment. The mean number of decayed/filled surfaces in Group I was significantly less, which indicates that the fluoride decreases caries incidence.

To appreciate fully the data analysis, a few important points must be discussed. Because the majority of the data were collected from dental records, the chance of error in record keeping must be taken into account. The subjects were examined at different periods of time and by different examiners. Also, by the nature of the clinic system, many different practitioners performed the treatment. This can lead to differences in diagnosis and treatment philosophies, i.e., many extracted teeth could have been filled and many decayed teeth are overlooked in clinical judgement. These factors must be weighted before any conclusive evidence can be appreciated from this study.

As far as economic efficiency is concerned, if the results of this study can be considered indicative of fluoride's effect, the fluoridation effort, then, is very successful. With an initial expenditure of less than $8,000 and an average annual expenditure of less than $23,000, any reduction in caries would be very cost-efficient. Maintenance of the fluoridation system is very inexpensive; thus, as a long-term caries-prevention program, fluoridation is ideal and effective.

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Case reports

Hereditary hypophosphatemia rickets: an important awareness for dentists

Frank L. Herbert, DDS

Rickets is a disorder of developing bone, in which a disturbance of vitamin D metabolism occurs to produce a physiologic discrepancy between the production of bone matrix and the rate of mineralization of the matrix. This results in an accumulation of unmineralized matrix.[1] At one time, the simple expedient of supplying therapeutic doses of vitamin D was considered sufficient to stimulate remineralization of the organic matrix and produce normal bone.

Recognizing that some patients with rickets, however, did not respond to vitamin D therapy, Albright, Butler, and Bloomberg in 1937, described in detail a refractory condition, which they called vitamin D-resistant rickets (VDRR). This condition was resistant to the usual doses of vitamin D, but responded to massive doses of the vitamin.[2]

Reabsorption of phosphorus by the renal tubules was suggested by Robertson, Harris, and McCune as responsible to the pathogenesis of VDRR.[3]

Several other studies of large kindreds afflicted with the disease revealed hypophosphatemia to be the essential trait of the disorder, and rickets, when it occurred, to be secondary to this abnormality.[4-6] This form of hypophosphatemia, not correctable by small doses of vitamin D, is the result of a metabolic defect transmitted as a dominant characteristic through a gene of the X chromosome. Other forms of refractory rickets are reported in the literature, and at least thirteen rachitic

Dr. Herbert is Associate Professor, Department of General Dentistry, Louisiana State University School of Dentistry, LSUSD Box 127, 1100 Florida Avenue, New Orleans, LA 70119-2799.

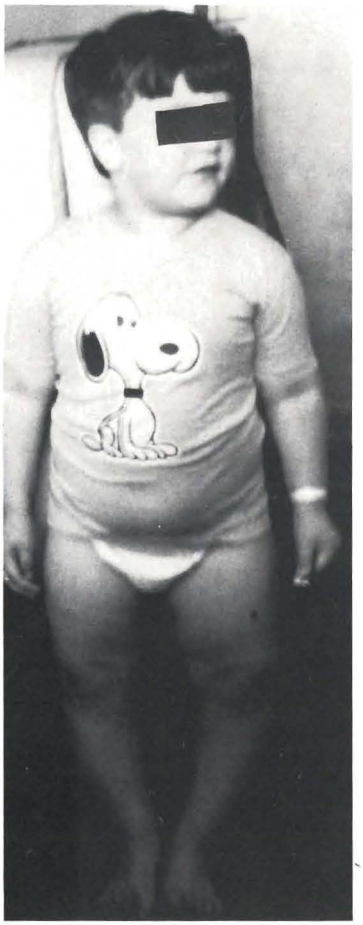

Figure 1. Physical appearance of patient.

conditions involving a variety of pathogenic mechanisms were reported by Fraser and Salter in 1958.[7]

In 1960, Harris and Sullivan first reported a case describing dental sequelae in the primary dentition of a patient with vitamin D-resistant rickets.[8] The well-cared-for, caries-free dentition of a seven-year-old boy mysteriously demonstrated multiple draining periapical abscesses that necessitated removal of the teeth. They postulated an association between the child's dental problems and his rickets, correlating the deficiencies in dentin formation with the well-defined bony changes.

Archard and Witkop corroborated this postulation with their report in 1966 of an apparently well-developed, well-nourished five-year-old boy who had recurrent gingival abscesses and proven hereditary hypophosphatemia.[9] Their report described distinctive dental findings similar to those reported by Harris and Sullivan:

- ☐ Multiple abscesses not associated with caries or trauma.
- ☐ Radiographic evidence of unusually large pulp chambers with long pulp horns extending into the cusp tips.
- ☐ Histologic evidence of abnormal dentin formation with large tubular clefts or voids extending to the dentinoenamel junction in the areas of the pulp horns.
- ☐ Invasion of the pulp by microcoorganisms directly through these large voids in the dentin.

Numerous investigators since have verified the histologic evidence of these abnormalities in dentin formation, and Sauk and Witkop, using the scanning electron microscope, confirmed the basic dentin defect to be elongated pulp horns extending into the incisal edge just below the enamel.[10-13]

Hypophosphatemia is a necessary finding to establish the diagnosis, and Seow lists ten more case reports confirming these skeletal and dental findings in hereditary hypophosphatemic vitamin D-resistant rickets.[14]

Because VDRR is inherited through an X-linked dominant gene, affected males are mutant hemizygous.[15] The hypophosphatemic trait is expressed uniformly in those who inherit the mutant allele, but the severity of bone disease is quite variable, and there is not consistent correlation between the severity of hypophosphatemia and the severity of bone disease in females. This is one distinction between VDRR and vitamin D-dependent rickets that is inherited as an autosomal trait and may, therefore, affect females as severely as males.[15]

CASE REPORT

In 1979, a six-year-old male child was referred by a dentist for "treatment of abscessed teeth." No other information was given over the telephone.

