ASDC
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN
JOURNAL OF DENTISTRY FOR CHILDREN

MAY—JUNE 1995

GALLERY OF THE INNOCENT

AGES 4-8

It is my firm belief that the most important single thing we can do — as parents, teachers, and health professionals — is to reinvent our adulthood. Perhaps because of the heady discovery of the stages of adulthood and the parallel denigration of the stages of childhood and adolescence, the all-important sense of difference between children and adults has diminished.

Children are the young of the species and, like the young of all species, they need adult guidance, direction, and protection. While we cannot reinvent adulthood for everyone, we can do it for ourselves as parents, teachers, and health professionals.

Asserting one's adultness does not mean being an ogre or a drill sergeant. It does mean that we appreciate that children, adolescents, and even young adults may not yet have a set of internalized rules and standards, nor a good set of controls over their emotions and behavior. The only way they are going to get these internalized rules and standards is from us.

— David Elkind

OF ALL THE ACTIVITIES CHARACTERISTIC OF A LITERATE FAMILY ENVIRONMENT, IT WAS SHARING OF STORIES THAT WE FOUND TO BE MOST IMPORTANT.

—Susan Engle

ASDC
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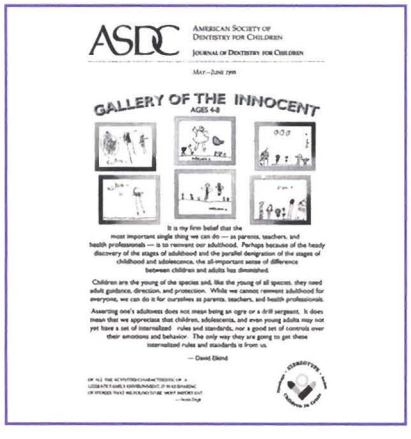

It is time that parents regain control of their children's lives by exercising intelligent leadership. Children need, and most of them want, guidance, direction, and protection.

Art and design by Sharlene Nowak-Stellmach

SPEECH

CLINIC

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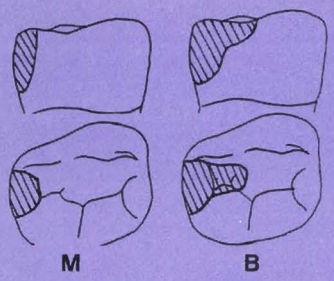

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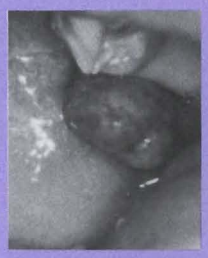

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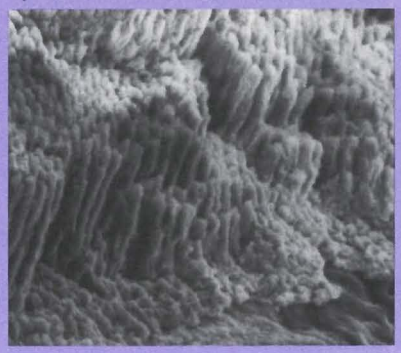

DEMOGRAPHICS

CASE REPORTS

Calendar

1995

ASDC Annual Meeting, Palm Springs, CA, October 11-15
Florida ASDC Unit, Gainesville, FL, September 8 & 9
Iowa ASDC Unit, Lake Okoboji, July 27-29

1996

ASDC Annual Meeting, Westin Canal Place, New Orleans, LA, October 23-27

1997

ASDC Annual Meeting, site to be determined, late October

For the busy reader

Premature loss of the maxillary primary incisors: Effect on speech production—page 173

In light of the growing prevalence of nursing caries and the devastation of the maxillary primary incisors it can cause, the authors studied the relationship of the premature loss of those teeth and speech production. They did show statistically significant differences in the percentages of misarticulations of six sounds produced by the children with missing or abnormal central incisor teeth and the children with normal teeth.

Requests for reprints to: Ms. Theresa Ott Gable, Northern Kentucky Easter Seals, 212 Levasor Avenue, Covington, KY 41014.

Effect of chewing gums on plaque pH after a sucrose challenge—page 180

Because of the widespread use of sweeteners and the limited information available regarding their relative acidogenic or anticariogenic properties, the authors were interested in determining whether chewing gum sweetened with these agents differ in their ability to reduce an acidogenic response of dental plaque from a sucrose challenge.

Requests for reprints to: Dr. Kichuel K. Park, Indiana University School of Dentistry, Oral Health Research Institute, 415 N. Lansing Street, Indianapolis, IN 46202-2876.

The effect of caries scoring systems on the association between dental caries and streptococcus mutans—page 187

The authors examined the effect of different systems of scoring caries on the association between caries indices and salivary *S. mutans* counts in a group of preschool children. Another objective was to determine whether eliminating filled surfaces from caries indices altered the degree of statistical association between caries prevalence and *S. mutans* levels.

Requests for reprints to: Dr. Lorne D. Koroluk, Division of Orthodontics, University of Minnesota, School of Dentistry, 6-320 Moos Health Science Tower, 515 Delaware Street S.E., Minneapolis, MN 55455.

Influence of fluoride in saliva during the early cariogenic changes in the enamel of boys and girls—page 192

The authors explain the failure of the traditional DMF index to indicate the clinical processes of demineralization and remineralization, because it refers to the cummulative caries prevalence in the past. To overcome this problem, they scored the cariogenic changes clinically observable within a short interval of time, and were, thus, able to study the effect of the actual F^- concentration in the saliva on caries development.

Requests for reprints to: Dr. J.H.M. Wöltgens, ACTA, Department of Oral Cell Biology, Van der Boechorststraat 7, 1081 BT Amsterdam, The Netherlands.

Effect of cavity form on the durability of glass ionomer cement restorations in primary teeth: A three-year clinical evaluation—page 197

The authors evaluated the durability of Class II glass ionomer cement restorations in primary molars in two types of cavities: a tissue-saving proximal microcavity and a slightly modified Black's Class II cavity. The quality of the restorations was assessed and scored according to the USPHS criteria described by Ryge and Snyder, and after six, twelve, twenty-four, and thirty-six months, or until exfoliation or failure.

Requests for reprints to: Dr. Ingrid E. Andersson-Wenckert, Dept. of Pedodontics, Umeå University, S-901 87, Umeå, Sweden.

Traumatic herniation of the buccal fat pad: Report of case—page 201

Traumas and lacerations of buccal mucosa are commonly seen in young children who fall with rod-like objects held in the mouth. The authors report the diagnosis and treatment of a twenty-month-old patient who was injured while holding a toothbrush in the mouth.

Requests for reprints to: Dr. Yasuharu Takenoshita, Second Dept. of Oral and Maxillofacial Surgery, Faculty of Dentistry, Kyushu University 61, 3-1-1, Maidashi, Higashiku, Fukuoka, 812 Japan.

Almost four million children with disabilities—page 205

The presentation reviews the prevalence of disabled children in the United States based on the 1991-92 Bureau of the Census reports and on reports from a variety of federal support programs. The regulations of the *Americans with Disabilities Act* has prompted a greater appreciation of the populations faced with a wide range of disabilities.

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

Is your pediatric practice keeping pace with the improving picture of dental economics?—page 210

The overall economics of dental practices continue to improve. Between 1980, 1990, and 1993, National expenditures for dental services increased from $14.4 billion to $34 billion to $41.0 billion. Some of these increases were due to inflation. In terms of constant dollars since 1990 there has been no change in the National spending level for dental services in the country despite an increase of more than 10 million residents.

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

Primary tooth ankylosis: Report of case with histological analysis—page 215

The authors present a case report of a patient with two ankylosed primary molars. The patient was observed from nine to fourteen years of age.

Requests for reprints to: Dr. Paolo Romagnoli, Dept. Human Anatomy and Histology, Section "E. Allara", Viale Pieraccini, 6, I-50139 Florence, Italy.

Latent fluorides: Report of case—page 220

The authors report on a case of dental fluorosis induced by a long-term consumption of a highly fluoridated (6.02 ppm/F) mineral water. Two children (siblings) drank only the mineral water for approximately the previous nine years.

Requests for reprints to: Dr. Jean R. Jasmin, Laboratory of Biomaterials and Experimental Odontologia (Director Pr. J. Exbrayat), Dental Faculty, Avenue Joseph Vallot, 06034 Nice Cedex, France.

SPEECH

Premature loss of the maxillary primary incisors: Effect on speech production

Theresa Ott Gable, MA
Ann W. Kummer, PhD
Linda Lee, PhD
Nancy A. Creaghead, PhD
Lisa J. Moore, MA

Children younger than five years of age often must have their maxillary anterior teeth extracted due to dental caries.[1-6] Trask contended that the nursing bottle is the largest cause of cavities in young children and is the result of liquids such as milk (both bovine and formula) and juices bathing the teeth of babies who go to sleep with bottles.[4] Even human milk and nonnutritive sucking habits have been implicated as factors in this form of dental decay.[6-9] Nursing caries, sometimes referred to as nursing-bottle caries, baby-bottle caries, or the condition of baby-bottle syndrome or milk-bottle syndrome, is described in the literature as a specific pattern of dental caries involving the maxillary primary incisors, followed by the maxillary and mandibular canines and first molars.[6,9,10] The four mandibular incisors are usually unaffected, because during sucking they are protected by the tongue and washed by saliva.[6,9,11]

The investigations by Dilley *et al* and Johnsen *et al* revealed that the maxillary primary incisors are the most severely affected by caries associated with "prolonged" or "excessive" nursing habits.[2,5] These teeth are at greater risk than other teeth because they are among the first to erupt and are maximally exposed to liquids.[9] The caries pattern is usually not identified by a professional or noticed by the parents until approximately eighteen to twenty-two months of age, although the caries may begin before twelve months of age.[2,3,5] Only brown, decayed root stumps may be left in advanced cases.[6] Extraction is required when the caries process is not treated and the teeth decay extensively.[11]

The effect of missing maxillary incisors on speech production has been the subject of only a few research investigations, some of which were conducted as long as thirty years ago. Snow studied the effect of missing maxillary primary central incisors on articulation of the sounds /f,v,θ,ð,s,z/.[12] In her study, the articulation of 438 first-grade children was evaluated. None of the children had an organic limitation and none had received previous speech therapy. Snow compared the production of /f,v,θ,ð,s,z/ by children with normal maxillary central incisors to productions by children with missing or grossly defective maxillary central incisors. The results of this investigation revealed statistically significant differences in the percentages of misarticulations of all six sounds produced by the children with missing or abnormal central incisor teeth and the children with normal teeth.

The authors are grateful to Ernest Weiler, PhD, for his guidance with the statistical analysis. The contributions of Murray Dock, DDS; Michael P. Berry, DDS and Nick Murphy, DDS, are also appreciated.

The study was made in the Division of Pediatric Dentistry and Speech Pathology Department Children's Hospital Medical Center, Cincinnati, OH. Ms. Gable is with Northern Kentucky Easter Seals; Dr. Kummer is with Children's Hospital Medical Center, in Cincinnati; Dr. Lee and Dr. Creaghead are with the University of Cincinnati. Ms. Lisa J. Moore is deceased.

Although the differences in productions between the two groups were statistically significant, approximately 75 percent of the children with missing or abnormal dentition produced the test sounds correctly. In addition, the children with normal teeth produced misarticulations with an average frequency of 18 percent. Snow summarized that in some children, a causal relationship between abnormal dentition and disordered articulation of the sounds tested may exist. She cautioned, however, against conclusions that assume a direct correlation between deviations in the dentition and disordered patterns of articulation. Overall, Snow concluded that correct articulation for the sounds studied was not negatively influenced by defective incisor teeth.

Bankson and Byrne hypothesized that if the open spaces, due to missing teeth, cause changes in the acoustic characteristics of sound production, developing and establishing sounds may be affected. Hence these investigators conducted a study on the effects of the loss of maxillary and mandibular primary incisors and canines on articulation of the fricatives /s,z,f,ʃ/. It should be noted that, "the term missing teeth included all or any one or combination of the upper and lower medial and lateral incisors or canine teeth".[13] Bankson and Byrne tested 304 children, in May 1960, at a mean age of six years and one month and retested these same children four months later, in September 1960. At the time of the pretest all of the children included in the statistical analysis had all twelve incisors and canines intact. Upon the pretest these children were then assigned to one of two groups:

- ☐ Those with correct sound production and teeth intact.
- ☐ Those with incorrect production and teeth intact.

They did not find any significant differences for the productions of /z,f,ʃ/, following loss of maxillary and mandibular primary incisors and canines in children who had produced the sound correctly on the pretest.[13] Results of their study indicated, however, "a statistically significant relationship between production of the initial, medial, and final /s/ and presence or absence of incisors and canines among children who had used the sound correctly on the pretest".[13] Despite this significant finding, the authors noted that a large number of the children tested continued to produce accurate /s/ sounds. They postulated that the changes from correct to incorrect productions of /s/ were probably related to loss of the incisor teeth, not maxillary or mandibular canines, since only three children in the entire subsample were missing canines. It was found, however, that among children who had lost a combination of two or more teeth, those whose productions changed from correct to incorrect were in higher proportion than those whose productions did not change. These investigators did not examine the relationship between specific missing teeth and articulation errors.

Like Bankson and Byrne, Reikman and ElBadrawy were interested in examining the longitudinal effect of missing dentition on sound production. Where Bankson and Byrne, however, studied articulatory patterns of children who had lost their maxillary and mandibular incisors and canines under normal maturational conditions, Reikman and ElBadrawy examined the speech of children who had lost their maxillary primary incisors due to premature extraction as a result of nursing caries. Subjects for their study included fourteen children with a mean age of nine years and one month who had their maxillary primary incisors extracted at a mean age of two years and ten months. An average of six years, three months had elapsed between the time of extraction and the assessment of articulation. In general, these researchers found there were no apparent long-term effects of premature loss due to extraction on speech development of the children they tested, with the exception of four children (40 percent of the sample) who were younger than three years of age at the time of their extractions. Of these four children, two were evaluated as having some mild speech distortions and two were diagnosed with severe distortions of speech.

Occlusion may also be an important element in articulation, but again, this has not been extensively researched. Witzel, Ross and Munro examined the effect of facial osteotomies to correct occlusal defects on articulation.[14] These investigators assessed articulation both before and after surgery. They hypothesized that in apertognathic or prognathic conditions labiodental sounds may be affected; and in prognathic, retrognathic, or apertognathic conditions, sibilant sounds, /s,z,ʒ,ʃ,ʧ,ʤ/ may be affected. Subjects were grouped and articulation examined according to occlusal conditions. Preoperatively, these investigators found that subjects in all of the above occlusal groups produced error productions of sibilants, but nearly half of the subjects demonstrated no articulation errors. Postoperatively, a significant improvement in the sibilant sounds was found for all groups, as a result of surgical correction of occlusion.

Kummer *et al* also studied the effect on articulation of surgically improving occlusion.[15] In their study of patients undergoing LeFort I maxillary advancement for midface and occlusal abnormalities, eleven of sixteen patients demonstrated articulation errors preoperatively. Postoperatively, seven of eleven patients showed an im-

provement in articulation, presumably due to the normalization of occlusion.

Although the effect of missing dentition on articulation was the subject of three of the above investigations, only one study, that of Reikman and ElBadrawy, looked at the effect of premature loss of dentition on speech production.[1,12,13] In addition, there were variations across investigations, in terms of the numbers and types of missing teeth that were examined in relationship to articulation and in terms of the speech sounds studied. Reikman and ElBadrawy were exclusionary in their definition and only included subjects with premature loss secondary to extraction of the four maxillary primary incisors. Because of their small sample size of fourteen subjects, these authors were cautious in their conclusions. In summary, although they stated that the premature loss of these four teeth did not appear to affect developing or established patterns of speech, they also suggested that premature loss in children younger than three years of age may result in residual speech problems.

Dentists and speech-language pathologists often need to counsel parents about the possible effects of early incisor extraction on speech production. Such questions as "How will my child's speech be affected?" or "Will my child need speech therapy before or after the permanent teeth erupt?" are not easily answered based on the present literature. The purpose of the present investigation was to examine further, therefore, the effect of premature extraction of maxillary primary central and lateral incisors on the development of speech.

METHOD

Subjects

Twenty-six children, with a history of premature loss due to extraction of the central and lateral maxillary incisors, were selected from the dental records of the Division of Pediatric Dentistry of Children's Hospital Medical Center in Cincinnati, Ohio. Criteria for selection of these subjects included a history free of medical complications. All twenty-six subjects had lost the four maxillary primary incisors by means of extraction before the age of five. All subjects had their four maxillary permanent incisors at the time of testing. The subjects' ages ranged from eight years and one month to ten years and eleven months at the time of this study with a mean age of nine years and five months. The mean age at the time of extraction was three years and five months, three years and ten months, and three years and two months for the eight-, nine- and ten-year-olds, respectively.

Twenty-six subjects with a history of normal exfoliation of their four maxillary primary incisors served as the comparison group. All of these subjects also had their maxillary permanent incisors at the time of testing. These subjects were randomly selected at the Division of Pediatric Dentistry and outpatient departments of Children's Hospital Medical Center in Cincinnati, Ohio and ranged in age from eight years and one month to ten years and eleven months, with a mean age of nine years and four months. Means and standard deviations of subjects' ages when tested are presented in Table 1 for both the group with premature loss and the comparison group.

Two of the twenty-six subjects with premature loss had received speech therapy, both for less than three months. Five of the twenty-six subjects with normal exfoliation of their incisors had received speech therapy for an average of two years, six months.

Procedures

Subjects with premature loss of their incisors were contacted through letters, followed by telephone calls. The subjects with normal exfoliation of their incisors were selected from the Division of Pediatric Dentistry and outpatient departments following their appointments. Parents of children from both groups were informed of the purpose of this study and that their child would receive a free articulation evaluation, hearing screening and dental screening, through their participation. Written informed consent was obtained from all participants.

All testing took place in the Speech Pathology Department and Division of Pediatric Dentistry of the Children's Hospital Medical Center. For inclusion in the

Table 1 ☐ Means and standard deviations of subject ages when tested for both subject groups.

Subject age at testing	Mean age at testing	Standard deviation
Age 8		
Premature loss group	8:5	3.25 mo
Comparison group	8:5	3.27 mo
Age 9		
Premature loss group	9:5	6.57 mo
Comparison group	9:5	4.08 mo
Age 10		
Premature loss group	10:7	3.33 mo
Comparison group	10:3	3.87 mo

Table 2 ☐ Classification of occlusion and abnormal bite exhibited by both subject groups. Some subjects exhibited both a malocclusion and an abnormal bite and are represented more than once.*

	Premature loss group			Comparison group		
	Age 8	Age 9	Age 10	Age 8	Age 9	Age 10
Occlusal relationship						
Class I	5	4	6	5	8	6
Class II	1	2	1	1		
Class III		1				1
Mixed	2	3	1	1		
Variant incisor relationship						
Deep overbite	1	1				
Crossbite	2	1			2	2
Openbite	1	3	1			

*Four subjects from the comparison group were not included.

study, subjects had to pass a puretone hearing screening.

The occlusion was evaluated by a dentist and recorded according to incisor relationship and Class I, Class II, or Class III molar relationships.[16] For the purpose of this study, asymmetrical occlusal classifications such as Class I/Class II were catergorized under the heading of mixed occlusal relationships. Incisor relationships of deep overbite, crossbite, and openbite were also noted. This information is presented in Table 2.

Articulation was evaluated by two graduate students. To establish rater reliability, approximately 40 percent of the subjects were also rated by a speech-language pathologist with a PhD degree and over fifteen years of experience. These ratings were in full agreement.

The Photo Articulation Test was administered in order to assess the production of all consonant sounds (/s,z,ʃ,ʧ,ʤ,t,d,n,l,θ,r,k,g,f,v,p,b,m,w,h-w,ð,h,ɧ,j,ʒ/) as well as three consonant blends (/s/ blends, /l/ blends and /r/ blends).[17] Most consonants were tested in the initial, medial, and final word positions, with the exception of /w/ (only tested in the initial and medial positions), /hw/ (only tested in the initial position), /h/ (only tested in the initial position), /ɧ/ (only tested in the medial and final positions), /j/ (only tested in the initial and medial positions) and /ʒ/ (only tested in the medial and final word positions), since these phonemes only occur in these word positions in the English language. A total of seventy-six possible phonemic productions were assessed on this test.

Assessment of articulation at the sentence level was also conducted (see Appendix). Subjects were asked to either read or repeat (if unable to read) sentences containing the following target phonemes in the initial, medial and final word positions: / f,v,ʃ,ʒ,θ,ð,s,z,ʧ,ʤ,t,d,n/. All phonemes were tested in the initial, medial, and final word positions with the exception of /ʒ/ which was only targeted in the medial and final word position (since it does not occur in the English language in the initial word position). On this sentence test, a total of thirty-eight phonemic productions were tested. Fewer phonemes were targeted at the sentence level, therefore, than on the Photo Articulation Test and no blends were tested. The phonemes targeted on the sentence test were only those that would be expected to be produced in error by subjects with missing dentition, due to the placement in the oral cavity when these sounds are articulated.

For analysis of both word and sentence tests, phonemes were categorized according to the definitions of Edwards and Shriberg.[18] Error productions were coded as sibilant (/s,z,ʒ,ʃ,ʧ/ʤ/) distortions, substitutions, or omissions. A production was tabulated as an error for each word position in which it was misarticulated on the Photo Articulation Test. If a phoneme was misproduced more than one time in any one word position on the sentence test, it was counted as only one error.

Data analysis

Six t-tests for related measures were used to compare data from the two groups. Tests were conducted for word and sentence contexts separately, one for each age level (eight-, nine- and ten-year-olds).

RESULTS

Types and number of articulation errors

The types and numbers of articulation errors produced by the subjects with premature loss of their four maxillary primary incisors (premature loss group) and the subjects with normal exfoliation (comparison group) are presented in Tables 3 and 4. Twelve of the twenty-six subjects with premature loss and thirteen of the twenty-six comparison subjects produced some type of articulation error. Subjects with premature loss demonstrated more total articulation errors (fifty-one at the word level and forty-seven at the sentence level, compared with forty and twenty-one for the comparison group, respectively). T-test comparisons revealed, however, that these differences were not statistically significant ($p>.05$).

For both word and sentence contexts, distortions of sibilants /s,z,ʒ,ʃ,sp,st,sk,tʃ,dʒ/ were the most commonly occurring errors for subjects in the premature loss group. No errors of distortion occurred in any other

Table 3 ☐ Types and number of error productions in at least one word position on the Photo Articulation Test*. The number of subjects is recorded in parentheses. Some subjects may be represented more than once across error categories. The actual number of subjects (# Ss) is represented in total at the bottom of the table.

	Premature loss group			Comparison group		
	Age 8	Age 9	Age 10	Age 8	Age 9	Age 10
Total N =	(8)	(10)	(8)	(8)	(10)	(8)
	Number of errors					
Sibilants/s, z, ʒ, ʃ, sp, st, sk, ʧ, ʤ/ Distortions	29 (3)			8 (1)		
Fricatives/θ, ð, f, v/ Substitutions	3 (2)	2 (2)		4 (2)	11 (5)	2 (1)
Omissions	1 (1)	2 (2)	4 (4)	1 (1)	1 (1)	
Stops/t, d, g/ Omissions	2 (1)	2 (1)	6 (3)		2 (2)	
Liquids/r, br, kr, tr/ Substitutions				11 (2)		
Total errors and (# Ss)	35 (5)	6 (3)	10 (4)	24 (6)	14 (6)	2 (1)

*Not all error types were seen in each sound category tested.

Table 4 ☐ Types and number of error productions in at least one word position on the sentence test.*. The number of subjects is recorded in parentheses. Some subjects may be represented more than once across error categories. The actual number of subjects (# Ss) is represented in total at the bottom of the table.

	Premature loss group			Comparison group		
	Age 8	Age 9	Age 10	Age 8	Age 9	Age 10
Total N =	(8)	(10)	(8)	(8)	(10)	(8)
	Number of errors					
Sibilants/s, z, ʒ, ʃ, ʧ, ʤ/ Distortions	23 (3)		14 (1)	6 (1)		
Fricatives/θ, ð, f, v/ Substitutions	6 (4)	2 (1)	1 (1)	5 (2)	7 (5)	2 (1)
Stops/t, d, g/ Omissions	1 (1)				1 (1)	
Total errors and (# Ss)	30 (5)	2 (1)	15 (1)	11 (3)	8 (6)	2 (1)

*Not all error types were seen in each sound category tested.

sound category. The second most commonly produced errors for this group were in the form of substitutions and omissions of fricatives /θ,ð,f,v/.

The two most frequently produced error patterns by the comparison group were the same as those produced by the premature loss group, but their relative frequency was in reverse order. The majority of errors produced by the comparison group for both word and sentence contexts were substitutions of fricatives /θ,ð,f,v/. The second most frequently produced errors were distortions of sibilants /s,z,ʒ,ʃ,sp,st,sk,ʧ,ʤ/. These errors of distortion were produced, however, by only one subject.

Effect of age on articulation errors

The eight-year-old subjects in both groups produced more total errors than the other two age-groups combined, at both word and sentence levels. There were no statistically significant differences between the premature loss and comparison groups at any age level. Out of seventy-six phonemic productions on the word test, eight-year-old subjects in the premature loss group produced thirty-five total errors for word level testing, while nine- and ten-year-old subjects combined produced only sixteen errors. Eight-year-old subjects in the comparison group produced a total of twenty-four errors at the word level, whereas the nine- and ten-year-old subjects together produced just sixteen errors. Out of thirty-eight phonemic productions on the sentence test, eight-year-old subjects in the premature loss group produced a total of thirty errors. Nine- and ten-year-old subjects in the premature loss group only produced a total of seventeen errors on this test. For the comparison group, the eight-year-old subjects produced eleven errors at the sentence level, while the nine- and ten-year-olds combined error total was only ten errors for the sentence context.

Occlusal classification

Class I occlusions were found in the majority of subjects in both groups (Table 2). In the premature loss group, fifteen subjects had Class I, four had Class II, one had Class III molar relationships, and six had mixed occlusal relationships. Of those subjects in the comparison group, nineteen had Class I, one had Class II, one had Class III molar relationships, and one had mixed occlusal relationships. Variant incisor relationships, that is deep overbite, crossbite and openbite, were observed in ten of twenty-six (38 percent) subjects with premature loss and in four of twenty-two (18 percent) subjects with normal exfoliation of their incisors. Information about occlusion was unobtainable from four of the subjects with normal exfoliation of their incisors.

Fifty-five percent of the subjects in the premature loss group who presented with a malocclusion produced no articulatory errors. Four other members of this group presented with just a few errors. Only one subject, who

had a mixed occlusal relationship, produced one sibilant distortion. Three subjects in the comparison group were found to have malocclusions. One of these subjects, who had a mixed occlusal relationship, produced a few sibilant distortions; and another, who had a Class II molar relationship and three years of speech therapy, produced a liquid /l,r/ substitution.

DISCUSSION

Previous investigations of children with normal exfoliation of their teeth have consistently shown that the majority manage to compensate and produce correct sounds.[12,13] Although some children produce distortions of fricatives in the presence of missing incisors, parents are rarely concerned with these sound errors, correctly assuming that they will disappear once the permanent teeth erupt. Loss due to extraction of the incisors during the time of speech acquisition, however, is a more complex issue for parents, dentists, and speech-language pathologists.

The results of the present investigation showed no significant differences in the articulatory abilities of children with histories of premature loss of the four maxillary incisors and those with histories of normal exfoliation. Both subject groups revealed a maturation effect, in that the number of articulation errors decreased as the subjects aged. Very few residual problems remained by the time the children were ten years old. Even in the presence of malocclusion, the majority of subjects produced normal articulation. These results are similar to those of the previous investigation of speech production following premature extraction.[1]

Dentists and speech-language pathologists who must counsel parents about the effects of premature loss of teeth can refer to two bodies of literature. Studies of approximately 700 children with normal exfoliation of their incisors reveal that most will still manage to produce normal sounds, and most of those who have some sound distortions will self-correct their errors when the missing teeth are replaced.[12,13] Fourteen children with premature extraction of various incisors, and twenty-six from the present study with extraction of all four incisors, showed similar patterns. It appears that children who have their incisors extracted before the age of five years will probably make no greater number of articulation errors, once the permanent dentition erupts, than children with normal exfoliation. In addition, the majority may be expected to acquire normal speech, without therapy intervention, by the age of ten years.

Further research is needed to determine whether children who fail to acquire normal speech sounds after early loss of teeth display special characteristics that could allow one to predict early speech problems. In these exceptional cases, speech therapy or prosthetic intervention may be considered during the time between extraction and development of permanent dentition.

Conclusions

- ☐ At least one-half of the subjects tested in both groups produced articulation errors.
- ☐ The premature loss group produced more articulation errors overall than the group with normal exfoliation.
- ☐ Sibilant distortions and fricative substitutions were the most commonly produced errors by both groups.
- ☐ No statistically significant differences in error production were found between subject groups.

Appendix

Sentence Test with Target Sound Underlined.

Father has five fingers.
I drink coffee at the office.
One knife will be enough.

Valerie drove her van.
There were seven beavers.
They didn't want to leave the cave.

Sam will turn six.
The castle was messy.
Bess went to her house.

Our zoo has one zebra.
Susie likes music.
Please don't hurt Liz.

She went shopping.
We were washing dishes.
I wish Joe had some cash.

The garage is beige.
I usually watch television.

Thank you for thinking of me.
Something is better than nothing.
Both children need a bath.

They were over there.
I'd rather play the other game.
Bathe your skin until it's smooth.

Chocolate cookies are on this chair.
The ketchup is in the kitchen.
Rich was at lunch.

Jane is playing jumprope.
Roger is a magician.
We made a huge batch of fudge.

Ten children asked for tacos.
We saw nineteen guitars.
My seat is wet.

Dad and I went out for dinner.
Judy sang louder.
The bread was good.

Neither of us knew what to do.
The pennies fell out of the canoe.
Ann just turned ten.

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OTITIS MEDIA AND SPEECH DEVELOPMENT

Otitis media (OM) is one of the most common reasons for an illness visit to pediatricians. In 1990 it was estimated that 24.5 million visits were made to physician's offices in which the principal diagnosis was OM. In the past 15 years, office visits for OM in the United States have increased 150 percent. Each year, health expenditure for the treatment of OM in the United States is estimated to be close to $2 billion. Infants with recurrent OM (ROM) may suffer resultant hearing loss, which may affect normal speech development during a crucial period in their lives.

The following risk factors for ROM have been identified: being male, being formula fed, having siblings, attending day care, having parents with allergies or with a history of ROM, and cold weather months.

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CLINIC

Effect of chewing gums on plaque pH after a sucrose challenge

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A number of studies have shown that the consumption of fermentable carbohydrates promotes a plaque pH-drop to values far below the critical pH at which enamel starts to demineralize.[1] Since this bacterial production of acids in dental plaque is a key component in the development of dental caries, the use of alternative sweeteners for sucrose has received serious attention during the last two decades.[2,3] The information on the role of alternative sweeteners on plaque metabolism, however, is relatively limited. The polyols (sorbitol and xylitol) and the "high-intensity sweeteners" (aspartame, saccharin, and acesulfame-K) have been used in many foods and are widely used by the manufacturers of products such as confectionery, chewing gums, and soft drinks.[3-6]

During the past two decades a number of reports have appeared in the literature regarding the influence of various alternative sweetening agents upon the metabolism of selected oral microorganisms as well as dental plaque. In several instances the impact of the agents upon dental caries has been evaluated using both experimental animal models and controlled clinical trials.[6-12] Alternative sweetening agents that have been evaluated in this manner and are relevant to this investigation include saccharin, xylitol, aspartame, and acesulfame-K. All of these agents have been shown to be essentially noncariogenic, and reports have suggested that some of the agents may actually exert anticariogenic properties.

In view of the widespread use of these sweeteners and the limited information available concerning their relative acidogenic or anticariogenic properties, the objective of this investigation was to determine, using an in situ wire telemetry system, whether chewing gums sweetened with these sweetening agents differ in their ability to reduce an acidogenic response of dental plaque from a sucrose challenge.

MATERIALS AND METHODS

Informed consent letter/health history forms were approved and implemented under the guidelines of the Institutional Review Board of Indiana University - Purdue University at Indianapolis. The consent forms were provided to each volunteer, and only those panelists who completed this form and had a negative health history

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were accepted. Subjects for this project had been screened and selected according to the general selection criteria established by the American Dental Association's Health Foundation consensus report.[13] Eight adult subjects participated in this project. The mean age of the panelists was thirty-eight years, with a range from twenty-five to forty-six years, and their average DMFT and DMFS scores were 11.0 and 22.2, respectively. Their appliances, each containing an interproximal wire-telemetry pH sensor, were prepared for a missing mandibular molar as previously described.[14]

The test design was a seven-period randomized block. Test regimens consisted of five commercially-available gums (from the U.S., Canada or Europe) sweetened with sucrose (BeechNut® Peppermint), or high-intensity sweeteners aspartame (Extra®, Peppermint), saccharin (Trident® Peppermint), or acesulfame-K (Trident® Original), or xylitol (Sugarless X® Peppermint). All gums were peppermint or peppermint-like flavor. Gums sweetened with the high-intensity sweeteners also contained bulking agents such as sorbitol. In addition, paraffin and no-gum chewing regimens were included as controls. Panelists used one of the randomly selected test regimens for the first test period and similarly used the other test regimens during the subsequent six test periods. Each panelist used each chewing regimen once.

Before beginning the study, the subjects brushed and flossed, inserted their appliances and refrained from any oral hygiene procedures in the area of the appliance for seventy-two hours. They were asked to fast for at least twelve hours before each test period. Upon participant arrival at the Institute on the morning of each test, the electrode-containing appliance was removed, and the electrode was connected to a preamplifier, which was channelled to an IBM-PC through a Techmar Data Acquisition System (LabMaster). The connection joint then was sealed with a mixture of thermoplastic resins and waxes (dental prosthetic sticky green compound), and the appliance was reinserted into the mouth. Conductive electrode cream (Cor-gel electrolyte gel) was placed on the participant's cheek on the same side as the indwelling system, and the flexible reference electrode was taped in place using surgical tape. To aid in data analysis, event marks were placed on the x-axis of the plaque pH curves to identify each event that might affect the plaque pH response.

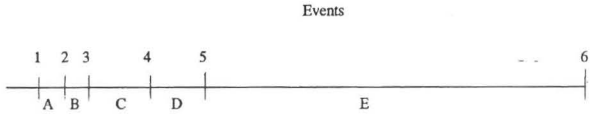
At the beginning of each test, the participants were asked repeatedly to rinse their mouths thoroughly with a total of 50 ml of warm tap water. They then chewed paraffin, and their plaque pH was allowed to stabilize for approximately two to three minutes. The stable baseline pH value was recorded for approximately five minutes.

After baseline pH responses were recorded, the test sessions were initiated. The participants were asked to rinse with 10 percent sucrose solution for two minutes and swallow (or expectorate). The plaque pH response was then monitored for ten minutes to confirm an acidogenic response. The panelists were then asked to use their specified treatment regimen for ten minutes. An effort was made by the panelists to distribute the test solution and to chew the gum as uniformly about the entire mouth as possible. The appliances had an all metal pontic, which permitted gum chewing directly in the area of the electrode. None of the panelists indicated any problem with the gum sticking to the partial denture. At the end of the gum chewing period, they were instructed to continue their normal swallowing behavior during the remainder of the experiment (93 minutes). The time of usage of the test regimens for each participant was indicated with an event mark. At the end of each test session, the monitoring screen was shifted to a new screen, and an intraoral calibration of the indwelling pH electrode system was performed by exposing the electrodes to reference buffer solutions of pH 7.0 and 4.0. The responses obtained from the two reference buffers were used for calibration of the test pH responses from the mV readings.

Following each test the indwelling pH system was disconnected, and the reference electrode removed. The plug was then replaced in the connector receptacle on the pontic. During this period, the subjects brushed and flossed. They then inserted their plaque-covered appliances and refrained from any oral hygiene procedures to the appliances until the next scheduled test. Two more test periods were scheduled on the next two consecutive days, thus completing the first part of the study. The following week the second part of the study was completed after a second plaque accumulation period. This procedure kept the plaque age between three and six days for all test periods.

A composite graph for each test regimen was plotted by combining the eight individual curves obtained at each data point and calculating the average and variation. The mean values, plus or minus one standard error, of the eight pH responses were plotted at one-minute intervals for the two-hour test period. Data analyses were performed for the following periods:

- ☐ The response period after 10 percent sucrose challenge (10 minutes).
- ☐ Gum chewing (10 minutes).



Event identification

Event #1	Initiate recording of resting plaque pH.
Event #2	Initiate rinsing with 10% sucrose solution.
Event #3	Complete using the sucrose rinse and initiate monitoring sucrose response.
Event #4	Complete monitoring sucrose response and initiate using test gum chewing (when gum chewing occurred).
Event #5	Complete chewing gum and initiate monitoring test response (when gum chewing occurred).
Event #6	Complete test session.

Time interval identification

Interval A	5 minutes	Baseline recording
Interval B	2 minutes	Sucrose challenge
Interval C	10 minutes	Monitor sucrose response
Interval D	10 minutes	Gum chewing
Interval E	93 minutes	Monitor effect of gum chewing

Figure 1. Sequence of events during test session.

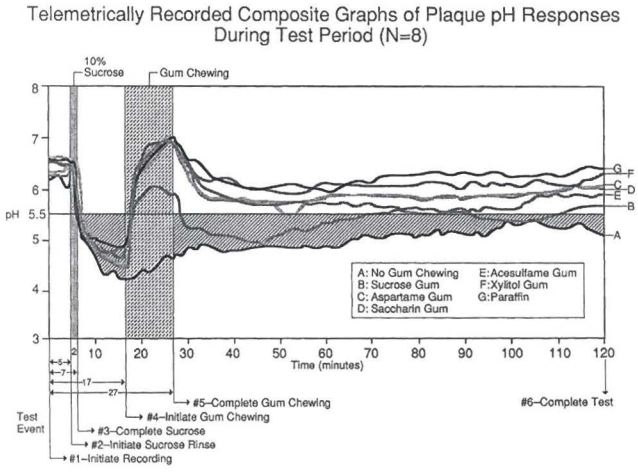


Figure 2. Summary of plaque pH curves over the test period.

☐ After gum chewing (93 minutes) as well as
☐ The combined periods of gum chewing plus the response period after gum chewing (103 minutes).

The plaque pH values at different event marks were then calculated from their respective millivolt readings. A diagram of the different time periods and events is shown in Figure 1. The numerical data of the response curves for each period were analyzed for:

☐ The area (pH X Time) of the response curve below pH 5.5 (AUC).
☐ The beginning, minimum and ending plaque pH attained.
☐ The maximum plaque change following each test regimen.
☐ The time pH remained below pH 5.5.

The areas under pH 5.5, in terms of pH X Time units, were derived by computer-processed geometric integration.

All analyses used the SAS GLM procedure to run a randomized block analysis of variance model using subjects as the blocking factor. A Student Newman-Keuls (SNK) analysis was performed for each dependent measure in which a significant treatment effect was detected. Values for the AUC were transformed to logarithms before analysis, because of the substantial heterogeneity of variance of the data.

RESULTS

The results were analyzed within different phases (Figure 1) in relation to events that occurred during the two-hour test session to highlight the response to the gum chewing independent of the response to the sucrose challenge. The pH responses, before, during, and after gum chewing were analyzed in order to clearly delineate any effect of gum chewing.

Figure 2 compares the average pH responses to the seven test regimens over the two-hour period. The mean resting plaque pH values ranged from 6.29 to 6.71 among the seven test regimens and were not significantly different. The average pH response to the 10 percent sucrose rinse in all test groups reached below the critical value of 5.5 during the ten-minute response period. The average pH response to the sucrose with no subsequent gum chewing (A) stayed below pH 5.5 during the entire two-hour test period. The sucrose gum chewing (B) raised the mean plaque pH above 5.5, while the test gum was chewed; it fell back below 5.5 after the gum chewing ceased, however, and gradually increased to above pH 5.5 (about forty minutes after gum chewing ceased). All plaque pH responses to sugarless chewing gums and paraffin were above 5.5. For the most part, the pH values after the sugarless gums were maintained above 5.5 after the gum chewing ceased.

The numerical data extracted from the response curves are summarized in Tables 1 through 3. Table 1 compares the AUC during different periods of the two-hour test session. The mean AUC for the 10 percent sucrose rinse (Period C) among the seven test regimens were not significantly different, indicating that all plaques were responding similarly to the 10 percent sucrose rinse. The AUCs during the test gum chewing periods (Period D) for all of the chewing gums were not significantly dif-

Table 1 ☐ Area (pH/Time) below plaque pH 5.5 during different periods.

Test regimens	Area under curve (AUC) during various intervals: Sucrose challenge (C)		During gum chewing (D)	After gum chewing (E)	During & after gum chewing (D & E)
None	1849 ± 346*	**	1996 ± 366	8052 ± 2909	10048 ± 3032
Sucrose	1108 ± 416		550 ± 265	5618 ± 2314	6168 ± 2408
Acesulfame K	1225 ± 269		175 ± 54	2549 ± 961	2724 ± 992
Saccharin	1230 ± 313		237 ± 85	1813 ± 995	2050 ± 1038
Aspartame	1503 ± 418		172 ± 49	941 ± 410	1113 ± 420
Paraffin	1151 ± 298		214 ± 72	371 ± 253	585 ± 238
Xylitol	1274 ± 274		111 ± 42	156 ± 106	268 ± 146

* Mean ± SEM (N = 8)
** Values within brackets do not differ significantly ($p > 0.05$) as determined by the ANOVA or SNK procedures performed on logarithms of the data.
Note: An average numeric AUC might not be depicted on the composite curves in Figure 2 because, in the averaging process, the individual low pH values would not occur at the same time after the challenge and therefore the average pH at that specific time period would be higher.

Table 2 ☐ Pleque pH during the study.

Test regimens	End of sucrose challenge		Minimum during gum chewing	End of gum chewing	Minimum post gum chewing	End of study
None	4.27 ± 0.21*	**	4.06 ± 0.20	4.83 ± 0.21	4.40 ± 0.18	5.27 ± 0.14
Sucrose	4.85 ± 0.25		4.69 ± 0.24	5.82 ± 0.42	4.73 ± 0.25	5.71 ± 0.22
Acesulfame K	4.60 ± 0.21		4.60 ± 0.19	6.93 ± 0.33	5.17 ± 0.19	6.02 ± 0.25
Saccharin	4.69 ± 0.20		4.53 ± 0.19	6.83 ± 0.11	5.29 ± 0.20	6.03 ± 0.24
Aspartame	4.62 ± 0.20		4.49 ± 0.18	7.00 ± 0.17	5.28 ± 0.19	6.33 ± 0.27
Paraffin	4.77 ± 0.28		4.77 ± 0.28	7.01 ± 0.20	5.70 ± 0.21	6.31 ± 0.29
Xylitol	5.02 ± 0.18		4.88 ± 0.15	7.02 ± 0.11	5.55 ± 0.12	6.35 ± 0.17

* Mean ± SEM (N = 8)
** Values within brackets do not differ significantly ($p > 0.05$) as determined by the ANOVA or SNK procedures.

Table 3 ☐ Plaque pH changes during gum chewing.

Test regimens	pH change (lowest to final)	
None	0.76 ± 0.08*	**
Sucrose	1.13 ± 0.34	
Acesulfame K	2.33 ± 0.29	
Saccharin	2.30 ± 0.16	
Paraffin	2.14 ± 0.16	
Xylitol	2.25 ± 0.25	
Aspartame	2.51 ± 0.17	

* Mean ± SEM (N = 8)
** Values within brackets do not differ significantly ($p > 0.05$) as determined by SNK procedures.

ferent; the AUCs during all gum-chewing regimens were significantly less, however, than when no gum was chewed. When the mean AUCs following the gum chewing sessions (Period E) were compared, there were no significant differences among gums; only xylitol gum and paraffin demonstrated a significant residual effect, however, over no gum. When the AUCs during the gum chewing period and the subsequent post gum chewing period were combined (i.e., total period from beginning of gum chewing to end of test session; Periods D & E) all of the sugarless gums, but not the sucrose-sweetened gum, significantly reduced the AUC compared with no gum. Although all of the sugarless gums had numerically less AUCs compared with sucrose gum, only the xylitol gum and paraffin significantly reduced the AUC compared with sucrose gum. No statistically significant differences were detected among the different sugarless gums, and all were effective in reducing the AUCs. The results of the time below pH 5.5 data were not qualitatively different from the AUC data and are thus not presented.

Table 2 summarizes the average plaque pH values during different time periods and at the end of these periods. No significant differences in pH were detected among the groups at the end of the sucrose challenge

(first column) or in the minimum pH during the gum chewing period (second column). The minimum pH values during the gum chewing period are all from the very first part of the period before much saliva was stimulated. At completion of gum chewing (third column), all of the sugarless chewing gums, but not the sucrose-containing gum, significantly raised the plaque pH as compared to the no-chewing regimen. The sugarless gums and paraffin were not significantly different, however, from the sucrose-containing gum. In the period after completion of gum chewing, no significant differences in minimum plaque pH were detected among the sugarless gums (fourth column). Only the pH after sucrose-gum was not different from the no-gum regimen, and only xylitol-gum and paraffin chewing resulted in pH values significantly higher than sucrose-gum. At the end of the study (fifth column), there were no significant differences in pH among any of the gums. Only the plaque pH after the aspartame, paraffin, and xylitol regimens were significantly higher than the no-gum regimen.