Upon arrival, a very recalcitrant, short child with severely bowed legs and distended abdomen was greeted by the staff (Figure 1). The child was loudly and vigorously resistant.

Both parents were embarrassed and apologetic, ex-

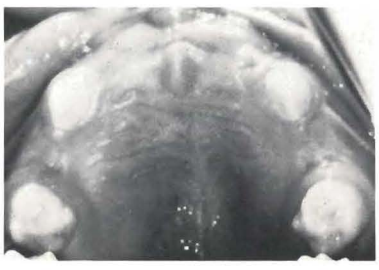

Figure 2. Partially edentulous maxillary arch resulting from earlier removal of abscessed teeth. No caries present.

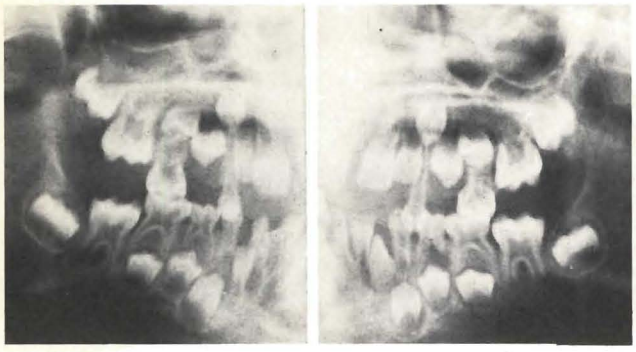

Figure 3. Panoramic radiograph of patient.

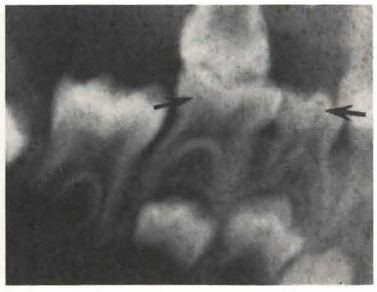

Figure 4. Magnified view of right side of panoramic radiograph. Note large stellate pulps and elongation of pulp horns into cusp tips (arrows).

plaining that the child was generally unmanageable and uncommunicative, and was receiving therapy for a major communication problem. They also reported that he was being treated for rickets. Because the child was adopted, they could provide no family history, despite many investigative attempts on their part.

The child had to be physically restrained by the father to permit intraoral dental examination. There was no evidence of dental caries or dental restorations. Maxillary right and left central and lateral incisors and first primary molars were absent, having been removed "because they developed gum boils," when the child was approximately two years of age (Figure 2).

An intact caries-free primary dentition was present in the mandibular arch, with permanent central incisors erupting normally into the space created by exfoliation of the primary central and lateral incisors. A parulis was apparent in the buccal mucosa adjacent to the lower left first primary molar.

Intraoral radiographs were not possible because of the child's vigorous resistance. A good quality panoramic radiograph (Figure 3) was obtained through the friendly persistence of a dedicated staff member who gave the child several "rides" in the panorex chair with the radiation switch off until he became comfortable enough with the equipment for the switch to be covertly turned on and thus to expose the film in the cassette.

The radiograph disclosed a radiolucency in the apical area of the lower left first primary molar, and large stellate pulp chambers with multiple pulpal horns extending close to the enamel and to the cusp tips in the molar teeth (Figure 4).

Consultation with a pediatric endocrinologist confirmed a biochemical picture consistent with a diagnosis of hypophosphatemia vitamin D-resistant rickets and not the vitamin D-dependent form.

Following a thorough physical and laboratory evaluation by the pediatrician and with clearance for a general anesthetic, the patient was admitted to the hospital for comprehensive dental care under general anesthesia. All ten remaining primary teeth were covered with stainless steel crowns. Tooth preparation was minimal and performed very carefully to avoid pulp exposure. Prior to cementation of the crowns the enamel surfaces of the teeth were coated with several layers of calcium hydroxide. The rationale was to prevent penetration of the defective tooth structure by the acid in the cementing medium (zinc phosphate cement) and subsequent pulp damage. Zinc oxide pulpectomy procedures were performed on the lower right and left first primary molars before covering them with the steel crowns. (Note: Reexamination of the mouth after the child was asleep revealed a small draining fistula in the area of the right molar.)

The child recovered uneventfully with complete remission of the fistulae. No further abscesses occurred for a period of twelve months, after which the patient moved and contact was lost.

SUMMARY

Because X-linked hypophosphatemia VDRR is reported to be the most comon form of rickets in the United States today, it is important for dentists to be aware of this condition.[14] Characteristic dental findings are often the first clinically noticeable signs of the disease. The confirmation of hypophosphatemia with dental findings will permit early diagnosis and prevent crippling rachitic deformities, otherwise certain to follow.

Reports in the literature confirm the validity of conservative, prophylactic full coverage restorations in patients with VDRR.[1617]

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EFFECT OF UNILATERAL EXTRACTION OF PRIMARY MOLARS ON CASES OF BILATERAL INFRACLUSION

After unilateral extraction of primary molars in infraclusion in bilateral cases, the successors were in most cases seen to erupt about a year earlier on the extraction side than on the nonextraction infracluded side. Earlier eruption occurred more often in children who were older at the time of extraction. On the nonextraction side, all infracluded primary molars exfoliated normally and the successors reached the normal position within the normal time range. In all cases a rapid normalization of alveolar bone height was noted after eruption of the succedaneous teeth.

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