The plaque pH changes resulting from the test-gum chewing regimens are summarized in Table 3. All sugarless gums, but not the sucrose-gum, significantly increased the plaque pH during the chewing phase over no-gum chewing. Among the sugarless gums, only the aspartame-containing gum resulted in a significantly greater increase in plaque pH compared with the sucrose-gum.

DISCUSSION

A variety of factors that control salivary stimulation play an important role in determining the degree and rate of mineral loss from enamel. It is apparent that all gums tested in this study had some ability to reduce the acidogenicity of a sucrose challenge. This effect is likely attributable to salivary stimulation.[2,15,16] Plaque pH studies have investigated the effect of stimulus sequence and have demonstrated that when cheese or sorbitol gum are used after a sugar challenge, the pH response to that challenge can be cancelled.[14,17-19]

The question in this study was whether or not different sweetening agents in chewing gum would have different effects on the modification of plaque pH responses by the gum. A difference was considered possible because of reported potential antimicrobial effects or varying gustatory stimulation of saliva. In order to minimize possible differences in flavor-related gustation effects, we kept the flavors of the gums as similar as possible (primarily peppermint), but that was difficult to control with commercial products.

The results of this study demonstrate that all of the gums containing alternative sweeteners, i.e., high-intensity sweeteners (aspartame, saccharin, or acesulfame-K) or a polyol (xylitol), were significantly more effective in reducing the effects of a cariogenic challenge compared with no-gum chewing. There were no statistically significant differences among the gums sweetened with the alternative sweeteners. Although not statistically significant from no-gum, sucrose-gum was numerically intermediate between no-gum and the sugarless gums in its ability to reduce the effects of a cariogenic challenge.

With sucrose-containing gum, the potential anticariogenic effect of salivary stimulation may be offset by release of sucrose from the gum. Clinical studies of caries increments in subjects who chewed sucrose-containing gums versus non-gum chewing controls have given equivocal results, with some findings of a higher incidence of caries in the chewing-gum group, while others found no significant difference between the groups.[20,21] In these studies the gum was not chewed immediately after a meal or snack, however, which was suggested to have the greatest benefit.[9] No clinical trials using the latter regimen have been reported. In plaque pH studies, the interproximal plaque pH of subjects returned to neutrality very rapidly, when either a sugar-containing gum or a sugar-free gum was chewed for twenty minutes after a fall in plaque pH, caused by the consumption of meals.[22] The pH remained elevated, furthermore, after the cessation of chewing either type of gum. The authors argued, therefore, that both sugar-free and sugar-containing gums could be used for elevation of plaque pH, if chewed for twenty-minute periods after meals or snacks, and that both types of gums, when used in this manner, should be considered for use by individuals at risk of dental caries. This work has been confirmed by Fröhlich *et al.*[23] The same positive effect of sugar-gum has been reported in saliva.[24] On the other hand, these findings have not been confirmed by other workers, and the results are surprising, since the use of sugar-containing gum has been shown to result in a fall in plaque pH.[25-28] It has also been reported that interproximal plaque pH was not significantly elevated by the use of either sugar-free gum or sugar-containing gum following a meal.[29] With an *in vitro* simulation study, when the plaque pH has fallen after the exposure to fermentable carbohydrate, the rate of pH return to neutrality was much faster during simulation of the use of sugar-free chewing gum than during simulation of a gum-containing sucrose.[26] More laboratory and clinical studies are required, therefore, before the effects of sugar-contain-

ing gums on plaque pH profiles and enamel de-/remineralization can be definitively elucidated.

The different high-intensity sweeteners and xylitol tested in this study showed some numeric ranking trends of effectiveness; they were not, however, significantly different. Although the xylitol gum was numerically more effective than the others in reducing AUC after a sucrose challenge, such trends could be related to differences in pH, between the groups at the beginning of the gum chewing period. For example, the pH at the beginning of gum chewing ranged from 4.60 for acesulfame-K gum to 5.02 for xylitol-gum. The lack of a significant difference among the sugarless chewing gums encourages the view that gums containing different high-intensity sweeteners should exert similar clinical effects.[29-31] Interestingly the next most effective test regimen in our study was the paraffin. This is most likely due to the absence of other ingredients present in the gums containing the high-intensity sweeteners, such as hydrogenated corn syrup, sorbitol, mannitol, or other sugar alcohols, which may be slightly fermentable by the oral microorganisms. This same result has been observed by Jensen.[32]

It should be remembered that a challenge from a sucrose rinse is a rather mild challenge in that it is not retentive. It would appear that most of the effect seen in this study resulted from the act of chewing and saliva stimulation. If the challenge had been more substantial, like eating a cookie or another sucrose-containing snack, more effect of the chewing gums might have occurred.

Nevertheless, this study demonstrates that all of the sugarless chewing gums containing various high-intensity sweeteners or xylitol were effective in significantly reducing the area of the curve under pH 5.5 after a sucrose challenge as compared to no-gum chewing, probably by stimulating salivary flow and thus increasing removal or buffering of the acids produced by plaque microorganisms. Further, the different alternative sweeteners showed some numeric ranking trends of effectiveness, but no statistically significant differences were observed. The sucrose-containing gum was not significantly different from no-gum chewing or the sugarless gums in general. Future investigations of chewing gum sweetened with different alternative sweeteners should consider the use of more retentive acidogenic substrates to increase the acidogenic challenge.

SUMMARY AND CONCLUSIONS

The objective of this study was to determine whether sugarless chewing gums sweetened with different sweeteners differ in their ability to reduce an acidogenic response from a 10 percent sucrose-rinse challenge. Five commercially available chewing gums and two control regimens ("no gum" or paraffin) were tested using a plaque pH telemetry system. The gums were sweetened with sucrose, high-intensity sweeteners (aspartame, saccharin, or acesulfame-K), or a polyol (xylitol). Using a seven-period randomized block design, eight adult panelists were challenged with a 10 percent sucrose solution and then randomly used one of the test regimens during each of the seven test sessions. Each two-hour test session was divided into five periods: resting baseline (five minutes); sucrose rinse challenge (two minutes); postsucrose challenge (ten minutes); gum chewing (ten minutes); post gum chewing (ninety-three minutes). The factors analyzed were: the area of the curve (pH X Time) below pH 5.5, the minimum plaque pH attained, the changes in plaque pH over relevant intervals, and the length of time the plaque pH remained below pH 5.5.

The various response variables showed a similar pattern of statistically significant differences. All of the sugarless gums were effective in significantly increasing plaque pH and in reducing the area under the curve after the sucrose challenge compared with "no gum" treatment. No statistically significant differences were noted among the sugarless gums. The response to sucrose gum was intermediate between sugarless gums and "no gum" but was not statistically different from "no gum" or three of the sugarless gums. The areas under the curve after chewing paraffin and xylitol gums were significantly less than after sucrose-gum. The results indicated that, regardless of evaluation factor, chewing any of the sugarless gums or paraffin resulted in a beneficial effect on plaque pH response after a sucrose challenge.

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EFFECTS OF SODIUM BICARBONATE ON CARIOGENIC BACTERIA

Studies have indicated that the bacterial composition of dental plaque on tooth surfaces is more important to the development of caries than the amount of plaque accumulated. The presence of certain microorganisms such as mutans streptococci and lactobacilli is generally considered to increase the susceptibility to caries [Barenie and Bibby, 1981; Tanzer, 1989]. This increase in susceptibility is a result of some phenotypic traits that are clearly determinants of the cariogenicity of plaque; these include: intracellular polysaccharide synthesis, acid tolerance, and lactic acid production. These virulence characteristics provide mutans streptococci and lactobacilli with an ecological advantage on the surfaces of teeth over other bacteria and alter the chemistry of plaque such as to enhance the development of caries [Clarke and Dowdell, 1976; Tanzer, 1989]. Therefore, it would be a therapeutically useful strategy to alter the composition and chemistry of plaque, so as to reduce the quantity of cariogenic bacteria and the degree of acid production from those that remain.

The results from this study show that the daily use of a bicarbonate-based dentifrice significantly reduced the numbers of mutans streptococci and produced a trend toward lower numbers of lactobacilli over a 4-week period.

Legier-Vargas, K. *et al*: Effects of sodium bicarbonate dentifrices on the levels of cariogenic bacteria in human saliva. Caries Res, 29:143-147, March-April 1995.

The effect of caries scoring systems on the association between dental caries and streptococcus mutans

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Numerous authors have reported positive associations between levels of *Streptococcus mutans* in plaque and/or saliva and dental caries.[1-10] The microbiological techniques used in these investigations were standardized and highly uniform. The criteria used in the diagnosis of caries in some of these studies, however, varied significantly.

When visible cavitation is present, diagnosis of clinical caries diagnosis is usually straightforward. For smaller and incipient lesions, however, there can be considerable difficulty in the diagnosis of dental caries.[11,12] A number of scoring systems have been developed to aid in the diagnosis of dental caries. One of the most widely used scoring systems is that of the World Health Organization (WHO).[13] In this system, the stages of caries that precede cavitation, as well as other conditions similar to the early stages of caries are excluded, because they cannot be diagnosed reliably. To increase reliability, all questionable lesions are coded as sound. Other authors have described similar protocols.[14]

In other systems, questionable lesions are included in a separate category and are not classified as being sound. Moller and Poulsen described criteria for caries diagnosis on occlusal surfaces, whereby two categories were added to describe questionable lesions.[15] Category 1 was defined as being discolored with no definite sticking of a dental explorer, while category 2 was described as sticking of a dental explorer with or without discoloration in the absence of dentinal involvement. Similar criteria have been used by other authors.[16,17]

In investigations where *S. mutans* levels and caries prevalence were found to be associated, some investigators utilized diagnostic criteria similar to those of the WHO, while others adopted a system in which questionable lesions were scored as carious.[5,7,9,17]

The purpose of this investigation was to examine the effect of different systems of scoring caries on the association between caries indices and salivary *S. mutans* counts in a group of preschool children. Another objective was to determine whether eliminating filled surfaces from caries indices altered the degree of statistical association between caries prevalence and *S. mutans* levels.

MATERIALS AND METHODS

Subjects

Ninety-eight preschool children, three to five years of age, from seven randomly selected preschool programs in Saskatoon, Canada, were examined for dental caries. The community water supply was fluoridated to an optimal level. Informed parental consent and a question-

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naire-based health history were obtained before conducting the clinical examinations. Children who had taken antibiotics within thirty days before the examination were excluded from the study to prevent any effects of antibiotics on the oral microflora.

Dental examinations were performed in the preschool classrooms using a portable dental chair and fiberoptic dental light. A dental mirror and explorer were used to examine each subject for dental caries, but no radiographs were taken. All children examined had only primary teeth present. One author (LK) examined all the children for dental caries. The examinations took place either in the early morning or early afternoon.

Dental Caries Criteria

Dental caries was recorded using the WHO criteria and a modified scoring system (MC) based on the work by Shimono *et al.*[13,18] Using the WHO criteria, all questionable lesions were scored as sound. Teeth with the following defects were scored as sound: white or chalky spots; discolored or rough spots; and stained pits or fissures in the enamel that caught an explorer, but did not have a detectably softened floor, undermined enamel, or softened walls.

Using the modified system (MC), questionable lesions were scored as carious. Using this system caries was scored as follows:

S: Sound

C1: Stained or unstained, pits or fissures in the enamel that catch the explorer, but did not have a detectably softened floor, undermined enamel or softening of the walls.

C2: Obvious explorer catch with soft walls, softened floor or undermined enamel.

C3: Caries exceeds C2 and involvement of the pulp exists. For this category a fistula, abscess, or hyperplastic pulpitis must have been clinically present.

C4: Crown is destroyed by the caries process, retained roots present clinically.

A caries severity index (csi) was calculated for each subject, using the above modified system, to obtain a representation of caries severity. Scores were obtained by assigning numerical values to the various caries criteria as follows: S = 0, C1 = 1, C2 = 2, C3 = 3 and C4 = 4.

A csi (caries severity index), as modified from Shimono *et al*, was then calculated for each subject using the following equation:

$$\text{csi(caries severity index)} = \frac{\text{Sum of the caries scores for all surfaces}^{18}}{\text{Number of carious, filled, or extracted teeth}}$$

Higher csi values indicated a patient who had unrestored teeth with advanced caries involvement.

Microbiological procedures

Samples of whole saliva were obtained from all the subjects examined. Paraffin-stimulated whole saliva was collected in an ice-chilled glass container (100 ml). The samples were brought to the microbiology laboratory and processed for *S. mutans* count as soon as possible. All the microbiological procedures were concluded within two hours after salivary collection of the saliva. After agitation for 30 seconds in a Vortex Mixer (McGraw Park, Illinois, USA), the saliva was serially diluted in phosphate buffer. From each of the dilutions, a 0.1 ml sample was placed, in duplicate, on Mitis-Salivarius agar (Difco, Detroit, Michigan, USA) containing sucrose and bacitracin (MSB).[19] The prepared samples were spread over the MSB agar using an L-shaped glass rod. All MSB agar plates were incubated anaerobically (10 percent CO2, 10 percent H2, 80 percent N2) at 37°C for 48 hours. The number of colony forming units (CFU) were then counted and the CFU of *S. mutans*/ml of saliva was then calculated.

Statistical analysis of the data was done using a statistical program designed for Macintosh computers (Apple Computers Inc., 1987; Cupertino California, USA).

RESULTS

In this group of preschool children the mean dft (WHO)=0.69 ± 1.85 (Standard Deviation, SD) and dfs (WHO)=1.17 ± 3.70 (SD) using the WHO criteria. Using the modified system, the mean dft (MC)=1.03 ± 2.10 (SD) and dfs (MC)=1.50 ± 3.84 (SD). The dft(WHO) was significantly less ($p<0.0001$, Wilcoxon signed-rank test) than dft(MC). Similarly dfs(WHO) was significantly less ($p<0.0001$, Wilcoxon signed-rank test) than dfs(MC). (Table 1)

When using the WHO criteria, 66.7 percent of the subjects were caries free and had a dft and dfs equal to zero. These children were caries free and had no restored teeth or tooth surfaces.

Restored surfaces were then eliminated from both indices to obtain dt and ds values for both caries-scoring systems. The mean dt(WHO)=0.35 ± 0.99 (SD) and ds (WHO)=0.53 ± 1.63 (SD) while mean dt (MC)=0.71 ± 1.40 (SD) and ds (MC)=0.91± 1.95 (SD). The dt(WHO) was significantly less ($p<0.0001$, Wilcoxon signed-rank test)than dt(MC) and ds(WHO) was significantly less ($p<0.0001$, Wilcoxon signed-rank test) than ds(MC). (Table 1)

Table 1 ☐ Caries prevalence using two different scoring systems.

	WHO criteria			Modified criteria	
	Mean value	Standard deviation		Mean value	Standard deviation
dft	0.69	1.85	dft	1.03	2.10
dfs	1.17	3.70	dfs	1.50	3.84
dt	0.35	0.99	dt	0.71	1.40
ds	0.53	1.63	ds	0.91	1.95

(All WHO indices significantly lower than corresponding modified indices) ($p \leq 0.0001$, Wilcoxon signed-rank test)
(n=98)

Table 2 ☐ Streptococcus mutans distribution.

S. mutans group	Number of subjects	Mean S. mutans count
O	60	0 (0)*
A	18	4.64×10^4 (3.37×10^4)*
B	16	4.18×10^5 (2.83×10^5)*
C	4	1.12×10^7 (1.72×10^7)*

*(Number in parenthesis represents the Standard Deviation)
O = <400 CFU/ml of saliva, A = 400 – 10^5 CFU/ml, B = 10^5 – 10^6 CFU/ml and C = >10^6 CFU/ml

Table 3 ☐ Relationship between S. mutans and caries prevalence (WHO criteria).

Index	S. mutans group	Mean dft	S. D.	p-value
dft (WHO)	O	0.37	1.48	
	A	0.78	2.21	
	B	1.69	2.24	0.001*
	C	1.25	2.50	
dfs(WHO)	O	0.57	2.80	
	A	1.61	5.65	
	B	2.69	3.59	0.0007*
	C	2.25	4.50	
dt(WHO)	O	0.12	0.59	
	A	0.28	0.83	
	B	1.06	1.39	0.0001**
	C	1.25	2.50	
ds(WHO)	O	0.15	0.69	
	A	0.28	0.83	
	B	1.81	2.69	0.0001**
	C	2.25	4.50	

(*Significant at $p \leq 0.001$, **Significant at $p \leq 0.0001$; Kruskal-Wallis test)
O = <400 CFU/ml of saliva, A = 400 – 10^5 CFU/ml, B = 10^5 – 10^6 CFU/ml and C = >10^6 CFU/ml
(n=98)

Table 4 ☐ Relationship between S. mutans and caries prevalence (modified system).

Index	S. mutans group	Mean dft	S. D.	p-value
dft (MC)	O	0.50	1.51	
	A	1.33	2.57	
	B	2.56	2.66	0.0009*
	C	1.50	2.39	
dfs(MC)	O	0.68	2.80	
	A	2.17	5.71	
	B	3.50	3.85	0.0007*
	C	2.75	4.86	
dt(MC)	O	0.32	0.85	
	A	0.83	1.72	
	B	1.88	1.75	0.0003*
	C	1.50	2.38	
ds(MC)	O	0.35	0.94	
	A	0.83	1.72	
	B	2.63	2.80	0.0002*
	C	2.75	4.86	

(*Significant at $p<0.001$; Kruskal-Wallis test)
O = <400 CFU/ml of saliva, A = 400 – 10^5 CFU/ml, B = 10^5 – 10^6 CFU/ml and C = >10^6 CFU/ml
(n=98)

Table 5 ☐ Relationship between caries severity index(csi) and S. mutans counts.

S. mutans	Mean csi	S. D.	p-value
O	0.20	0.49	
A	0.33	0.57	0.0002*
B	1.28	1.46	
C	1.04	1.50	

*Significant at $p \leq 0.001$; Kruskal-Wallis test
O = <400 CFU/ml of saliva, A = 400 – 10^5 CFU/ml, B = 10^5 – 10^6 CFU/ml and C = >10^6 CFU/ml
(n = 98)

The *S. mutans* count was found to be significantly related to the prevalence of dental caries, using both the WHO criteria and the modified scoring system. The *S. mutans* count was significantly related to dft(WHO) ($p=0.0007$) and dfs(WHO) ($p=0.0006$)(Spearman rank correlation coefficient analysis). Similarly the *S. mutans* count was significantly related to dft(MC) ($p=0.0002$) and dfs(MC) ($p=0.0002$)(Spearman rank correlation coefficient analysis).

After restored surfaces were eliminated from the caries indices, the *S. mutans* count was also found to be significantly related ($p<0.0001$, Spearman rank correlation coefficient analysis) to dt (WHO), ds (WHO), dt (MC) and ds (MC).

Streptococcus mutans counts were also grouped into four categories as follows: O = < 400 CFU/ml of saliva, A = 400 - 10^5 CFU/ml of saliva, B = 10^5 -10^6 CFU/ml of saliva and C = >10^6 CFU/ml of saliva. Data in Table 2 show the number of individuals in each of these categories and the mean *S. mutans* count for each group.

The mean dft, dfs, dt and ds were significantly different between the four *S. mutans* groups for both WHO and modified criteria (Tables 3 and 4).

The *S. mutans* count was found to be significantly related to the caries severity index ($p<0.0001$, Spearman-rank correlation coefficient analysis). Also when the *S. mutans* count was grouped as previously described, the mean csi varied significantly between the four *S. mutans* groups ($p< 0.001$, Kruskal-Wallis test) (Table 5).

No significant difference ($p=0.09$, Kruskal-Wallis test) was found between the mean *S. mutans* count for the age of the subjects, when they were grouped as follows: three to four years of age, four to five years of age, and five to six years of age.

DISCUSSION

In this investigation the mean dft, dfs, dt and ds obtained by using the WHO criteria were all significantly less than the same caries indices obtained using the modified system. These differences could be expected as questionable lesions were scored as carious in the modified system and would result in a higher number of

carious teeth or surfaces as compared to the WHO - based data.

One disadvantage of using the modified system in epidemiological studies is the lack of reliability which results from the difficulty in diagnosing and scoring questionable lesions.[12-14] In this study the modified criteria were well defined, and only one examiner was used to examine and score all children for dental caries. If multiple examiners were used, the calibration and reliability of results using the modified criteria would be more difficult due to the increased complexity.

The *S. mutans* count was found to be significantly related to the caries prevalence (dft and dfs), using both the WHO criteria and the modified system. These results are in accordance with many other studies in which a similar association, was found.[1-10] Previous studies have found a similar association using WHO criteria and modified WHO criteria.[5,7,9] Other investigators have also found a positive association, using criteria similar to the modified system used in this study.[17,18,20] By scoring questionable lesions as carious, the caries prevalence remains significantly related to the *S. mutans* count. Loesche *et al* showed that teeth destined to become decayed exhibited a significant increase in the proportions of *S. mutans* from six to twenty-four months before the clinical diagnosis of dental decay could be made, when the criterion was that softness or a definite break in enamel surface continuity had to be detected with an explorer.[20] This study confirmed that pre-carious or questionable carious lesions do harbor higher levels of *S. mutans* than do sound surfaces. If this is the case, one would expect *S. mutans* counts and caries prevalence using the modified system to be more closely associated than is the case when using the WHO criteria.

When restored surfaces and teeth were eliminated from the caries indices using both the WHO criteria and the modified system, the *S. mutans* count remained associated with the caries prevalence (dt and ds). Similar results have been reported elsewhere.[21,22] By eliminating restored surfaces from the caries indices, the level of significance, using the Kruskal-Wallis test, increased for both caries criteria systems. This can be expected, as restored surfaces replace carious surfaces that harbor *S. mutans*. Hence, the mean dt and ds are then identifying only actively carious surfaces.

The caries severity index was also found to be significantly related to the *S. mutans* count. These results are similar to other studies which have found that both *S. mutans* and lactobacillus play an important role in the initiation and progression of the caries process.[23]

The *S. mutans* counts in sixty percent of the subjects were low (<400 CFU/ml of saliva). They were also found not to be significantly different with respect to the age of the subjects. Catalanotto *et al* has previously shown that as the number of primary teeth increased in young children, the prevalence of *S. mutans* also increased.[24] The greatest observed frequency occurred in patients with a complete primary dentition and contacts between the molar teeth. All the children examined in the current study had a full complement of primary teeth. Any age related differences seen in other studies due to the eruption of primary teeth would not be seen in these subjects.

The low *S. mutans* levels in this sample of preschool children may be due to the low caries rate observed in which 66.7 percent of the children were caries free and had no restored surfaces, using the WHO criteria. Carlsson *et al* showed that *S. mutans* did not become established in the mouths of infants during their first year even with the eruption of the anterior teeth.[25] This effect could also carry over into preschool-age children, explaining the low counts observed in this study.

CONCLUSIONS

From this study the following conclusions can be drawn:

- ☐ The *S. mutans* count and caries prevalence remain significantly associated, whether using WHO criteria in which questionable carious lesions are scored as sound or a modified system in which such lesions are scored as carious.
- ☐ When restored surfaces are eliminated from caries indices, the *S. mutans* count remains significantly associated with the caries index. By doing so, the level of significance is also increased.
- ☐ Caries severity is significantly associated with the *S. mutans* count.

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DETECTION OF DEVELOPMENTAL AND BEHAVIORAL PROBLEMS

Conservative estimates suggest that 12 percent to 16 percent of American children have behavioral and emotional disorders. The Committee on Psychosocial Aspects of Child and Family Health of the American Academy of Pediatrics has urged pediatricians to be concerned with the early detection of children's behavioral problems. Recent initiatives such as Bright Futures, a collaborative project of the Maternal and Child Health Bureau, the Medicaid Bureau, and the American Academy of Pediatrics to develop national guidelines for child health supervision, have emphasized the importance of early detection of psychosocial risks.

Glascoe, F.P. and Dworkin, P.H.: The role of parents in the detection of developmental and behavioral problems. Pediatrics, 95:829-836, June 1995.

Influence of fluoride in saliva during the early cariogenic changes in the enamel of boys and girls

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Several years ago it was claimed that the fluoride (F^-) concentration in the enamel surface is the main caries reducing factor.[1] More recent studies have failed to establish a clear relationship, however, between F^- content in the enamel and caries inhibition either in vitro or in vivo.[2,3] On the other hand, microbiological experiments provided increasing evidence that F^- in saliva also was able to inhibit the formation of and the acid production in dental plaque.[4-6] More recently the F^- concentration in the saliva, similar to that in the enamel, was linked with caries reduction in vivo.[7,8] Until now it has been shown in vitro only that F^- in concentrations comparable to that found in saliva can either inhibit demineralization or stimulate remineralization.[9,10]

Clinically, processes of demineralization and remineralization occur in the enamel at the very beginning of caries development and cannot be determined using the traditional D(ecayed) M(issing) F(illed) index because this index refers to the cumulative caries prevalence in the past and is thus not suitable for measuring the cariogenic changes that can take place in the enamel at the moment that the ionic F^- concentration of the saliva is measured. To overcome this problem we scored the cariogenic changes clinically observable within a short interval of time and in this way, studied more specifically the effect of the actual F^- concentration in the saliva on caries development.

MATERIALS AND METHODS

Procedures

A total of 185 Dutch children with ages ranging from six to eighteen years (104 boys with mean age of 11.4 years $\pm$2.8 (SD) and 81 girls with mean age of 11.6 years $\pm$2.5 (SD) participated in our study. At two visits with a six-month interval starting in 1986, their dental status, including white-spots of buccal and lingual smooth surfaces and fissures (plus buccal/palatinal pits) in primary and permanent teeth, was registered by two examiners after supervised toothbrushing followed by airdrying of the teeth for a few seconds. Mesial and distal surfaces were not included in this study because, for safety reasons, bite-wing radiographs were taken only once a year or when indicated. The examinations were performed by two examiners with the aid of a dental

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operation lamp, a multifunctional syringe and mirror and probe. To prevent fissure damage, the probe was only used to clean the fissures and no pressure was applied for testing cavity formation in fissures or the integrity of the smooth-surface enamel.

Dental status

A white spot was defined as demineralized enamel with an intact surface, whereas a cavity was defined as demineralized enamel without an intact enamel surface. A white spot was differentiated from other buccal enamel defects by its localization near the gingival margin, following the gingival curve. In the occlusal enamel surfaces (pits and fissures) apart from white spot enamel, most lesions showed a brownish discoloration in the depth of the pits and fissures.[11] Within the six-month interval the following changes in the enamel of smooth surfaces and fissures were noted by the two examiners (Figure 1):

- ☐ Sound enamel becomes a white spot (=initiation)
- ☐ A white spot becomes a cavity (=progression)
- ☐ A white spot remains a white spot (=stabilization)
- ☐ A white spot disappears (=regression)

The interexaminer reliability, calculated with the aid of Cohen's Kappa was between 0.81-1 for the fissures (very good) and for the smooth surfaces between 0.57-0.81 (satisfactory to good); the intraexaminer reliability for each examiner, calculated according to Wiegman, was 75 percent for fissures and 92 percent for smooth enamel surfaces.[12,13]

Fluoride determinations in saliva

At the second visit, to reduce diurnal variations, saliva was collected always in the morning as soon as the children entered the clinic before dental status was determined or dental treatment occurred. Unstimulated saliva was collected in a five-minute period. Stimulated saliva was collected after continuous chewing of paraffin pellets in a five-minute period. The sample was weighed and the ionic F^- concentrations determined with an Orion F^--sensitive electrode (accuracy: 1-2 percent). In both the first and second visits for each child it was also registered how often and in which forms F^- was applied in the preceding six-month period. During the study, a very intensive F^- application program was conducted. The F^- concentration of the nonfluoridated drinking water was 0.2-0.3 ppm. The program consisted of the following treatments:

- ☐ F^- toothpaste, three times a day, containing (0.025 percent F^-).
- ☐ F^- tablets (0.25 mg F^- each), for children over four years of age, four times daily, one each time, or two times daily, two each time.
- ☐ Cleaning the teeth with F^--containing prophylactic paste, every six-month visit at the dental school.
- ☐ F^- fluid application (1.25 percent F^-), twice a year.
- ☐ A fluoride (0.1 percent F^-) rinse program at school once a week (not always applied).

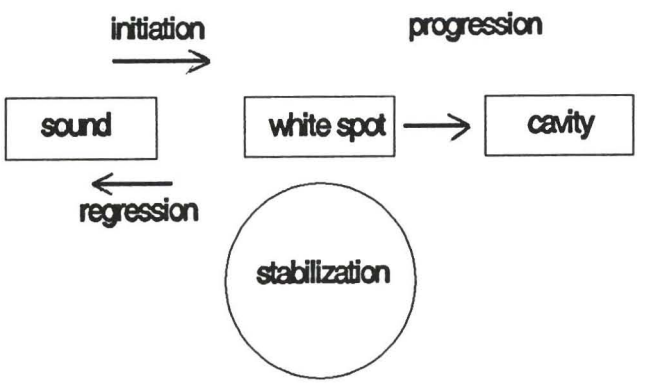

Figure 1. Schematic representation of the various cariogenic events occurring in the enamel surfaces.

Calculations

Based on the clinical data, the children (boys and girls separately) were divided in two groups: one in which a particular cariogenic event had taken place in the six-month interval and a second group in which that cariogenic event had not taken place, i.e., children with initiation against children without initiation, children with progression against children with regression and stabilization, children with stabilization against children with progression and regression, and children with regression against children with progression and stabilization. Then, between both groups the concomitant ionic F^- concentrations in stimulated and unstimulated saliva from the children were compared with each other. It should be mentioned that by reducing the interval of cariogenic events to six months and using the clinical factors mentioned above, it is justifiable to relate the cariogenic changes taking place in the enamel with a single determination of F^- concentration in the saliva at the end of the interval. Moreover, it should be kept in mind that various cariogenic changes in enamel can take place simultaneously in one child. So one child can belong to more than one group concurrently.

Statistical analysis

To determine the significance of the relationship between F^- concentrations in the saliva and the cariogenic enamel changes, the X^2-test for frequency distribution of qualitative systems was used. The reliability for this test was 95 percent.

Figure 2. Distribution of the percentage of various combinations of F^- use at the first visit (T0) at the start and the second visit (T1) at the end of the investigation for boys (A) and girls (B). (P = F^--containing toothpaste, T = F^--containing tablets, S = F^--containing rinse solution, A = topical F^- application). Most frequently the following combinations were noticed:

- *Combination of P, T and A (PTA) was utilized by boys for 61 percent at T0 resp. 65 percent at T1 and by girls for 59 percent at T0 resp. 64 percent at T1.*
- *Combination of P and A (PA) was utilized by boys for 16 percent at T0 resp. 22 percent at T1 and by girls for 18 percent at T0 resp. 19 percent at T1.*
- *Combination of P, T and S (PTS) was utilized by boys for 5 percent at both visits and by girls for 4 percent at T0 resp. 3 percent at T1.*

RESULTS

Before relating F^- concentrations in the saliva, determined at the end of the experimental period with cariogenic events occurring in advance, we must be sure that the use of various F^- combinations did not change during the investigation. We looked first for possible variations in the use of various F^- combinations, between the start and the end of the experimental period. Figure 2 shows the frequency distribution of the use of various F^- combinations applied in the six months preceding the visits to our clinic, for boys (Figure 2A) and girls (Figure 2B). About 60-65 percent of the children used F^- containing tooth paste and F^- local applications together with tablets, and 20 percent without F^- tablets. Only ± 5 percent of the children rinsed their teeth with a F^- containing solution in addition to using both F^- containing tooth paste and receiving tablets. The frequency distribution of the use of F^- in the foregoing six months at the second visit did not change significantly from that of the first visit for both sexes. At both visits also no sig-

Table 1 ☐ The percentage distribution of the presence or absence of various cariogenic events observed on the enamel surfaces of all teeth in boys and girls within six months.

	Boys		Girls	
	number	%	number	%
		Initiation		
with	83	80	60	74
without	21	20	21	26
Totals	104	100	81	100
		Regression		
with	78	76	56	71
without	24	24	23	29
Totals	102	100	79	100
		Stabilization		
with	96	94	75	95
without	6	6	4	5
Totals	102	100	79	100
		Progression		
with	6	6	9	11
without	96	94	70	89
Totals	102	100	79	100

Table 2 ☐ Mean values of F^- concentration in unstimulated and stimulated saliva of boys and girls, measured during the last visit.

	Fluoride concentration (μMol/L)			
	Boys (n=104)		Girls (n=81)	
Saliva	mean	sd	mean	sd
Unstimulated	10.4	11.6	9.9	10.1
Stimulated	6.3	5.0	5.3	4.3

The values for boys did not differ significantly from girls according to the Student t test.

The mean amount of unstimulated saliva, produced within five minutes was 3.6 ml for boys and 2.8 ml for girls and of stimulated saliva 6.8 ml and 4.9 ml for boys and girls, respectively. Consequently the total amount of F^- produced within five minutes from unstimulated saliva was 37.8 nM for boys and 29.1 nM for girls or by stimulated saliva 42.5 nM for boys and 31.0 nM for girls.

Table 3 □ Analysis of the ionic F^- concentration in unstimulated and stimulated saliva of boys and girls with a certain cariogenic event occurring within six months, tested with the X^2-test for $p<0.05$ against the ionic F^- concentration in saliva of boys and girls without this event.

Saliva	Ionic F^- concentration	
	Boys (n=104)	Girls (n=81)
	Initiation	
Unstimulated	ns	significantly higher
Stimulated	ns	significantly higher
	Regression	
Unstimulated	significantly higher	significantly higher
Stimulated	ns	ns
	Stabilization	
Unstimulated	ns	ns
Stimulated	ns	ns
	Progression	
Unstimulated	ns	significantly lower
Stimulated	ns	significantly lower

ns = not significant

nificant differences in frequency distribution could be found between boys and girls.

On examining the cariogenic events that had occurred during the six months interval, girls had developed more new cavities (progression) than boys (Table 1). The ionic F^- concentration in saliva, determined at the second visit, was in unstimulated saliva 10.4 ± 11.6 μM (SD) for boys and 9.9 ± 10.1 μM (SD) for girls; in stimulated saliva these values were 6.3 ± 5.0 μM (SD) for boys and 5.3 ± 4.3 μM (SD) for girls (Table 2). There were no significant differences between the sexes and the values were age independent. By comparing the total amounts of F^- present in unstimulated and stimulated saliva, it turned out that only 10 percent more F^- was secreted under stimulation in both boys and girls. On the other hand, apparently due to higher flow rates, boys produced within five minutes about 30 percent more F^- in unstimulated saliva and stimulated saliva (37.8 nM/5min respectively 42.5 nM/5min) than girls (29.1 nM/5min respectively 31.0 nM/5min). Finally, F^- concentrations in saliva from children with various cariogenic events were compared with those of children without the events (Table 3).

Development of new white spots was only observed in those girls who also had significantly higher F^- levels in both the stimulated as well as unstimulated saliva (Table 3). On the other hand those girls who developed new cavities (progression) had lower F^- levels in both stimulated and unstimulated saliva (Table 3). Children (boys and girls) who had white spots turned into sound enamel (regression) had significantly elevated F^- concentrations in unstimulated but not in stimulated saliva (Table 3).

No clear relation was observed between F^- levels in saliva and stabilization of caries.

DISCUSSION

By calculating, from our data, the D_3 (cavity formation) MFS of children with an age of ten years at the first visit, we found a mean value of 2.18 ± 2.93 (SD). This value agrees well with that for the children of similar age, as determined by Truin *et al* in The Hague.[14] Consequently the children visiting our dental clinic did not differ from other Dutch children regarding their caries prevalence.

The frequency distribution for various combinations of F^- preparations taken by the children in the foregoing six months did not differ, either between boys and girls or between the first and second visits. So it is unlikely that the F^- concentration in the saliva at the beginning of the experiment (which was not determined at the first visit) will differ from that at the end. Accordingly, we think that it is allowable to compare the F^- concentration of saliva of boys and girls, determined at the end of a relatively small test period of six months, with early cariogenic changes in the enamel within both visits.

Intake of F^- through the application of F^--containing preparations (Figure 2) and F^- levels measured in saliva (Table 2) did not differ between boys and girls. Girls differed from boys, however, as they developed more new cavities (progression) than boys in the test period (Table 1). Girls also differed from boys in significance of correlations in salivary F^- and cariogenic events (Table 3). Because these phenomena can not be explained by differences in the use of F^-, they may be interpreted in terms of sugar consumption, which in these girls is higher than in boys.[15] A higher sugar consumption leads to a higher acidogenic oral environment, resulting in a higher percentage progression (Table 1). On the other hand, remineralization of dental enamel is stimulated by F^- at acidic pH.[16] Higher acidic environment would explain why for girls, a high F^- concentration in both types of saliva resulted in a significant reduction of progression. The positive relationship for girls between white spot formation (initiation) and F^- concentrations in both types of saliva seems paradoxical at first sight. But it should be mentioned here that in our investigation, we could not discriminate between white spots that had not yet stabilized and those that did under the influence of salivary F^-. Some of these white spots may have turned into cavities in children with low salivary F^- levels, but are stabilized, instead of forming a cavity in girls with high salivary F^- levels, particularly in a more acidic environment. This may explain the positive relationship between initiation and salivary F^- in girls. An increase in white spots has also been observed by Groeneveld in an area with fluoridated drinking water.[11]

For both sexes, the F^- concentration in the unstimulated saliva was significantly positively related with the disappearance of white spots converted to sound enamel (regression) only. Stimulation of regression (remineralization) must be the main caries reducing effect of F^-, because regression occurs about eight times more frequently than progression. In this respect F^- concentration in unstimulated saliva must be more important, because teeth are in contact with this type of saliva most of the day.

Regression is highest just after tooth eruption, as we have shown for premolars in vitro as well as in vivo.[17-19] Consequently F^- applications will be most effective in children during and just after tooth eruption.

Summarizing, with the aid of more adequate clinical factors than used in other investigations, it became possible to emphasize in this study the importance of F^- concentrations in the oral fluid for reducing caries by its action also on early processes and in particular regression, during caries development.[7,8,20]

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UNINTENTIONAL INJURIES

For the past few decades, unintentional injuries have been the leading cause of death and disability for children older than age one in this country. Prevention of those injuries is of critical importance to those interested in the health and well-being of children.

Understanding the origins of unintentional injuries and measuring their impact are key parts of the prevention process. Correctly understood and used, measurements of the causes and results of unintentional injuries will assist in determining what the problems are and how they can best be solved.

Documenting the effectiveness of prevention programs is not always straight-forward. Sometimes, detailed examination of the data and the context of the program is required to understand the potentially confounding effects of factors other than the prevention strategy. However, prevention strategies used but not evaluated cannot show their effectiveness and are more difficult for others to adopt credibly. Measurement is important only insofar as it informs the policymaking process. The purpose of data collection is understanding, so that events leading to unintentional injuries can be changed, and the injuries themselves can be prevented.

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Effect of cavity form on the durability of glass ionomer cement restorations in primary teeth: A three-year clinical evaluation

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The first glass ionomer cements were introduced about twenty years ago.[1] Although inadequate mechanical properties limited their use as dental restorative materials for many years, their potential to replace amalgam as a restorative material has increased. In so far that glass ionomer cements adhere to mineralized tooth tissue, the removal of sound tooth structure can be kept to a minimum.[2] Their ability to leak and absorb fluoride decreases the rate of secondary caries and protects adjoining enamel surfaces.[3-5] The physical properties of glass ionomer cements have been much improved, but low tensile and compressive strengths and the sensitivity of the material to technique are still a problem. In clinical studies the longest survival rates for glass ionomer cement restorations have been shown in low stress areas as Class V and Class III restorations.[6-8]

As the functional life of restorations is limited in the primary dentition, glass ionomer cements have been recommended also for Class II restorations. The durability of Class II glass ionomer cement restorations varies in earlier reported studies, from 67 percent failures after six months to 9 percent after twelve to thirty-two months.[9-18] In a five-year longitudinal study the median survival time for Class I and Class II glass ionomer restorations in primary molars was 33.4 months compared to 41.4 months for amalgam restorations.[18] In that study the cavity preparation for glass ionomer cement restorations simply consisted of caries removal and finishing of the margins perpendicular to the surrounding enamel. No complementary retention was prepared.

The cavity form should be chosen to match the physical properties of the restorative material. According to Croll and Phillips, narrow isthmuses and regions of minimal cement bulk should be avoided in proximal glass ionomer cement restorations. They claim that well-designed preparations that include mechanical retention will enhance the success of an adhesively bonded glass ionomer cement restoration.[11] Wilson and McLean stressed the importance of avoiding thin sections, shallow keyways and narrow isthmuses for Class II cavities in primary molars. They also described a tissue saving microcavity with an occlusal approach.[6] The failure rate of glass ionomer cement restorations with special reference to cavity form is rarely mentioned in earlier studies. More often, higher failure rates have been reported in studies where conventional Black's Class II cavity forms were used compared to those employing microcavities.[9-18]

The aim of this study was to evaluate intraindividually the durability of Class II glass ionomer cement restorations in primary molars in two types of cavities: a) a

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tissue-saving proximal microcavity and b) a slightly modified Black's Class II cavity.

MATERIALS AND METHODS

Twenty-five children attending two Public Dental Health clinics in the city of Umeå, Sweden, each with at least one pair of proximal carious lesions in one primary molar and its contralateral, participated in the study. Their mean age was about eight years (range of six to ten years). A total of twenty-eight pairs of Class II glass ionomer restorations (ChemFil II, DeTrey/Dentsply, Konstanz, Germany) were placed by two dentists (IAW, RS). Two types of Class II cavities were prepared:

- ☐ A proximal microcavity where the external angles were rounded after caries excavation (M).
- ☐ A modified Black's Class II preparation with rounded inner and outer line angles and broad isthmus (B), (Figure).

In order to evaluate the two cavity types intraindividually, each patient received at least one cavity of each type.

After preparation of the cavities the operation field was isolated with cotton rolls and a saliva ejector was placed. A steel matrix band was placed around the tooth and wedged. The cavities were cleaned with 40 percent polyacrylic acid (Durelon Liquid, ESPE, Seefeld/Oberbay, Germany) for ten seconds and then rinsed with liberal water spray. In deep cavities small amounts of calcium hydroxide cement (Dycal, DeTrey/Dentsply, Konstanz, Germany) were applied to the pulpal wall. Chemfil II or, in a few cases Chemfil junior, was hand mixed according to the producer's recommendations and placed in the cavity with a syringe tip (Centrix, Hawe Neos, Gentilino, Switzerland). The material was slowly injected to a surplus, compressed and shaped occlusally with a preformed cervical foil (Hawe Neos). Large surplus was removed with a scaler or sharp excavator. After a total setting time of five minutes, the occlusal foil and matrix were removed and a protecting varnish immediately applied. Occlusion was checked and if necessary adjusted with either a round steel bur under protection of petroleum jelly or by high-speed diamond and generous water spray. A varnish was finally applied in two thin layers and then covered with petroleum jelly. Final polishing was done at the following dental visit after at least one week. The buccolingual, the mesiodistal, and the incisocervical dimensions of the cavities were recorded; the treatment time was also noted (from the start of the preparation to the final application of the protecting varnish).

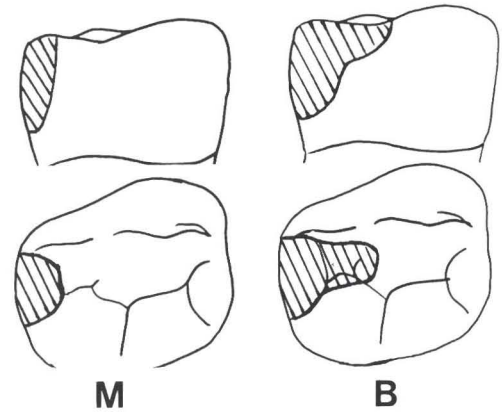

Figure. Cavity types studied; M: a microcavity, where after caries excavation the external angles were rounded, B: a modified Black's Class II preparation with rounded inner and outer line angles and broad isthmus.

Evaluation

The quality of the restorations was assessed and scored according to the USPHS criteria described by Ryge and Snyder 1973 at baseline (directly after finishing) and after six, twelve, twenty-four, and thirty-six months or until exfoliation or failure.[19] Caries, anatomical form, marginal adaptation, color match, marginal discoloration and surface roughness were evaluated. A score of A (Alpha) or B (Bravo) was considered acceptable, while a restoration with score of C (Charlie) or D (Delta) for any of the variables was considered unacceptable. If a primary molar had been exfoliated, the assessments at the last examination were used. Teeth exfoliated during the first year of the study were excluded.

Statistical analysis

Intraindividual comparisons of the durability and quality of the restorations in the two cavity types were performed at the end of the evaluation period. The two cavity types were rank ordered within each patient. The best ranked restoration obtained three points and the second ranked, one point. If the restorations had the same ranking scores, they received two points each. In this way each patient served as a statistical unit. The sums of the ranks of the cavity types were then tested using Friedman's two way analysis of variance test.[20]

RESULTS

Of the fifty-six restorations included in the trial, thirty-eight could be evaluated clinically at the twenty-four

Table 1 ☐ Total number of evaluated teeth, cumulative rate of exfoliated teeth and number of dropouts for other reasons at the recalls for the microcavity group (M) and for the modified Black's Class II cavity group (B).

	Number of teeth		Number of exfoliated teeth		Drop-outs for other reasons	
	M	B	M	B	M	B
Baseline	28	28	0	0	0	0
6 months	21	21	1	1	6	6
12 months	24	24	2	2	1	1
24 months	18	20	7	6	1	1
36 months	9	12	14	9	1	3

Table 2 ☐ Total number of evaluated teeth, number of failed restorations, reasons for failure and the cumulative failure rate at the recalls for the microcavity group (M) and for the modified Black's Class II cavity group (B).

	Number of teeth		Number of failures		Cumulative failure rate percent	
	M	B	M	B	M	B
Baseline	28	28	0	0	0	0%
6 months	21	21	1*	1[▫]	5	5%
12 months	24	24	1[@]	0	8	4%
24 months	18	20	2[*,#]	3[▫,@,#]	16	16
36 months	9	12	1*	2[*,▫]	25	32

* Totally lost restoration
▫ Isthmus fracture
@ Secondary caries
# Partially dissolved

Table 3 ☐ Mean values of the buccolingual (b-l), mesiodistal (m-d), incisocervical (i-c) dimensions of the two cavity types studied.

	First primary molar		Second primary molar	
	M	B	M	B
b-l	2.6	2.6	2.4	3.1
m-d	1.9	3.7	1.9	4.4
i-c	2.7	3.1	3.1	3.3

month recalls and twenty-one at the thirty-six-month recalls. The numbers of drop-outs at the different recalls are shown in Table 1. A total of twenty-three teeth exfoliated during the three-year evaluation period. All nine teeth exfoliated between twelve and twenty-four months were scored as acceptable at the twelve-month examination, and all ten teeth exfoliated between twenty-four and thirty-six months were scored as acceptable at the twenty-four-month examination. The numbers of failed restorations, cumulative failure rates (percent) and reasons for failure are shown in Table 2. At the twenty-four-month examination, four restorations placed in microcavities (M) and four in modified Black's cavities (B) were considered unacceptable according to the USPHS criteria. At the thirty-six-month evaluation the cumulative number of failed restorations increased to five and six, respectively. Excluding the exfoliated teeth in the failure rating changed the percentage of cumulative failures after twenty-four months from 16 percent to 20 percent in group M and from 16 percent to 19 percent in group B. For the registrations at thirty-six months, the cumulative failure rating changed from 25 percent to 39 percent in group M and from 32 percent to 38 percent in group B.

The unacceptable restorations included three that were totally lost, two in group M and one in group B. Three isthmus fractures were registered in group B. Secondary caries was found in two cases at the outer axial angle of the proximal part of the cavity in connection with what seemed to be a void in the restoration. In both cases the lesions were superficial. Three restorations were partially dissolved, with a C score for marginal adaptation. In these cases, replacement was clinically not indicated, because of anticipated exfoliation in the near future. In the intraindividual comparisons of the durability and quality of the restorations in the two study groups, no significant difference was found at either the twenty-four-month or the thirty-six-month evaluation.

Mean treatment time for the restorations in groups M and B, from the beginning of preparation to final application of the protecting varnish, was 20 and 23 minutes, respectively. The size of the two restoration types is given in Table 3 as the mean values of the buccolingual, mesiodistal and incisocervical dimensions.

DISCUSSION

In this study an attempt was made to evaluate one of the factors said to be of importance for the durability of glass ionomer cement restorations, namely cavity form. A great advantage of glass ionomer cements is their adhesive property, which makes it possible to save tooth substance. A saucer-shaped microcavity for proximal lesions has been suggested, therefore, by several authors.[6,13,17] On the other hand, Croll and Phillips recommended that a narrowly prepared isthmus and regions of minimal cement bulk should be avoided to compensate for the low tensile strength and fracture toughness of glass ionomer cements.[11] Östlund *et al* compared class II restorations of amalgam, composite and glass ionomer cement in primary molars, using a small standard Class II cavity with rounded proximal angles, a broad isthmus and no proximal extension for prevention.[14] They reported a high failure rate for glass ionomer cement of 60 percent, mainly due to fractures in the isthmus area, and concluded that the cavity design they used was not suitable for glass ionomer cements. Walls *et al* used a cavity form where sufficient tissue was

removed to enable excavation of carious dentin.[12] No attempt was made to create retentive undercuts. After two years, their failure rate for glass ionomer cement restorations was 14 percent. After five years the failure rate was 32.8 percent compared with 16 percent failures after three years in our study.[18] Unfortunately, the authors do not report whether exfoliated teeth were included. Forsten and Karjalainen used much the same cavity designs as in the present study, with rounded outer and inner angles, comparing restorations with or without "dove-tail" on the occlusal surface.[17] After a follow-up period of five to eighteen months, they reported failure rates of 19 percent and 21 percent, respectively, for the two cavity types, in agreement with the results of our study.

The most frequent reason for drop-out in this study was exfoliation of the experimental primary molars. All nine teeth exfoliated after one year, but before two years, and the ten teeth exfoliated after two years, but before three years were scored as acceptable at the previous examination. Exfoliated teeth pose a particular problem in clinical evaluations in the mixed dentition. Including these teeth in the results gives a truer clinical picture, which in this study resulted in a higher durability rate.

A restoration with score C for anatomical form or marginal adaptation is normally registered as a failure according to the USPHS criteria. Clinically, however, for a primary tooth where exfoliation is anticipated within about a year, this is usually not considered an indication for replacement.

It can be concluded from the results of this study that the extension of the cavity form, to create a bulk of restorative material, is not of major importance for the durability of Class II glass ionomer cement restorations in primary molars, as long as outer and inner angles are well rounded. A saucer-shaped preparation is to be preferred when possible, with conservation of sound tooth substance and a reduced risk of pulpal involvement and cuspal fractures, apart from being simple and time saving.

The physical properties of conventional glass ionomer cements are inferior to those of amalgam and composite resins. Composite resins show better mechanical and physical properties with a higher strength. On the other hand, a higher incidence of secondary caries has been found contiguous to these restorations. Recently, hybrid restoratives, like the light-cured, resin-reinforced, glass ionomer cements, have been developed. Advantages of glass ionomer cements and composite resins are now combined in a single restorative material. The new materials seem to be less technique sensitive, have a faster setting-time and better physical properties than the conventional glass ionomer cements and probably do not need initial protection of the new restoration. These materials are clinically promising and may offer a more durable solution for the treatment of primary molars.

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Traumatic herniation of the buccal fat pad: Report of case

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The buccal fat pad lies within the cheek, lateral to the buccinator, and deep to the masseter muscle. It is a well-formed, subcutaneous, circumscribed, rounded, biconcave body of fatty tissue. The anterior part extends in front of the anterior border of the masseter muscle. The main function of this fat pad is thought to act as a cushioning tissue.

A minor tear of the buccinator extending through the oral mucosa can result in herniation of a large volume of the fat into the mouth. It is not uncommon, when operating in this region, to encounter fat, if the buccinator is incised. Thus the herniation of the buccal fat pad is secondary to the rupture of the buccal mucosa and buccinator muscle.

Traumas and lacerations of buccal mucosa are commonly seen in young children who fall with rod-like objects held in the mouth. This paper describes the clinical features and treatment of a case of a twenty-month-old child with traumatic prolapse of the buccal fat pad into the oral cavity.

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CASE REPORT

A twenty-month-old girl was referred to the Dental Clinic of Kyushu University for evaluation of an intraoral tumorous mass. This otherwise healthy child had been seen three days before in the other pediatric clinic after she had fallen with a tooth brush in the mouth. Following the accident, her mother had noticed slight bleeding after removing the tooth brush. The day after the accident, mother had observed slightly diffused facial swelling and evidence of a prolapse on the right buccal mucosa in the child's mouth, which apparently had not been present earlier. She also had noticed that the patient did not eat the usual food, because of the discomfort caused by the prolapse.

Gross intraoral examination revealed the presence of a firm, purple-yellow, freely mobile, pedunculated projection from the right buccal mucosa at the level of the occlusion of the primary molar teeth. There was no evidence of free bleeding. The mass appeared to be encapsulated and a small part of the tip was white-yellow. No other mucosal wound could be detected, and the dentition of sixteen primary teeth was intact. Based on those findings, a diagnosis of *traumatic herniation of the right buccal fat pad* was made.

Because of the child's rigorous objection to treatment, using local anesthesia, the excision was planned for four days later, using general anesthesia. A signed informed consent was obtained from her mother, permitting treat-

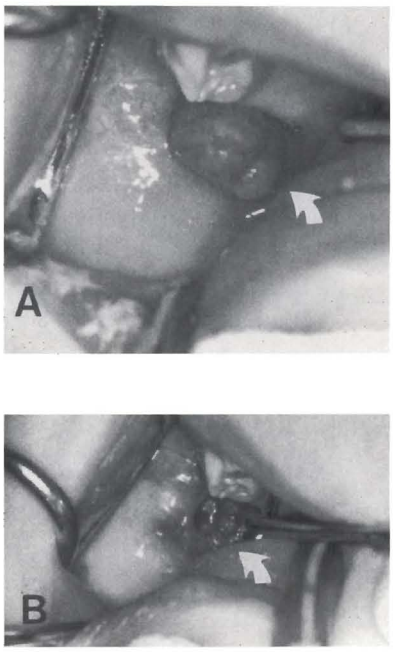

Figure 1A. Intraoral view showing the herniated fat pad in the right buccal mucosa in a twenty-month-old female. B. The injury of buccal mucosa and the perforation of the buccinator muscle. (Arrows)

ment, and using general anesthesia. Clinical and routine blood examinations revealed no abnormality, other than the soft tissue prolapse. After premedication with trichloral and atropine, the child was taken to the operating room. Vecuronium was used as a relaxant. Nitrous oxide, oxygen, and isoflurane were administered in a semi-closed circuit via an orotracheal intubation.

The discolored tip of the original swelling had exfoliated spontaneously. It seemed to originate from a narrow base very close to the aperture of the right parotid duct (Figure 1, A). Radiological examination using general anesthesia disclosed no radiopaque foreign body. The depth of the wound was evaluated, however, by inserting a gutta-percha point (Figure 2).

Then a half cartridge of 2 percent lidocaine hydrochloride with 1 :80,000 epinephrine was infiltrated at the base of the lesion. It was now seen to arise from the buccal mucosa adjacent to the right maxillary tuberosity region. The pedicle of the lesion penetrated the mucous membrane through a long slit and was attached deeply to the buccal fat pad. The prolapse was excised under the silk ligation at the base. The laceration wound was located distal to the parotid papilla at the occlusal level (Figure 1, B). Three Dexon sutures were placed to close

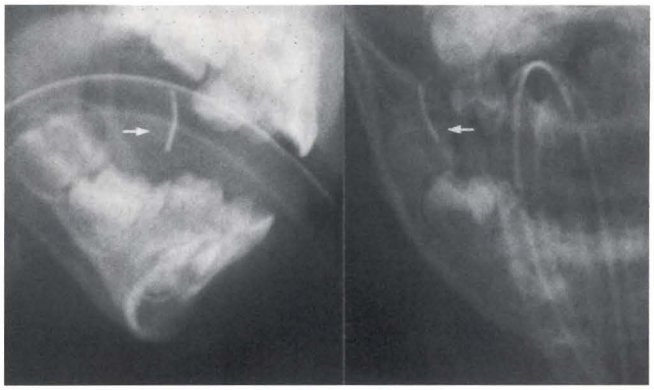

Figure 2. Radiograms showing the location and depth of the buccal wound. A gutta-percha point was introduced.

the wound. An antibiotic cover was maintained for three succeeding days.

The mass was covered with a normal-looking connective tissue capsule. It was grayish yellow in color, firm, and had a smooth surface. The specimen consisted of a firm nodule and measured 1 cm × 1.2 cm × 1.5 cm. The patient was discharged from the hospital on the third day after admission. One month after the operation, there was no masticatory discomfort or facial swelling, and the wound had healed satisfactorily. Salivary flow had not been disturbed.

Histopathological examination revealed fat tissue with some fibrous tissue, and with acute inflammatory cell invasion, without epithelial cover (Figure 3). The fat cells were mature with no evidence of atypia and were contained by a moderate supporting fibro-vascular stroma. These features confirmed the clinical diagnosis of traumatic herniation of the buccal fat pad.

DISCUSSION

The buccal fat pad is described as a rounded, biconvex structure, limited by a thin, but distinct, capsule and located in the so-called masticator space. The anterior part of the masticatory fat pad fills the space between the masseter and buccinator muscles.[1,2] Although the function of the fat pad is questionable, it is believed that this fatty tissue functions more as a cushioning and fill-in tissue. Its importance in masticatory function is best illustrated in the infant, where it acts as an aid in sucking.[3] It contributes to the bulging of the infant's cheeks and usually persists in adults. The body of the fat pad and the buccal extensions are largely responsible for cheek contour in adults. Thus, the buccal fat pad is cur-

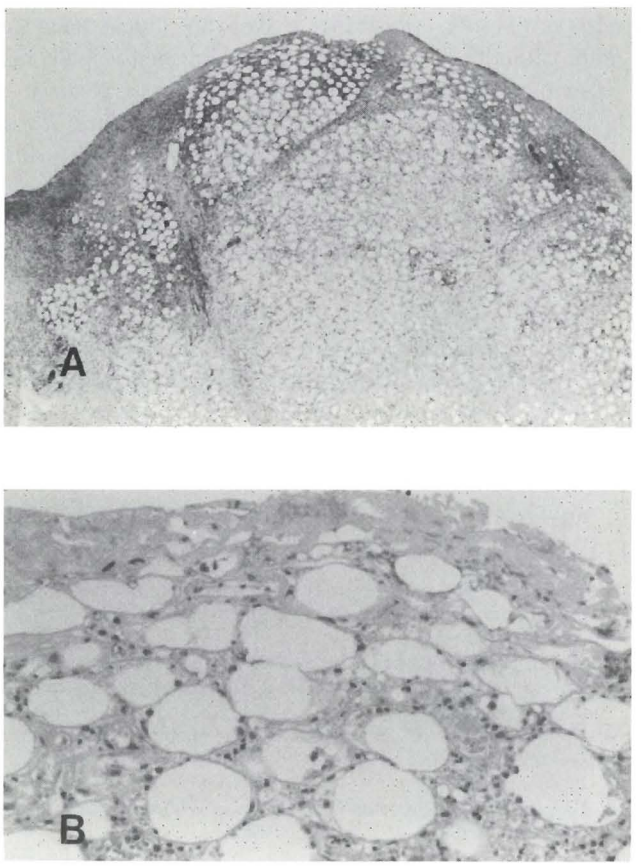

Figure 3. Photomicrograph of lesion. 3A. A section of the excised material showing lobules of adipose tissue with fibrous septae and an inflammatory infiltration without epithelial cover. (Hematoxylin eosin, magnification × 5) B. Histologic appearance showing mature fat cells and absence of capsule. (Magnification, × 20.)

rently of interest in esthetic surgery, such as buccal lipectomy in the adult, to modify the contour of the face.[3]

The buccal fat pad is sometimes encountered in the course of surgical procedures in the region of the anterior ramus of the mandible or the maxillary tuberosity.[1,4-6] Review of the literature suggests, however, that traumatic intraoral herniation of the buccal fat pad is rare. Only Brooke has referred to this condition as traumatic pseudolipoma, to distinguish it from other benign fatty tumors of the oral cavity.[2,7] All cases reported, except one twelve-year-old boy, were in infants or young children, with an age-range from five months to five years.[7,8] There was a history of trauma in every case. In such cases, two contributing factors are proposed: the buccal fat pad is relatively prominent in infants and young children, who frequently investigate foreign objects by placing them in their mouths.[4,9] Infants and young children may also have burned their lips by biting or inserting a live electric plug in the mouth. They are also prone to fall and thus a sharp object is likely to result in a puncture wound of the buccal mucosa. Further sucking action could have resulted in the fat pad being pulled out.[6,9] The lesions varied in size up to a maximum diameter of 2.5 cm.[7]

The most characteristic aspect of this lesion is that the mucosal injury or perforation is very small compared to the size of the extruded mass. Peacock described a case of a ten-month-old boy who showed an increase in size of the buccal fat pad, possibly contributed to by sucking.[10] Fleming claimed that the sucking activity of an infant might encourage the herniation of the fat pad through the wound into the mouth, and also mentioned the risk of respiratory embarrassment.[9] In the case of Haria *et al*, volume of the intraoral mass was observed to have increased after five days.[4] The majority of cases with some exceptions involved a foreign object in the mouth, which subsequently caused the penetrating injury through the buccal mucosa and buccinator muscle.[8,10] Similarly palatal lacerations are commonly seen in children who fall on to objects such as pens and pencils held in the mouth.[11]

The buccal mucosa can be traumatized by the teeth in an accident such as falling off a bicycle.[4] An external blow may also cause rupture of the buccal mucosa. Leopard reported a case in which a blow with a cricket bat produced a moderate degree of external bruising, ruptured the buccal mucosa and thus allowing the buccal fat pad to prolapse.[12] Messenger *et al* described a case in which a four-year-old boy struck his face in falling off a scooter toy.[1] Fleming described a case of a twelve-year-old boy struck on his left cheek by a fist.[9] Marano *et al* reported a case in which herniation of the buccal fat pad into the maxillary sinus occurred, associated with the fracture of the lateral sinus, following a blow to the face.[13]

In the case reported by Wolford *et al* there were dark-colored, necrotic-appearing areas where the lesion was obviously being traumatized by the occlusion of the teeth.[8] Two days after the accident in the case reported in this paper, the findings were similar. Additionally chewing was causing moderate discomfort.[4]

Initially bleeding is brisk, but spontaneous arrest usually occurs. As for the treatment, repositioning or excision of the prolapsed fat tissue, following suturing of the wound is advocated. An alternative to excision is to replace the herniated buccal fat pad to its original position

and suture the puncture wound. In a recent case reported by Clawson *et al*, the fat pad was gently pushed through the laceration and into its capsule.[14] When extrusion of the buccal fat pad occurs, as in some osteotomies, the tissue may be pushed back and repaired by sutures. In some cases, the tissue was replaced in its normal anatomic position by primary closure.

Infection due to salivary contamination can be a problem. In a case reported by Fleming, bacterial aggregates were found at the site of the lesion within three hours after the trauma occurred.[9] Necrotic tissue and bacteria must be removed before the fat pad is returned to its normal position. In the present case, because of the intense inflammatory reaction with necrosis, it was decided to excise the extruded fat pad and close the perforation. In a case of partial prolapse, one method of treatment is partial excision, followed by closure of the buccal mucosa, as described in this case report. Removal of the pad may produce a change in facial contour, reducing cheek fullness, highlighting the malar eminences, and giving a more sculptured look to the face.[3] Thus, a conservative excision is indicated. Complications have proven minimal. Nevertheless, it should be emphasized that the resection is limited to the fat that protrudes into the mouth.

The buccal fat pad is intimately related to the masticatory muscles, facial nerve, and parotid duct. In the excision of the prominent tissue and closure of the wound, great care must be taken to avoid injury to the parotid duct. An attempt to express saliva from the parotid duct should be made to identify the location of the parotid papilla. As the parotid gland is contained within a very dense fibrous capsule, it is easy to identify the duct. No evidence of damage to Stensen's duct has been reported.[14] Exploration for foreign bodies and irrigation are important before closing the wound.

Although a benign tumor must be included in the differential diagnosis, the history of trauma and absence of prolapse before the injury rule out such lesions.[2,4] It may be speculated that if healing had occurred, the lesion would have beco e covered with epithelium, making it clinically indistinguishable from lipoma. In cases where there is a delay in seeking treatment or proper treatment, as in the case reported here, the inflamed, indurated or necrotic appearance makes the clinical diagnosis more difficult.[2,6,8] In case of herniation of the pad, microscopic examination reveals mainly adipose tissue in a light to moderate fibrous stroma with bleeding, fibrin deposition, and acute inflammation. Characteristically, no capsule is demonstrated histologically.[2,15] It was described as a necrotic lesion consisting of fatty tissue with a heavy infiltration of polymorphs.[10] These features mimic a panniculitis.

In conclusion, traumatic herniation of the buccal fat pad is a clinical entity that appears on the cheek mucosa as a pseudolipoma. Including the patient in this report, there has been no report of recurrence of herniation.

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DEMOGRAPHICS

Almost four million children with disabilities

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The recent publication by the Bureau of Census of its report, *Americans with disabilities: 1991-92* provides an extended summary of the demographic characteristics of the 48.9 million individuals (19.4 percent of the total population not living in nursing homes and other institutions) with disabilities in this country—including 3.9 million children (5.8 percent of all children less than 18 years of age).[1] Approximately 30,000 households were interviewed for purposes of this study.†

Previous reviews by this writer in the *Journal of Dentistry for Children* emphasized:

- ☐ Special pediatric population groups and their use of dental services in the mid 1980s.[2]
- ☐ The prevalence of developmental, learning and emotional problems in the general population of children in the late 1980s.[3]
- ☐ The prevalence of chronic illnesses reported for the children in our nation in the late 1980s.[4]

The following presentation will review the prevalence of disabled children in our country based on the 1991-92 Bureau of the Census reports as well as reports from a variety of federal support programs (Head Start, Supplemental Security Income [SSI] and Education for the Handicapped). As individual dental practitioners and general communities increasingly respond to and work within the regulations of the Americans with Disabilities Act of 1990 (ADA), it is essential to develop a greater appreciation of the populations faced with a wide range of disabilities.

DEFINING DISABILITIES

The term "disability" can be defined narrowly or broadly depending on the need for and use of the data. The Social Security Disability Insurance Program uses the narrow definition of disabled persons as individuals who are "unable to engage in substantial gainful activity." The broader definition in the Americans with Disabilities Act of 1990 considers an individual to be disabled, if the person a) has a physical or mental impairment that substantially limits one or more major life activities, b) has a record of such an impairment, or c) is regarded as having such an impairment.

Other more extensive definitions include specific limitations in performing socially defined roles and tasks in such areas as personal relationships, family life, education, recreation, self-care and work. Still other definitions emphasize the relationship between the individual and his/her environment. Thus it is necessary to distinguish between:

- ☐ Disability as the functional limitation within the individual caused by physical, mental, or sensory impairments; and
- ☐ Handicap as the loss or limitation of opportunities to take part in the normal life of the community on

†Unless otherwise specified, data in this presentation were drawn from the 1991-92 Bureau of the Census report.

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an equal level with others due to physical and social barriers.[1]††

IN GENERAL

There is a progressive increase with age in the percent of the population that is disabled and severely disabled, increasing from 5.8 percent and 1.3 percent, respectively, for children less than eighteen years of age to 84 percent and 68 percent, respectively, for individuals eighty-five years and older (Figure). (Note: unless otherwise specified, numbers and percent of severely disabled are included within the general category of the disabled.)

Disability rates are somewhat lower among males than among females. In the general population, males have a disability rate of 18.7 percent and a severely disabled rate of 8.1 percent. By comparison, female rates are 20.2 percent and 11.0 percent, respectively. Part of the explanation of the differences between males and females has to do with the overall population age structure (i.e. greater numbers of older age females) and the fact that disability rates increase with age.

By contrast for children, boys have somewhat higher disability and severe disability rates than girls. In addition, the percent of boys that have limited ability to do regular school work is twice that of girls (7.1 percent vs. 3.5 percent) (Table 1). (Note: children were classified as having a severe disability if they a) used a wheelchair or had used another special aid for 6 months or longer, b) were unable to perform one or more functional activities or c) needed assistance (beyond normal levels for their age) with activities of daily living (e.g. bathing, dressing, toileting) or d) had a selected condition including autism, cerebral palsy or mental retardation.)

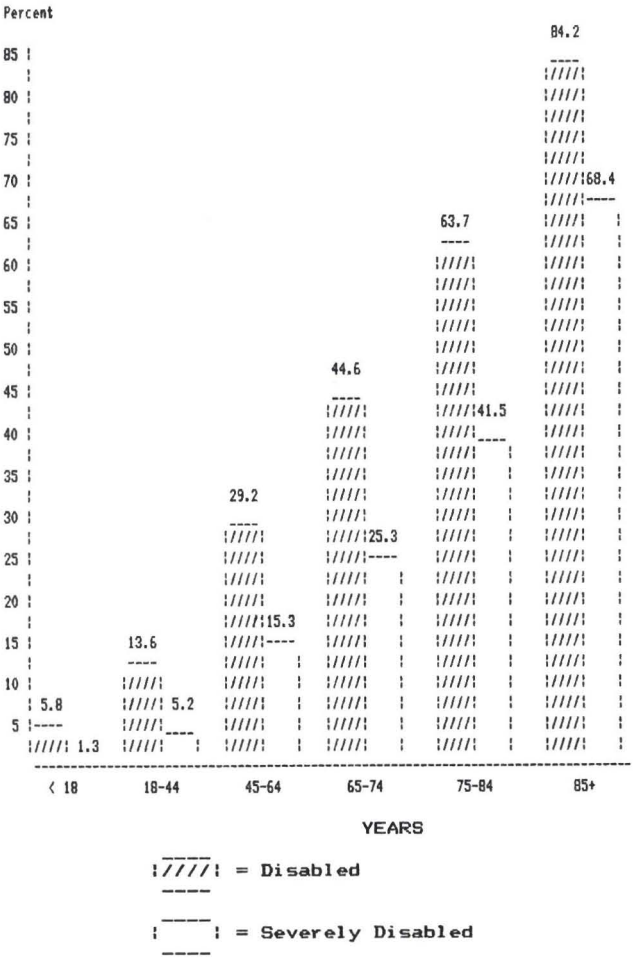

Figure. Percent of persons with disabilities and with severe disabilities, by age: 1991-92.[1]

BY RACE AND ETHNICITY

General population

Data for persons of all ages show that the overall disability rates among whites (19.7 percent), blacks (20.0 percent) and American Indians, Eskimos and Aleuts (21.9 percent) were not statistically different, but the rate of 15.3 percent among persons of Hispanic origin (who may be of any race) was lower than the rates for the first three groups. In addition, the rate among Asian-Americans and Pacific Islanders (9.9 percent) was lowest of all groups.

Similarly, severe disability prevalence rates differed among race and ethnicity groups, 9.4 percent among whites, 12.2 percent among blacks, 8.4 percent among persons of Hispanic origin. Asian-Americans and Pacific Islanders had the lowest prevalence rate, 4.9 percent.

Children

In 1991-92, there were more than 3.1 million white, 0.6 million black and 0.3 million Hispanic disabled children.

††For purposes of the Bureau of the Census study, childhood disabilities included the following health conditions: asthma, autism, blindness or vision problems, cancer, cerebral palsy, deafness or serious trouble hearing, diabetes, drug or alcohol problems or disorders, epilepsy or seizure disorders, hay fever or other respiratory allergies, head or spinal cord injury, heart trouble, impairment or deformity of back, side, foot or leg, impairment or deformity of fingers, hands or arms, learning disability, mental or emotional problem or disorder, mental retardation, missing legs, feet, toes, arms, hands or fingers, paralysis of any kind, speech problems, and repeated ear infections.

Table 1 ☐ Children with disabilities and severe disabilities by age and gender: 1991–92.[1]

	Male	Female
	(in 000s)	
< 3 yrs		
Disabled	133	121
Severely disabled	32	8
3-5 yrs		
Disabled	370	228
Severly disabled	54	21
6-14 yrs		
Disabled	1,373	689
Severly disabled	250	163
Limited in ability to do regular school work	1,197	567
15-17 yrs		
Disabled	588	374
Severely disabled	159	150
	Percent of children	
< 3 yrs		
Disabled	2.2%	2.1%
Severely disabled	0.5	0.1
3-5 yrs		
Disabled	6.2	4.1
Severely disabled	0.9	0.4
6-14 yrs		
Disabled	8.2	4.3
Severely disabled	1.5	1.0
Limited in ability to do regular school work	7.1	3.5
15-17 yrs		
Disabled	10.8	7.7
Severely disabled	3.1	3.1

Table 2 ☐ Disabilities by age, race and Hispanic origin: 1991–92.[1]

	Totals*	White	Black	Hispanic**
		(in 000s)		
< 3 yrs	254	203	45	17
3-5 yrs	597	498	80	35
6-14 yrs	2,062	1,702	302	151
15-17 yrs	933	702	184	104
Totals	3,846	3,105	611	307
		Percent of children		
< 3 yrs	2.2%	2.2%	2.5%	1.2%
3-5 yrs	5.2	5.5	4.3	2.5
6-14 yrs	6.3	6.5	5.9	4.1
15-17 yrs	9.3	8.9	10.9	8.5

*Includes other races not specified
**May be of any race

Table 3 ☐ Children with severe disabilities by age, race and Hispanic origin: 1991–92[1]

	Totals*	White	Black	Hispanic	Asian-American & Pacific Islander
			(in 000s)		
< 6 yrs	116	94	16	5	6
6-14 yrs	412	357	44	22	6
15-17 yrs	309	207	94	28	na
Totals	837	658	154	55	na
			Percent of children		
< 6 yrs	0.5%	0.5%	0.4%	0.2%	0.8%
6-14 yrs	1.3	1.4	0.8	0.6	0.5
15-17 yrs	3.1	2.6	5.5	2.3	na

*Includes other races not specified

There were small variations in the prevalence ratios between white and black children of various ages. In most age-groups, Hispanic children had the lowest prevalence rates (Table 2).

In the same period, there were more than 0.8 million severely disabled children, including 0.6 million white, 0.1 million black, and 55 thousand Hispanic children. While the severe disability prevalence rates increased with the age of the children, there were no consistent major differences between children in the various racial and ethnic groups (Table 3).

SUPPORT PROGRAMS

Head Start program

The Head Start program provides comprehensive educational, medical, nutritional, dental and mental health and other social services for children from low-income families, along with parent involvement and social services for their parents. The Head Start Act requires that no less than 10 percent of the total number of enrollment opportunities in Head Start programs in each state shall be available for handicapped children. In the 1989-90 program year, Head Start provided comprehensive developmental services to 521,266 children, 69,267 (13.3 percent) were children with disabilities. In 1990, 15.5 percent of the children with disabilities enrolled in the programs had multiple disabling conditions (Table 4). Between 1980 and 1989-90, there was an increase of more than 25 thousand children with disabilities enrolled in the programs (a 58.5 percent increase). The increase in the number of children was reported for Head Start programs in every state and territory of the country (Table 5).

Supplemental security insurance program

The Supplemental Security Insurance (SSI) Program is limited to low income persons who are aged, blind or disabled. For children under eighteen years of age, the disability must be of comparable severity to that of an

Table 4 ☐ Head Start program: types of disabling conditions of children: 1989–90.[5]

Disabling condition	Number of children	Percent
Speech impairment	46,549	67.2%
Health impairment	7,611	11.1
Learning disabilities	4,109	5.9
Serious emotional disturbance	2,898	4.2
Physical disability	2,724	3.9
Mental retardation	2,616	3.8
Hearing impairment	1,331	1.9
Visual impairment	1,244	1.8
Blindness	95	0.1
Deafness	90	0.1
Total	69,267	100%

Note: speech impairment does not include conditions of a transitional nature resulting from the developmental processes of the child

adult.‡ In 1989, 6,700 blind and 258,107 disabled children less than eighteen years of age received benefits under the SSI program. More than 13 thousand disabled children in Florida, Ohio and Pennsylvania and more than 21 thousand disabled children in California, New York and Texas received benefits (Table 5). (Note: individual state data are for recipients less than twenty-one years of age.)

Education of the handicapped

The Elementary and Secondary Education Act of 1965 and the Education of the Handicapped Act provide an opportunity for a free education of all handicapped students. In 1989-90, 4.2 million children between six and twenty-one years were enrolled in schools under this program. At the state level, there were almost 300,000 children in Texas and almost 400,000 in California enrolled under this program (Table 5).

BEYOND NUMBERS

If we are to comprehend some of the consequences and impact on individual family members of the increasing numbers of disabled children identified in federal surveys and reports, consider some of the caregiver services necessary for these children that may require additional

‡For example, eligibility for disability benefits requires inability to engage in any substantial gainful activity by reason of any medically determinable physical or mental impairment which can be expected to result in death or which lasted or can be expected to last for a continuous period not less than 12 months.[6] Note: This review of SSI recipients does not include the 590,360 disabled children eighteen years and older (in 1989) of either retired or Social Security Disability Insurance recipient parents.

Table 5 ☐ Children with disabilities in Head Start, Supplemental Insurance, and Education for the Handicapped Programs: 1980–1990.[5,6]

	Head Start (age < 7 yrs)		Supplemental Security Income - 1989 (Age <21 yrs)		Education of the Handicapped (Age 6-21 yrs)
	1980	1989-90	Blind	Disabled	1988-1989
					(in 000s)
Alabama	1,033	1,377	105	8,111	94.6
Alaska	79	146	12	332	12.9
Arizona	355	502	79	3,622	51.4
Arkansas	677	959	122	5,049	43.2
California	2,687	5,191	1,372	26,804	397.3
Colorado	545	743	62	3,281	48.7
Connecticut	353	537	77	1,940	58.1
Delaware	112	131	11	693	12.2
Dist. Columbia	114	244	12	913	6.6
Florida	1,072	1,697	290	13,299	193.8
Georgia	1,163	1,662	181	9,205	86.7
Hawaii	136	181	24	509	11.5
Idaho	241	259	36	1,110	18.3
Illinois	1,996	3,022	280	12,671	222.2
Indiana	806	1,350	169	5,775	101.2
Iowa	425	754	159	2,606	51.6
Kansas	488	719	60	2,140	39.4
Kentucky	1,281	1,854	132	6,636	67.3
Louisiana	1,108	1,485	234	11,856	62.5
Maine	217	729	29	1,066	25.1
Maryland	538	829	84	3,549	83.0
Massachusetts	728	1,414	470	5,516	133.8
Michigan	1,918	2,496	242	9,176	147.6
Minnesota	490	1,050	120	2,732	73.1
Mississippi	3,350	2,804	102	7,706	54.0
Missouri	1,150	1,521	130	5,912	96.1
Montana	135	286	19	887	13.9
Nebraska	301	314	43	1,580	28.7
Nevada	75	99	58	795	14.5
New Hampshire	112	115	11	488	16.4
New Jersey	828	1,603	130	7,297	158.7
New Mexico	342	539	54	2,192	30.7
New York	2,134	4,329	392	25,209	269.6
North Carolina	909	1,938	208	7,730	106.0
North Dakota	76	118	11	501	11.2
Ohio	2,192	3,995	354	13,113	190.4
Oklahoma	895	1,296	109	3,618	58.9
Oregon	385	609	79	2,336	45.8
Pennsylvania	2,106	3,324	308	13,706	190.4
Rhode Island	186	255	26	992	18.1
South Carolina	666	908	172	5,547	68.8
South Dakota	105	157	29	1,016	12.5
Tennessee	1,029	1,450	174	7,773	94.5
Texas	1,934	3,061	636	21,096	296.4
Utah	227	381	64	1,450	40.0
Vermont	97	150	11	440	11.7
Virginia	551	985	136	5,117	96.6
Washington	582	697	95	4,138	66.8
West Virginia	537	695	74	2,892	41.5
Wisconsin	623	948	116	5,963	68.6
Wyoming	73	112	7	303	9.3
Puerto Rico	1,623	2,737			10.0
Virgin Islands	22	188			1.2
Pacific Territories	59	156			2.8
Indian Programs	983	2,152			
Migrant Programs	840	2,055			
Total	43,689	69,267	7,910	288,388	4,186.6
			Children < 18 yrs		
		Total	6,700	258,107	

time and financial expenditures.

- ☐ Personal care (helping with the child's bathing, grooming, eating).
- ☐ Providing medical care (medications, tube feedings, treatment of wounds).
- ☐ Preparing special or extra meals and clean up.
- ☐ Extra or special shopping and errands (including medications).
- ☐ Extra household chores (in particular, cleaning and straightening out).
- ☐ Escorting the child for health care services.
- ☐ Escorting the child to nonmedical services beyond those necessary for a healthy child (social and entertainment).
- ☐ Managing the child's medical conditions (appointment with physicians, nurses and social workers).
- ☐ Managing the child's finances (dealing with insurance companies, finding special programs, filling out forms).
- ☐ Waiting time in health practitioner offices.
- ☐ Monitoring medical equipment.

FROM THE PERSPECTIVE OF THE DENTIST

"...it is the pediatric dentist who increasingly is being called upon to treat the dental needs of a 'new' special patient population, the survivors of medical conditions who previously succumbed to a seemingly endless range of morbidities: hydrocephalus, epilepsy, spinal cord injuries, spina bifida, cerebral palsy, major cardiovascular anomalies, neonatal abnormalities, kidney dialysis, a wide range of blood dyscrasias, immunosuppressant diseases (including AIDS), muscular dystrophy, multiple sclerosis, juvenile diabetes and complex endocrine abnormalities." [2]

It was not that long ago that the existence of disabled (both children and adults) was "hidden" from general public. Case in point was the press picture blackout of President Franklin Roosevelt's physical limitation. Other than the advertising gimmick used by "Hathaway Shirt Man" (who wore a patch over one eye) virtually all models, actors (except Lionel Barrymore, who used a wheelchair in his older years - but now I'm beginning to date myself) and actresses seemed to be in perfect health. Only recently have I seen advertisements in magazines and on television that include children and adults in wheelchairs. Maybe the passage of the Americans with Disabilities Act has sensitized Madison Avenue to the realities that not all of us are in the Hollywood image of perfect health. But more than likely, advertisers now consider the enlarging population of disabled youngsters and adults as a potential increasing market for sales.

As dentists expand their services to more diverse populations groups they too will be marketing their services to populations with which many practitioners have had limited experiences. Modifications may be necessary in the physical layout of dental offices. Treatment planning may require added consultations with a broad range of health practitioners and social service personnel. Indeed, it may take added time, patience, and understanding in caring for a disabled child. But aside from the fact that there are increasing numbers of disabled youngsters in your community, and we are the only professionals trained to provide the need care, the satisfaction is worth the effort.

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Is your pediatric practice keeping pace with the improving picture of dental economics?

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It is only natural to wonder, "how am I doing" compared to other dentists. The peculiarities of private practice arrangements all too often limit the interaction of practitioners, particularly when it comes to economics. But while we cannot tell you how "doctors A and B" down the street from your office are doing, we can assure you that the overall economics of dental practices continue to improve into the early 1990.

A previous review in the *Journal of Dentistry for Children* provided an overview of the evolving economics of dental and general health care through the mid and late 1980s.[1] The following presentation will follow these trends into the first third of the 1990s.

SOURCE OF DATA

Information from the annual reports from the Office of National Health Care Statistics of the Health Care and Financing Administration, the American Dental Association (ADA) Survey of Dental Practice, and the dental practice business receipt data from the Internal Revenue Service (IRS), providing different "readings" on the economics of practice.[2-10]

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NATIONAL EXPENDITURES AND PRACTITIONER INCOME

Population spending

Between 1980, 1990, and 1993, national expenditures for dental services increased from $14.4 billion to $34 billion to $41.0 billion. Much of these increases were a reflection of the effects of inflation. In terms of constant dollars, (i.e. removing the effects of inflation) since 1990 there has been no change in the national spending level for dental services in the country despite an increase of more than 10 million residents. Since 1990, the combination of level spending and an increasing population produced a decline in constant dollar dental expenditures per individual (Table 1). The hold on dental expenditures was a reflection of the economic uncertainties, during the recession of the early l990s. Spending for dental services was placed on the proverbial "back burner." Family funds were being used for perceived "more important" needs.

Practitioner income

Expenditures per professionally active dentist increased from $120 thousand in 1980 to $288 thousand in 1993. (Not all professionally active dentists are in full-time active practice, e.g. educators, administrators and researchers. This approach would provide a relatively conservative estimate of practitioner income.) The increasing current dollar expenditures per dentist re-

Table 1. ☐ Population expenditures for dental services: selected years 1980–1993.[2–4, 11–14]

	1980	1985	1990	1991	1992	1993
Current dollars						
National expenditures (billions)	$14.4	$23.3	$34.0	$36.6	$38.7	$41.0*
National population (millions)	226.5	238.2	248.7	252.2	255.1	257.6
Expenditures per individual	$63.58	$97.82	$136.71	$145.12	$151.71	$159.16
Dental Price Index (1982–1984=100)	80.1	114.3	155.8	167.4	178.7	188.1
Constant dollar						
National expenditures (billions)	$17.9	$20.4	$21.8	$21.8	$21.6	$21.7
Expenditures per individual	$79.37	$85.58	$87.75	$86.69	$84.89	$84.62

*Estimate

Table 2 ☐ Dental practitioner income based on national expenditures: selected years 1980–1991.[3–5, 13, 14, 16–18]

	1980	1985	1990	1991	1992	1993
National expenditures (billions)	$14.4	$23.3	$34.0	$36.6	$38.7	$41.0*
Number of professionally active dentists	120,483**	134,201**	140,699	141,327	141,915	142,395
Expenditures per dentist	$119,518	$173,620	$241.650	$258,973	$272,698	$287,931
Net income as a percent of gross receipts	40.1%***	36.0%**	33.2%	33.2%	33.7%	33.7%****
Net income	$47,927	$62.503	$80,227	$85,979	$91,899	$97,032
Consumer Price Index (1982–84=100)	82.4	107.6	130.7	136.2	140.3	144.5
Constant dollar net income	$58,163	$58,088	$61,382	$63,127	$65,502	$67,150

*Estimate
**Estimate based on ADA data
***1981 datum
****Based on 1992 datum

flected, 1) the increases in inflation, 2) real increases in expenditures during the 1980s, and 3) a more than one-third decrease in the annual numbers of dental school graduates.[15]

But in the intervening period, the costs of doing business increased significantly. Net income as a percent of gross receipts decreased from 40 percent to between 34 percent and 33 percent. Nevertheless, current dollar net income per professionally active dentist (based on national expenditure data) increased from $48 thousand to $97 thousand. In addition, when eliminating the effects of inflation,

- ☐ Constant dollar practitioner net income in the 1990s has been greater than earnings in the 1980s.
- ☐ Constant dollar practitioner net income has increased annually during the 1990s (Table 2).

Note: The Consumer Price Index (CPI) reflects the cost of purchasing an ongoing "market basket" of various goods and services. The component item costs do not increase (or decrease) at uniform rates. Thus the overall CPI represents the average of all component changes. Health care costs, including dental services, historically have increased at rates well in excess of the overall CPI (Figure). While the overall CPI increased by 10.5 percent between 1990 and 1993, the Dental Price Index (the cost of purchasing particular dental services, e.g. an extraction or a filling — both of which are included in the Dental Price Index) increased by 20.7 percent (Tables 1 and 2).

Different index deflators are used, therefore, to eliminate the effects of inflation. For example,

- ☐ To determine an individual's (or the general public's) constant dollar cost for dental care the more rapidly increasing Dental Price Index component of the CPI is used.
- ☐ To determine dental practitioners' constant dollar net income the overall CPI index is used, since dentists will be purchasing the total "market basket" with their incomes.

Thus while constant dollar national and individual expenditures for dental services have remained level or decreased, (based on the relatively greater inflationary increases of dental care costs) practitioner constant dollar net income during the same period continued to increase (reflecting the more moderate inflationary rate of the overall CPI).

ADA REPORTS ON PRACTITIONER INCOME

Reports from the ADA Survey of Dental Practice on the income of dentists mirrored the general improvements

in practitioner current and constant dollar income during the 1980s and early 1990s. The reports were developed using national dental expenditure rates. The mean constant dollar income rates, which remained level during the late 1980s and early 1990s, once again increased in 1992. In the same year, the median income of practitioners did not maintain parity with the rat of inflation. Practitioner constant dollar mean and median incomes consistently were higher in the early 1990s, however, than in the first half of the 1980s (Table 3).†

IRS BUSINESS RECEIPT REPORTS

Reports by the Internal Revenue Service on the business receipts of solo, partnership (per partner) and corporate dental practice arrangements consistently have been lower than gross receipt data reported by the ADA and national expenditure data per practitioner that were developed for this presentation. Nevertheless, between 1980s and the early 1990s, (despite changing numbers of the various practice arrangements) current dollar IRS business receipt data for dental practices consistently increased each year.[6-10]

In an effort to simplify the varied and complex series of accounting procedures used in determining practitioner net incomes in the series of IRS reports, for purposes of this presentation, practitioner current and constant dollar net incomes were developed using,

☐ IRS reported gross receipt data for sole proprietorships, partners and corporations.

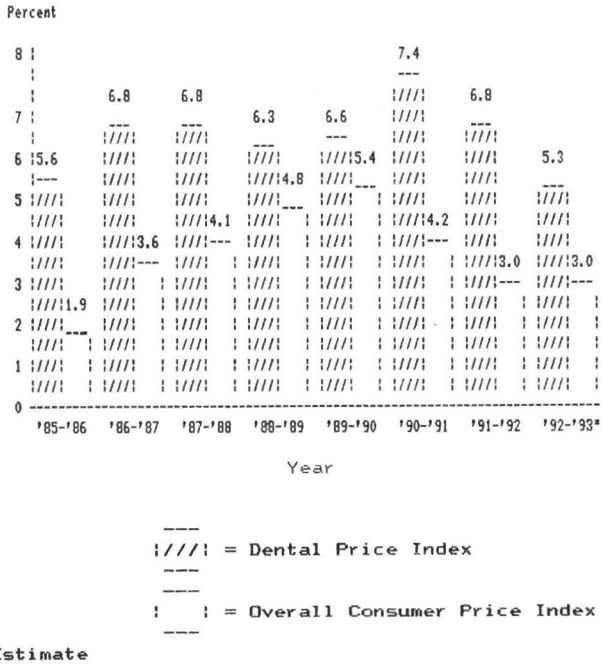

Figure. Percent increase from previous year of Dental Component Index and overall Consumer Price Index 1985-1993.[19]

☐ ADA reported net income-to-gross receipt ratios for all independent practitioners.‡

Reflecting the reduced gross receipt data reported to the IRS, (except for corporate income levels—for which the numbers of practitioners employed per corporation are not available) practitioner current and constant dollar net incomes consistently were lower than (in some

†The surveys and reports on practitioner earnings carried out by the commercial publication *Dental Economics*, e.g. "Median income outpaces inflation" were not considered in this review because of the consistently low respondent response rates to their past and present economic surveys. In 1993, the response rate for an elective mail-in form was approximately 1 percent of all dentists who receive the publication.[20]

‡An independent dentist owns or shares in the ownership of a dental practice (including sole proprietorship, partnership and corporate arrangements). In 1992, 90.6 percent of all dentists in private practice were reported as independent dentists.[5]

Table 3 ☐ ADA SURVEY DATA: Independent dentist mean and median net income: selected years 1981–1992.[5, 11, 14]

Year	Current dollar Mean	Current dollar Median	Consumer Price Index (1982–84=100)	Constant dollar Mean	Constant dollar Median
1981	$56,232	$50,000	90.9	$61,861	$55,005
1985	69,980	64,600	107.6	65,037	60,037
1989	91,220	80,000	124.0	73,545	64,516
1990	96,500	85,000	130.7	73,833	65,034
1991	100,540	90,000	136.2	73,817	66,079
1992	107,220	90,000	140.3	76,421	64,148

Note: As compared to mean income data, median income data are affected less by high and low income extremes.

Table 4 ☐ **Tax return data:** *Current dollar* dental practice *Net income* for sole proprietorships, partnerships and corporations: selected years 1980–1991.[5,6–10]

	ADA Survey Report Net income as a percent of gross income *	Sole Proprietor	Per partner	Per corporation
1980	44.6%**	$38,252	$18,718	$107,504
1985	34.5	39,935	34,478	114,750
1989	33.4	44,688	39,854	123,493
1990	33.2	51,729	40,270	141,289
1991	33.2	59,506	57,494	

*The ADA Survey of Dental Practice does not report net to gross receipt ratio data for different practice modalities. Throughout this report, the percent net to gross income is based on ratios reported for all independent practitioners.

**Percent of net to gross income is based on ADA Survey reports in prior and following years

Note: Beginning in 1982, data for corporations represent fiscal year periods between July 1st and June 30th (e.g. 1990 data represent information for the period July 1, 1990 to June 30, 1991)

Table 5 ☐ **Tax return data:** *Constant dollar* dental practice *Net income* for sole proprietorships, partnerships and corporations: selected years 1980–1991.[5,6–10]

Year	Consumer Price Index (1982–84=100)	Sole Proprietor	Per partner	Per corporation
1980	82.4	$46,422	$22,716	$130,466
1985	107.6	37,114	32,043	106,645
1989	124.0	36,038	32,140	97,247
1990	130.7	39,578	30,811	108,101
1991	136.3	43,690	42,212	

Note: for a more detailed review of IRS business receipt data and related practitioner income, see a previous review.[21]

Table 6 ☐ Dental school graduates, graduates per million population and number of dentists per 100,000 population: selected years 1950–2020.[14–16, 22]

Year	Number of graduates	Graduates per million pop.	Dentists per 100,000 pop.
1950	2,565	16.8	49.8
1960	3,253	18.0	47.0
1970	3,749	18.3	47.0
1980	5,256	23.1	53.5
1983	5,756*	24.5	
1990	4,233	17.0	56.6**
1993	3,778	14.6	55.6
Projections			
2000	3,800***	13.9	47.8****
2010	3,800***	12.8	46.0***
2020	3,800***	11.8	39.7****

*Greatest number of graduates
**1987 datum (highest ratio)
***Using 1993 graduation levels
****Based upon American Association of Dental School projections

cases less than half) 1) ADA net income reports, and 2) practitioner incomes that were developed using national dental expenditure rates. Nevertheless, even at these reported reduced levels, in almost all years practitioner incomes continue to increase in the early 1990 (Tables 3 and 4).

COMPARISONS

Yes, there are different readings of the economic pulse of dentistry. But in each scenario, the national economic picture of the profession continues to improve. No doubt in particular areas and communities there are ongoing difficulties as a result of economic reverses, concentration of dental practitioners and other factors in some locales. But as we review the economics for pediatric dentistry, two facts should be emphasized,

- ☐ As a result of the continuing increase in the size of the population in this country and the control in the production of dentists, the dentist-to-population ratio that peaked in 1987, will continue to decrease into the first decades of the next century, reaching levels that were last observed at the end of the nineteenth century (Table 6).
- ☐ This material is being prepared in the summer of 1994 as the Congress is enmeshed in the efforts to establish a workable system for national health insurance. While the specific outcome is far from certain, the various plans have recognized the need to ensure that dental care for children is included in the required package of services.

Consider then an already favorable dental economic picture in the 1990s, a future with decreasing number of dentists and dentist-to-population ratios, and an effort to guarantee dental services financially for many of the underserved youngsters. Sounds great, but is your pediatric dental practice keeping pace with this improving picture? Probably yes, but if not, the developing set of circumstances bodes well for your future.

Addendum

Results from the 1993 ADA Survey of Dental Practice – Specialists in Private Practice (1992 data) indicate that the average net income from pediatric dental private practice was $145,020, compared to the average net income of $153,700 for all independent specialists. Specialist average net income ranged from $119,570 for prosthodontists to $171,840 for oral and maxillofacial surgeons.[23]

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IN TERMS OF INSURANCE, IS DENTISTRY ANY DIFFERENT FROM MEDICINE?

While there is some evidence that judiciously applied managed care is demanded by excesses of American medical care, the same case cannot be made for intrusion in dentistry. From 1960 to 1991, the average annual increase in U.S. health care expenditures was 11.3 percent. Dental care costs rose significantly over this same period, but dental increases were substantially outstripped by hospitals, physicians, and other general health care cost hikes. A cost/benefit analysis concludes that the dramatic recent improvement in American's oral health is unparalleled in the rest of the health care system, despite a drop in dental expenditures over the same period. The dental profession has failed to adequately communicate its successes in prevention and primary care, and failed to take credit for an extraordinarily cost effective delivery model. To insert dentistry into a costly and too often ineffective medical model would be an economic mistake and clinical travesty.

Because dentistry represents such a small slice of the American health care pie, it is too often mingled indiscriminately with medical care by public policy makers and independent health care analysts. Current dental care costs are 4.2 percent of U.S. health care costs compared to 6.2 percent in 1975 and 7.4 percent in 1960.

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CASE REPORTS

Primary tooth ankylosis: Report of case with histological analysis

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Tooth ankylosis may be defined as a fusion of alveolar bone with cementum and/or dentin, which occurs either before or after tooth eruption.[1] Primary teeth are ankylosed much more frequently than permanent ones.[2-4]

Ankylosis of primary teeth upon their eruption into the oral cavity results in a fixed occlusal level, while the eruption of neighboring teeth continues simultaneously with the vertical growth of the alveolar bone. This clinical condition is called submersion, infraclusion, secondary retention or hypotrusion.[5-10]

The incidence of ankylosis of primary molars varies among different reports between 1.3 percent and 38.5 percent.[6,10-13] This process affects most frequently primary mandibular molars, more precisely the second molars, according to some authors.[2-4,14-18] Other authors report the condition to be predominant in the first molars.[1,19] Although more frequent, the infraclusion of mandibular primary molars is often mild, compared with that observed for maxillary primary molars.[1] The extent of infraclusion is directly correlated with the rate of facial growth.[5] The prevalence of infraclusion is age-related, with a maximum in the eighth and ninth years of age.[7] Spontaneous reactivation of the eruption of ankylosed molars occurs occasionally, as a consequence of resorption of the area of ankylosis.[17, 20]

Several conditions have been proposed as causes of ankylosis:

- ☐ Genetic tendency.[6]
- ☐ Traumatic injury to the bone and/or periodontal ligament.[21,22]
- ☐ Deficient local vertical bone growth.[23]
- ☐ Disturbed local metabolism with disorder in the process of normal resorption and hard tissue repair.[4]

Congenital absence of permanent teeth has also been proposed as the cause of primary tooth ankylosis, but it cannot be accepted as sufficient, because such absence is not always associated with ankylosis of the corresponding primary molars.[1,19] The exact changes occurring in the periodontal ligament of primary teeth during shedding, which can lead to ankylosis or normal exfoliation, are still unknown.[24]

Histological examination of teeth in infraclusion generally shows areas of fusion between cementum or dentin and alveolar bone , with signs of resorption.[16,25] These areas are most frequently localized in the interradicular area.[18] Periodontal ligament remnants are highly fibrotic, contain only a few cells and are devoid of mucopolysac-

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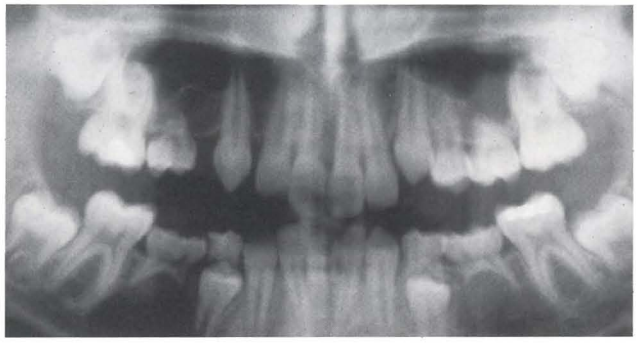

Figure 1. Panoramic radiograph of the patient at nine years of age. The agenesis of several permanent teeth may be recognized. The roots of primary second molars are clearly distinguishable from the surrounding bone.

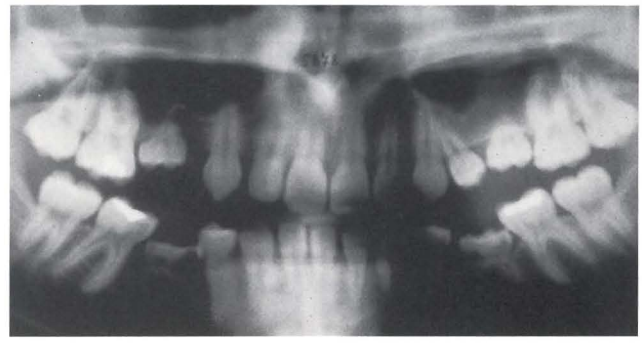

Figure 2. Panoramic radiograph of the patient at 14 years of age. The four second primary molars are in infraclusion, which is especially severe for the mandibular ones.

charidase activity. This latter activity is present in the periodontal ligament during the resorption of the primary tooth root and is believed to be in part responsible for resorption itself.[24]

The purpose of this paper is to present the case of a subject with two ankylosed primary molars, who was observed from nine to fourteen years of age.

CASE REPORT

Clinical findings

A nine-year-old girl was referred to the orthodontic clinic for consultation and treatment. The medical history was within normal limits, except for numerous unerupted permanent teeth. Clinical and radiographic (Figure 1) examination revealed the congenital absence of the following permanent teeth and their respective tooth germs: four second premolars, the right first maxillary premolar and the left central mandibular incisor. Radiographic examination showed that primary second molars' roots were homogeneously radiopaque and clearly distinct from the surrounding bone (Figure 1).

Subsequently, furthermore, the congenital absence of all third molars became evident; moreover the four second primary molars did not exfoliate and underwent progressive infraclusion; radiographic examination showed that root resorption, which was detectable at nine years of age, slowed down in two teeth; stopped completely in the other ones; and that there was a progressive disappearance of the periodontal space (Figures 2 and 3). In fact, as early as nine years of age, this space was visible only in some root areas. Furthermore the roots became progressively less radiopaque and less distinguishable from the surrounding bone (Figures 2 and 3). At fourteen years, the occlusal surfaces of the mandibular second primary molars were at the level of the cervical third of the crown of the contiguous teeth, while the occlusal surfaces of the maxillary second primary molars were under the line of the crown of the contiguous teeth; all these teeth had a sharp, solid sound on percussion, as is characteristic of ankylosed teeth.[12,26] Extraction of these teeth was necessary for therapeutic reasons. Immediately before extraction, the patient's erupted dentition included the permanent anterior teeth with the exception of the left mandibular central incisor; the first premolars with the exception of the right maxillary; the first and second permanent molars; and the four ankylosed primary second molars.

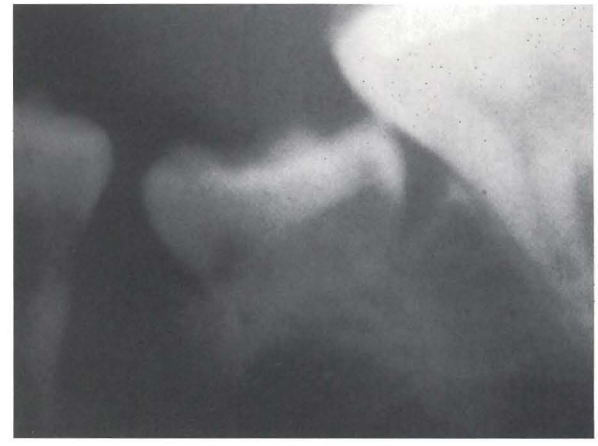

Figure 3. Detail of the mandibular left second primary molar at fourteen years of age. Because of the fusion of the root and the alveolar bone, a distinct outline of the root is no longer apparent. Signs of previous resorption are seen within the crown as relatively radio-transparent areas with irregular shape.

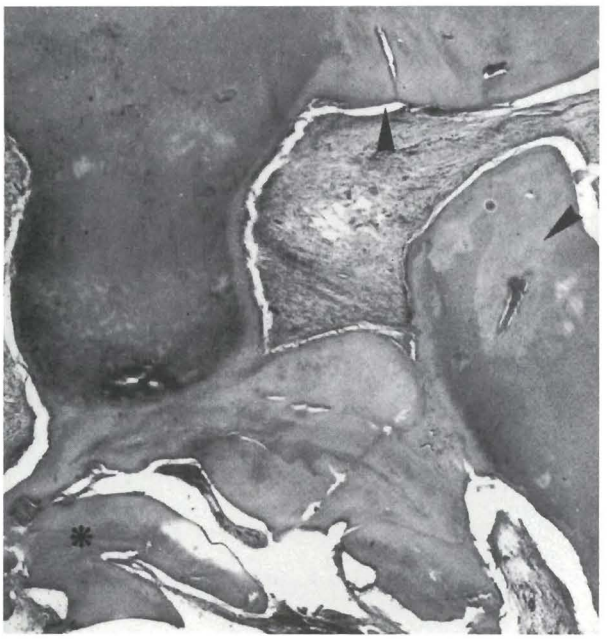

Figure 4. Section of the mandibular left second primary molar. Tooth tissues are continuous with bone trabeculae (asterisk). The dentin has been in part resorbed along the pulp chamber and repaired by cellular cementum (arrowheads) including strips of loose connective tissue with blood vessels. Hematoxylin and eosin; x20.

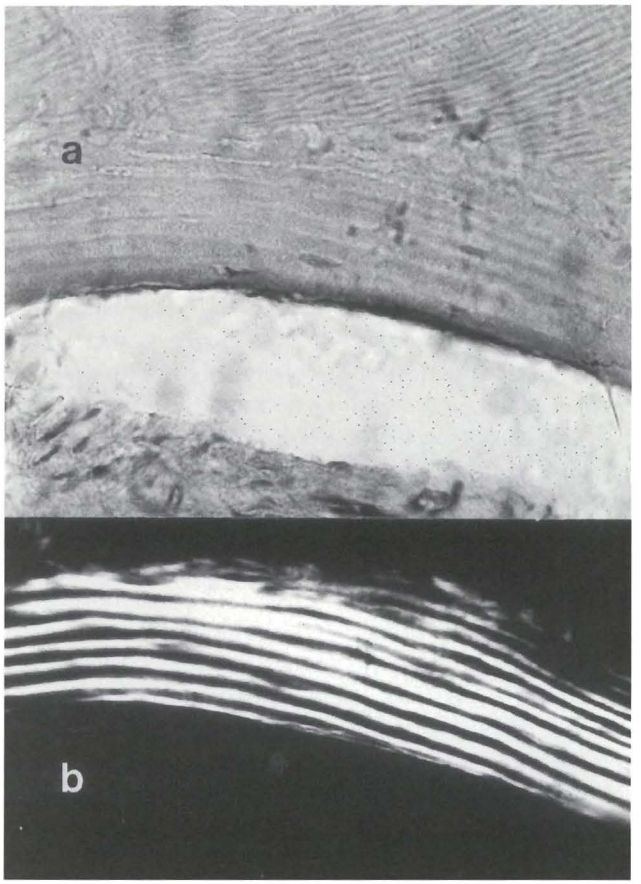

Figure 5. Lamellar bone is in contact with dentin in the wall of a tunnel within the crown of the same tooth as in Figure 4.a: hematoxylin and eosin; b: polarization microscopy; x300.

Histological findings

The four second primary molars were extracted at fourteen years of age for therapeutic reasons. Those from the left side were fixed with 10 percent formol in 0.1 mol/L phosphate buffer, pH 7.4, for two days, decalcified with 3.2 mol/L formiate buffer, pH 2.5, and embedded in paraplast; sections were cut parallel to the tooth axis, stained with hematoxylin and eosin and observed by bright field and polarization microscopy.[27]

The two teeth prepared for microscopy showed one root more extensively resorbed than the other. The root surfaces that had undergone resorption revealed signs of previous linear and lacunar resorption; osteoclasts were rare, but not totally absent. Lacunar resorption was characterized by the presence of Howship's lacunae, either apparently empty or containing odontoclasts, and was assumed to be an expression of active resorption.[28] Linear resorption was characterized by the presence of smooth dentinal surfaces, without cementum, and was assumed to be an expression of cessation or marked slowing down of resorption.[28,29]

The resorbed roots also showed signs of repair: lacunar resorption repaired with cellular cementum; and areas of dentin with signs of resorption or cemental repair were fused with bone lined by osteoblasts on some surfaces (Figures 4 and 5). Both simple lamellar and osteonic bone with osteocytes were present; some bone lamellae were found also in tunnels in coronal dentin (Figure 5).

An inflammatory infiltrate of plasma cells, lymphocytes, macrophages, and granulocytes was found in the pulp of one tooth, which was also devoid of odontoblasts. No alterations were seen within the pulp of the other tooth. A mild inflammatory infiltrate of macrophages, lymphocytes, and plasma cells was found in the remnants of the periodontal ligament of both teeth.

DISCUSSION

Ankylosis, fusion of dentin and bone, caused the infraclusion of primary teeth in the case presented here. A

similar histological condition is present in most cases of infraclusion, although other possible causes exist.[1] In this case the primary molars showed an extensive, but incomplete, root resorption, in spite of the congenital absence of the succedaneous teeth. It is possible that pressure from adjoining permanent teeth at the time of their eruption had caused such resorption, but earlier radiological documentation did not address this issue. Signs of remodeling at the moment of extraction were present, however, on the surfaces of the teeth examined here. These signs were represented by osteoclasts adhering to Howship's lacunae and osteoblasts adhering to bone lamellae, which, in turn, adhered to dentin. These findings suggest that mechanical trauma, possibly a consequence of a mixed dentition, and a previous partial resorption of the roots, had stimulated hard tissue resorption and redeposition continuously. Periods of active resorption and of suspension or marked slowing of resorption, with redeposition of a mineralized tissue, alternate during the root resorption of primary teeth. The hard tissue, which is redeposited during this process, is usually cellular cementum.[30,31] Ankylosis of the primary molars can be envisaged, therefore, as the result of a disturbance of root resorption in which repair processes prevail leading to excessive deposition not only of cementum, but also of bone and to the disappearance of the periodontal ligament.[24]

Deposition of bone contacting dentin has been observed also in permanent teeth.[32] That finding and those presented here accentuate the bone-inductive ability of dentin, which is expressed much less frequently than cementum-inductive ability, but is nonetheless a characteristic of dentin and gives hope to its attempted use as a therapeutic agent.[33,34] Factors regulating resorptive and reparative processes may be produced by the cells responsible for the formation of hard tissues and incorporated into the extracellular matrix before calcification; these factors would be liberated at resorption surfaces and be diffused in the surrounding tissue. Because resorption requires cessation of hard tissue deposition and mineralization of the surface destined to be resorbed (i.e., disappearance of the precementum in cases of root surfaces), it suggests that such a process had occurred, for some reason, in the teeth studied here (presence of adjacent permanent teeth, occlusal trauma or other) and had played a prominent role in the initiation of the events leading ultimately to ankylosis. The occurrence of a remodeling activity on part of the surface of the ankylosed roots may explain why ankylosis may be spontaneously reversed, with consequent reactivation of resorption.

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DENTAL ANXIETY AND THOUGHTS RELATED TO TREATMENT

The aim of the current study was to explore the relationship between dental anxiety and thoughts related to dental treatment in a sample of Dutch students.

The results clearly indicate that dental anxiety is associated with the tendency to experience negative or threatening thoughts. That is, the more apprehensive a patient is about dental treatment, the more likely this person is to report that he or she ruminates on the condition of his or her teeth or on negative possible outcomes of treatment, particularly pain. In addition, highly anxious individuals seem to overestimate the intensity of a potential aversive event in the situation or the impending situation. For example, highly anxious dental patients are more likely to think "My teeth can break off" instead of "It's not as bad as I thought." It may be that the tendency of highly anxious dental patients to make negative and unrealistic predictions about a forthcoming dental treatment is the construct underlying the anticipatory anxiety that anxious patients experience while having the prospect of a dental appointment. Clearly, the present results should be viewed conservatively, given the self-report nature of the measures and the use of a student population. The results, however, are in accordance with many other studies among people suffering from divergent forms of anxiety, indicating that anxiety is associated with a negative cognitive content. The findings will be used to develop a measure for assessing peoples' degree of negative and catastrophic thinking. Such a cognitive assessment instrument may be useful for both the improvement of knowledge on the concept of dental anxiety and for the evaluation of treatment procedures upon cognitions.

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Latent fluorides: Report of case

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Nicole Ionesco-Benaiche, DCD, DU
Michele Muller, DCD, DU

Dental fluorosis is a defect of enamel induced by fluoride ingestion during tooth formation. Clinically this condition varies from white spots to large lesions to which all teeth in the process of forming enamel are prone. Histologically, dental fluorosis is a condition of permanent hypomineralized change, with increased surface and subsurface porosity of enamel resulting from excessive fluoride reaching the developing tooth before eruption.[1] The severity and distribution of fluorosis depend on the fluoride concentration, the duration of exposure to fluoride, the stage of ameloblast activity, and individual variations in susceptibility.[2] The fluoride content in drinking water represents the largest single contribution to the daily fluoride intake. Many surveys have endorsed the association between the severity of fluorosis and the level of fluorides in drinking water.[3,4] In France, fluoridation of the water supply does not exist and many families drink exclusively bottled mineral waters, the fluoride levels of which range between 0.15 to 9. 20 ppm/F.

This case reports the clinical and scanning electron microscopy (SEM) study of a dental fluorosis induced by a long-term consumption of a highly fluoridated (6.02 ppm/F) mineral water.

CASE REPORT

A thirteen-year-old Caucasian male and his eleven-year-old sister without any previous medical history presented at Lenval Children's Hospital of Nice for dental examination and treatment. The boy needed extractions of the four first premolars for orthodontic purposes. The clinical examination showed that his maxillary central incisor, the mandibular lateral incisors, and the first permanent molars presented lines and cloudy areas of opacity. The canines and the premolars displayed marked opacities on one third to half of the labial surface; some pits were present near the occlusal border of the premolars (Figure 1A). The sister's examination revealed white patches of lusterless enamel on every tooth. The incisors and the first molars showed pits in their incisal parts, while pronounced perikymata were evenly distributed over the enamel surface. The incisal edge of the maxillary central incisors presented a brown discoloration (Figure 1B). Questioning of the children's mother revealed that since her son was approximately four and her daughter approximately three, they drank only mineral water with a fluoride content of 6.02 ppm/F. Because no history of amelogenesis imperfecta existed in this family, (the children had received no tetracycline medication), and the enamel defects were

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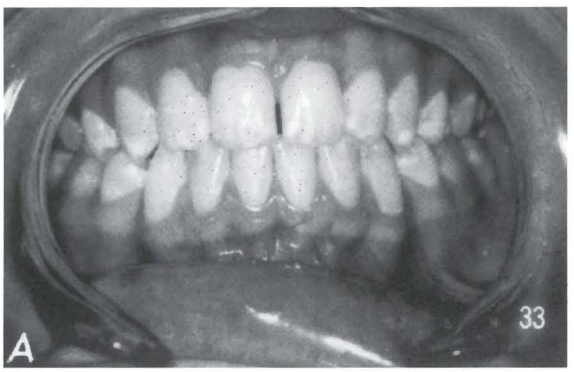

Figure 1A. Intraoral photograph of the male, which shows the typical fluorosis pattern. This case has been classified as mild to moderate.

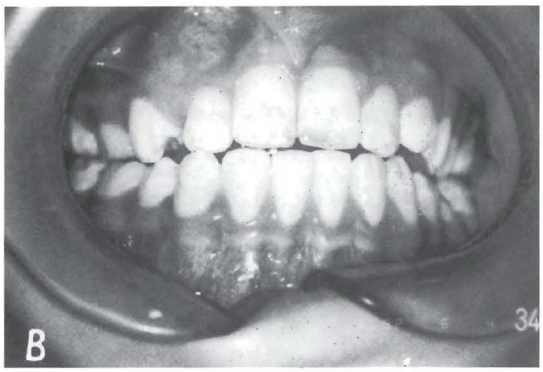

Figure 1B. Intraoral photograph of the female. This case was classified as moderate to severe due to the pits and the brown stainings of the teeth.

symmetrical, the condition was classified as enamel fluorosis.

Following the extractions of the boy's first premolars, the teeth were immersed in a 10 percent solution of sodium hypochlorite and simultaneously sonicated for ten minutes to remove the superficial organic material. After rinsing and air-drying, samples were fixed in a 70 percent ethanol solution and prepared for SEM examination according to standard techniques.[5,6] All specimens were sputter-coated with gold for examination under the SEM (Jeol JSM 35C) at 15 KV.

RESULTS

The surface of the teeth exhibited accentuated perikymata (Figure 2). In the occlusal and the central surface zones, enlargement of the enamel sheets was observed (Figure 3). Pits were present on the occlusal third of the teeth; the bottoms of the pits were irregular (Figure 4). At the dentinoenamel junction enamel and dentine were normal (Figure 5).

DISCUSSION

In this case, the dental fluorosis resulted from the fluoride in the mineral water, which appeared to be the children's only exposure to fluorides. In addition, the symmetrical distribution of white spots, pits, and brown stains on the enamel surface confirms the diagnosis of dental fluorosis. Because all teeth do not exhibit the same clinical features, these cases can be classified as moderate fluorosis for the boy and as severe fluorosis for the girl, according to Dean's Index.[7]

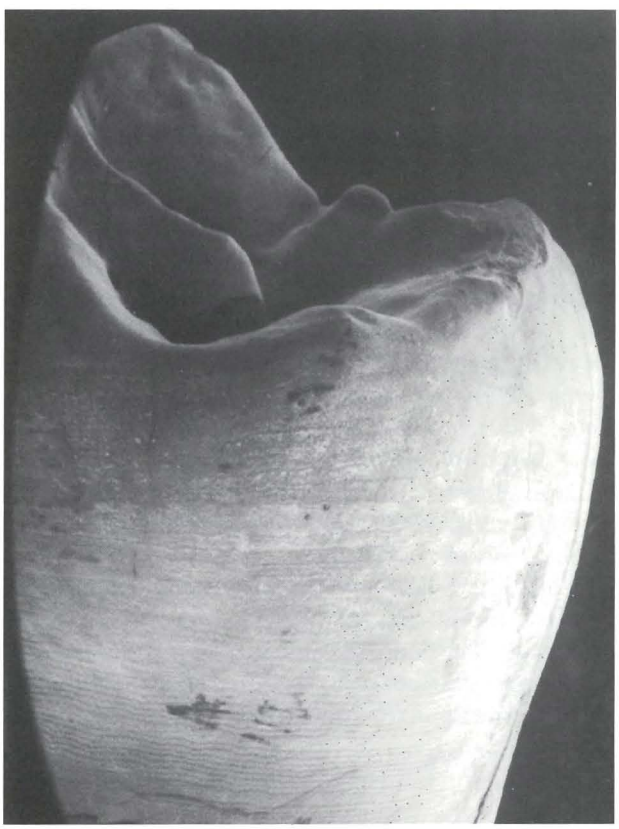

Figure 2. SEM presentation of an extracted first premolar. Perikimata are accentuated. Mag: original 12 ×.

The girl's clinical condition corroborates previous studies that found the same degree of fluorosis in children who had been drinking water containing 7.8 ppm

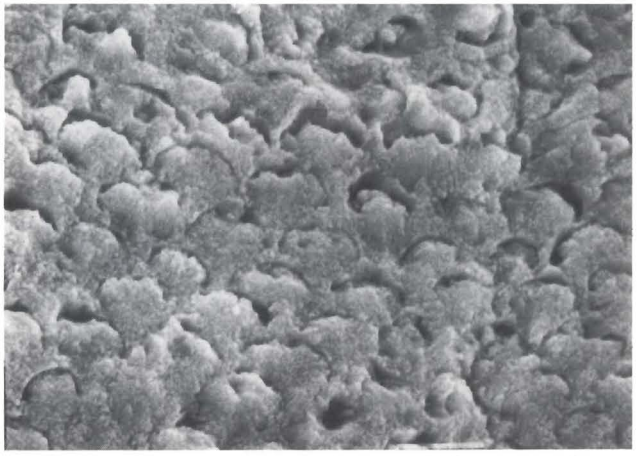

Figure 3. SEM. Enlargement of the enamel sheets near the occlusal border. Mag: original 1500 ×.

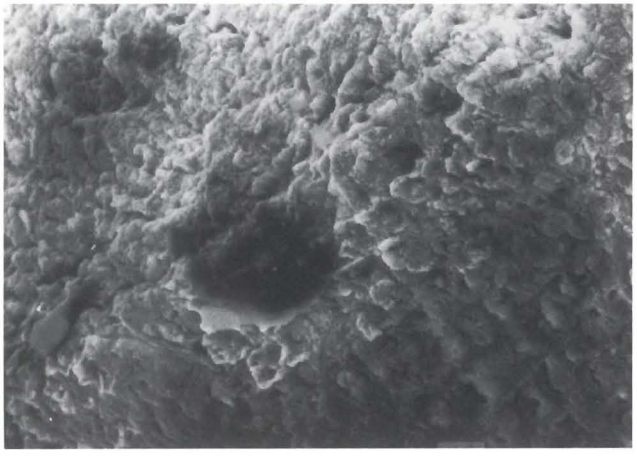

Figure 4. SEM. A pit located on the occlusal third of the enamel surface. The bottom looks irregular. Mag: original 750 ×.

fluoride at ages thirty-five through forty-two months.[8] In the boy, there was an increase in severity from the anterior to the posterior teeth, indicating that the spectrum of macroscopic changes was more pronounced for teeth whose enamel maturated after age four. In both cases the clinical appearance of fluorotic enamel followed the calendar of tooth eruption; this gives support to previous studies that confirmed that the pathogenic mechanism of dental fluorosis occurs during the maturation state of enamel formation.[9,10]

Our SEM study shows findings similar to those presented in other studies, i.e: the accentuation of perikymata, an insufficient closure of intercrystalline spaces, and a focal loss of surface enamel that corresponds to macroscopically visible pits.[11,12] These findings confirm the diagnosis of enamel fluorosis.

Dental fluorosis results from a long-term intake of a high level of fluoride during enamel formation. The severity of dental fluorosis increases with the amount of fluoride ingested during the period of tooth formation and the duration of fluoride ingestion. The Environmental Protection Agency in 1986 defined 4.0 mg/L fluoride as the maximum Contaminant Level permitted for drinking waters in the United States.[13] Signs of skeletal fluorosis may appear with higher levels of fluoride intake (8-10 ppm or more in drinking water) for approximately ten years or more.[14] In France, the consumption of bottles of mineral water is important. The fluoride content of mineral water ranges between 0.15 ppm to 9.20 ppm. The ionic concentration of these waters is generally written on the label in very small letters and few consumers, if any, read these dosages. Moreover, few people are

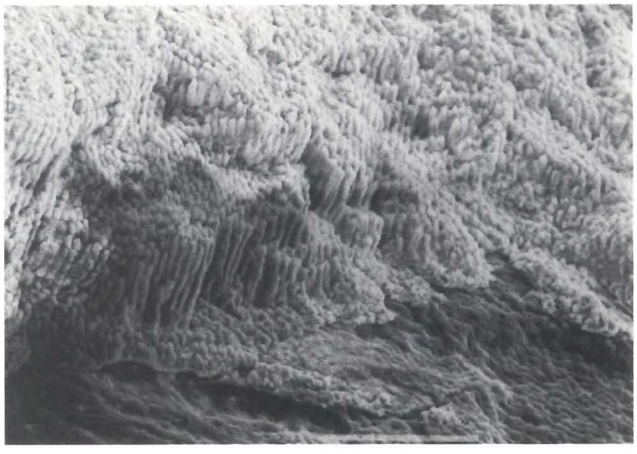

Figure 5. SEM. The dentinoenamel junction is normal. Mag: original 270 ×.

aware of the side effects induced by fluorides. In the same way recent data indicate that patterns in fluid consumption among North Americans have changed dramatically over the last thirty years, with soft drinks contributing an ever increasing proportion of total fluid intake and a major source of fluoride for consumers in the younger age-groups.[15]

Results from this study support advising the need for care when children under eight years drink daily highly fluoridated mineral water. It suggests, as recommended by Newbrun, an explicit labeling of the fluoride content, and a warning of dental fluorosis risks.[16] Such labeling seems to be the only effective measure to prevent un-

acceptable cosmetic and disfiguring dental fluorosis for people using alternative water supplies that contain 1 ppm or more of fluorides.

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CARBOHYDRATE ABSORPTION FROM FRUIT JUICE IN YOUNG CHILDREN

Fruit juice has become a significant part of the diets of young children. Many factors may be responsible for this, among them children's innate preference for sweetness, parents' perception of fruit juice as a "natural" and "healthful" food, and the convenience of a juice bottle or box. Unpublished surveys by juice manufacturers report that about 90 percent of all infants consume fruit juice by 1 year of age. On average, infants drink 5 ounces of juice per day, but about 1 percent consume more than 20 ounces daily. Children under 5 years of age average 9 gallons of juice per year, of which approximately 50 percent is apple juice.

The major carbohydrates present in juice are fructose, glucose, and sorbitol. Glucose is rapidly absorbed by an active process; fructose is absorbed by two separate mechanisms: (1) facilitated transport, a slow process, and (2) co-transport with glucose, a fast process. Sorbitol is poorly absorbed by a passive process.

The carbohydrate composition of apple juice and white grape juice differ in several aspects, which may account for the increased malabsorption following apple juice. First, there is much more glucose in white grape juice. Second, there is a significant amount of sorbitol in the apple juice but none in white grape juice. Both a higher ratio of fructose compared with glucose and the presence of sorbitol have been implicated as factors that increase fructose malabsorption.

Smith, M.M. *et al*: Carbohydrate absorption from fruit juice in young children. Pediatrics, 95:340-344, March 1995.

ABSTRACTS

Gable, Theresa Ott; Kummer, Ann W.; Lee, Linda; Creaghead, Nancy A.: Premature loss of the maxillary primary incisors: Effect on speech production. J Dent Child, 62:173-179, May-June 1995.

The residual effect of premature loss due to extraction of the four maxillary primary incisors on speech production was studied. The articulation of twenty-six subjects who had their teeth extracted before the age of five years was evaluated at eight, nine, or ten years old and compared with the articulation of an age-matched comparison group with normal exfoliation of their incisors. T-tests for related measures revealed no statistically significant differences between the group with premature loss and the comparison group. These results suggest that loss of maxillary incisors in children younger than five years is not likely to result in defective articulation while the teeth are missing or when the permanent dentition is acquired.

Speech articulation; Loss of maxillary primary incisors

Park, Kichuel K.; Hemandez, David; Schemehorn, Bruce R. *et al*: Effect of chewing gums on plaque pH after a sucrose challenge. J Dent Child, 62:180-186, May-June 1995.

The objective of this study was to determine whether sugarless chewing gums with different sweetening agents (aspartame, saccharin, acesulfame-K or xylitol) differ in their ability to reduce an acidogenic response from a 10 percent sucrose rinse challenge in eight panelists using a plaque pH telemetry system. All of the sugarless gums were effective in increasing plaque pH and in reducing the area under the curve after the sucrose challenge compared with "no gum" treatment. No statistically significant differences were noted among the sugarless gums. The response to sucrose gum was intermediate between sugarless gums and "no gum" and was not statistically different from either "no gum" or three of the sugarless gums. The areas under the curve after paraffin and xylitol gums were significantly less than after sucrose gum. The results indicated that chewing any of the sugarless gums or paraffin resulted in a beneficial effect on plaque pH response after sucrose challenge.

Sugarless chewing gum; Aspartame; Saccharin; Acesulfame-K; Xylitol

Koroluk, L.D.; Hoover, J.N.; Komiyama, K.: The effect of caries scoring systems on the association between dental caries and streptococcus mutans. J Dent Child, 62: 187-191, May-June 1995.

The purpose of this study was to examine the effect of caries scoring systems on the association between caries indices and salivary counts of *S. mutans* in a group of ninety-eight three to five-year-old preschool children in Saskatoon, Canada. Caries was recorded using the WHO (1987) criteria and a modified scoring system(MC) in which questionable lesions (no soft walls or floor) were included as carious. Whole stimulated saliva was collected from each subject and *S. mutans* counts were determined (CFU/ml of saliva). Both dft(MC)=1.03 and dfs(MC)=1.53 were significantly different ($p<.0001$) from dft(WHO)= O.69 and dfs(WHO)=1.17, respectively. The *S. mutans* count was found to be significantly ($p<.001$) correlated with dft(MC), dfs(MC), dft(WHO) and dfs(WHO). When questionable carious lesions are included in caries indices, they appear to remain well associated with salivary *S. mutans* counts.

Caries scoring systems; *S. mutans*

Wöltgens, J.H.M.; Etty, E.J.; Gruythuysen, R.J.M. *et al*: Influence of fluoride in saliva during the early cariogenic changes in the enamel of boys and girls. J Dent Child, 62:192-196, May-June 1995.

In boys and girls cariogenic changes in the dental enamel in relation to fluoride (F^-) concentrations in stimulated and unstimulated saliva were studied in a six-month period. Also the use of various types of applications of F^- was assessed. No difference in the use of F^- between boys and girls before and after the interval was observed. Also no clear differences were found between boys and girls in the levels of F^- in both types of saliva, determined at the end of the six-month period.

The most important finding was that for all children, a significantly positive relationship was found between the disappearance of white spots turning into sound enamel (regression) and the F^- concentration in unstimulated saliva. In addition, girls who developed new white spots had higher levels of F^-, but those who developed new cavities had lower F^- levels in both types of saliva.

Apparently F^- can prevent dental caries by acting very early on remineralization and demineralization processes in enamel surfaces.

Saliva; Fluoride ion; Remineralization; Demineralization

Andersson-Wenckert, Ingrid E.; van Dijken, Jan W.V.; Stenberg, Roger: Effect of cavity form on the durability of glass ionomer cement restorations in primary teeth: A three year clinical evaluation. J Dent Child, 62:197-200, May-June 1995.

The effect of cavity form on the durability of glass ionomer cement restorations in primary molars was studied in a clinical, intraindividual, three-year longitudinal study. Proximal restorations were placed in either a microcavity or a modified Black's Class ll cavity with rounded internal and external angles. A total of twenty-eight pairs were assessed. After two years the cumulative failure rate was 16 percent for each of the cavity types and after three years 25 percent for the microcavity and 32 percent for the modified Black's Class ll cavity. No statistical difference was found in the longevity of the restorations placed in the two different types of cavities.

Cavity form; Microcavity; Black Class ll

Takenoshita, Yasuharu; Shimada, Makoto; Kubo, Shuro: Traumatic herniation of buccal fat pad: Report of case. J Dent Child, 62:201-204, May-June 1995.
Traumatic herniation of buccal fat pad in a case of twenty-month-old girl is presented. The lesion has been caused by the fall on the tooth brush in the mouth, and treated with excision and wound sutures.
Buccal fat pad; Herniation

Waldman, H. Barry: Almost four million children with disabilities. J Dent Child, 62:205-209, May-June 1995.
A summary is provided for a series of federal agency reports on the prevalence and demographic characteristics of the disabled children in our communities.
Disabled children; Prevalence; Demographics

Waldman, H. Barry: Is your pediatric practice keeping pace with the improving picture of dental economics? J Dent Child, 62:210-214, May-June 1995.
A review is provided of dental economics based on national dental expenditure data, ADA economic reports and IRS business receipt information. The favorable series of economic reports, together with continuing projected decreases in the dentist-to-population ratios points to a continuing favorable economic picture for pediatric dental practitioners.
Pediatric dental practice; Economics

Mancini, Giovanni; Francini, Elisabetta; Vichi, Marco *et al*: Primary tooth ankylosis: Report of case with histological analysis. J Dent Child, 62:215-219, May-June 1995.
Two primary molars in infraclusion showing clinical and radiographic signs of ankylosis, were subject to histological examination by bright field and polarization microscopy. The roots revealed signs of ongoing resorptive and reparative processes and in some areas dentin with signs of resorption-or repair cementum-were fused with simple lamellar or osteonic bone. These results suggest that ankylosis of primary molars can result from a disturbance of root resorption, with repair processes prevailing over resorptive ones and leading to excessive deposition of bone besides cementum-as a consequence of bone-inductive properties of dentin.
Infraclusion; Primary molars; Resorption; Repair

Jasmin, Jean R.; lonesco-Benaiche, Nicole; Muller, Michele: Latent fluorides: Report of case. J Dent Child, 62:220-223, May-June 1995.
This case report examines the effects of a long-term exposure to fluoride on the teeth of two children who started to drink a highly fluoridated mineral water when they were three years and four years of age, respectively. A clinical and SEM study was conducted to supply evidence of the harmful effect of fluoride ingestion at above optimal levels and to ask for an explicit labeling of drinks that contain more than 1 ppm/F.
Fluoride; Fluorosis; Fluoridated water

REMEMBRANCE *continued from page 169*

In the fall of 1985, I took a group of Dentists to China on a People to People Program. This project established by President Eisenhower encouraged an exchange of professionals in underprivileged or so-called Third World countries with the United States. Knowing the newer techniques required dental supplies not available in China, I brought with me a huge amount of disposable anesthetic needles, anesthetic solution, and composite resins. It was interesting to see how advanced our country was in techniques and equipment and the general approach to patient care. But the desire of dentists in China to catch up with the rest of the world was enormous. They were just beginning early investigations in the use of lasers in cavity preparation. Many of their bright teachers were in the United States and in the Scandinavian countries learning and then returning to China. These teachers were China's hope to catch up with the rest of the dental world. On this People to People trip, a stop was made in Hong Kong, at the Prince Philip Hospital and Dental School, whose Dean was an old friend, Stephen Wei. Steve recently served as President of The International Association for Dental Research. Undoubtedly Hong Kong will be the research center and provide advanced schooling for China, when Hong Kong reverts to China in 1997.

A few years ago, Irv Eichenbaum, George Teuscher and I were reminiscing at breakfast about the changes that have occurred in our profession and about the friendships that were made and have endured over these many years. We have come a long way over these almost fifty years. I have seen many changes in my lifetime, from seeing a man land on the moon to computers being part of a young child's school training. And as I look back to what was my Golden Age, I ask myself, will there be another Golden Age that present dentists will experience? Will the future bring the same pleasures of treating our patients and each experience be a landmark in our learning? Will there be that special person who was not only a teacher, but a caring friend and colleague who shared our joys, our highs, and our lows over the years? These were the gentle people of our generation. I can only wonder and hope the best for the future.°

° Retired from private practice October 1, 1994. Upon learning of the retirement of Dr. Album from active clinical practice, the editors asked him to reflect on his personal experiences and to record them for publication in JDC.